Universitat Autònoma de Barcelona

ADULT ATTACHMENT STYLES IN AMERICAN AND SPANISH PATIENTS WITH FIBROMYALGIA AND RHEUMATOID ARTHRITIS: A CROSS-SECTIONAL STUDY

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Universitat Autònoma de Barcelona

Ph.D. Thesis

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Tesi doctoral

ADULT ATTACHMENT STYLES IN AMERICAN AND SPANISH PATIENTS WITH FIBROMYALGIA AND RHEUMATOID ARTHRITIS: A CROSS-SECTIONAL STUDY

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Men ought to know that from the brain and from the brain only arise our pleasures, joys, laughter, and jests as well as our sorrows, pains, griefs and tears. ... It is the same thing which makes us mad or delirious, inspires us with dread and fear, whether by night or by day, brings us sleeplessness, inopportune mistakes, aimless anxieties, absent-mindedness and acts that are contrary to habit…

Hippocrates

Alice: "Would you tell me, please, which way I ought to go from here?"
The Cheshire Cat: "That depends a good deal on where you want to get to."
Alice: "I don't much care where—"
The Cheshire Cat: "Then it doesn't much matter which way you go."
Alice: "—So long as I get somewhere."
The Cheshire Cat: "Oh, you're sure to do that, if only you walk long enough."

Lewis Carroll, Alice in Wonderland

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And finally, to all health professionals who, like the above and my father, have dedicated their life as doctors to treat other human beings with compassion and intent and have paved the way to follow in their footsteps.

Table of Contents

List of Abbreviations

FM	Fibromyalgia
FMS	Fibromyalgia Syndrome
ACR	American College of Rheumatology
WPI	Widespread Pain Index
SS	Symptom Severity
FS	Fibromyalgia Symptom Scale
PSD	Polysymptomatic Distress Scale
SSRI	Selective Serotonin Reuptake Inhibitors
SNRI	Serotonin and Noradrenaline Reuptake Inhibitors
RA	Rheumatoid Arthritis
RF	Rheumatoid Factor
ACPA	Anti-Citrullinated Protein Antibody
anti-CCP	Anti-Cyclic Citrullinated Peptide
HLA	Human Leukocyte Antigen
PTPN22	Protein Tyrosine Phosphatase, Non-receptor type 22
STAT4	Signal Transducer and Activator of Transcription 4
TRAF-1/C5	Encoding Tumor Necrosis Factor Receptor-Associated Factor 1 and encoding Complement Component 5
DMARDs	Disease-Modifying Antirheumatic Drugs
MTX	Methotrexate
DAS28	Disease Activity Score in 28 joints
SJC	Swollen Joint Count
TJC	Tender Joint Count
ESR	Erythrocyte Sedimentation Rate
CRP	C-Reactive Protein level
VAS	Visual Analog Scale
PIP	Proximal Interphalangeal joints
MCP	Metacarpophalangeal joints
MTP	Metatarsophalangeal joints

EULAR	European League Against Rheumatism
DSM	Diagnostic and Statistical Manual of Mental Disorders
SSD	Somatic Symptom Disorder
PHQ-15	15-Symptom Checklist
AAS	Adult Attachment Scale
RAAS	Revised-Adult Attachment Scale
AHQ	Attachment History Questionnaire
AORI	Attachment and Object Relations Inventory
ASQ	Attachment Style Questionnaire
CAS	Continued Attachment Scale
CATS	Client Attachment to Therapist Scale
ECR	Experiences in Close Relationships
ECR-R	Experiences in Close Relationships-Revised
MAQ	Measure of Attachment Qualities
MFPS	Mother Father Peer Scale
MSAS	Maternal Separation Anxiety Scale
PAQ	Parental Attachment Questionnaire
PASAS	Parents of Adolescents Separation Anxiety Scale
PBI	Parenting Bonding Instrument
RAQA	Reciprocal Attachment Questionnaire for Adults
RQ	Relationship Questionnaire
RSQ	Relationship Scales Questionnaire
R-IPA	Revised Inventory of Parental Attachment
VASQ	Vulnerable Attachment Style Questionnaire
ESEMeD	European Study of the Epidemiology of Mental Disorders
ICD	International Statistical Classification of Diseases and Related Health Problems
BDI	Beck Depression Inventory
HADS	Hospital Anxiety and Depression Scale
HRSD	Hamilton Rating Scale for Depression
MADRS	Montgomery Asberg Depression Rating Scale

PHQ-9	Brief Patient Health Questionnaire
PRIME-MD	Primary Care Evaluation of Mental Disorders Procedure
CES-D	Centre for Epidemiology Studies Depression Rating Scale
MAOI	Monoamine Oxidase Inhibitors
TCA	Tricyclic Antidepressants
NDRI	Norepinephrine-Dopamine Reuptake Inhibitors
SARI	Serotonine Antagonists and Reuptake Inhibitor
NaSSA	Noradrenergic and Specific Serotonergic Antidepressant
NRI	Norepinaphrine Reuptake Inhibitor
5-HT	Serotonin
NE	Norepinephrine
IL	Interleukin
CRH	Cortisol Releasing Hormone
HPA	Hypothalamus-Pituitary-Adrenal axis
HRQoL	Health-Related Quality of Life
WHO	World Health Organization
WHO ICF	World Health Organization International Classification of Functioning, Disability, and Health
QALYs	Quality-Adjusted Life Years
SF-36	36-Item Short-Form Health Survey
COOP	Dartmouth Primary Care Cooperative Information Charts
NHP	Nottingham Health Profile
SIP	Sickness Impact Profile
EQ-5D	Euroqol Five-Item Questionnaire
HUI	Health Utilities Index
QWB	Quality of Well-Being Index
PCS	Physical Component Summary
MCS	Mental Component Summary
IMMPACT	Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials
RMQ	Roland-Morris Back Pain Questionnaire
WOMAC	Western Ontario and McMaster University Osteoarthritis Index

HAQ	Stanford Health Assessment Questionnaire
MDHAQ	Multidimensional Health Assessment Questionnaire
AIMS	Arthritis Impact Measurement Scales
(S)FIQ	(Spanish) Fibromyalgia Impact Questionnaire
FFS	FibroFatigue Scale
RADAI	Rheumatoid Arthritis Disease Activity Index joint count
PRO	Patient-Reported Outcomes
RAPID	Routine Assessment of Patient Index Data
ROS	Symptom Checklist Review of Systems
ICF	International Classification of Functioning
CUPID	Cultural and Psychosocial Influences on Disability
AIS	*Àrea d'Atenció Integral de Salut*
AERA	American Educational Research Association
APA	American Psychological Association
NCME	National Council on Measurement in Education
CAA	*Cuestionario de Apego Adulto*
BPI	Brief Pain Inventory
PSS	BPI's Pain Severity Score
PIS	BPI's Pain Interference Scale
RDCI	Rheumatic Disease Comorbidity Index
ANS	HADS Anxiety Subscale score
DEP	HADS Depression Subscale score
FN	RAPID3'S Physical Function component
PN	RAPID3's Pain component
PTGL	RAPID3's Patient's Global Estimate of Status component

List of Tables

List of Figures

Abstract

Introduction and aims. Fibromyalgia is defined by criteria related on symptomatic range and severity. In the American College of Rheumatology 1990 criteria, musculoskeletal pain is the cardinal symptom evaluated, while in the updated 2010 criteria, only one of the five criteria items directly concerns musculoskeletal pain. The etiology is yet unclear, although there is a frequent established association with psychosocial stressors. Adult attachment style is an internal representation of self and others, yielding from early childhood experiences of relationships with primary caregivers. Attachment style determines how individuals relate to each other and is associated to strategies for managing threatening situations, with precedents of distinctive traits within Spanish population. Given that attachment is a relatively stable, trait-like characteristic, it served as framework for this research. In this study, the main aim was to obtain more detailed clinical, attachment, and psychosocial information through a comparison between fibromyalgia and another rheumatic condition of a mainly organic, autoimmune, etiology: rheumatoid arthritis. Also, a cross-cultural report was added to examine the Spanish attachment particularities.

Material and methods. The study consisted of a cross-sectional design with a fibromyalgia group and a rheumatoid arthritis comparison group, both in Barcelona (67 and 70 patients, respectively) and New York (16 and 15 patients). All subjects submitted demographic and clinical information, as well as complied psychological questionnaires on attachment, depression, functional status, pain-related dimensions, quality of life, and anxiety.

Results. Fibromyalgia patients showed higher levels of depression, anxiety, pain intensity and interference, somatic symptoms, and worse functional status than the rheumatoid arthritis sample. Insecure attachment was prominent in both conditions, albeit it yielded significant differences only in the cross-cultural comparison. In general, the hostile fearful style reported the worse health status. Most significant differences remained when comparing both medical conditions across nationalities. Attachment was found to predict pain-related outcomes in fibromyalgia; as well as rheumatoid arthritis quality of life, pain-related variables, functional status, and somatic symptoms. In fibromyalgia, changes in quality of life and pain interference were explained by anxiety and depression; whereas pain intensity, functional status, and somatic symptoms were predicted only by anxiety. In rheumatoid arthritis, most variables were also predicted by both anxiety and depression; while the physical component of quality of life and pain intensity were predicted only by depression.

Conclusions. The findings highlight the importance of overall adult attachment and mood disorders contributing to the burden of fibromyalgia and rheumatoid arthritis, despite the latter being an inflammatory disease associated to severe disability and premature mortality. Investigation in the direction of adapting psychological interventions stressing these components will help, striving for an improvement in quality of life and function of these medication conditions that still require palliative treatment and hold no definitive cure.

Resum

Introducció i objectius. La fibromiàlgia està definida per criteris relacionats amb rang simptomàtic i severitat. Als criteris de l'*American College of Rheumatology* del 1990, el dolor musculoesquelètic és el símptoma cardinal avaluat, mentre que als criteris actualitzats al 2010, només un dels cinc ítems diagnòstics hi està vinculat directament. L'etiologia roman indefinida, malgrat sovint s'estableix una associació amb estressors psicosocials. L'estil de vincle adult és una representació interna d'un mateix i dels altres, producte d'experiències primerenques de relació amb cuidadors primaris. L'estil de vincle determina com els individus es relacionen i s'associa a estratègies de gestió de situacions amenaçadores, amb precedents de trets distintius en població espanyola. Donat que el vincle és una característica relativament estable que funciona com a tret, serveix de marc per aquesta recerca. En aquest estudi, l'objectiu principal era obtenir més profunditat d'informació clínica, psicosocial i del vincle a través de la comparació entre fibromiàlgia i una altra malaltia reumàtica d'etiologia bàsicament orgànica i autoimmune: l'artritis reumatoide. Així mateix, es va afegir una comparativa intercultural per tal d'examinar les particularitats espanyoles del vincle.

Material i mètodes. L'estudi va consistir en un disseny transversal amb un grup de fibromiàlgia i un grup de comparació d'artritis reumatoide, tant a Barcelona (67 i 70 pacients, respectivament) com a Nova York (16 i 15 pacients). Tots els participants van lliurar informació sociodemogràfica i clínica, així com van omplir qüestionaris psicològics del vincle, depressió, capacitat funcional, dimensions relacionades amb el dolor, qualitat de vida i ansietat.

Resultats. Els pacients amb fibromiàlgia van mostrar nivells més alts de depressió, ansietat, intensitat i interferència del dolor, símptomes somàtics i pitjor capacitat funcional que la mostra d'artritis reumatoide. El vincle insegur va ser prominent en ambdues malalties, tot i que va derivar en diferències significatives només a la comparació intercultural. En general, l'estil temerós hostil va reportar el pitjor estat de salut. La major part de les diferències significatives van persistir en la comparació de nacionalitats entre ambdues condicions mèdiques. El vincle va predir les variables del dolor a la fibromiàlgia; així com qualitat de vida, dimensions del dolor, capacitat funcional i símptomes somàtics a l'artritis reumatoide. A la fibromiàlgia, els canvis en qualitat de vida i interferència del dolor van ser explicats per l'ansietat i la depressió; mentre que la intensitat del dolor, la capacitat funcional i els símptomes somàtics van ser predits només per l'ansietat. En artritis reumatoide, la majoria de variables van ser predites només per l'ansietat i la depressió; mentre que el component físic de qualitat de vida i la intensitat del dolor van ser predites només per la depressió.

Conclusions. Les troballes ressalten la importància del vincle adult general i els trastorns de l'estat d'ànim com a contribuents de la càrrega que suposen la fibromiàlgia i l'artritis reumatoide, malgrat que aquesta última sigui una malaltia inflamatòria associada a discapacitat severa i mortalitat prematura. Pot resultar útil la recerca en la direcció d'adaptar intervencions psicològiques que posin especial èmfasi en aquests components, intentant abastar una millora en qualitat de vida i funció d'aquestes afeccions que segueixen requerint de tractament pal·liatiu i no tenen cura definitiva.

Introduction

Fibromyalgia

Historical overview of the disease

The first accounts of musculoskeletal aches and pains date back to 1592, when the French physician Guillaume de Baillon coined the term "**rheumatism**" to describe a condition of muscular pain and acute rheumatic fever. In 1806, Edinburgh surgeon William Balfour identified nodules as fibrous thickenings in chronic muscular rheumatism, which caused pain. In 1824, he proposed the tender points as referred focal pain when pressed. In 1880, the American neurologist George Miller Beard clustered together widespread pain, fatigue and psychologic disturbance in what he named "**neurasthenia**"/"**myelasthenia**".

However, it was in 1904 that British neurologist Sir William R. Gowers firstly suggested the term "**fibrositis**" as a lumbago-like pain of the arm, stemming from inflammation of the fibrous tissues of the muscle. He also included fatigue and sleep disturbances. In the same year, Stockman provided a pathologic etiology for Gower's theory, by publishing the findings from seven biopsy studies of excised "fibrositic nodules", that he reported inflamed due to "small colonies of microbes". Nevertheless, these were found to be invalid later by Collins at the Mayo Clinic because of a lack of appropriate design. Not long after, in 1913, Llewellyn and Jones wrote "Fibrositis", the book that popularized and classified the illness, depicting its inflammatory characteristic, as well as aggravating factors such as climate changes and over-exertion. Slocumb was already referencing fibrositis in 1943, as the most common form of rheumatism to have a socioeconomic impact on the British economy, costing 60% worth of rheumatic disease insurance cases.

Australian Michael Kelly (1946) is responsible for the first mention of the CNS involvement in muscle pain disorders: he explained tender points as the origin of referred pain through the somatovisceral reflex theory, according to which central connections of neurons triggered heterotopic pain mechanisms. World War II brought a spike in the prevalence of rheumatic soldiers and diagnoses of fibrositis, which in the absence of an organic etiology and the clear stress and depression association, favored views such as the one of Edward Boland and William Corr (1947), who named the condition "**psychogenic rheumatism**". This organic versus psychogenic contention is still present today, peaking in

1937 through James Halliday, who argued that muscular rheumatism was symptomatic of chronic psychoneurotic state.

In 1968, Traut described fibrositis very closely to how it is currently considered: almost exclusive to the female gender, generalized aching and stiffness, fatigue, colitis, headaches, excessive worrying, poor sleep, and presence of tender points on physical examination. He considered regional pain as well, such as carpal tunnel syndrome, and brought forth axial pain as essential to the modern classification criteria of fibromyalgia. He shed light on the mind-body interaction in the pathogenesis of fibromyalgia. Further, it was in the 1970s that Smythe conceived working diagnostic criteria and specific tender points, many of which were reused in the 1990 American College of Rheumatology (ACR) criteria. He joined efforts with Dr. Harvey Moldofsky, whose unpublished electroencephalogram (EEG) findings proved absent stage-4 and scarce stage-3 sleep, thus highlighting the role of sleep in a leading manner. These findings were successfully confirmed in later studies, and added to Smythe's myriad contributions in providing a pathophysiologic basis for fibromyalgia (Inanici & Yunus, 2004).

In 1976, Hench published the term "**fibromyalgia**" as a type of nonarticular rheumatism in a review in Arthritis and Rheumatism. Later, in 1981, Yunus published the first controlled study of the clinical characteristics of the syndrome, confirming known symptoms and finding that the number of tender points was increased in fibromyalgia patients; and also discovering other symptoms (e.g. paresthesia, associated migraine and irritable bowel syndrome and restless leg syndrome) that were more common in fibromyalgia, meriting the condition a syndrome consideration. The data-based criteria born from this study were used until the 1990 ACR criteria. Already in 1999, Bennett published evidence of **central sensitization** in fibromyalgia on the basis of previous articles; and in 2000, Yunus extended the concept of central sensitization to other overlapping syndromes, such as temporomandibular disorder, chronic fatigue syndrome, myofascial pain syndrome, and migraine. He subsequently created the notion "**central sensitivity syndromes**" as a group terminology for these overlapping syndromes, that have grown with further studies to include conditions like multiple chemical sensitivity, giving an important significance to the concept nowadays. In the decade before the adoption of the 1990 criteria, there were contending definitions and diverse diagnostic propositions. These were examined in a blinded

study, performed by a multicenter criteria committee coordinated by Frederick Wolfe, that produced the official ACR criteria, later revised in 2010 and serving as the current clinical guidelines. The issue of histologic findings in fibromyalgia is still disputed, which makes for a syndrome that has been fraught with equivocacy in diagnosis and pathophysiology, and difficulties for physicians in managing it.

Concept and diagnostic criteria

Fibromyalgia (**FM** or **FMS** when considered as a syndrome) is a chronic health problem, a controversial but increasingly recognized syndrome since the **American College of Rheumatology (ACR)** produced the classification criteria in **1990**. These criteria formally established a diagnostic requirement of tenderness on pressure (**tender points**) in at least 11 of 18 specified sites and the presence of widespread pain (Wolfe et al., 1990), defined as axial pain, left- and right-sided pain, and upper and lower segment pain. Therefore, it allowed for **chronic widespread pain** to become the cornerstone sign of the syndrome, notwithstanding the multiple objections to a symptom-based diagnosis and the tender point count.

The American College of Rheumatology 1990 criteria (Wolfe et al., 1990)

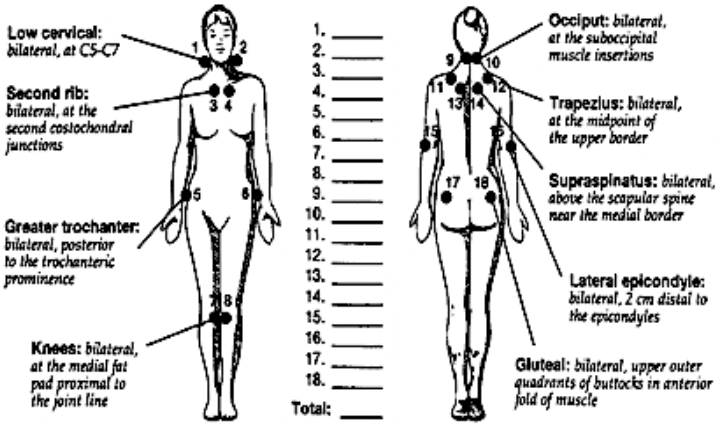
1. History of widespread pain, present for at least 3 months.
2. Pain in 11 of 18 tender point sites on digital palpation (see Figure 1).

Digital palpation should be performed with an approximate force of 4 kg/1.4cm².

For a tender point to be considered "positive", the subject must state that the palpation was painful.

Both criteria must be satisfied for a fibromyalgia diagnosis.

Figure 1. Tender points, adapted from Wolfe et al., 1990.



The 1990 criteria sparked the ignition and growth of clinical era for FM, and a scientific body and study began to grow insofar there were many neurobiologic findings, particularly the recognition of central pain sensitization. Many more symptoms and co-diagnoses such as chronic fatigue and irritable bowel syndrome were identified; some became increasingly acknowledged and known as key FM features (e.g. fatigue, cognitive symptoms). Moreover, a number of experts believed that tender point examination was blinding specialists to important considerations and an erroneous impression that FM is a "peripheral musculoskeletal disease" (Crofford & Clauw, 2002) was created. The tender point count was seldom carried out in primary care, where most FM diagnoses were performed, and when done it was frequently incorrect (Buskila, Neumann, Sibirski, & Shvartzman, 1997). This definition viewed FM as an almost exclusively female condition, due to the fact that women have notably more tender points than men: population-based studies have found that women are 10 times more likely to have 11 tender points in comparison to men (Wolfe, Ross, Anderson, & Russell, 1995). Some physicians considered that FM was more a **spectrum disorder** than could be described by dichotomous criteria (Wolfe & Michaud, 2009).

Ultimately, in **2010**, a revised definition of FM came into being (Wolfe et al., 2010). It was the culmination of many conjoint efforts to find **non-tender point** diagnostic criteria and integrate self-reported, **severity** scale-based symptoms on the basis of characteristic features of FM.

ACR 2010 revision (Wolfe et al., 2010)

Criteria

A patient satisfies diagnostic criteria for FM if the following 3 conditions are met:

1) Widespread pain index (WPI) ≥ 7 and symptom severity (SS) scale score ≥ 5
   or
   WPI 3-6 and SS scale score ≥9.
2) Symptoms have been present at a similar level for at least 3 months.
3) The patient does not have a disorder that would otherwise explain the pain.

Considering somatic symptoms in general, indicate whether the patient has:*

0 = no symptoms

1 = few symptoms

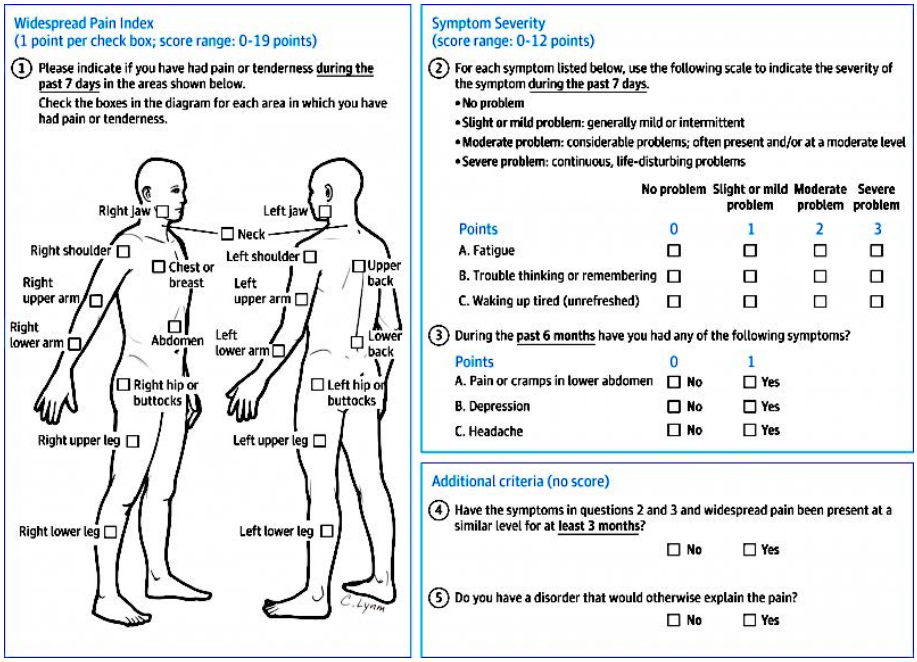
2 = a moderate number of symptoms

3 = a great deal of symptoms

The SS scale score is the sum of the severity of the 3 symptoms (fatigue, waking unrefreshed, cognitive symptoms) plus the extent (severity) of somatic symptoms in general. The final score is between 0 and 12. WPI score will be between 0 and 19 (see Figure 2).

* Somatic symptoms that might be considered: muscle pain, irritable bowel syndrome, fatigue/tiredness, thinking or remembering problem, muscle weakness, headache, pain/cramps in the abdomen, numbness/tingling, dizziness, insomnia, depression, constipation, pain in the upper abdomen, nausea, nervousness, chest pain, blurred vision, fever, diarrhea, dry mouth, itching, wheezing, Raynaud's phenomenon, hives/welts, ringing in ears, vomiting, heartburn, oral ulcers, loss of/ change in taste, seizures, dry eyes, shortness of breath, loss of appetite, rash, sun sensitivity, hearing difficulties, easy bruising, hair loss, frequent urination, painful urination, and bladder spasms.

Figure 2. ACR2010 criteria. The 2011 survey criteria for FM



Thus, FM became defined in its symptom spectrum through the **WPI** and composite **SS** scale. However, the study conducted to achieve the ACR 2010 revision proved that approximately 25% of patients diagnosed with FM did not satisfy the ACR classification criteria, due to the fact that diagnosis is based on severity assessments. This is a major difference from other illnesses such as rheumatoid arthritis, where the condition continues to exist even if patients don't meet diagnostic criteria, despite it possibly resulting of some health improvement. The SS scale was taken as the solution to this diagnostic problem, operating as a quantifier of FM symptom severity. Also, mood was not included for its evaluation difficulty and the inability to define it as a primary or a secondary feature of the illness. Hence, the conceptualization of the condition shifted towards a **dimensional** condition at the end of a **spectrum of polysymptomatic distress** (Wolfe & Walitt, 2013).

More recently, the ACR2010 criteria have been further simplified in another study by the same authors to a patient-driven survey format for use in **epidemiological** research. The physician's estimate of the extent of somatic symptoms was eliminated, and, in its place, the sum of specific self-reported symptoms was suggested. That is, the external assessment of somatic symptoms was substituted by the Symptom Severity Scale, or the sum of the severity (0-3) of the 3 symptoms (fatigue, waking unrefreshed, cognitive symptoms) plus the sum of the number of headache, pain or cramps in lower abdomen, and depression (0-3), during the previous 6 months, remaining the final modified SS score between 0 and 12. In addition, the WPI was added to the modified SS scale, creating a 0 to 31 FM symptom scale (FS), also known as "**fibromyalgianess scale**" (Wolfe et al., 2011). According to the study's findings, the FS was the best univariate predictor of FM: a score of 13 or higher classified 93% of patients correctly, with both sensitivity and specificity over 90%. However, the authors pointed out the limitations of relying on patients' self-diagnosis, and vouched for further epidemiological and clinical studies to assess these criteria's acceptance, reliability, and validity.

The FS was subsequently termed "**polysymptomatic distress scale**" (**PSD**), providing a continuous examination of FM intensity and content, and correlating with all general measures of distress (Wolfe, Brähler, Hinz, & Häuser, 2013). PSD allows for a flexible application in patients with any rheumatic disease and in the general population, and

in Wolfe's findings it became clear that a variable degree of polysymptomatic distress bears an influence in clinical outcomes (Wolfe et al., 2016).

2016 Changes to modified ACR FM diagnostic criteria

This revision makes the following changes to the FM criteria shown in the ACR 2010/2011 revision:

1) Changes criterion 1 to "widespread pain index (WPI) ≥ 7 and symptom severity scale (SSS) score ≥ 5 OR WPI 4–6 and SSS score ≥ 9" (WPI minimum must be ≥4 instead of previous ≥3).
2) Adds a generalized pain criterion (criterion 2), and one that is different from the 1990 widespread pain definition. The definition is: "Generalized pain is defined as pain in at least 4 of 5 regions. In this definition, jaw, chest, and abdominal pain are not evaluated as part of the generalized pain definition."
3) Standardizes and makes 2010 and 2011 criterion (criterion 3) wording the same: "Symptoms have been generally present for at least 3 months."
4) Removes the exclusion that regarding disorders that could (sufficiently) explain the pain (criterion 4) and adds the following text: "A diagnosis of FM is valid irrespective of other diagnoses. A diagnosis of FM does not exclude the presence of other clinically important illnesses."
5) Adds the FM symptom (FS) scale as a full component of the FM criteria.
6) Creates one set of criteria instead of having separate physician and patient criteria by replacing the physician estimate of somatic symptom burden with ascertainment of the presence of headaches, pain or cramps in lower abdomen, and depression during the previous 6 months.

The FM symptom (FS) scale is also known as the polysymptomatic distress (PSD) scale.
These combined, 2010 and 2011, single set of dual purpose criteria can continue to serve as diagnostic criteria when used in the clinic, but also as classification criteria when used for research.

Epidemiology and other relevant clinical aspects

FM is a chronic pain disorder affecting 0.5% to 5% of the general population, **more frequently in women** and common between the ages of 20 and 50 (Branco et al., 2009; White & Harth, 2001; Wolfe, Ross, Anderson, Russell, & Hebert, 1995). Nowadays, it is the **second most prevalent** disease within the chronic pain conditions, nearing an average of 2.3%-2.4% in Spain (Queiroz, 2013; Weiser et al., 2011). By using either the 2010 or the 2011 diagnostic criteria, notably more men are diagnosed, with the female/male ratio changing with respect to the 1990 criteria and being approximately 2:1 (Vincent et al., 2013). This ratio comes closer to those seen for the vast majority of chronic pain conditions in general (McBeth & Jones, 2007). The **etiology** of FM is yet to be elucidated, although there

is a growing number of experimental studies identifying myriad abnormalities in these patients, such as lower thermoalgesic and mechanical thresholds, increased sensitivity to multiple types of painful stimuli, and alterations in pain modulatory mechanisms (Ceko, Bushnell, & Gracely, 2012). Evidence now indicates that tender points are normally more sensitive to pressure pain in any given individual, and FM patients also have an enhanced pressure sensitivity at non-tender-points (Gracely, Grant, & Giesecke, 2003). However, to date there is **no clear biological signature**, and any underlying CNS and/or neurobiological involvement in the pathogenic explanation are yet to be fully ascertained (Walitt, Ceko, Gracely, & Gracely, 2016; Williams & Clauw, 2009). Its course seems to be influenced by a large number of physical, psychological, and environmental factors, and due to limited comprehension of causes, there are few widely accepted treatment approaches, excepting the recommendation of a **multidisciplinary** intervention, encompassing pharmacological, physical and psychological treatments. Clinical practice guidelines emphasize recommendations that have recently proved to be evidence-based, such as aerobic exercise, cognitive-behavioral therapy, amitriptyline, selective serotonin reuptake inhibitors (SSRI), serotonin and noradrenaline reuptake inhibitors (SNRI), anticonvulsive drugs, and mild opioids (Ángel García, Martínez Nicolás, & Saturno Hernández, 2016).

Following the proved effectiveness of an interdisciplinary strategy, recent literature has shown that priority must be given to **nonpharmacological** treatment, due to decreased risk, possibility of being maintained indefinitely, control of symptoms, and improvement of quality of life (Kumar & Jim, 2010; Nielson & Weir, 2001; Stanos, 2012). With respect to drug therapy, short-term results, collateral damage, limited and moderate results at best, and controversial or low level evidence make them adequate only for very specific situations, such as uncontrolled symptoms or intense and irruptive pain. In the case of opioids, there is increased mortality and important secondary effects, leading to them presenting a considerable public health problem in the United States, where their use has proliferated in nonmalignant chronic disease (Centers for Disease Control and Prevention, 2012).

Rheumatoid arthritis

Historical overview of the disease

The first clinical description of rheumatoid arthritis (RA) was made in 1800 by Augustin Jacob Landré-Beauvais. He noticed that the condition commonly affected poor women more often than men, and posited that these patients had a previously unknown illness he named *Goutte Asthénique Primitive*, or "**Primary Asthenic Gout**", opening research in the field of bone and joint disorders to study this disease. In 1859, English physician Alfred Garrod distinguished gout from other arthritic conditions in his *Treatise on Nature of Gout and Rheumatic Gout*, where he describes finding an excess of uric acid in the blood of these patients. He further differentiated RA as a condition, which he termed "**Rheumatic Gout**", thus leading research on its distinct etiology.

The fourth son of Alfred Garrod, Archibald Garrod, wrote in 1890 the *Treatise on Rheumatism and Rheumatoid Arthritis*, finally coining the term "**Rheumatoid Arthritis**". His work laid the groundwork for the Ancient Origin school of thought with respects to the illnesses' etiology, due to the fact that he claimed —albeit without supporting evidence— that there were skeletal findings with RA-related damage from around the world and dating as far back as ancient Egypt. His paleopathological theories led him to suggest that RA was not a modern era disease, but already existing in our ancestors. In the 20th century, American physician Charles Short refuted Archibald Garrod's **Ancient Origin** posit and reexamined the skeletal samples, finding diagnoses of ankylosing spondylitis, osteoarthritis, and gout but none for RA. Thus, Short's work became the most commonly credited basis of the **Recent Origins** perspective of RA, initiating a debate between both views that continues today.

Concept and epidemiology

RA is a chronic, systemic, progressive **inflammatory** disease whose cardinal features are swelling and tenderness of joints, as well as destruction of synovial joints, encompassing severe disability and premature mortality (Pincus et al., 1984; D. L. Scott, Symmons, Coulton, & Popert, 1987; Wolfe, 1996). Its worldwide prevalence ranges from 0.5% to 1%; it is **predominant in the female gender** (3:1) and its incidence peaks in the age range of 40 to

50 years, albeit in men it can have a later onset. In the Spanish adult population, its prevalence is 0.5%, and the incidence is 20 to 50 cases per 100,000 inhabitants per year (Aletaha et al., 2010; Carmona, Cross, Williams, Lassere, & March, 2010; Helmick, Felson, Lawrence, & al., 2008; Kvien, Uhlig, Ødegård, & Heiberg, 2006; Silman & Pearson, 2002).

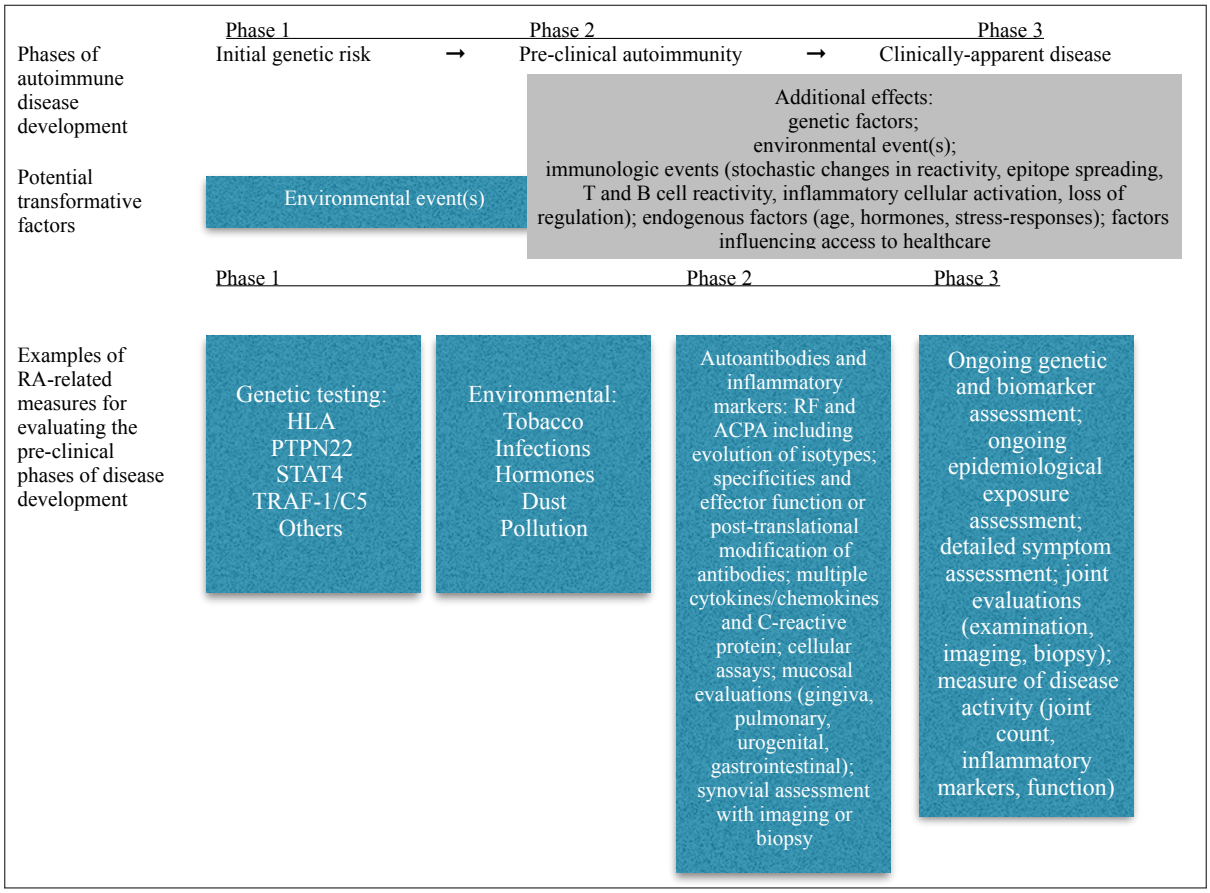
RA is considered an **autoimmune disease** (Firestein, 2003; Smolen, Aletaha, Koeller, Weisman, & Emery, 2007), given the presence of autoantibodies that can be a prelude to the clinical manifestation by many years, namely **rheumatoid factor (RF) and anti-citrullinated protein antibody (ACPA)**, which is tested as **anti-cyclic citrullinated peptide (anti-CCP)** (Aho, Heliövaara, Maatela, Tuomi, & Palusuo, 1991; Rantapaa-Dahlqvist et al., 2003). ACPA occurs in 50% to 80% of cases, thus facilitating a broad pre-clinical stage of RA that may exist with different phases, including systemic autoimmunity without symptoms, symptoms in the absence of arthritis, and unclassified arthritis (Gerlag et al., 2012). Risk factors may also differ according to the phase. Moreover, the destructive progression of the disease stems also from systemic and articular inflammatory load, although joint damage accumulates over time and is rarely apparent in the very early stages. These structural changes are the distinguishing trait of RA over other arthritic disorders, and can be evidenced through conventional radiography or other imaging techniques (Van der Heijde, 1995; Wolfe & Sharp, 1998).

However, the **causes** of RA remain largely **unknown**, existing universal agreement for only **three risk factors**: female gender, family history of RA and smoking tobacco, all of which cannot sufficiently describe the overall risk for the condition (Aho & Heliövaara, 2004; Symmons et al., 1997). A good candidate risk factor for the development of RA is **psychological stress**, given for instance that stressful life-events have preceded the onset in 86% of cases, and higher stress at the onset predicts worse disease prognosis (Cutolo & Straub, 2006; Feigenbaum, Masi, & Kaplan, 1979; Rimon & Laakso, 1985; J. G. Walker, Littlejohn, McMurray, & Cutolo, 1999). Additionally, enhanced psychological symptoms in RA are associated with worse patient outcomes, such as increased pain (Kojima et al., 2009), fatigue (Matcham, Ali, Hotopf, & Chalder, 2015), healthcare use (Joyce, Smith, Khandker, Melin, & Singh, 2009), and increased risk of premature mortality (Ang, Choi, Kroenke, & Wolfe, 2005).

Hence, the link between mental and physical health is bidirectional, as can be

expected from a chronic, painful medical condition that shares these **similarities with FM**: the experience of psychological distress may inflate the subjective severity of self-reported symptoms (Baumeister, Balke, & Härter, 2005), and it may also condition health outcomes by impacting health behaviors such as medication adherence and smoking (DiMatteo, Lepper, & Croghan, 2000; Pratt & Brody, 2010). Moreover, decreased physical activity can lead to loss of natural endorphins and increased pain (Covic, Adamson, Spencer, & Howe, 2003); and **common** mental disorders in this illness, such as **depression** and **anxiety**, are associated with immune dysregulation (Barnes & Adcock, 2009; McAllister-Williams, Ferrier, & Young, 1998). There is also evidence that suggests an association between depression and anxiety and RA, in which they impact perceptions and behaviors (Matcham, Ali, Irving, Hotopf, & Chalder, 2016).

Figure 3. Model of RA development*, adapted from Kolfenbach J et al. A prospective approach to investigating the natural history of preclinical arthritis (RA) using first-degree relatives of probands with RA. Arthritis Rheum. 2009; 61(12):1735-40



* Based on studies on RA and on prospective studies in other autoimmune diseases (e.g. type 1 diabetes mellitus), transition between phases may be due to interactions between genetic and environmental factors, and/or changes in immune reactivity. RF: Rheumatoid factor; ACPA: Antibodies to Citrullinated Protein Antigens; HLA: Human Leukocyte Antigen; PTPN22: Protein Tyrosine Phosphatase, Non-receptor type 22; STAT4: Signal Transducer and Activator of Transcription 4; TRAF-1/C5: encoding tumor necrosis factor receptor-associated factor 1 and encoding complement component 5.

The last decade (Figure 4) has brought an optimal use of d**isease-modifying antirheumatic drugs (DMARDs)**, in particular **methotrexate (MTX)**, which together with the discovery of **new biologic agents** have dramatically increased the success of the illness management (Doan & Massarotti, 2005; Pincus, Yazici, Sokka, Aletaha, & Smolen, 2003; Smolen et al., 2007). Additionally, an early therapeutic intervention improves clinical outcomes and decreases joint damage and disability (Van der Heide et al., 1996; Van Dongen et al., 2007). Severity of RA is commonly assessed using the **Disease Activity Score in 28 joints (DAS28)**, which is a composite score entailing clinician report of signs, patient self-report, and biochemical measures. To this end, it combines scores for 3 main areas: swollen joint count (SJC) and tender joint count (TJC), a biologic marker of inflammation (erythrocyte sedimentation rate, ESR, or C-reactive protein level, CRP), and a visual analog scale (VAS) score of global wellbeing. It was created to monitor RA activity and is used as a standard measure of therapeutic response, although it has recently risen as a main criterion to determine treatment options, in particular in the transition from traditional DMARDs to therapy with biologic agents (Prevoo et al., 1995). Nevertheless, the performance of individual measures of the DAS28 has yet to be fully explored, with the patient-reported VAS accounting for more than 25% of the overall score (Cordingley et al., 2014); whereas, evidence from other inflammatory conditions shows that different aspects of the illness may be more responsive to different treatments, with an increasing body of literature evidencing that the subjective patient perceptions of their RA have a strong influence on disease outcome (Hale, Treharne, & Kitas, 2007; Hill, Dziedzic, Thomas, Baker, & Croft, 2007; Kirby, Fortune, Bhushan, Chalmers, & Griffiths, 2000). Furthermore, not all RA patients respond to antiinflammatory treatment, proving that other pain mechanisms may be important: **noninflammatory pain mechanisms** in RA may comprise variations in central pain processing, or similar pathology, and they may share some common features with those found in other arthropaties, such as osteoarthritis, in which increased pain sensitivity may underline augmented central pain processing (Suokas et al., 2012). Indeed, **augmented pain processing** linked with pain and distress in people with established RA has been found to muddle assessment of inflammatory disease activity when using DAS28 (Joharatnam et al., 2015).

Figure 4. ACR medication recommendations for RA. Adapted from Singh, J. A. et al., 2015 American College of Rheumatology Guideline for the Treatment of Rheumatoid Arthritis. Arthritis Care & Research. 2016, 68(1): 1-26.

DMARDs	**Biologic Agents**		**Others**
	Non-TNF	**Anti-TNF**	
Hydroxychloroquine	Abatacept	Adalimumab	Tofacitinib
Leflunomide	Rituximab	Etanercept	Glucocorticoids (mainly
Methotrexate	Tocilizumab	Infliximab	prednisone)
Sulfasalazine		Certolizumab pegol	
		Golimumab	

DMARDs: disease-modifying anti rheumatic drugs; TNF: tumor necrosis factor. Anakinra, minocycline, cyclosporine, azathioprine, and gold were not included in the recommendations due to lack of new data and/or infrequent use.

Diagnostic criteria

The diagnosis of RA is often challenging, given the wide array of presentations, changes in illness course, and, especially, lack of a clinical or laboratory gold standard to establish the presence or absence of disease. Several attempts at the creation of classification criteria have been made, albeit with considerable limitations in application to the clinical setting (Sokolove & Strand, 2010). For instance, the classification criteria recommended by the ACR in **1987** had an elevated sensibility (94%) and specificity (89%) to diagnose the illness in advanced stages, with a consolidated presence of joint damage, but had a scarce capacity to identify early RA: its use in the clinical practice could postpone diagnosis and treatment (Arnett et al., 1988; MacGregor, 1995).

ACR 1987 revised criteria (Arnett et al.)

1. Morning stiffness	Morning stiffness in and around the joints, lasting at least 1 hour before maximal improvement
2. Arthritis of 3 or more joint areas	At least 3 joint areas simultaneously have had soft tissue swelling or fluid (not bony overgrowth alone) observed by a physician. The 14 possible areas are right or left PIP, MCP, wrist, elbow, knee, ankle, and MTP joints
3. Arthritis of hand joints	At least 1 area swollen (as defined above) in a wrist, MCP, or PIP joint
4. Symmetric arthritis	Simultaneous involvement of the same joint areas (as defined in 2) on both sides of the body (bilateral involvement of PIPs, MCPs, or MTPs is acceptable without absolute symmetry)
5. Rheumatoid nodules	Subcutaneous nodules, over bony prominences, or extensor surfaces, or in juxtaarticular regions, observed by a physician
6. Serum rheumatoid factor	Demonstration of abnormal amounts of serum RF by any method for which the result has been positive in <5% of normal control subjects
7. Radiographic changes	Radiographic changes typical of RA on posteroanterior hand and wrist radiographs, which must include erosions or unequivocal bony decalcification localized in or most marked adjacent to the involved joints (osteoarthritis changes alone do not qualify)

For classification purposes, a patient shall be said to have RA if he/she has satisfied at least 4 of these 7 criteria. Criteria 1 through 4 must have been present for at least 6 weeks. Patients with 2 clinical diagnoses are not excluded. Designation as classic, definite, or probable RA is *not* to be made.
PIP: proximal interphalangeal joints. MCP: metacarpophalangeal joints. MTP: metatarsophalangeal joints. RF: rheumatoid factor.

The recent advent of highly specific **biomarkers** for RA, not available in 1987, facilitates **early diagnosis** in the disease process: ACPA —or anti-CCP, in assays— have been identified as important for diagnosis and prognosis; as opposed to RF, IgG, IgA, and IgM auto-antibodies directed against the Fc portion of IgG, all of which have been considered the main serologic marker for the diagnosis of inflammatory arthritis for decades (Arnett et al., 1988; Nishimura et al., 2007). Also, acute phase reactants such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are inflammation markers non-specific for RA, which albeit helpful when elevated and being the best validated biomarkers to date, will never become a diagnosis gold standard due to their absence in 40% of patients with active RA and their presence in many inflammatory states (Wolfe & Michaud, 1994). Additionally, up to 30% of RA patients do not have usual biomarkers of RF, anti-CCP, elevated ESR, or CRP, which means that a considerable population of patients would be missed if too much attention were focused on current clinically available biomarkers (Sokka & Pincus, 2003; Wolfe & Michaud, 1994). Due to these early diagnosis limitations and because most RA patients in the United States are referred from primary care physicians, it

became essential that diagnostic criteria expedite early referral to rheumatologists (Nell et al., 2004), in order to also ensue early aggressive therapy.

Ultimately, these limitations and motivations spurred a combined task force of experts from the ACR and European League Against Rheumatism (EULAR) to work together towards developing the combined ACR/EULAR classification criteria for the diagnosis of RA. Thus, the new scoring system of the **2010 ACR/EULAR** criteria was created, as an endeavor not to develop diagnostic but **classification criteria**, including four domains: (1) symptom duration, (2) number and (3) types of joints involved, and (4) laboratory biomarkers of inflammation and autoimmunity. These new criteria target early disease, before joint imaging can reveal synovitis and erosions typical of RA, although they are suitable for diagnosis of established RA (Aletaha et al., 2010).

This classification aims to identify the patient subset that presents an otherwise unexplained inflammatory arthritis of a peripheral joint(s), for whom the risk of symptom persistence or structural damage is enough to be selected for DMARDs intervention (Aletaha et al., 2010). Insofar that these criteria take into account anti-CCP antibodies, which predict an increased disease activity and risk for radiographic progression, it potentially identifies a population with a more aggressive and erosive RA, who can therefore benefit more from an early and intensive therapy (Sokolove & Strand, 2010).

The 2010 ACR/EULAR classification criteria for RA (Aletaha et al., 2010)

	Score
Target population: Patients who	
1) have at least 1 joint with definite clinical synovitis (swelling)	
2) with the synovitis not better explained by another disease	
Classification criteria for RA (score-based algorithm: add score of categories A-D; a score of ≥6/10 is needed for classification of a patient as having definite RA)	
A. Joint involvement	
1) 1 large joint	0
2) 2-10 large joints	1
3) 1-3 small joints (with or without involvement of large joints)	2
4) 4-10 small joints (with or without involvement of large joints)	3
5) >10 joints (at least 1 small joint)*	5
B. Serology (at least 1 test result is needed for classification)**	
1) Negative RF *and* negative ACPA	0
2) Low-positive RF *or* low-positive ACPA	2
3) High-positive RF *or* high-positive ACPA	3
C. Acute-phase reactants (at least 1 test result is needed for classification)***	
1) Normal CRP *and* normal ESR	0
2) Abnormal CRP *or* normal ESR	1
D. Duration of symptoms	
1) <6 weeks	0
2) ≥6 weeks	1

Although patients with a score of <6/10 are not classifiable as having RA, their status can be reassessed and the criteria might be fulfilled cumulatively over time. "Joint involvement" refers to any swollen or tender going on examination, which may be confirmed by imaging evidence of synovitis. Distal interphalangeal joints, first carpometacarpal joints, and first metatarsophalangeal joints are excluded from assessment. Categories of joint distribution are classified according to the location and number of involved joints, with placement into the highest category possible based on the pattern of joint involvement. "Large joints" refers to shoulders, elbows, hips, knees, and ankles. "Small joints" refers to the metacarpophalangeal joints, proximal interphalangeal joints, second through fifth metatarsophalangeal joints, thumb interphalangeal joints, and wrists.

*In this category, at least 1 of the involved joints must be a small joint; the other joints can include any combination of large and additional small joints, as well as other joints not specifically listed elsewhere (e.g., temporomandibular, acromioclavicular, sternoclavicular, etc.).

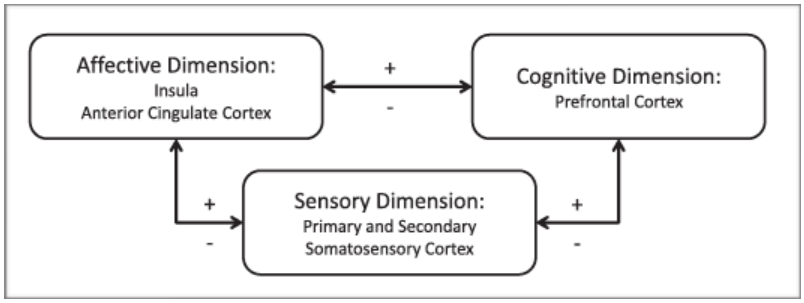
**Negative refers to IU values that are less than or equal to the upper limit of normal (ULN) for the laboratory and assay; low-positive refers to IU values that are higher than the ULN but 3 times the ULN for the laboratory and assay; high-positive refers to IU values that are 3 times the ULN for the laboratory and assay. Where rheumatoid factor (RF) information is only available as positive or negative, a positive result should be scored as low-positive for RF. ***Normal/abnormal is determined by local laboratory standards. Duration of symptoms refers to patient self-report of the duration of signs or symptoms of synovitis (e.g., pain, swelling, tenderness) of joints that are clinically involved at the time of assessment, regardless of treatment status.

ACPA: anticitrullinated protein antibody. CRP: C-reactive protein; ESR: erythrocyte sedimentation rate.

Chronic musculoskeletal pain and psychological aspects

Chronic pain encompasses a wide array of conditions, including arthritis and generalized pain conditions such as FM. Chronic pain is a complex experience that comprises myriad **sensory** and **emotional** facets and may range widely between individuals with context and meaning of the pain, as well as the **psychological** state (Bushnell, Ceko, & Low, 2013). Current models view chronic pain as the result of intricate interactions between **biological** and **psychosocial** factors, and whether or not it has an underlying organic cause in terms of medical pathology, it will pervasively have physiological and psychological consequences (Flor & Hermann, 2004). Indeed, the links between cognitive and emotional factors and pain perceptions can be explained by the connectivity of the **brain regions** controlling pain perception, attention or expectation, and emotions (Figure 5). Neuroimaging findings have shown that the activity of afferent and descending pain pathways is conditioned, among other unrelated factors to the pain stimulus, by attentional state and positive and negative emotions. The sophisticated framework of altered and interconnected brain areas in cognitive and emotional modulation of pain is known as a risk factor of chronic pain and **central amplification of pain;** it could underlie why chronic pain patients develop anxiety and depression, and also explains the physiology of central pain amplification (Bushnell et al., 2013).

Figure 5. Brain regions implicated in pain, from Crofford, 2015, Psychological aspects of chronic musculoskeletal pain.



Central pain amplification can be defined as perceived pain that cannot be fully explained through somatic or neuropathic processes and that results from physiologic alterations in pain transmission or descending pain modulatory pathways (Crofford, 2015a). Chronic regional pain is present in 20% to 25% of the population and chronic widespread

pain is present in approximately 10% of the population (McBeth & Jones, 2007). Thus, the physiological hallmark of centralized pain, central sensitization, or FM is **increased CNS pain processing**: in FM, this constitutes being diffusely tender to palpation, or diffuse hyperalgesia and allodynia co-occurrent to no identifiable diffuse peripheral inflammatory process of any body tissues. This would entail that the CNS, that is, the spinal cord and the brain, is augmenting pain processing somehow (Clauw, 2015). It is becoming increasingly evident that therapies best suited for peripheral nociceptive pain (e. g. NSAIDs, opioids, surgical procedures, and injections) are less likely to be effective in these patients (C. J. Woolf, 2011). It is common for one pain condition to develop the other, more centralized form of pain, as in the example of patients with inflammatory or degenerative joint disease that are almost four times as likely to also have FM, the paradigm of musculoskeletal central pain amplification syndrome (Haliloglu, Carlioglu, Akdeniz, Karaaslan, & Kosar, 2014).

Table 1. Underlying mechanisms in chronic pain states, adapted from Clauw, 2015.

Peripheral (nociceptive)	Neuropathic	Centralized
Pain due to inflammation or mechanical lesion in tissues	Damage or entrapment of peripheral nerves	Central alteration in pain processing leading to diffuse hyperalgesia/allodynia
Nonsteroidal anti-inflammatory drugs and opioids are responsive	Responds to peripheral and centralized pain interventions	Responsive to central nervous system-acting drugs operating on neurotransmitters involved in pain, sleep, and mood disturbances
Responds to procedures	Entrapment responds to surgery or injection	Paradigmatic examples • FM • Irritable bowel syndrome • Temporomandibular joint disorder • Tension headache
Paradigmatic examples • Acute pain due to injury • Osteoarthritis • Rheumatoid arthritis • Cancer pain	Paradigmatic examples • Diabetic neuropathic pain • Postherpetic neuralgia	

However, when talking about chronic pain, central pain amplification, and the organic and psychological underpinnings, other important issues beg the question. The affair of meaning and relationship of **somatization syndromes** and FM and RA remains debated and challenging, especially in the current context set forth in 2013 by the DSM-5. In the **Fifth Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5)**, the

American Psychiatric Association replaced the category of somatoform disorders with the new "**somatic symptom disorder**" (**SSD**). Patients have an SSD diagnosis if they have at least 1 severe somatic symptom (e.g. joint pain, headache, etc.) and at least one of the following: (1) Disproportionate and persistent thoughts about the seriousness of the symptoms, (2) Persistently high level of anxiety about health or symptoms, or (3) Excessive time and energy devoted to these symptoms or health concerns (American Psychiatric Association, 2013).

Research and clinical data direct attention on the current difficulty to prove whether fibromyalgia and rheumatoid arthritis with fibromyalgia patients exaggerate their reports; and doubt still hangs over validity and reliability of DSM-5 SSD to ascertain mental illness, particularly when diagnosis comes most often from generalists and this application to rheumatic disorders, such as rheumatoid arthritis and fibromyalgia, was not tested (Wolfe, Walitt, Katz, & Häuser, 2014). This is a lingering problem already in 1980, with somatization as one of the somatoform disorders being officially recognized a psychiatric diagnosis in version 3 of the DSM. Since then (DSM-III (American Psychiatric Association, 1980), DSM-IV (American Psychiatric Association, 1994), DSM-IV-TR (American Psychiatric Association, 2000)), somatization as a disorder became insufficient in its attempt to recognize "**medically unexplained symptoms**", symptoms not sustained by pathological findings and inevitably leading to the idea of inauthenticity. Moreover, DSM-IV criteria justified other symptoms as being potentially exaggerated, and the "Undifferentiated Somatoform Disorder" criteria were too easy to meet, resulting in a **lack of diagnostic acceptance** and coding in the clinic of both DSM-IV illnesses (Dimsdale et al., 2013).

In a broader perspective of this matter, symptom disorders are inextricably bonded to the idea of **somatization**, which organizes and attributes sense to symptoms. Conflict arises in elucidating the role of psychological factors when defining somatization. All rheumatic patients have symptoms, and it can be complicated to assess whether and why they would be reporting excessive symptoms or have disproportionate concerns regarding their health (Hidding et al., 1994). However, fibromyalgia seems to fit what Kurt Kroenke has termed a "**physical symptom disorder**", and it is being suggested as a simpler diagnosis than several current somatoform diagnoses. Physical symptom disorder "would consist of one or more physical symptoms currently present, not fully explainable by another medical or psychiatric

disorder (with the exception of functional somatic syndromes), causing functional impairment. Duration must be at least 6 months, and severity could be graded as mild, moderate, or severe using a 15-symptom checklist (PHQ-15) (Kroenke, 2006). This prospect, additionally to the concept of **polysymptomatic distress scale** and the finding of its high correlation (0.74) to PHQ-15 due to the shared somatic dimension, highlight the idea that **FM** is a **pain-predominant somatic symptom disorder** (Wolfe et al., 2014).

Patients with FM and other forms of central pain amplification are more susceptible to other **psychiatric disorders**, in particular depression, post-traumatic stress disorder and other anxiety disorders, and bipolar disorder (Arnold et al., 2006). Overall, patients with chronic musculoskeletal pain of any etiology are prone, in variable degrees, to heightened emotional, cognitive, and behavioral responses to chronic pain due to centrally amplified pain and pain-related SSD. This means that a patient with FM or rheumatoid arthritis (RA) or systemic lupus erythematous may coexist with SSD that could be treated comprehensively, for instance with **cognitive behavioral management strategies** (Crofford, 2015b). Indeed, many of the pathways important to chronic widespread pain and FM coincide with pathways also essential to mood; for instance, the **adrenergic and the serotonin pathways**, associated with FM features like autonomic unbalance, altered pain processing and modulation, sleep dysregulation, and anxiety. Furthermore, personality and affective traits such as depression, somatic awareness, and anxiety are linked to genetic changes in the serotonin pathway, albeit they are also related with the risk of chronic pain (Diatchenko, Fillingim, Smith, & Maixner, 2013). With respect to negative attentional bias in chronic pain, or broadened sensitivity and detection of aversive or unpleasant stimuli, it has been associated to the hippocampal formation and its connections to the anterior cingulate cortex and posterior insula. Pain catastrophizing is related to the anterior cingulate cortex, amygdala, and lateral prefrontal cortex. These findings all seem to lead to the idea that the dorsal anterior cingulate cortex and anterior middle cingulate cortex serve to the integration of negative affect, pain, and cognitive control (Shackman et al., 2011).

Mention must also be made of the **social** and **psychological** variables that have been found to produce poor outcomes in chronic musculoskeletal pain and that are known as risk factor for chronic widespread pain and FM. An important contributor to chronic pain is **perceived stress and stress response systems**: a history of childhood stress and current

psychosocial stress increases the risk for developing chronic centrally maintained pain (Gupta et al., 2007). For instance, in a population-based study to determine psychosocial factors that predicted new-onset chronic widespread pain, researchers selected a random sample of subjects from socio-demographically varied backgrounds then identified more than 3,000 who did not have pain at baseline and more than 300 that had new widespread musculoskeletal pain at follow-up examination. They found that the strongest predictors were premorbid somatic symptoms, illness behaviors, and sleep problems (Gupta et al., 2007). Moreover, longitudinal epidemiologic studies have shown in chronic pain and other somatic symptoms a history of childhood abuse and traumas, low educational level, social isolation, depression, and anxiety (Nicholl et al., 2009). In chronic pain, there is a negative link with pain, psychological status, disability, and quality of life that has been found independent from depression (Outcalt et al., 2015). Pain is associated with enhanced stress as well as decreased resilience and coping capability regarding traumatic life events (Van den Berg, Grievink, Stellato, Yzermans, & Lebret, 2005). Chronic pain inevitably can lead to social isolation, increased dependence, work disability and high healthcare expenses, and lower income can also be linked to more pain problems (Krueger & Stone, 2008; Poleshuck & Green, 2008).

In fact, psychological characteristics, such as a tendency to catastrophize, or the strength of one's belief in the ability to effectively respond to pain (known as "pain self-efficacy") have proved to be stronger predictors of pain outcomes than medical diagnosis or pain intensity (Arnstein, 2000). Despite the **many explanatory models** that have been developed to depict the complex interrelationships among the diverse pain-related factors, these models have thus far failed to explain the developmental origins of these individual differences (Meredith, Ownsworth, & Strong, 2008). Indeed, one of the core issues for any model that ascribes illness-vulnerability to relatively common events −trauma, loss, isolation or stress− lies in how the model explains individual differences in psychosocial exposure (Maunder & Hunter, 2001). Therefore, a stable, trait-like characteristic could serve as framework to explore many aspects of chronic pain, as has been the case recently, since attention has focused on linking **adult attachment and illness**, such as features of the pain experience (Mikulincer & Shaver, 2007a). **Attachment theory** may fill this gap, as it represents a convincing, evidence-based model for understanding the development of social and personality factors that may contribute to resilience or vulnerability regarding pain, and

comprehending the individual's past and present social environment and how people are likely to experience the therapeutic relationship (Meredith, 2016).

Attachment theory

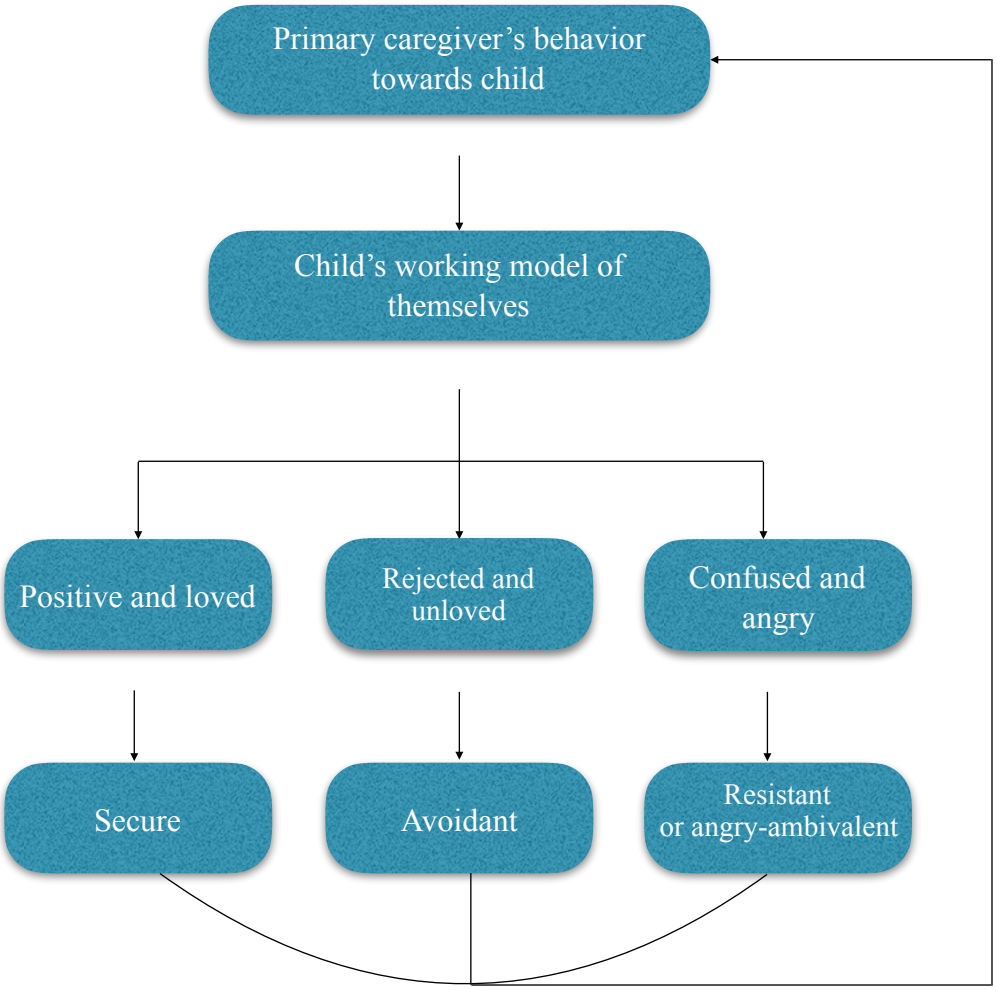
John Bowlby (Bowlby, 1982) postulated that humans are born with a **psycho-biological system** that motivates them to seek proximity to significant others, especially the mother, when confronted with need, with the aim of achieving a feeling of security. Thus, he provided a biological basis for understanding close, protective relationships, and posited that this **desire for proximity** has been selected in **evolution** as a fundamental need. Attachment behavior can be understood as a set of strategies that have been learned to achieve optimal proximity: this system includes a variety of non-verbal expressions of neediness and desire for proximity (e.g., crying, vocalizing), as well as active approaching behaviors to reestablish and maintain closeness (e.g., clinging, approaching) (Ainsworth, Blehar, Waters, & Wall, 1978). An infant increases its **chances of survival**, defenseless on its own, through this complex system of communications and behaviors, which are ultimately the normal and adaptive response of a mammal to threat. **Organized patterns of attachment behavior** emerge, or are learned, at a relatively fixed time for each species, which in humans is the second half of the first year (Ainsworth et al., 1978). Prior to learning the organized attachment behavior, signaling of needs is **innate** and responsive to stimuli. Therefore, in this pre-attachment period, appropriate proximity depends on **parental anticipation and sensitivity of infant needs and signals**, and hence increasingly relies on patterns of approach and withdrawal that are reinforced by the parent. This operant conditioning is the underlying framework of procedural memory in the infant, or learning “how to”; since the neurological systems involved in the development of declarative memories will reach their full form by age 2 to 3, the behavioral patterns learned at this critical stage are not available for conscious recall (Kandel, 1999). Thus, these patterns result from the interaction of environmental, parental in particular, and genetic factors in early development (Belsky, Rosenberger, & Crnic, 1995; Lakatos et al., 2000) and are **quite stable throughout life** (Ainsworth et al., 1978; E. C. Klohnen & Bera, 1998; Waters, Merrick, Treboux, Crowell, & Albersheim, 2000; Weinfield, Sroufe, & Egeland, 2000).

Early attachment relationships between a child and the primary caregivers enable the development of the child’s capacity for effective **social interaction** and for **mentalization**, which can be understood as the ability to infer the motivations, beliefs and

desires of others. Hence, children develop schemas, or sets of expectations that allow for rapid predictions about key situations, and most children learn to use internal and external resources to calm down after a stressor and learn how to tell a measured and precise personal story (Maunder & Hunter, 2009) .

Attachment behaviors occur in typical clusters that Mary Ainsworth classified in an attachment typology based on the standardized **Strange Situation**. In it, the child is presented with stressful situations that include being alone with a stranger, separation from the primary caregiver, and reunion (Ainsworth et al., 1978). ***Avoidant*** children scarcely cry during separation, treat the stranger in a similar manner as the parent, and refrain from contact on reunion; whereas ***secure*** infants experience distress at separation and actively try to obtain contact during reunion; and ***angry-ambivalent*** children also show separation distress and proximity-seeking on reunion, but combine seeking proximity with angry resistance (Ainsworth et al., 1978). Finally, the ***disorganized*** attachment category was termed later to describe subjects who present none of these coherent patterns, typically following interrupted or inconsistent parental care: once it is obtained, proximity is often not soothing and is accompanied by persistent anxiety (Bowlby, 1977). Altogether, these attachment phenotypes result from a sophisticated rapport between parent, child, and environment. In this context, individual differences in attachment behavior develop as stable patterns that preferentially highlight, for instance, expression versus suppression of separation protest, or a greater or lesser tendency towards seeking proximity (Maunder & Hunter, 2016).

Figure 6. Ainsworth's attachment model in children.



Ultimately, and when applying this concept to adult studies, an attachment relationship is any relationship in which proximity to the other has an impact on the felt security, therefore including the relationship of patient and clinician. According to Bowlby's theory (Figure 7), what makes the attachment style significantly stable over time is the notion of the **internal working model**, or **models of self and other** that range from very positive expectations (resiliency of self, responsiveness of other) to very negative expectations (fragile or unable self, unreliable other), which influence appraisals of, and behavior in, subsequent relationships. Since these expectations are relatively independent, a **four-category and two-dimensional model of attachment** can be drawn from the position of an individual's attachment status on each of these dimensions (Bartholomew & Horowitz, 1991; Bowlby, 1973; K. A. Brennan, Clark, & Shaver, 1998; Mikulincer & Shaver, 2007a). Thus, a person who has positive expectations of both self and other is ***secure***, with an internalized sense of meriting care, of being effective in bidding care when needed, and a notion of

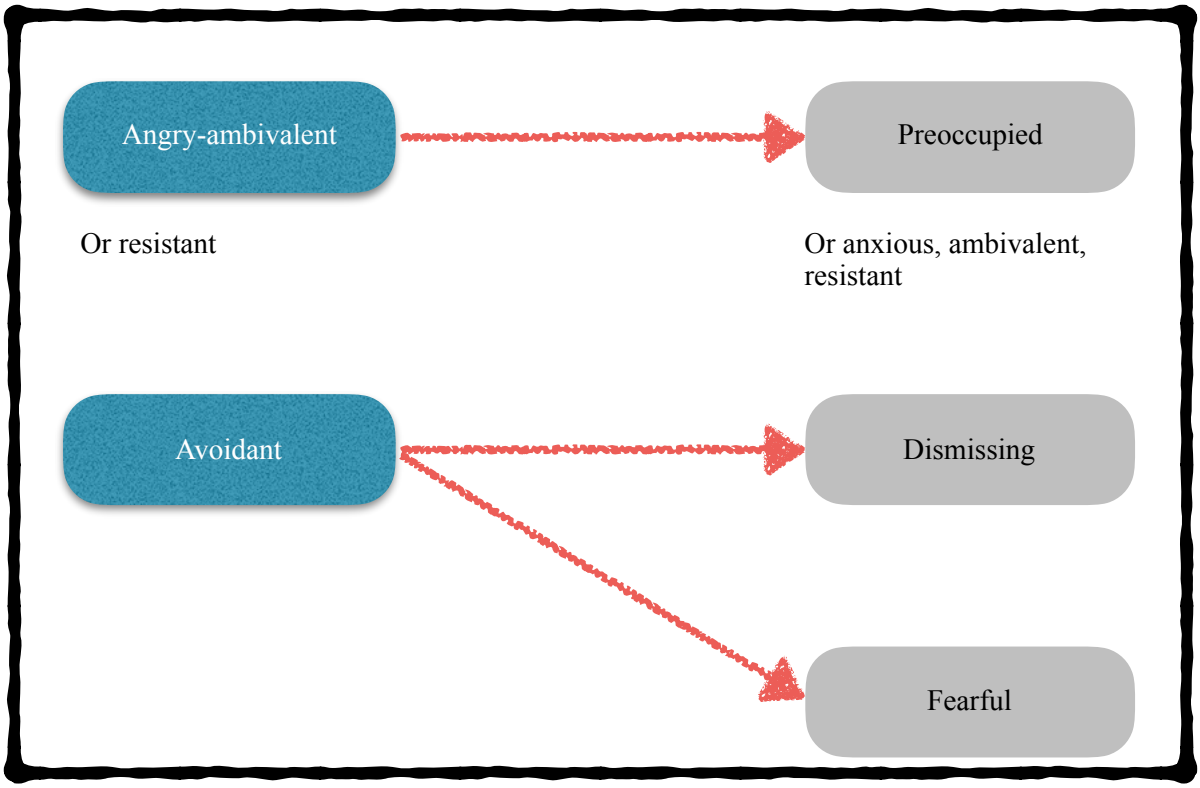
personal efficacy in managing most stressors independently (M. L. West & Sheldon-Kellor, 1994). Secure subjects are adaptable, capable, trusting, and understanding; they keep an adequate balance between emotional needs and personal autonomy (E. Klohnen, John, OP, 1998; Mayseless, 1996).

On the other hand, **insecure** attachment encompasses the categories of preoccupied, dismissing, and fearful. A ***preoccupied*** person expects to inadequately cope with stress but expects more positively of others; this kind of attachment is linked with excessive care-seeking, separation protest, and fear of loss (M. L. West & Sheldon-Kellor, 1994). Despite the care-seeking, the resulting appeasement from contact is partial and transitory. Preoccupied individuals are described as dependent, anxious, emotional, impulsive, and approval-seeking (E. C. Klohnen & John, 1998). Further, a ***dismissing*** person distrusts the effectiveness of social support but has a positive view of themselves, asserting their independence. People in this category highly value self-sufficiency, but the associated lack of trust and avoidance of intimacy conveys the underlying insecurity. Crisis may arise in situations that involve surrendering control and depending on others, such as hospitalization. The dismissing type is characterized by coldness and disregard of interpersonal relationships, and being success-oriented and competitive (Bartholomew & Horowitz, 1991; Feeney, Noller, & Hanrahan, 1994; Mayseless, 1996). Lastly, ***fearful*** attachment was introduced by Kim Bartholomew in 1991, who described it as the combination of negative expectations of both self and other. A fearful person is featured as doubting, cautious, shy, self-conscious, and mistrustful (E. C. Klohnen & John, 1998). Most studies do not distinguish between fearful and avoidant attachment, commonly joining them in a composite category of "**avoidant**".

Figure 7. Bowlby's attachment theory.

Internal working of others (avoidance)		Internal working model of self (dependence)	
		Positive (low dependence)	Negative (high dependence)
	Positive (low avoidance)	Secure	Preoccupied
	Negative (high avoidance)	Dismissing	Fearful

Figure 8. Adult correlates of infant insecure attachment categories.



As per the **prevalence** of attachment styles in different age groups, a 31-year longitudinal study indicated that there may be trends over time in individuals, with a modest decline in preoccupied attachment (approximately 20% in college to 5% to 8% in middle age), a similar increase in secure attachment, and stable avoidance scores throughout adult life (about 20% to 25%) (E. C. Klohnen & John, 1998; Mickelson, Kessler, & Shaver, 1997). One study also analyzed the distribution of adult attachment representations in North American non-clinical mothers using the Adult Attachment Interview (George, Kaplan, & Main, 1985) classifications. The results showed 23% of dismissing attachment, 58% secure mothers, 19% preoccupied attachment representations, and 18% coded for unresolved loss or other trauma (Bakermans-Kranenburg & van IJzendoorn, 2009).

Since an attachment pattern is the product of an internal working model, which neurologically has developed due to highly salient conditioning in the first attachment, attachment type is thus understood as a **disposition** toward certain perceptions of others and self and certain preferred strategies that will be **triggered** by a perceived **threat**. In other words, the origin of attachment behavior is chiefly context dependent, a state phenomenon,

but the internal working model that provides consistency to the type of behavior that emerges is a trait (Maunder & Hunter, 2001). Therefore, these models are considered to be an essential source of continuity in the functioning of the attachment system throughout life (Mikulincer & Shaver, 2003).

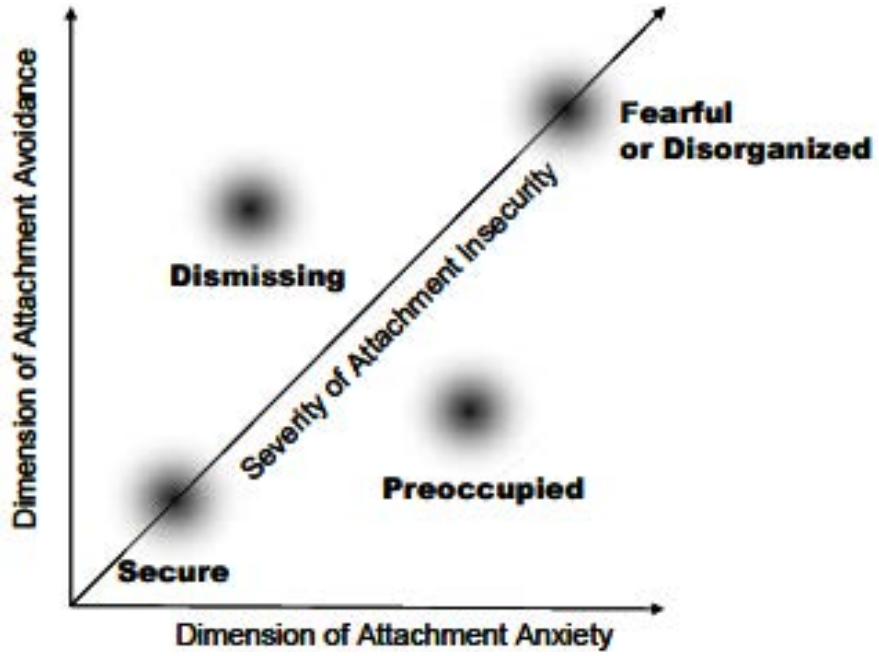
Importantly, in attachment between parents and the child and in romantic relationships, an attachment figure is not replaceable. That is, **attachment functions** are provided by specific people, and unlike material support, which may come from anyone who behaves in the appropriate manner, safe haven and secure base functions may only be offered by a small group of individuals (Maunder & Hunter, 2016). Adult attachment figures can be identified by their three functions: being the one to whom there is a **desire of closeness**, the person one turns to at times of adversity (**a safe haven**), and the person on whom one relies on to go out and explore independently (**a secure base**) (Hazan & Shaver, 1994). Proximity-seeking functions are activated at times of attachment strain or threat, such as when a person is separated from those who offer attachment functions or when someone is in pain or unable to fend for themselves. Therefore, these are conditions that are commonly present when illness requires investigation and treatment (Hunter, Maunder, & Lan Le, 2016).

Individual differences in adult attachment have been described differently, according to **theoretical and measurement considerations** (Maunder & Hunter, 2014). Attachment measures assign individuals to categories of attachment style or estimate the degree to which various dimensions of attachment style are present. These dimensional models of adult attachment, such as work by Brennan, Clark, and Shaver (1998), conceptualize attachment theory into **two axes of insecurity** (Figure 9), or relatively orthogonal dimensions: **attachment anxiety** (or negative sense of self) and **attachment avoidance** (negative sense of others). Attachment anxiety features a separation expectation, abandonment, or insufficient love; a preoccupation with how available and responsive others are; and hyperactivation of attachment behavior. On the other hand, attachment avoidance is characterized by devaluation of the importance of close relationships, elusion of intimacy and dependence, self-reliance, and relative deactivation of attachment behavior (K. A. Brennan et al., 1998).

In the instance of **dimensional** approaches, if standard and consented cutoff points are established, categories can be produced from dimensional scales. **Categorical** measures are criticized from a theoretical perspective, due to the fact that they assume differences among

people within a category are “unimportant or do not exist” (Mikulincer & Shaver, 2007b, p. 85), and, from an analytical view, for their limited statistical power in comparison to dimensional measures. However, the categorical approach is often preferred in clinical settings due to their resemblance to illustrative “textbook cases” (Maunder & Hunter, 2009). Additionally, and in analytical terms, when a categorical construct is measured using a dimensional scale, part of the observed variance is spurious: the question of whether attachment phenomena are inherently categorical or dimensional remains debated (Ravitz, Maunder, Hunter, Sthankiya, & Lancee, 2010).

Figure 9. Patterns of adult attachment, from Maunder, R. G. and Hunter, J. J., A prototype-based model of adult attachment for clinicians. Psychodynamic Psychiatry. 2012;40(4):549-73.



Moreover, **Bartholomew and Horowitz’s four-category model** helpfully reconciles categorical and dimensional models. Drawing on the theory of Bowlby, it posits two types of internal working models, one of the self and one of others, each dichotomized as positive or negative to result in four theoretical attachment styles (Bartholomew & Horowitz, 1991). It also defines categories that correspond to combinations of polarized positions on the dimensions of attachment anxiety and attachment avoidance. Hence, the subsequent four-category scheme has *secure*, *preoccupied*, *avoidant/dismissing*, and *avoidant/fearful* categories. *Secure* attachment can be understood as a relative absence of attachment anxiety

and attachment avoidance; the *preoccupied* style is postulated as a relative absence of high attachment anxiety and low attachment avoidance; the *dismissing* pattern is the combination of high attachment avoidance and low attachment anxiety, and *fearful* attachment is conceptualized as high insecurity on both dimensions of attachment avoidance and anxiety.

However, none of the **measures** of adult attachment currently used were developed for psychosomatic research (Ravitz et al., 2010). Adult attachment is becoming increasingly important in psychosomatic research because attachment affects many biopsychosocial phenomena, such as social functioning, stress response and coping, psychological wellbeing, health behavior, and morbidity (Ciechanowski & Katon, 2006; Ditzen et al., 2008; Maunder & Hunter, 2001; Maunder, Lancee, Hunter, Greenberg, & Steinhart, 2005; Maunder, Lancee, Nolan, Hunter, & Tannenbaum, 2006; Meredith, Strong, & Feeney, 2006a; Schmidt, Nachtigall, Wuethrich-Martone, & Strauss, 2002; Waller, Scheidt, & Hartmann, 2004b), but it is complicated to ask of patients with serious medical conditions to report on attitudes towards relationships to significant others. In effect, when choosing an attachment measure, the relative relevance of questions or items to the situations of the evaluated subjects must be taken into consideration (Table 2).

Table 2. Self-report questionnaires

Scale	Authors	Relationship focus	Categories/dimensions measured
Adult Attachment Styles	Hazan and Shaver	Intimate relationships	Secure, avoidant, anxious/ambivalent
Adult Attachment Questionnaire (AAQ)	Simpson Simpson et al.	Partner	Attachment anxiety, attachment avoidance
Avoidant Attachment Questionnaire for Adults (AAQA)	West and Sheldon-Kellor	General	Maintains distance in relationships, priority on self-sufficiency, attachment relationship is a threat to security, desire for close affectional bonds
Adult Attachment Scale (AAS) and Revised-Adult Attachment Scale (RAAS)	Collins and Read Collins	Partner	Comfort with closeness, comfort with depending on others, anxious concern about abandonment

Scale	Authors	Relationship focus	Categories/dimensions measured
Attachment History Questionnaire (AHQ)	Pottharst	Partner	Categories: secure, insecure; dimensions: secure attachment, parental discipline, peer system
Attachment and Object Relations Inventory (AORI)	Buelow et al.	Parents, peers, partners, and self	View of self as: warm, secure, interdependent, not anxious versus distant, dependent/ preoccupied, anxious; view of others as: emotionally accessible, responsive versus not accessible, unresponsive
Attachment Style Questionnaire (ASQ)	Feeney et al.	Close relationships	Discomfort with closeness, need for approval, preoccupation with relations, viewing relationships as secondary to achievement, lack of confidence
Continued Attachment Scale (CAS)	Berman et al.	Parents	Cognitive and behavioral components of parental attachment
Client Attachment to Therapist Scale (CATS)	Mallinckrodtet et al.	Therapist	Secure, avoidant/fearful, preoccupied/merger
Experiences in Close Relationships (ECR) and Experiences in Close Relationships-Revised (ECR-R)	Brennan et al. Fraley and Shaver	Partner (or general)	Attachment anxiety, attachment avoidance
Measure of Attachment Qualities (MAQ)	Carver	General	Security, avoidance, ambivalence/worry, ambivalence/merger
Mother Father Peer Scale (MFPS)	Epstein	Parents and peers	Acceptance/rejection, independence/ overprotection, defensive idealization
Maternal Separation Anxiety Scale (MSAS)	Hock et al.	Child	Maternal separation anxiety, perception of separation effects on child, employment-related separation concerns
Parental Attachment Questionnaire (PAQ)	Kenny	Parents (of adolescents)	Affective quality of relationships, fostering of autonomy, provision of emotional support
Parents of Adolescents Separation Anxiety Scale (PASAS)	Hock et al.	Adolescent children	Anxiety about adolescent distancing, comfort with secure base role

Scale	Authors	Relationship focus	Categories/dimensions measured
Parenting Bonding Instrument (PBI)	Parker et al.	Parents	Parental care, parental protection
Reciprocal Attachment Questionnaire for Adults (RAQA)	West et al., West and Sheldon, and West and Sheldon-Kellor	Most important attachment figure	Proximity seeking, separation protest, feared loss, perceived availability, angry withdrawal; compulsive: care giving, self-reliance, and care seeking
Relationship Questionnaire (RQ)	Bartholomew and Horowitz	Partner (or general)	Secure, preoccupied, dismissing, fearful
Relationship Scales Questionnaire (RSQ)	Griffin and Bartholomew	Partner (or general)	Categories: secure, preoccupied, fearful, dismissing; dimensions: model of self and model of other
Revised Inventory of Parental Attachment (R-IPA)	Johnson et al.	Children	Trust/avoidance, symptom distress, social role, interpersonal relations, physical aggression
Vulnerable Attachment Style Questionnaire (VASQ)	Bifulco et al.	Support	Insecurity, proximity seeking
Adapted from Ravitz, P. et al., 2010.			

In current **psychosomatic research**, the most commonly used self-report scales are the ECR-R, the RQ, the AAS, and the ASQ (Ravitz et al., 2010). **Self-report measures** directly evaluate conscious attitudes regarding separation, loss, intimacy, dependence and trust, all of which constitute nuclear features of the predictive schemas that are believed to support the stability of attachment styles over time (Maunder & Hunter, 2009).

In spite of this, there are also neurobiological and genetic hypotheses supporting the notion of attachment (Crawford et al., 2007; Donnellan, Burt, Levendosky, & Klump, 2008; Dutton, 2011; Gillath, Shaver, Baek, & Chun, 2008). This would entail a basis for the development of stable emotional bonds that would transcend the social interactions in childhood and thereafter.

However, it must be noted that **internal working models can change** due to interpersonal and emotional relevant life circumstances, despite the essential continuity of the attachment system (Bowlby, 1982; Davila & Cobb, 2004). This entails that psychotherapy, for instance, offers a significant emotional experience which may change conflictive working

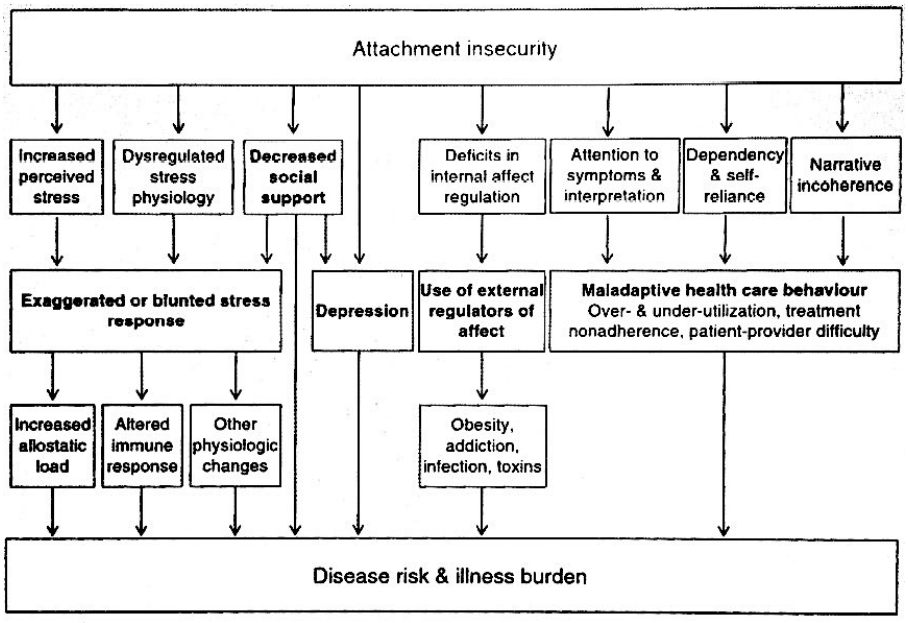
models (Bowlby, 1988). In this regard, a review of studies investigating changes in attachment style over the therapeutic course has found that increases in attachment security or decreases in attachment insecurity are linked with a better outcome (Mikulincer, Shaver, & Berant, 2013). Ultimately, there is a growing body of evidence supporting the theoretical associations between the **quality of patient-provider relationship**, healthcare utilization, and other medical outcomes (Ditzen et al., 2008; Dozier, Cue, & Barnett, 1994; Dozier, Stovall-McClough, & Albus, 2008; Gunnar, Brodersen, Nachmias, Buss, & Rigatuso, 1996; Maunder & Hunter, 2016; Maunder, Lancee, et al., 2006; Maunder, Panzer, et al., 2006; Meredith et al., 2006a; Meredith, Strong, & Feeney, 2007; Schmidt, Strauss, & Braehler, 2002; Waller, Scheidt, & Hartmann, 2004a). That is, in an analogous way to how the parent's secure attachment organization provides the internal resources to respond to the infant appropriately and empathetically, so it seems that a clinician's secure attachment organization may provide the necessary resources to respond sensitively to patients (Dozier et al., 1994). Furthermore, health outcomes are understandably linked to the patient-provider relationship since they each are a manifestation of underlying attachment dynamics: at times of health-related threat or distress, individuals engage in attachment attitudes and behaviors (proximity seeking or avoidance, trust or distrust, expression or suppression of distress) with healthcare providers in a similar way than within the context of a romantic relationship (Ciechanowski, Walker, Katon, & Russo, 2002; Maunder & Hunter, 2016). In effect, attachment behavior is always activated at times of sufficient stress, and the typical motives of the majority of healthcare interactions −illness, injury, and loss− are nuclear triggers of attachment behavior (Hunter & Maunder, 2016).

Research on patterns of adult attachment in medical patients

When an individual's attachment figures are not reliable and supportive, proximity seeking doesn't relieve distress, the safe haven is undermined, negative models of self and others emerge, and the likelihood of emotional problems and maladjustment increases with time. Insofar this attachment theory has been tested in studies of adults, there has been a focus on what Hazan and Shaver termed **attachment style** (Hazan & Shaver, 1987), or the systematic pattern of relational emotions, expectations, and behavior yielded by one's attachment history. Mikulincer and Shaver (Mikulincer & Shaver, 2007a) reviewed hundreds

of cross-sectional, longitudinal, and prospective studies of clinical and non-clinical samples and found a common link between attachment insecurity and a wide array of **mental disorders**, such as depression (Catanzaro & Wei, 2010), clinically significant anxiety (Bosmans, Braet, & Van Vlierberghe, 2010), obsessive-compulsive disorder (Doron, Moulding, Kyrios, Nedeljkovic, & Mikulincer, 2009), post-traumatic disorder (Ein-Dor, Doron, Solomon, Mikulincer, & Shaver, 2010), suicidal tendencies (Gormley & McNiel, 2010), eating disorders (Illing, Tasca, Balfour, & Bissada, 2010), and as a key feature in many **personality disorders** (Crawford et al., 2007; Meyer & Pilkonis, 2005). However, other than this causal link between attachment and psychopathology, which has been proved bilateral as psychological problems can also increase attachment insecurity (Mikulincer & Shaver, 2012), researchers have also amply described how **attachment insecurity** may contribute to **disease and health** (Ciechanowski, Walker, et al., 2002; Feeney, 1995; Maunder & Hunter, 2001; McWilliams & Bailey, 2010).

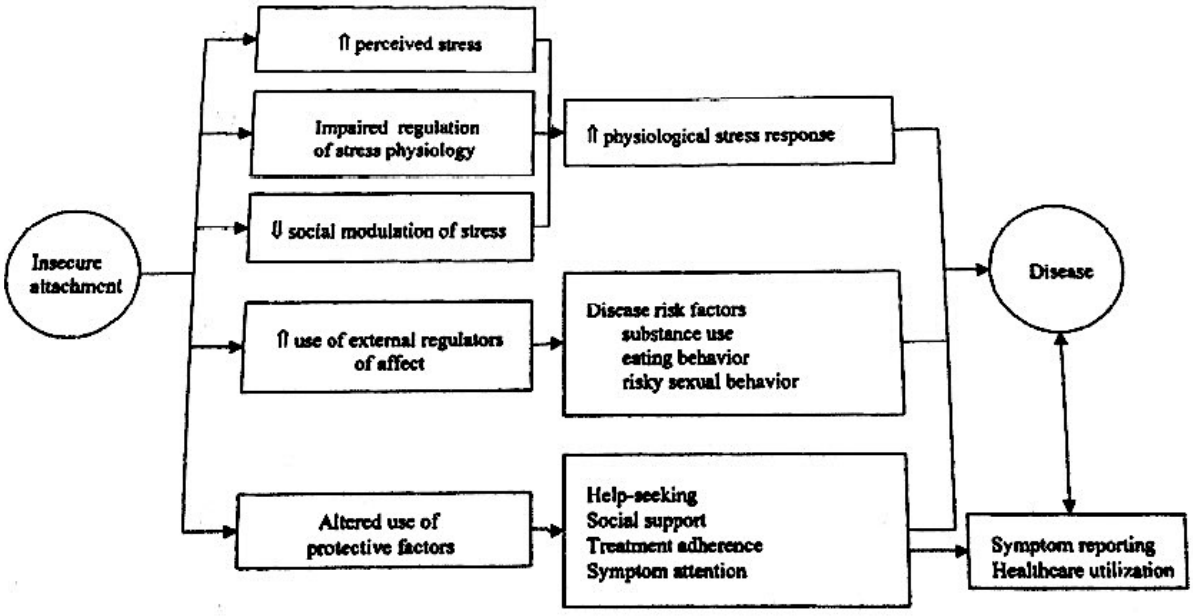
Figure 10. Mechanisms by which insecure patterns of attachment may contribute to disease, from J. J. Hunter and R. G. Maunder (2016).



Authors such as Maunder and Hunter have theorized a model of insecure attachment contributing to disease, which they have furnished over time (Figure 10 is the most recent, Figure 11 is from 2001) through reviewing the literature. This most recent model strives to

show the intricacy of relationships, how they work and their applications in provider-patient interactions, and how experiencing insecurity in relationships over a lifetime increases the risk of sickness. Relationship patterns linked to feeling insecure enhance the likelihood of chronic illness in **several possible ways**: by contributing to exaggerating physiological responses to stress, through ineffective stress management by social support, and also by using mood-altering drugs to buffer emotional discomfort. Other behaviors closely associated to insecure attachment patterns that also contribute to poor health outcomes are maladaptive health behaviors, such as excessive help-seeking, and nonadherence to treatment recommendations (Hunter et al., 2016).

Figure 11. Model of hypothesized mechanisms by which attachment security could contribute to disease, from Maunder, R. G. and Hunter, J. J., 2001.



On the other hand, the **general model** from which the current one stemmed (Figure 11) shows how insecure attachment may follow **three different paths** that could account for contributions to disease: disturbances of stress regulation, use of external regulators of affect, and nonuse of protective behaviors (Maunder & Hunter, 2001).

According to the authors, the first path of stress is relevant due to the developmental link between **attachment and stress,** in which the stress response is evolutionally selected to be triggered by an external threat and the attachment system is meant to increase security when there is an environmental threat. The authors conceive three ways in which attachment

molds individual differences in the stress response, the first being that insecurity may increase perceived stress. This may be illustrated in the fact that preoccupied attachment involves a self-perception of vulnerability, which could explain a lower threshold for attachment behavior; whereas avoidant attachment entails an attitude of interpersonal distrust that may lead to intimacy or interdependence situations being experienced as threatening. Secondly, attachment insecurity may also influence the intensity or duration of the physiological stress response. An example of this relationship is found in the association between attachment insecurity and the intensity of the cardiovascular and cortisol response to acute stress in children and in adults, in a study by Sroufe and Waters via the Strange Situation (Sroufe & Waters, 1977). Thirdly, attachment pattern may condition the success of social support in buffering stress, as well. In spite of the lingering debate concerning association and mechanism (Uchino, Cacioppo, & Kielcolt-Glaser, 1996), social support has been repeatedly found to be a mediator of illness and is beneficial to a variety of health outcomes (S. Cohen & Wills, 1985; House, Landis, & Umberson, 1988). Additionally, the theory of attachment suggests that attachment relationships provide "felt security" when there is a threat or a loss, thus being a possible effective mechanism for reducing stress. Also, attachment style determines the degree to which social contact is sought at times of stress. Importantly, the social support received may be conditioned by the nature of the medical condition; in this regard, there is research showing that chronic fatigue syndrome and FM patients may have less social support, and perceive more negative social relationships, than the general population (Anderson & Ferrans, 1997; Davis, Zautra, & Relch, 2001).

The second path regards insecurity altering the **use of external regulators of affect**. It is known that insecure attachment yields deficits in internal affect regulation (Kobak & Sceery, 1999; Mikulincer, 1999), and therefore it is expected to be linked to a greater use of external regulators. There are several behavioral strategies used to regulate dysphoric affect, such as soothing, distracting, or exciting, that also constitute risk factors of disease: smoking tobacco, drinking alcohol, using other psychoactive drugs, eating in excess or scarcely, and engaging in unsafe sexual activity (e.g. Bassman, 1991; DeFronzo & Pawlak, 1993; Feeney & Raphael, 1992; Magai, 1999; Sharpe et al., 1998; Springs & Friedrich, 1992).

The third and last path indicates that insecure attachment may also lead to **failure or nonuse of protective factors of health**. Among the most important protective factors, there

is the aforementioned social support, but also treatment adherence and symptom reporting. Diabetes is one epitome of treatment adherence becoming essential, and Viederman and Hymowitz (1988) theorized a model of **diabetic** control in which the success of patients engaging and participating in their treatment is determined by early relationship experiences. On a related note, it is possible to find examples of attachment linked to relationship with care providers in diabetic patients (Ciechanowski & Katon, 2006; Ciechanowski, Russo, et al., 2006). Also, another study used HbA1c (a well-known index that is elevated in poorly controlled diabetes) as a measure of diabetic control in 15 type 1 diabetics, and described that avoidantly attached subjects had considerably higher levels of HbA1c than in secure or preoccupied attachment (Ciechanowski, Hirsch, & Katon, 2002). As per symptom reporting, there are many studies evidencing its association to attachment insecurity (e.g. Ciechanowski, Walker, et al., 2002).

Indeed, recent reports show that insecure attachment is particularly associated with impaired stress regulation (Flor & Hermann, 2004), increased symptom reporting (Ciechanowski, Walker, et al., 2002), medically unexplained symptoms (Ciechanowski, Katon, Russo, & Dwight-Johnson, 2002), and somatoform disorders (Waller et al., 2004b). Insecurely attached subjects without chronic pain conditions also proved to have increased catastrophizing hypervigilance, decreased pain thresholds and self-efficacy to episodes of acute pain or experimentally induced pain (Martínez, Miró, Sánchez, Mundo, & Martínez, 2012; Meredith, Strong, & Feeney, 2006b; C. L. Wilson & Ruben, 2011). In light of this evidence, it has been suggested that attachment insecurity is related to the development of chronic pain through dysfunctional reactions to episodes of acute pain (Porter, Davis, & Keefe, 2007).

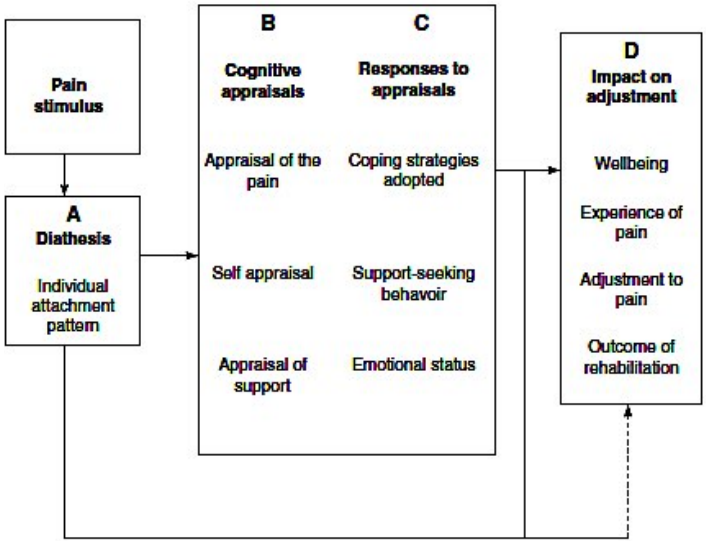
Attachment-based theoretical approaches of pain began to emerge in the 1980s and early 1990s: researchers mainly contended the pain experience as a form of threat activating the attachment system (N. E. Andrews, P. J. Meredith, J. Strong, & G. F. Donohue, 2014). This would lead to a cascade of behaviors that feature insecurely attached individuals as being at greater risk for chronic pain and being less able to cope with established chronic pain (Andreson & Hines, 1994; Kolb, 1982; Mikail, Henderson, & Tasca, 1994).

Nevertheless, results are mixed concerning direct links between attachment patterns and pain intensity and disability among **chronic pain** patients (Porter et al., 2007): some

authors found no direct association between insecure attachment and pain intensity (Ciechanowski, Sullivan, Jensen, Romano, & Summers, 2003; Meredith et al., 2006a) or disability (Meredith et al., 2006a) in samples with diverse chronic pain conditions, while others reported more varied results with homogeneous subsamples and different attachment measures. For instance, romantic anxious attachment was related to pain intensity and disability in arthritis patients (L. McWilliams, B. Cox, & M. Enns, 2000), and romantic fearful attachment was associated to pain severity in lung cancer patients (Rumble, Keefe, Porter, Miller, Davis, Scipio, & et al., 2006); whereas in chronic widespread pain patients, general preoccupied attachment was linked with disability and number of pain sites, but not with pain intensity (Davies, Macfarlane, McBeth, Morriss, & Dickens, 2009). In chronic pain patients, general insecure attachment has been associated to catastrophizing (Ciechanowski et al., 2003; McWilliams & Asmundson, 2007), lower self-efficacy (Meredith et al., 2006a), and depression (Ciechanowski et al., 2003; Meredith et al., 2007).

In the face of this complex interplay of psychosocial factors linking attachment theory and the pain experience, Meredith (Meredith et al., 2008) conceptualized an **attachment-diathesis model of chronic pain** (Figure 12), theorizing that insecure attachment is both a vulnerability factor for the development of chronic pain as well as for poor outcome of chronic pain. Further, this author has successfully linked attachment to a myriad of pain experiences, such as acute and experimental pain (Meredith et al., 2006a, 2006b). Notably, there is also evidence showing that insecure attachment is more prevalent in medically unexplained pain compared to pain with a clear organic cause, and that poorer outcome in chronic pain is associated to insecure attachment independently of organic pathology (Schroeter et al., 2015). Attachment insecurity, and fearful and dismissing attachment in particular, is overrepresented in chronic pain populations (Davies et al., 2009; Kowal et al., 2015; Meredith, Strong, & Feeney, 2005; Meredith et al., 2006a; Schmidt, Nachtigall, et al., 2002). In other words, while there is evidence that approximately 65% of people in normative samples are securely attached and 35% are insecurely attached (Mickelson et al., 1997), in samples with pain patients these numbers are quite likely to be reversed (Kowal et al., 2015; Meredith, 2016).

Figure 12. The Attachment-Diathesis Model of Chronic Pain (from Meredith et al., 2008).



Nonetheless, the relevance of attachment to clinical populations has not been found to be limited to chronic pain, and the bounds of this research area seem to stretch further as evidence grows. It is possible to find examples of attachment linked to **chronic diseases**, such as ulcerative colitis (Maunder, Lancee, et al., 2006); **alopecia**, **leg ulcers**, and **breast cancer** (Schmidt, 2003; Schmidt, Nachtigall, et al., 2002); **somatization** (Stuart & Noyes, 1999; Waller et al., 2004b); **hypochondriacal** concerns (Schmidt, Strauss, et al., 2002); **idiopathic spasmodic torticollis** (Scheidt et al., 2000); and **use of healthcare** (Ciechanowski, Walker, et al., 2002).

Further, and bearing in mind that trauma can be conceived as the most provocative test of the child-caregiver system's resiliency, studying the **precursors of attachment insecurity** allows for causal inferences in the absence of prospective studies (Maunder & Hunter, 2001). There is strong evidence for an association between these precursors of attachment insecurity and adult diseases in a very large study of primary practice medical patients, in which the risk factors evaluated were psychological, physical, or sexual abuse, violence against mother, and living with family members who were substance abusers, mentally ill, suicidal, or imprisoned. The disease outcomes studied were cancer, ischemic heart disease, chronic lung disease, skeletal fractures, and liver disease. A graded relationship was found in 9,508 patients between the number of retrospectively reported adverse categories of childhood exposure and each of the adult health risk behaviors and diseases (Felitti et al., 1998).

Consequently to the notion of attachment insecurity and the vulnerability it entails, mention must be made again to the concept of **mentalization**. It emerged from the Ecole Psychosomatique de Paris and was further developed by theory of mind researchers (Leslie, 1987). The term was originally used in a more ample way by Peter Fonagy in 1989 (Fonagy, 1989) and has since grown together with the comprehension of many mental disorders. Mentalizing theory stems from Bowlby's attachment theory and its development by contemporary developmental psychologists, minding constitutional vulnerabilities as well (Bateman & Fonagy, 2010). The mentalizing process allows for individuals to make sense of each other and themselves with regards to subjective states and mental processes. Thus, it is a social construct due to the attention dedicated to the mental states of others in relation to oneself, implicitly and explicitly, at a physical or psychological level. It is therefore a general definition that allows for most mental disorders to ascribe, to some degree, to some difficulties with mentalization. Reasonably, mentalizing theory is being applied to a number of disorders (e.g.,post-traumatic disorder (Allen, 2001), eating disorders (Skarderud, 2007), and depression (Allen, Bleiberg, & Haslam-Hopwood, 2003)).

Depression

Attachment theory has thus proven to be a very prolific framework for studying emotion regulation and mental health. In effect, research on adult attachment processes and individual differences in attachment patterns has furnished strong evidence for the anxiety-buffering function of what Bowlby (Bowlby, 1982) termed the attachment behavioral system, as well as for the importance of attachment-related individual differences to coping with stress, managing distress, and maintaining psychological resilience (Mikulincer & Shaver, 2007a).

In the attachment literature, researchers have directed their interest toward understanding how attachment impacts psychosocial functioning: in particular, how attachment is related to depression (e.g. Catanzaro & Wei, 2010; Kobak & Sceery, 1999). Early work regarding the relationship between adult attachment insecurities, of both the anxious and avoidant varieties, and depression consistently found a positive association (e.g. Armsden, McCauley, Greenberg, Burke, & Mitchell, 1990). However, these first studies providing evidence of a positive direct association lacked an explanation of the mechanisms underlying this relationship (Roberts, Gotlib, & Kassel, 1996). In fact, the attachment-psychopathology liaison is moderated by a wide array of biological, psychological, and sociocultural factors, and mental disorders per se can undermine a person's sense of attachment security (Mikulincer & Shaver, 2012).

More recently, and according to attachment theory as it has been previously reviewed, research has established that the link between **attachment insecurities** and psychopathology in general —and depression, in this instance—, is mediated by several pathways, the most important of which are self-representations, emotion regulation, and problems in interpersonal relations. These pathways explore concepts such as lack of self-cohesion, unstable self-esteem, over-dependence on external approval, self-criticism, impairment of coping strategies due to absence of emotionally accessible and responsive others, emotion amplification and exaggeration of worries, and interference with the acquisition of social skills due to recurrent failure to obtain support from attachment figures (Mikulincer & Shaver, 2012).

Thus, the most important of the mental health problems that co-occur with insecure attachment is depression, due to its common incidence and its consistent, substantial negative impact on the burden and outcome of physical illness. Depression is not only associated with increased severity of physical symptoms, increased health-care costs, and reduced health-related quality of life (Evans et al., 2005), but also plays a significant part in increased mortality (Lemogne et al., 2013). Even in high-acuity settings such as the ICU, pre-existing depression is an independent risk factor for increased mortality (Wewalka et al., 2015).

Evidence corroborates Bowlby's prediction that factors that lead to insecure attachment also augment the risk of depression. A possible hypothesis might be that the developmental experience of attempting to relate to an unavailable parent and being thwarted yields learned helplessness, a state that consistently causes depression (Seligman & Maier, 1967). Another option is that attachment insecurity may increase the risk of depression by increasing vulnerability to the effects of stress. Additionally, insecure styles of attachment are often linked to deficits in self-esteem and self-efficacy (Mikulincer & Shaver, 2007a). For all these reasons, depression is common in those with insecure attachment, especially in the context of medical illness (Ciechanowski et al., 2003; Maunder et al., 2005). Hence the importance of recognition and management of depression, as sometimes it is the most malleable element of a vicious cycle of disease and the consequences of illness (Hunter et al., 2016).

Therefore, in order to adequately assess the concept of depression, it is important to consider the setting, i.e., clinical or non-clinical samples. Indeed, depression is commonly observed to coexist with chronic pain and is chiefly associated with higher levels of reported pain and increased functional impairment (Arnow et al., 2006; Bair, Robinson, Katon, & Kroenke, 2003; Demyttenaere et al., 2006), thus contributing to a challenging diagnosis due to overlapping somatic symptoms. Insomnia, fatigue, and change in activity constitute symptoms that can be related to both pain and depression, albeit according to DSM-5, symptom criteria that are fully attributable to the medical condition should not be included in the diagnosis (American Psychiatric Association, 2013).

Indeed, depression is quite complex in its symptomatology and etiology. The prevalence of this disorder varies according to country: the American National Comorbidity Survey Replication noted that 16.2% of people had major depression at some point in their

life and 6.6% in the last 12 months (Kessler et al., 2003). However, the European Study of the Epidemiology of Mental Disorders (ESEMeD) has featured the Spanish prevalence as lower than in other European countries, with a lifetime prevalence of a depressive episode of 10.6% and a yearly prevalence of 4.0%, although notably it has an earlier onset age and high rates of comorbidity and chronicity (Gabilondo et al., 2010). Furthermore, among hospitalized patients the prevalence of depression rises to 18.9% (Crespo, Gil, Porras-Chavarino, & Grupo de Investigación en Depresión y Psiquiatría de Enlace, 2001), and some groups, such as illegal immigrants, are particularly vulnerable, with a 40.7% of prevalence (Barro-Lugo, Saus-Arús, Barro-Lugo, & M., 2004).

Depression has high rates of comorbidity and mortality, and the association between depression and physical and mental illness, as well as with substance abuse and suicidal behavior, is considerable. The link between these disorders is intricate, since depression predisposes their condition while, at the same time, the presence of these disorders increases the likelihood of depression (World Health Organization, 2012).

Concept and epidemiology

Depression is conceived as a set of chiefly **affective symptoms** (pathological sadness, apathy, anhedonia, hopelessness, weakness, irritability, subjective feeling of distress and helplessness when faced with the demands of life). However, insofar there are varying degrees of cognitive, volitional, and somatic symptoms, it is possible to speak of an overall **physical and mental condition**, with particular emphasis on the affective sphere (Marcus, Yasamy, van Ommeren, Chisholm, & Saxena, 2012; National Collaborating Centre for Mental Health, 2009).

Diagnosing a depressive disorder is commonly based on not very specific observational data, such as physical slowness, a decline in demeanor and personal appearance, low voice, easy or spontaneous crying, decreased attention, sad expression, verbalization of pessimistic ideas (e.g. hypochondria, ruin, or guilt), sleep disturbances, and somatic complaints. The pathological quality of these changes is acquired by the persistence of the symptoms, their severity, and the degree of functional and social impairment.

It is often **difficult** to establish the **diagnostic autonomy** of depression from other psychopathological entities, as the association with anxiety is common, with various

symptomatic combinations being manifested. Depression may also be **concurrent** with the abuse of alcohol of other substances, some organic brain and systemic diseases (World Health Organization, 2013), eating behavior disorders, and some personality disorders (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). Moreover, it is more common among people with chronic physical illness, establishing a reciprocal relationship in which the physical problems may exacerbate depression and depression may in turn adversely affect the course of the physical condition. Additionally, depression is a **risk factor** for certain physical pathologies, such as cardiovascular disease (Egede, 2007).

Depression can start at any age, although the highest prevalence occurs in the 15 to 45 years age range, thus having a significant impact on education, productivity, performance, and relationships (Marcus et al., 2012; Patten et al., 2009). Symptoms may differ according to age: young people show mainly behavioral symptoms, while older adults have somatic symptoms more frequently (Hegeman, Kok, van der Mast, & Giltay, 2012; Serby & Yu, 2003).

Risk factors

Depression is a **multifactorial** and complex process, involving a wide array of risk factors whose totality and multiple interactions have not yet been completely established. The role of each in relation to the circumstances and time of life in which they develop is unknown (Butler et al., 2006). These variables that increase the risk of depression can be classified into **personal**, **social**, **cognitive**, **familial**, and **genetic**.

With regards to **personal** and **social** factors, the prevalence and incidence of depressive disorder is greater in females than in males (Bellón et al., 2008), and it is estimated that the burden of depression is 50% higher in women (Marcus et al., 2012).

Other important risk factors are **chronic diseases**, both physical and mental (National Collaborating Centre for Mental Health, 2009), and the possible link with consumption of alcohol and tobacco (Boden & Fergusson, 2011; Hamalainen et al., 2001). Further, there are also forms of psychopathology associated, especially anxiety disorders (Klein, Kotov, & Bufferd, 2011), which are mainly risk factors for the first episode of major depression. On the other hand, dysthymia (Eccleston & Scott, 1991) has been seen as an important predictor of later development of symptoms of major depression (Fogel, Eaton, & Ford, 2006).

An association between migraine and depression has also been described, according to which major depression patients have a higher risk of migraine and vice versa (Breslau, Lipton, Stewart, Schultz, & Welch, 2003). Moreover, the presence of heart disease and various endocrine diseases such as diabetes, hypo-or hyperthyroidism, Cushing's syndrome, Addison's disease, and hyperprolactinaemic amenorrhoea seem to increase the risk of depression (National Collaborating Centre for Mental Health, 2009).

Social circumstances attributed to the disorder are working circumstances and a low level of economic resources (National Collaborating Centre for Mental Health, 2009). Individuals who are unemployed or on sick leave have more frequent depressions (Haro et al., 2006). Also, marital status and chronic stress seem to be linked with a greater likelihood of developing depression (National Collaborating Centre for Mental Health, 2009); while it has also been observed that exposure to adversities throughout life is involved in the onset of depressive and anxious disorders (Turner & Lloyd, 2004).

As per the **cognitive** factors, research has been driven by how information is processed (Beck, 2008); as well as other factors, such as cognitive reactivity to negative events, the ruminative response style and attentional biases, which are considered key in the development and maintenance of depression (De Raedt & Koster, 2010).

Furthermore, **descendants** of patients with depression are a risk group for both somatic and mental disorders (Weissman et al., 2006). Hence, first-degree relatives of patients with major depressive disorder are twice as likely to have depression as the general population (P. F. Sullivan, Neale, & Kendler, 2000), while second-degree relatives are also significantly more prone (Weissman et al., 2005). However, these family studies are insufficient to determine how much risk comes from **genetic** factors and how much stems from the shared family environment.

Research of the genes involved in the development of depression has chiefly followed the approach analyzing the role of monoamines. A factor that may influence the development is the presence of a polymorphism in the gene encoding the serotonin transporter, which would lead to decreased transport of this neurotransmitter (Cervilla et al., 2006). This gene may be a response predictor to antidepressant treatment (Porcelli, Fabbri, & Serretti, 2012).

Ultimately, depression is one of the factors most associated with suicidal behavior (Grupo de Trabajo de la Guía de Práctica Clínica de Prevención y Tratamiento de la Conducta

Suicida, 2012; Hawton, Saunders, Topiwala, & Haw, 2013). The risk of **suicide** in people with depression has been estimated to be four times higher in comparison with the general population and 20 times higher for severe depression (Grupo de Trabajo de la Guía de Práctica Clínica de Prevención y Tratamiento de la Conducta Suicida, 2012). Some of the associated factors with increased risk of suicide in patients with depression are: depressive episode or period of partial remission (Sokero et al., 2005), male sex, family history of mental disorder, previous suicide attempt, more severe levels of depression, hopelessness, comorbid disorders —especially anxiety and alcohol of other drug abuse (Hawton, Casañas, Comabella, Haw, & Saunders, 2013)—, borderline personality disorder, and high levels of impulsivity and aggression (Dumais et al., 2005).

Diagnostic criteria

The most well used diagnoses of depression, both clinically and in research, are those of the **International Statistical Classification of Diseases and Related Health Problems (ICD)** (World Health Organization, 1992) and the aforementioned **DSM** classification from the American Psychiatric Association (Reed, J., Esparza, Saxena, & Maj, 2011).

As for the 5th edition of the DSM, it does not introduce significant changes to the DSM-IV-TR diagnostic criteria for a major depressive episode, except for some modifications and specifications to describe the current clinical status. Also, the specification of “chronic” is moved from major depressive disorder to persistent depressive disorder (dysthymia), excluding the requirement of DSM-IV-TR that the person must not have suffered a major depressive episode during the first 2 years of the disorder.

Major Depressive Disorder (DSM-5 Diagnostic Criteria)

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

Note: Do not include symptoms that are clearly attributable to another medical condition.

1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad, empty, hopeless) or observation made by others (e.g., appears tearful). (*Note:* In children and adolescents, can be irritable mood.)
2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation.)
3. Significant weight loss when not dieting or weight gain (e.g., a change of more that 5% of body weight in a month), or decrease or increase in appetite nearly every day. (*Note:* In children, consider failure to make expected weight gain.)
4. Insomnia or hypersomnia nearly every day.
5. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
6. Fatigue or loss of energy nearly every day.
7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The episode is not attributable to the physiological effects of a substance or to another medical condition.

Note: Criteria A-C represent a major depressive episode.

Note: Responses to a significant loss (e.g., bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability) may include the feelings of intense sadness, rumination about the loss, insomnia, poor appetite, and weight loss noted in Criterion A, which may resemble a depressive episode. Although such symptoms may be understandable or considered appropriate to the loss, the presence of a major depressive episode in addition to the normal response to a significant loss should also be carefully considered. This decision inevitably requires the exercise of clinical judgment based on the individual's history and the cultural norms for the expression of distress in the contest of loss.

D. The occurrence of the major depressive episode is not better explained by schizoaffective disorder, schizophrenia, schizophreniform disorder, delusional disorder, or other specified and unspecified schizophrenia spectrum and other psychotic disorders.

E. There has never been a manic episode or a hypomanic episode.

Note: This exclusion does not apply if all of the manic-like or hypomanic-like episodes are substance-induced or are attributable to the physiological effects of another medical condition.

Specify: With anxious distress, With mixed features, With melancholic features, With atypical features, With mood-congruent psychotic features, With mood-incongruent psychotic features, With catatonia, With peripartum onset, With seasonal pattern (recurrent episode only)

Persistent Depressive Disorder (Dysthimia)
(DSM-5 Diagnostic Criteria)

This disorder represents a consolidation of DSM-IV-defined chronic major depressive disorder and dysthymic disorder.

A. Depressed mood for most of the day, for more days than not, as indicated by either subjective account or observation by others, for at least 2 years. (*Note:* In children and adolescents, mood can be irritable and duration must be at least 1 year.
B. Presence, while depressed, of two (or more) of the following:
    1. Poor appetite or overating.
    2. Insomnia or hypersomnia.
    3. Low energy or fatigue.
    4. Low self-esteem.
    5. Poor concentration or difficulty making decisions.
    6. Feelings of hopelessness.
C. During the 2-year period (1 year for children or adolescents) of the disturbance, the individual has never been without the symptoms in Criteria A and B for more than 2 months at a time.
D. Criteria for a major depressive disorder may be continuously present for 2 years.
E. There has never been a manic episode or a hypomanic episode, and criteria have never been met for cyclothymic disorder.
F. The disturbance is not better explained by a persistent schizoaffective disorder, schizophrenia, delusional disorder, or other specified or unspecified schizophrenia spectrum and other psychotic disorder.
G. The symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g., hypothyroidism).
H. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Note: Because the criteria for a major depressive episode include four symptoms that are absent from the symptom list for persistent depressive disorder (dysthymia), a very limited number of individuals will have depressive symptoms that have persisted longer than 2 years but will not meet criteria for persistent depressive disorder. If full criteria for a major depressive episode have been met at some point during the current episode of illness, they should be given a diagnosis of major depressive disorder. Otherwise, a diagnosis of other specified depressive disorder or unspecified depressive disorder is warranted.

Specify if: With anxious distress, With mixed features, With melancholic features, With atypical features, With mood-congruent psychotic features, With mood-incongruent psychotic features, With peripartum onset.

Diagnostic criteria for a depressive episode according to ICD-10

A. The depressive episode must last at least two weeks.

B. The episode cannot be attributed to the abuse of psychoactive substances or an organic mental disorder.

C. Somatic syndrome: Some of the depressive symptoms may be very prominent and acquire special clinical significance. Typically, a somatic syndrome is considered present when at least four of the following features coexist:

- Marked loss of interest or pleasure in activities that were normally pleasurable.
- Absence of emotional reactions to events that normally produce a response.
- Waking up in the morning two or more hours before the usual time.
- Depression that is worse in the morning.
- Marked psychomotor retardation or agitation.
- Marked loss of appetite.
- Weight loss of at least 5% in the last month.
- Notable decrease of libido.

Severity criteria of a depressive episode, according to ICD-10

A. General criteria for depressive episode:
- The depressive episode must last at least two weeks.
- The episode cannot be attributed to the abuse of psychoactive substances or to an organic mental disorder.

B. Presence of at least two of the following symptoms:
- Clearly abnormal depressive mood for the subject, present during most of the day and almost every day, which is altered very little by environmental circumstances and which persists for at least two weeks.
- Marked loss of interest or ability to enjoy activities that were previously pleasurable.
- Lack of vitality or increased fatigability.

C. Up to three of the following symptoms are present:
- Loss of confidence and self-esteem, and feeling of inferiority.
- Disproportionate self-reproaches and feelings of excessive guilt or inadequacy.
- Recurrent thoughts of death or suicide, or any suicidal behavior.
- Complaints about or decreased ability to concentrate and think, accompanied by hesitation and a lack of decision.
- Changes of psychomotor activity, with agitation or inhibition.
- Sleep disturbances of any kind.
- Changes of appetite (decrease or increase) with the corresponding weight change.

D. There may or may not be somatic syndrome.

Mild depressive episode: Two or three of the symptoms of criteria B are present. Individuals with a mild episode can probably continue with their normal activities.
Moderate depressive episode: At least 6 symptoms from criteria B and C are present, with at least two from criterion B. The person with a moderate episode will probably have difficulty keeping up with their normal activities.
Severe depressive episode: At least 8 symptoms from criteria B and C are present, including all 3 from criterion B. People with this type of depression have marked and distressing symptoms, mainly a loss of self-esteem and feeling of guilt and worthlessness. Suicidal thoughts and actions are frequent with significant somatic symptoms. Psychotic symptoms can appear, such as hallucinations, delusions, psychomotor retardation or severe stupor. This case if called severe depressive episode with psychotic symptoms. Psychotic phenomena such as hallucinations or delusions may or may not be mood-congruent.

Cognitive and behavioral theories of depression

As depression is more constant and pervasive than anxiety, and it is not stimulus-bound in the way anxiety is, this can be seen as a problem in response and stimulus overgeneralization from the cognitive-behavioral perspective. Each theoretical model accounts for depression's diverse phenomena and takes a different approach to handling the problem. Hence, **four major theoretical models** of depression have been developed from the cognitive-behavioral perspective in clinical psychology.

Reinforcement theory

Charles Ferster (Ferster, 1973) was one of the first to apply a **behavioral** analysis to the issue of depression, and as such viewed it as a generalized decrease of rates of response to external stimuli. Therefore, behavior was no longer under the control of reinforcers that were once effective, thus establishing an analogy in learning terms with the process of extinction: major losses in life could be conceived as losses of important sources of reinforcement. Generalization of the loss's effects took place when other behavior was chained to or organized by the central source of reinforcement, in a manner where one response was dependent on a later response due to the fact that the first allowed access to the second.

In later elaborations, Ferster (Ferster, 1977, 1981) emphasized the analysis of verbal behavior as an important means for studying depression, as depression consists largely of complaints that are negatively reinforced by those around the depressed person.

Peter M. Lewinsohn developed a coherent theory from similar ideas and explored its applications in a clinical research program (Lewinsohn, 1974; Lewinsohn, Biglan, & Zeiss, 1976). According to Lewinsohn, depression is a response to a loss or lack of response-contingent positive reinforcement. Dysphoria and a reduction in behavior, which are the primary phenomena of depression, would stem from insufficient reinforcement in major domains of one's life, and other symptoms of depression such as low self-esteem and hopelessness would follow from the lower level of functioning. Furthermore, although in the short run depressive behavior elicits positive responses from others, maintained depression is aversive to these others and they begin to avoid the depressed person, thus reducing reinforcement yet again. Therefore, the depressed behavior is ultimately perpetuated on a thin

schedule of reinforcement, which is still not enough to overcome the depression in a self-recycling cycle.

Learned helplessness theory

Martin E. P. Seligman's (Seligman, 1974, 1975) learned helplessness theory of depression originated from an **animal model** for the disorder. Seligman observed the phenomenon in which animals in a shuttle box apparatus were exposed to unavoidable shock and became subsequently deficient in learning an escape or avoidance response (Seligman & Maier, 1967). He suggested that the animals had acquired a generalized helplessness, that is, a perceived lack of contingency between responses and outcomes. Following the model's conception, contingency is a critical factor, since animals with equivalent but response-contingent shock did learn eventually to escape and avoid like non-preconditioned animals.

Seligman drew an analogy from the animals' behavior to human depression, seeing induction by unavoidable shock as parallel to the traumatic loss that often yields depression. Also, the animals showed passivity, weight loss, lack of appetite, and dissipation of the learned helplessness effect with time, all of which coincided with the symptoms and behavior of normal depression in people. In conducting helplessness induction experiments with humans, findings were, in effect, similar.

However, there were conceptual and empirical issues with the animal learned helplessness model of depression. Such was the case of the paradox of guilt in depression, or the difficulty to explain why people perceive themselves responsible and to blame for bad outcomes if there is a perception of noncontingency between the person's behavior and resulting events (Abramson & Sackheim, 1977).

Thus, in 1978 an **attributional revision** of the learned helplessness theory was published (Abramson, Seligman, & Teasdale, 1978), which included the social psychological ideas about attribution of responsibility. People make inferences about the causes of life events, and these attributions can be categorized within a simple dimensional structure (Weiner et al., 1971): causes may be internal or external, depending on whether the event is caused by an trait of the person or an outside world aspect; and stable or unstable, if the causal factors continue to function over time or are relative to the particular time of the event. Furthermore, Abramson, Seligman, and Teasdale (Abramson et al., 1978) added another

dimension, according to which causes may also be global or specific. That is, global causes are general to many situations whereas specific causes only apply to restricted domains.

Therefore, following these hypothesis, people would develop consistent attributional styles and there would be a typical attributional style for people at risk for depression. This would entail a tendency to attribute negative outcomes to internal, stable, global causes and credit positive events to external, unstable, specific causes. Moreover, a person with this depressive style is likely to make a depressive attribution when a major aversive event occurs, hence perceiving themselves as helpless —as being unable to avoid failure and unable to produce success. The idea then is that an internal attribution determines whether the person's self-esteem is affected, a stable attribution establishes the depression's chronicity, and a global attribution conditions the generality of the depressive feelings. The intensity of the depression is linked not only by the event's aversiveness but by the resulting attributions.

Self-control theory

Models of self-control convey the ways in which people manage their behavior in order to obtain long-term goals (e.g., quit smoking, or start exercizing). In depression, people are hopeless about long-term goals and feel helpless in managing their behavior; thus, the depressed individual may not perform behavior without immediate consequences.

Rehm (Rehm, 1977) posited a self-control model of depression as an **integrative attempt** of theoretical notions of Lewinsohn, Beck, and Seligman, with a **self-control framework** that was an adaptation of Kanfer's (Kanfer, 1970) model of self-control. Kanfer described a three-stage feedback-loop process consistent of people's efforts at controlling their behavior towards long-term goals. The first step is when people see the need to change behavior to obtain a delayed goal and pay conscious attention to the relevant behavior. The monitored information is then contrasted to an internal standard and a process of self-evaluation is ignited, in which a judgement of the behavior's valence is made. At this point, the model was modified to add an attributional factor to self-evaluation, and therefore contemplated the premise of an initial internal attribution for the act to view self-evaluation of behavior as positive or negative. Thus, attributional judgements moderate self-evaluation.

The final phase in Kanfer's model is self-reinforcement. He premised that people can control and influence their own behavior through the same reinforcement principles

applicable to others' behavior. Therefore, self-reward and self-punishment would substitute for the environment's rewards and punishments and maintain behavior when external reinforcement is not immediate.

The self-control model of depression (Rehm, 1977) hypothesized that the behavior of depressed individuals featured one or more of six deficits in self-control behavior. First, depressed people selectively attend to negative life events and relatively exclude positive ones, a self-monitoring deficit described by Beck (Beck, 1972) as selective attention in depression. Second, depressed people selectively attend to the immediate rather than the delayed outcomes of their behavior. Third, a depressed person sets harsh self-evaluative standards, often being perfectionistic and applying more stringent standards for themselves than others. Fourth, depressed people make depressive attributions about their behavior: internal attributions for failure and external attributions for success. Later versions of the model incorporated the three-dimensional analysis of helplessness theory (Abramson et al., 1978), with the global-specific dimension discussed above. Fifth, depressed people self-administer insufficient contingent reward to maintain important areas of behavior, and sixth, they administer excessive self-punishment, which inhibits constructive behavior in many domains.

Ultimately, the self-control model is a **vulnerability model**, as the mentioned poor self-control skills would place individuals at risk for depression under disadvantageous conditions of external reinforcement. The overgeneralization that occurs with depression is integrated by positing that self-control skills would act like a control program that would manage all domains of behavior pursuing long-term goals. When self-control skills are required to readapt in a major life area, poor skills will yield maladaptation and repercussions of poor functioning in many areas.

Cognitive theory

Aaron T. Beck conceived a cognitive theory that originally focused on depression and has been extended to other areas of psychopathology and psychotherapy. Dissatisfied with his psychodynamic training, he read George Kelly's "The Psychology of Personal Constructs" (Kelly, 1955) and was appealed to the cognitive notion of unique construct systems through which each person construes the world. He also adopted the theoretical

concept of "**schema**" from modern cognitive psychology. Schemata are structural units of stored information through which new experience is interpreted, acting as templates to compare and incorporate new knowledge. Schemata range from representations of simple concepts, such as a chair schema, to complex interpretive rules, like a hotels schema that allows a person to see that the bellhop is waiting because he expects a tip.

Thus, Beck's (Beck, 1972) theory broke down depression into essential elements he termed the "**cognitive triad**": (1) negative view of self, (2) a negative view of the world, and (3) a negative view of the future. A depressed individual sees the world through an organized set of depressive schemata that negatively distort experience about self, the world, and the future.

Early in the development of the theory (Beck, 1963), a number of typical forms of cognitive distortion were designated, such as arbitrary inference, selective abstraction, magnification and minimization, and inexact labeling. In this case, as one of the basic principles of the cognitive approach is that a schematic interpretation always mediates between an experience and its corresponding emotional response, the negative and distorted cognitions that arise in a particular situation were termed "**automatic thoughts**". They were depicted as automatic due to the fact that the person is not aware of the interpretive process and may not be aware of the thoughts per se, but only of their emotional consequences. These automatic thoughts are formed by more basic interpretive rules, or underlying assumptions that help distinguish the specific thoughts. In the instance of depression, the theme of the automatic thoughts is the perception of loss: loss is the cognition linked to depression. Whereas perceptions of gain produce euphoria, perceptions of danger yield anxiety, and perceptions of offense lead to anger.

Depressive schemata are activated when a major loss is felt. As the person becomes depressed, an organized set of negative schemata, originated earlier in life when major losses were experienced, replaces nondistorted schemata, representing elaborated views of self, the world, and the future. The negative schemata may be replaced by more realistic schemata insofar they are used under usual life circumstances, but they remain intact as latent schemata, with the potential of being reactivated under circumstances of loss. Unless modified by some form of intervention, these schemata may again become latent with time

and improved circumstances. Thus, the characteristic overgeneralization in depression is due to the replacement of one broad network of schemata with another.

Evaluation and assessment instruments

In order to measure the severity of depression and response to treatment, scales and interviews with varying degrees of structure are used, aiming to evaluate patient symptoms within a specific timeframe by grading each item and yielding a final score. However, to form a proper diagnosis, a **clinical interview** is required to establish the psychopathological information.

In this section, different instruments to evaluate depression are discussed. However, due to the purposes of the present research, there are questionnaires, such as the Hospital Anxiety and Depression Scale, that have been included for their value in assessing the emotional state in medical patients, rather than their diagnostic value or exclusive use in depression.

Beck Depression Inventory

The widely used Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) was originally designed to measure the level of **depression** in patients **already having that diagnosis**. It has validated Spanish translations, from the original 1961 version (Beck et al., 1961) through the most recent 1996 version and the second edition (BDI-II) (Beck, Steer, & Brown, 1996), adapted by Sanz et al. (Sanz & García-Vera, 2013; Sanz, García-Vera, Espinosa, Fortún, & Vázquez, 2005; Sanz, Navarro, & Vázquez, 2003; Sanz, Perdigón, & Vázquez, 2003).

The BDI-II is a self-reporting instrument of 21 items in which there is a given choice between four alternative responses, describing the lowest to the highest severity, that best describe the subject's state during the previous two weeks. The four responses in each item are scored from 0 to 3 points, producing a total possible score range of 0 to 63 points.

Yet, the validity of assessing symptoms of **depression in medical illnesses** using self-report questionnaires such as BDI has been contested, as several items in the BDI can be attributed to the medical condition, like sleep problems, difficulties with concentration, and

fatigue, all of which may spuriously increase the sum score of the questionnaire (Knaster, Estlander, Karlsson, Kaprio, & Kalso, 2016).

Table 3. Cutoff points and BDI descriptors

	Minimal	Mild	Moderate	Severe
BDI-II original	0-13	14-19	20-28	29-63
Spanish modification	0-13	14-18	19-27	28-63

Compiled from various sources (Beck et al., 1996; Sanz, Gutiérrez, Gesteira, & García-Vera, 2013).

Hospital Anxiety and Depression Scale (HADS)

Nonetheless, other valid options have been researched: the Hospital Anxiety and Depression Scale (HADS) (Snaith & Zigmond, 1994) is a popular self-report scale, originally developed to measure **depression** and **anxiety** among **nonpsychiatric**, hospital clinic **outpatients**. Amid the positive qualities of the HADS there are the brevity, good reliability and validity, and efficiency in screening and case-finding (Bjelland, Dahl, Haug, & Neckelmann, 2002; C. Brennan, Worrall-Davies, McMillan, Gilbody, & House, 2010; Luckett et al., 2010). The HADS has been widely used as an effective tool to assess emotional distress in non-clinical populations. In spite of the many existing scales to assess depression and anxiety, the HADS is considered **useful** (Pascual López, García-Campayo, Lou, & Ibáñez, 2004) in **assessing fibromyalgia**. Due to the existing overlap between medical and psychological symptoms of the illness, this questionnaire is more appropriate as it focuses on evaluating the cognitive aspects of anxiety and depression (García-Campayo et al., 2006), thus additionally crediting its sensitivity to change (Chivite, Martínez, Pérez, & Peralta, 2008). Indeed, the psychological factors, such as anxiety and depression, must be compared to some severity and course criteria of the syndrome (Vallejo, Rivera, Esteve-Vives, Rodríguez-Muñoz, & Grupo ICAF, 2012).

Originally designed by Zigmond and Snaith (Zigmund & Snaith, 1983), it was translated and adapted to Spanish by Snaith, Bulbena, and Berrios, and was validated by Tejero and collaborators (Tejero, Guimera, Farré, & Peri, 1986).

It is a self-report of 14 items, composed by two subscales of 7 items: the odd items correspond to the anxiety subscale, and the even items to the depression subscale. The

anxiety items are selected from the analysis and review of the anxiety Hamilton Rating Scale, thus avoiding the inclusion of physical symptoms that might be confounded by the patient with the medical condition. The depression items, on the other hand, focus on the anhedonia, or loss of pleasure. The depression subscale does not contain items relative to the somatic components of mood depression, but is restricted to the mood hedonic and volitional aspects in order to avoid the bias of confounding somatic components with emotional ones (Torta, Pennazio, & Ieraci, 2014). The intensity or frequency of the symptom is assessed on a 4-point Likert scale, ranging from 0 to 3. Despite the questions being formulated in the present tense, the timeframe refers to the previous week. The score is obtained for each subscale by adding the respective items, and therefore the score ranges from 0 to 21 for each subscale, and from 0 to 42 in total.

The original scale suggested the same cut-off scores for both subscales, as seen in Table 4. Other authors have recommended the use of the total score instead of the separate subscales, due to the fact that there is no evidence of a good discriminative value between anxiety and depression, presenting cut-off scores that vary according to the different medical conditions.

Table 4. Cutoff points and HADS descriptors

	Normal	Moderate symptomatology; doubtful cases	Clinical problem
Original HADS	0-7	8-10	≥ 11
Medical patients with unexplained somatic symptoms	-	-	≥ 12
Palliative care patients	-	-	≥ 20

Compiled from various sources (Le Fevre, Devereux, Smith, Lawrie, & Cornbleet, 1999; Spinhoven et al., 1997; Zigmund & Snaith, 1983)

The HADS questionnaire has been amply used in samples of patients with arthritis, cancer, postpartum depression women, and traumatic brain lesion patients, amongst others, to evaluate the levels of anxiety and depression (Axford et al., 2010; Chivite et al., 2008; Cooper-Evans, Alderman, Kinht, & Oddy, 2008; Singer et al., 2008; Terol, López-Roig, Rodríguez-Marín, Martín-Aragón, & Pastor, 2007) with good psychometric properties

(Chivite et al., 2008; Herrero et al., 2003; Terol et al., 2007). It is useful to assess how the illness affects the emotional state in medical patients, and provides with dimensional measures of psychic distress, showing a good correlation with different severity aspects of the disease and other dimensional measures of quality of life. The HADS can be used to quantify changes in the course of illness or the response to different psychotherapeutic interventions (Herrmann, 1997).

Hamilton Rating Scale for Depression

The Hamilton Rating Scale for Depression (HRSD or HAM-D) (Hamilton, 1960) was created to evaluate the intensity or severity of depression by clinicians. It is one of the most **commonly** used questionnaires to **monitor** the evolution of depressive symptoms in clinical practice and research. The assessment corresponds to the timeframe of its application, excepting some items, such as sleep, which refer to the previous two days.

The original version was published in 1960 (Hamilton, 1960) and is composed of 21 items. There is a more brief version by the same author of 17 items (Hamilton, 1967); another version of 24 (Miller, Bishop, Norman, & Maddever, 1985); and one of 6 items, focusing on depressed mood, feelings of guilt, work and activities, inhibition, psychic anxiety, and the somatic symptoms from the 17-item version (Bech et al., 1981). The Spanish version's validation was carried out in 1986 (Ramos-Brieva & Cordero-Villafafila, 1986) and a psychometric assessment (Bobes et al., 2003) comparing versions 6, 17, and 21 was conducted later.

Adding the scores of each item yields a **global severity score** for depressive symptoms, with the cutoff points and descriptors of different degrees of depression shown on Table 5. Also, the score from the three factors or indices is obtained by adding the following: melancholy (items 1, 2, 7, 8, 10, and 13); anxiety (items 9-11); and sleep (items 4-6). These indices have no established cutoff scores.

Customarily, the response to treatment is considered equal to or greater than a 50% reduction of the initial score, a partial response is between 25 and 49%, and a non-response is defined as a reduction of less than 25% (Shelton, 2006). Remission is set to be achieved when the score is ≤ 7 (Bobes, Portilla, Bascarán, Saiz, & Bousoño, 2004).

Table 5. Cutoff points and Hamilton scale descriptors

APA 2000	No Depression	Mild	Moderate	Severe	Very Severe
NICE 2009	No Depression	Subclinical	Mild	Moderate	Severe
Score	0-7	8-13	14-18	19-22	> 23

Adapted from NICE (National Collaborating Centre for Mental Health, 2009)

Montgomery Asberg Depression Rating Scale

The Montgomery Asberg Depression Rating Scale (MADRS) (Montgomery & Asberg, 1979) is applied through an interview composed of 10 items that evaluate the severity of depressive symptoms. Despite the variety of self-rating versions that have shown a moderate/good correlation with expert ratings, the scale must be administered by a clinician (Cunningham, Wernroth, von Knorring, Berglund, & Ekselius, 2011).

The items include apparent sadness, reported sadness, inner tension, reduced sleep, reduced appetite, concentration difficulties, lassitude, inability to feel, pessimistic thoughts, and suicidal thoughts. Each item has a score varying from 0 to 6 points and the clinician can use patient information from different sources to determine the score. In contrast to the HRSD, it has the advantage of not counting with anxiety items, although it does have several **somatic or vegetative items** that **hamper** its use in patients with a predominance of physical symptoms. The assessment timeframe corresponds to the previous week or previous three days, and a Spanish version was validated in 2002 (Lobo et al., 2002).

As in the HRSD, response to treatment was established as a decrease in the initial score of ≥ 50%, partial response is between 25 and 49%, and non-response was defined as a reduction of < 25%. Remission is when the score ≤ 8-12 (Shelton, 2006).

The overall score ranges from 0 to 60 and is produced from the sum of the assigned scores in each item. Despite there not being established cutoff points, Table 6 shows the ones that are recommended.

Table 6. Recommended MADRS cutoff points and descriptors

	No Depression	Mild	Moderate	Severe
Recommended cutofff points	0-6	7-19	20-34	35-60
Other cutoff points in clinical trials	0-12	13-26	27-36	37-60

Adapted from Bobes et al., 2004 (Bobes et al., 2004).

Brief Patient Health Questionnaire and Wholley questions

The Brief Patient Health Questionnaire (PHQ-9) (Kroenke, Spitzer, & Williams, 2001) is the self-reported version of the depression module within the Primary Care Evaluation of Mental Disorders Procedure (PRIME-MD). It is composed by 9 items that assess the presence of depressive symptoms, according to DSM-IV criteria, over the previous 2 weeks. The diagnosis of major depression is defined by 5 of the 9 symptoms being present "for more than half the days" in the previous two weeks and one of the symptoms being associated with mood or anhedonia. The suicidal ideation item is considered positive for the diagnosis independently from its duration. Scores range from 0 to 27 and each of the items from 0 (never) to 3 (more than half the days). The questionnaire also includes an additional question to explore the degree of interference regarding symptoms in daily life. A Spanish version has been validated and proved to have similar properties to the original (Diez-Quevedo, Rangil, Sanchez-Planell, Kroenke, & Spitzer, 2001).

Table 7. Recommended PHQ-9 cutoff points and descriptors

	Minimal or mild depressive symptoms	Mild	Moderate	Severe
Cutofff points	<10	10-14	15-19	20-27

Adapted from Kroenke et al., 2001 (Kroenke et al., 2001).

The Whoolley questions (Whooley, Avins, Miranda, & Browner, 1997) are two items focusing on mood and anhedonia in the PHQ-9: "During the past month, have you often been bothered by feeling downhearted, depressed, or hopeless?" and "During the past month, have you often been bothered by little interest or pleasure in doing things?". However, in this version, the response is dichotomous (Yes/No) and has a cutoff point of 1. Due to its

psychometric properties, it has been recommended for identifying depression in patients with risk factors (National Collaborating Centre for Mental Health, 2009; U.S. Preventive Services Task Force, 2009).

Other questionnaires

Table 8 shows other questionnaires often used in the evaluation of depression.

Table 8. Other self-administered questionnaires used to assess depression

Instrument	Items	Features	Cutoff point	Adaptation and validation in Spanish
Zung Self-Rating Depression Scale (Zung, 1965)	20	Quantifies the frequency of depressive symptoms, with cognitive and somatic symptoms having greater weight (16 items).	50	Conde et al. (1970); Aragonès et al. (2001)
Centre for Epidemiology Studies Depression Rating Scale (CES-D)	20	Explores different symptoms of depression	16	Soler et al. (1997); Vázquez et al. (2007)

Sources: (Aragonès-Benaiges, Masdèu-Montalà, Cando-Guasch, & Coll-Borràs, 2001; Conde, Escribá, & Izquierdo, 1970; Radloff, 1977; Soler et al., 1997; Vázquez, Blanco, & López, 2007; Zung, 1965).

Treatment

Treatment of depression in adults should be **comprehensive** of all **psychotherapeutic**, **psychosocial**, and **pharmacological** interventions which may improve wellbeing and functional capacity. Therefore, the management of depression should include psychoeducation, individual and family support, coordination with other professionals, comorbidity care, and regular monitoring of mental and physical status. The selection of the mode and scope of treatment should be coherent with clinical findings and other factors, such as previous history, the availability of treatment, patient preference, and the possibility to offer support and containment in the environment.

Furthermore, psychotherapeutic treatment should be ensured for patients in need for it, and psychological interventions other than cognitive behavioral therapy, problem-solving therapy or interpersonal therapy should be considered when addressing comorbidity or the complexity of family/marital relationships, commonly associated with depression (Working

group of the Clinical Practice Guideline on the Management of Depression in Adults, Ministry of Health, & Galician Agency for Health Technology Assessment (avalia-t), 2014).

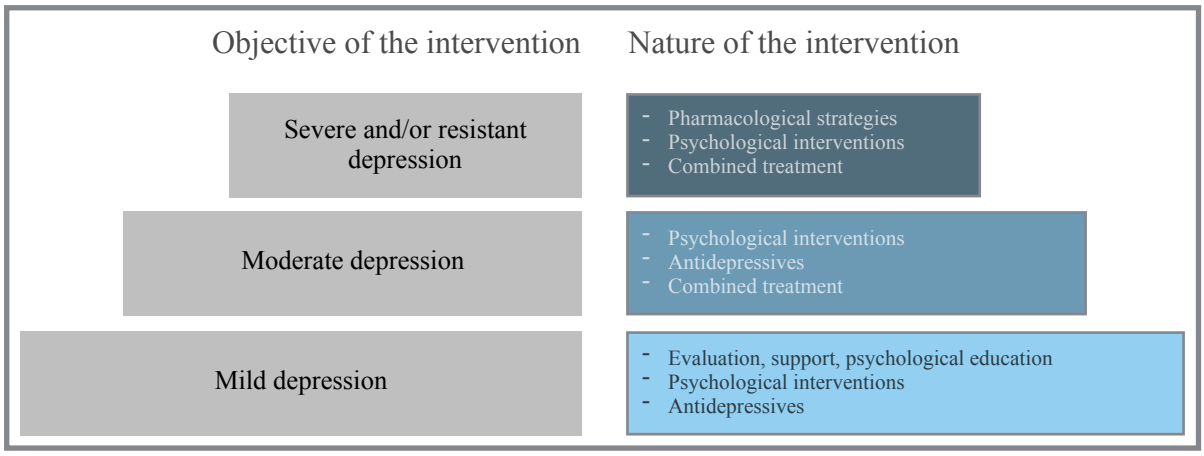
Among the myriad of models and interventions, designed to promote a shared care approach between primary and specialist care (Calderón et al., 2013; National Collaborating Centre for Mental Health, 2009), **stepped-care and collaborative care models** are likely the most promising. The management of depression in adults should be performed as a stepped care and collaboration model between primary care and mental health, promoting interventions and treatments tailored to the status and evolution of the patient. These two models will be discussed in this section. A brief mention will also be made to pharmacotherapy.

The stepped-care model

Stepped-care treatment models are intended to enhance effectiveness by providing less intensive interventions in accordance with patient status and development. In fact, stepped systems are an attempt to formalize ongoing care and maximize effectiveness (National Collaborating Centre for Mental Health, 2009), as **interventions** are implicitly **scaled** depending on complexity and severity of diseases in health care. Some of the premises of this model are a multidisciplinary work approach and collaboration between primary and specialized care, which is stratified so that the first proposed step is the least intensive intervention in primary care, while for those cases with an insufficient response to intensifying intervention, the most appropriate level of care is specialized (Palao, Pérez-Solà, Aragonés, & Jódar, 2010).

This treatment model is usually proposed by NICE, and the guidelines on depression in adults as well as depression with chronic diseases follow this model in their management recommendations (National Collaborating Centre for Mental Health, 2009). This organization model has also been suggested in Spain for the management of depression services (Palao et al., 2010).

Figure 13. Stepped-care model in depression management.



Adapted from various sources (National Collaborating Centre for Mental Health, 2009; Palao et al., 2010).

Collaborative care

Collaborative care models that follow the Chronic Care model have been implemented to improve the management of depression in primary care, where its effectiveness has proved to better the care process and clinical outcomes (Gilbody, Bower, Fletcher, Richards, & Sutton, 2006). This care model furthers interventions by care managers, often nurses, who mainly aim to improve the welfare and quality of life of depressed patients. It also facilitates a better structure in quality healthcare by planning nursing care that is paired to the patient's needs, while **integrating and coordinating** other personnel interventions (GPs, psychiatrists, psychologists, and other health professionals).

Pharmacotherapy

Antidepressants are aimed at improving symptoms associated with depression. They can be classified according to their chemical structure and mechanism of action (Table 9), and their therapeutic effects have a latency in the onset that ranges from 2 to 4 weeks, albeit some studies show an earlier response, especially in patients who achieve remission of their symptoms (Taylor, Freemantle, Geddes, & Bhagwagar, 2006). In general, the more severe the depressive symptoms, the more benefit provided by the drug treatment.

Table 9. Antidepressant drugs in Spain

Classic antidepressants		
Non-selective MAOI	Tranylcypromine	
MAO-A selective MAOI	Moclobemide	
Heterocyclics	Tricyclics: Imipramine, Clomipramine, Trimipramine, Amitriptyline, Nortriptyline, Doxepin	Heterocyclics: Amoxapine, Mianserin, Maprotiline
New generation		
SSRI	Citalopram, Fluoxetine, Fluvoxamine, Paroxetine, Sertraline, Escitalopram	
NDRI	Bupropion	
SNRI	Venlafaxine, Desvenlafaxine, Duloxetine	
SARI	Trazodone	
NaSSA	Mirtazapine	
NRI	Reboxetine	
Melatoninergic agonist	Agomelatine	

MAOI: Monoamine Oxidase Inhibitors; TCA: Tricyclic Antidepressants (reuptake inhibitors of 5-HT and NA); SSRI: Selective Serotonin Reuptake Inhibitors; NDRI: Norepinephrine-Dopamine Reuptake Inhibitors; SNRI: Serotonin-Norepinephrine Reuptake Inhibitors; SARI: Serotonine Antagonists and Reuptake Inhibitor (5-HT2 receptor and weak 5-HT uptake inhibitor); NaSSA: Noradrenergic and Specific Serotonergic Antidepressant; NRI: Norepinaphrine Reuptake Inhibitor.

Adapted from Working group of the Clinical Practice Guideline on the Management of Depression in Adults et al., 2014 (Working group of the Clinical Practice Guideline on the Management of Depression in Adults et al., 2014).

Depression and chronic pain

Depression is commonly observed in a **dyad** with chronic pain. Banks and Kerns (Banks & Kerns, 1996) introduced the **diathesis-stress model for pain and depression**, putting forth the idea that chronic pain patients who become depressed may suffer from a certain premorbid psychological predisposition toward developing depression. Multiple factors are involved in the depression-pain linkage, such as neurobiological, genetic, and precipitating environmental factors, also counting psychological, social, and cognitive influences (Bekkouche, Wawrzyniak, Whittaker, Ketterer, & Krantz, 2013; Covic et al., 2003; Gale, Deary, Cooper, & Batty, 2012; Goesling, Clauw, & Hassett, 2013; Mongini et al., 2009; Pulvers & Hood, 2013). Another model that helps explain the components involved in the

association between depression and pain is Engel's **biopsychosocial model** (Engel, 1977). On the basis of this approach, psychological stress, or the extent to which individuals feel that external demands exceed their ability to cope, has also shown to significantly correlate with pain and depression (Candrian, Farabaugh, Pizzagalli, Baer, & Fava, 2007; Kuiper, Olinger, & Lyons, 1986; Menzies, Lyon, Elswick, Montpetit, & McCain, 2013; Pizzagalli, Bogdan, Ratner, & Jahn, 2007). Other similar factors that have proved a link to pain include self-efficacy (E. Miró, Martínez, Sánchez, Prados, & Medina, 2011), mastery (Bierman, 2011), mental defeatism (Tang, Goodchild, Hester, & Salkovskis, 2010), catastrophizing, hopelessness and helplessness (Fahland, Kohlmann, Hasenbring, Feng, & Schmidt, 2012), and personal control (Q. Wang, Jayasuriya, Man, & Fu, 2015). Furthermore, an individual's attachment style also influences the relationship between depression and pain (Andersen, 2012; Martínez et al., 2012; Meredith et al., 2006a; Sockalingam et al., 2013; Tremblay & Sullivan, 2010), added to certain interpersonal problems that are correlated with a high prevalence of pain and depression: submissiveness and nonassertiveness, and self-sacrificing and friendly submissive behavior (Adler & Gattaz, 1993; Lackner & Gurtman, 2004).

Approximately, between 30% and 60% of chronic pain patients have comorbid depression (Goesling et al., 2013). The prevalence of a lifetime history of major depression or another mood disorder is even higher; furthermore, these data are muddled by a 50% prevalence of pain in patients with a primary diagnosis of depression (Crofford, 2015b). Depressed patients notably report more unexplained physical symptoms, such as pain and fatigue, and use more health resources than nondepressed patients (Bair et al., 2003). These findings highlight the conception of a **bidirectional relationship** between the presence and severity of pain and depression: in fact, a large longitudinal study of primary care patients with persistent pain of the back, hip, or knee stated that change in pain was a strong predictor of depression severity, and vice versa (Kroenke et al., 2011). There is extensive evidence of the high conjoint prevalence of mental health conditions with chronic pain (Bair et al., 2003; Banks & Kerns, 1996; Haythornthwaite, Sieber, & Kerns, 1991).

Indeed, depression and pain often coexist, respond to similar treatments, aggravate one another, and share biological pathways and neurotransmitters (Blier & Abbott, 2001; Gallagher & Verma, 1999). Depression has a direct effect on the development of pain and some studies have also shown indirect effects, or mediation, of depression on pain, whereas

other studies showed depression as an intervening variable (between another variable and pain) in path analysis. For instance, in the fear-avoidance model (Lethem, Slade, Troup, & Bentley, 1983), depression is posited as a mediator of prospective bonds between the fear-avoidance model and pain variables, achieving a better prediction of model variables (Seekatz, Meng, & Faller, 2013). Additionally, in the communal coping model of catastrophizing (Thorn, Ward, Sullivan, & Boothby, 2003), catastrophizing thought has a direct effect on pain intensity and predicted the affective component of pain through depression, as well.

Moreover, **pain** is even considered as a **somatic symptom of depression** (Kroenke, 2003; Seifsafari, Firoozabadi, Ghanizadeh, & Salehi, 2013). Symptoms of depression increase the risk of future episodes of pain, such as neck pain, low back pain, and cutaneous pain (Carroll, Cassidy, & Côté, 2004; Pinheiro et al., 2015); the greater the severity of depression, the higher the risk of pain (Pinheiro et al., 2015). Further, a systemic review accounted that symptoms of depression worsen the course of pain (Pinheiro et al., 2016). One study showed that depression was a robust and independent predictor for the onset of a neck and low back pain episode (Carroll et al., 2004), and another study found that symptoms of depression predicted the pain trajectory for up to 6 months (Schieir et al., 2009). Yet another study used a structural equation model to show that depression came before pain in cancer patients (Trudel-Fitzgerald, Savard, & Ivers, 2013).

Overall, the vast evidence of correlation can be **biologically** argued through the mediation of depression and pain by the neurotransmitters serotonin (5-HT), norepinephrine (NE), hormones, and cytokines through diverse albeit overlapping neuroanatomical pathways (Goldstein et al., 2004; Torta et al., 2014). A decline in 5-HT and NE, in particular in the limbic areas, is associated to the monoaminergic hypothesis of depressive disorders. Simultaneously, a similar decrease occurs in areas of the descendent inhibitory system, such as the periacqueductal griseum. Accordingly, the neurotransmitter deficit produces both a bad mood and an increased pain (Torta & Ieraci, 2013). At the same time, an alteration of the immune system, such as an increase of pro-inflammatory cytokines, can be related to depressive disorders and pain: the cytokine involvement in neurogenic inflammation and disease behavior is largely documented. For instance, cytokines seem to contribute in sensitizing deep tissue nociceptors of chronic widespread pain patients, and the combined

peripheral impulse input and enhanced central pain sensitivity may be the cause of widespread chronic pain disorders (Staud, 2011). It has also been suggested that the medial prefrontal cortex plays an important role in mediating the interaction between depressive symptoms and clinical pain severity in RA, perhaps by engaging brain areas significative to the processing of affective components (Schweinhardt et al., 2008). Additionally, elevated immune-inflammatory signaling is a relevant mechanism in the pathogenesis of mood disorders (Krishnadas & Cavanagh, 2012), which is confirmed as well by the fact that a simultaneous treatment with anti-inflammatory agents can sometimes enhance the therapeutic efficacy of antidepressants (Brunello et al., 2006). Pro-inflammatory cytokines [chiefly interleukin (IL)-2, IL-6, and tumor necrosis factor (TNF)-α] can affect mood via several paths: through the increase of the cortisol releasing hormone (CRH) and the global activation of the hypothalamus-pituitary-adrenal (HPA) axis, by glucocorticoid resistance due to a deficit of glucocorticoid receptor expression, by a decrease of T3, and by the excitotoxicity produced by the glutamate increase at microglial and astroglial level (Krishnadas & Cavanagh, 2012).

Indeed, there is a significant **interaction** between the **neurobiological** factors involved in **emotional** components, **rheumatic** diseases, and their relationship. The pathogenesis of mood, anxiety, sleep disorders, and pain in rheumatic diseases is multifactorial and often overlapping in certain factors, such as genetic, changes in the central nervous system and autonomic nervous system, inflammatory alterations, and environmental factors (Torta & Ieraci, 2013). This could help understand how pain in itself is correlated with anxiety, mood depression, and chronic stress (Torta & Ieraci, 2013). The depressive mood lowers the pain threshold and exacerbates pain perception both emotionally and cognitively; whereas chronic pain leads to strained relationships, decreases perceived self-efficacy, augments disability, and causes initial demoralization and then true depression (Torta & Ieraci, 2013; Torta & Munari, 2010).

Hence, from this point of view, many symptoms −e.g. fatigue, pain, cognitive impairment− must be seen from **two pathogenic perspectives**. While these symptoms are linked to the somatic disease itself, like FM, they also have an aspect which stems directly from mood depression. Therefore, the therapeutic approach has to encompass both

pathogeneses; for instance, by treating pain, when necessary, with analgesic and psychopharmacologic/psychological strategies (Torta et al., 2014).

As per depression in FM, a high prevalence of comorbidity has been largely documented (Arnold et al., 2006; Hudson, Arnold, Keck, Auchenbach, & Pope, 2004; Weir et al., 2006). The literature on the subject suggests an increase of comorbidity with depressive symptoms and a lifetime prevalence of major depressive disorder. Also, an association between FM and depression was found in epidemiological studies (Patten, Williams, & Wang, 2006). Several investigations corroborate the hypothesis of a predominance of negative rather than positive emotions in FM (Davis, Zautra, & Smith, 2004; Finnan, Zautra, & Davis, 2009; Gross & John, 2003; Sayar, Gulec, & Tppbas, 2004; Van Middendorp et al., 2008; van Middendorp et al., 2010; Zautra, Johnson, & Davis, 2005). Symptoms of depression are present in 26-71% of FM patients; a rate that appears very high in comparison to RA subjects, for instance, who are depressed in 14-23% of cases (Capraro et al., 2012; Murphy, Dickens, Creed, & Bernstein, 1999; Williams, 2003). In fact, antidepressant treatment used in FM has been found to be effective with both depression and pain (Häuser, Üceler, & Sommer, 2009), which has contributed to bring forth the hypothesis of an etiological link between pain and depression in FM. In a functional magnetic resonance imaging (fMRI) study, the neuronal activation pattern of the pain neuromatrix in FM was found to be modulated by comorbid depression, producing an increase of activation in the brain areas involved in affect processing, such as the cingulate cortex, the anterior insula cortex, and the amygdala (Giesecke et al., 2005). Additionally, the alterations in the HPA stress axis in FM are similar to those described in depression (Lund et al., 2006; McBeth et al., 2007; Wingenfeld et al., 2007). One study suggested that depression in FM chiefly determines pain perception, as opposed to RA where pain is rather due to peripheral stimuli (Scheidt et al., 2014); the main hypothesis is that anxiety and depression lower the pain threshold, thus exerting an influence on pain perception (Giesecke et al., 2005).

With regards to arthritis, the Land et al. (van't Land et al., 2010) study provides evidence of the damaging impact of depression on psychosocial functioning without serving as an independent cause for the development of arthritis, noting that disability could be a mechanism through which arthritis may lead to depression (van't Land et al., 2010). Furthermore, depression has been shown to contribute to adverse health outcomes in

previously diagnosed arthritis patients, for instance, exacerbating inflammatory processes, interfering with functioning, decreasing medical adherence, and aiding maladaptive health behaviors that create risk for greater disease activity and medical comorbidities (Nicassio, 2010; van't Land et al., 2010). Self-sacrificing tendencies have also been proved as moderators in the relationship between pain and physical symptoms in RA (Bai et al., 2009; Hyphantis, Goulia, & Carvalho, 2013), which is linked to depression (Chance et al., 1996). Certain psychological approaches have proven effective in randomized clinical trials in fostering adaptive coping and health behaviors, and palliating pain, disability, and mood disturbance in patients with RA and osteoarthritis (Astin, Beckner, Soeken, Hochberg, & Berman, 2002; Dixon, Keefe, Scipio, Perri, & Abernethy, 2007).

Consequently, authors such as Birtane et al. (Birtane, Uzunca, Tastekin, & Tuna, 2007) have depicted RA and FM as having a greater incidence of poor physical and psychological functioning in comparison to healthy controls, as well as they have identified higher depression levels in FM patients than in RA. In effect, FM is associated with the worst psychological functioning, given the frequency of depressive symptoms in individuals with FM (Wolfe et al., 2010). Thus, it is evident that addressing poor psychological status, and depression in particular, in musculoskeletal conditions is critical due to its impact on the condition. Coadjuvant relationships may occur between pain and depression and disability, yielding a cycle of poor mental and physical health (G. J. Walker & Littlejohn, 2007). Despite the fact that the pathophysiology of RA and FM are inherently different, the pain system still remains the common substrate, which can be functionally triggered by distress, as with the rest of chronic painful and disabling disorders. This is paramount evidence in advancing towards the comprehension of the causal mechanisms for FM, with its outcome of highly significant effect on the quality of life, and therefore also of improving the management of RA and related musculoskeletal conditions (G. J. Walker & Littlejohn, 2007).

Quality of life and health-related quality of life

Pain is the physical symptom-based condition most frequently reported in general population and primary care (Kroenke, 2003), while depression and anxiety are the two most common psychological conditions (Ansseau et al., 2004). Additionally, pain, depression, and anxiety are among the leading causes of functional impairment, disability related to work, and healthcare expenses (Greenberg et al., 1999; Institute of Medicine, 2011; W. F. Stewart, Ricci, Chee, Hahn, & Morganstein, 2003). Moreover, **pain**, **depression**, and **anxiety** commonly co-occur and have **additive and adverse effects** on **health-related quality of life (HRQoL)**, functional impairment, and response to treatment (Bair et al., 2003; Bair, Wu, Damush, Sutherland, & Kroenke, 2008; Lowe et al., 2008). Chronic pain influences all aspects of life, including work, quality of life, and functional ability (Gureje, Von Korff, Simon, & Gater, 1998; Katz, 2002), thereby reducing physical, mental, and social wellbeing (Becker et al., 1997). Furthermore, depression can lead to poor treatment compliance and increase morbidity and mortality (Ang et al., 2005; G. J. Walker & Littlejohn, 2007; K. B. Wells et al., 1989). In chronic pain patients, evaluating HRQoL is an important focus, and researchers have studied the effects of depression on HRQoL, revealing very poor scores (Becker et al., 1997; Elliot, Renier, Anderson, & Palcher, 2001; Hays, Wells, Sherbourne, Rogers, & Spritzer, 1995; Lenert & Kaplan, 2000; Spitzer et al., 1995; K. B. Wells & Sherbourne, 1999; K. B. Wells et al., 1989). Major depressive disorder patients have HRQoL scores among the lowest observed for many illnesses, below than those found in severe cardiopulmonary diseases or gastrointestinal disorders (Elliot, Renier, & Palcher, 2003). The impact of depression and chronic pain on reported HRQoL is a testimonial of how complex the relationships are amongst these three concepts (Becker et al., 1997; Elliot et al., 2001; Elliot et al., 2003; S. J. Wang, Fuh, Lu, & Juang, 2001).

Indeed, people with medical illnesses have reported increased rates of mental disorders, especially depression and anxiety, in contrast to those without them (Buist-Bouwman, de Graaf, Volleberg, & Ormel, 2005; Katon & Ciechanowski, 2002; K. M. Scott, Browne, McGee, & Wells, 2006). Findings have highlighted a poorer physical health status

among people with mental disorders relative to those without mental disorders (Harter, Conway, & Merikangas, 2003; Kendrick, 1996; K. M. Scott et al., 2006). Whereas physical (Sprangers et al., 2000; A. Stewart et al., 1989) and mental disorders (Beekman et al., 2002; Berardi et al., 1999; Bijl & Ravelli, 2000; Ormel et al., 1994) negatively affect disability, physical-mental comorbidity has been posited as even more disabling with regards to functioning (Arnow et al., 2006; Stang et al., 2006; M. Sullivan, LaCroix, Russo, & Walker, 2001). Research has shown that in the case of FM, there is a higher incidence of depressive symptomatology, a stronger impairment of quality of life, and worst illness perception, in particular with respect to RA patients (Capraro et al., 2012). In fact, a recent systematic review found that depression and self-efficacy are outcome predictors irrespective of intervention in self-management programs for chronic pain patients; therefore, these factors should be targeted at early stages in management programs, in order to prevent transition to chronic pain disability (Miles et al., 2011).

Moreover, there are findings that suggest a relative specificity between type of disorder and the area of HRQoL affected; that is, while chronic physical conditions mainly affect the physical component of a given measure, mental disorders affect both the physical and the mental component, especially the latter (Pinto-Meza et al., 2009). Hence, mental disorders tend to be more disabling than chronic physical conditions (Armenian, Pratt, Gallo, & Eaton, 1998; Hays et al., 1995; Ormel et al., 1998; Pinto-Meza et al., 2007), and mood disorders are particularly impairing (Pinto-Meza et al., 2007; Spitzer et al., 1995). However, it must be noted that what seems to be most disabling is chronic pain and its interaction with mood disorders: studies have shown that most of the HRQoL loss in the physical component of the measure stems from the presence of chronic pain and that mood disorders contribute to more impairment to chronic pain patients (Pinto-Meza et al., 2009). Also, chronic pain patients often report disruption in the HRQoL sleep domain (Björnsdóttir, Jónsson, & Valdimarsdóttir, 2014; Breivik, Collett, Ventafridda, Cohen, & Gallacher, 2006; Turk & Cohen, 2010; Wagner, DiBonaventura, Chandran, & Cappelleri, 2012), and studies show that pain catastrophizing (Buenaver et al., 2012) and negative mood (O'Brien et al., 2010) may foster sleep disturbances in this patient group. These findings are consistent with the literature on chronic pain patients, who often describe exacerbated negative consequences such as sleep-related problems, but also substantial pain intensity, depressive symptoms, increased

sick leave, and reduced emotional wellbeing, all conditions that result in suffering and large societal costs (Casarett, Karlawish, Sankar, Hirschman, & Asch, 2001; Lundberg & Gerdle, 2015; Robinson et al., 2005; Turk et al., 2008).

Definition

The World Health Organization (WHO) defines **quality of life** as individuals' perception of their own situation within the framework of their culture and value systems and with regard to their objectives, expectations, standards, and interests (World Health Organization, 1947). It is a vast ranging, fundamental but very subjective notion that entails the person's physical wellbeing, psychological state, autonomy level, social relationships, personal beliefs, and their interactions to salient features of the environment. As a concept, it innately merges sustained satisfaction with the diverse aspects of one's life (Fayers & Machin, 2007). Quality of life inherently encompasses the idea of **value**, or the way one state of existence is perceived as being superior to another (Patrick & Erickson, 1993).

The **World Health Organization** listed the domains and aspects that entail quality of life (Table 10), confirming the overall inclusive nature of the notion (Skevington, 1998).

Table 10. Domains and aspects of quality of life, as defined by World Health Organization Quality of Life Group 1995 (Skevington, 1998)

Domain I: Physical	
• Pain and discomfort • Energy and fatigue • Sexual activity	• Sleep and rest • Sensory functions
Domain II: Psychological	
• Positive feelings • Thinking, learning, memory, and concentration (cognitions) • Self-esteem	• Body image and appearance • Negative feelings
Domain III: Level of Independence	
• Mobility • Activities of daily living • Dependence on medication or treatment	• Dependence on non-medicinal substances • Communication capacity • Working capacity
Domain IV: Social Relationships	
• Personal relationships • Practical social support • Activities as provider/supporter	
Domain V: Environmental Health	
• Physical safety and security • Home environment • Work satisfaction • Financial resources • Health and social care services: availability and quality	• Opportunities of acquiring new information and skills • Participation in and opportunities for recreation and leisure activities • Physical environment (pollution, noise, traffic, climate) • Transport
Domain VI: Spirituality	
• Spirituality, religion, and personal beliefs	
General Facet	
• Overall perceptions of health and quality of life	

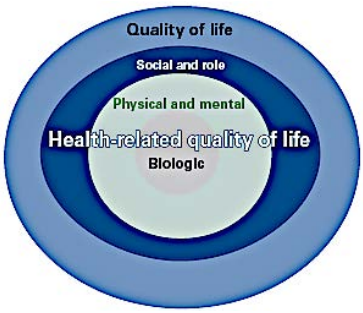
More specifically, HRQoL constitutes a **subjective outcome** measure chiefly used to assess the **impact of mental disorders and physical conditions**, thus denoting the quality of life aspects influenced by health status, and providing a multidimensional approach encompassing the physical, psychological, emotional, and social functioning of the patient (Pinto-Meza et al., 2009; Tander et al., 2008). It is therefore a concept that transcends traditional symptoms and includes the patient's subjective notions of wellbeing, satisfaction, functioning, and impairment. HRQoL is the value assigned to the quality of life after taking into account impairments, functional status, perceptions, and social opportunities, and as

influenced by illness, injury, treatment, or policy (Patrick & Erickson, 1993). It is becoming increasingly important to consider HRQoL in the domain of individuals with **chronic diseases**, in particular regarding resource allocation, design, intervention, and treatment (Currey, Rao, Winfield, & Callahan, 2003). The measurement of HRQoL provides additional relevant information about the subjective impact and thus burden of a chronic disease, in particular those causing pain and disability (Vetter, 2007). Consequently, HRQoL measures are commonly destined in **epidemiology** to quantify general health and functional status (Yamada, Matsudaira, Imano, Kitamura, & Iso, 2016).

HRQoL is a concept that emerged in **healthcare** with a focus in patients' wellbeing as a paramount element in both treatment and life support (Mceberg, 1993). Since its incorporation as a health status measurement, it has been widely and indistinctly used with the notion of **quality of life**, and there are very few authors that state a difference with the general term of quality of life. Thus, HRQoL is used to term health status, functional status, quality of life or needs assessment, which are often used as synonyms (Beckie & Hayduk, 1997; Gili & Feinstein, 1994; Guyat, Feeny, & Patrick, 1993; Nanda & Andresen, 1998; Urzúa, 2010). Some authors often identify HRQoL as a part of general quality of life, while others suggest that its use is undistinguishable of quality of life, therefore assuming that both constructs are measuring similar dimensions (Burke, 2001).

Despite this discrepancy, most authors coincide in the suggestion that quality of life must be differentiated from HRQoL. This is due to the fact that it is a medically used term for its interest in assessing the quality of changes as a medical intervention outcome (Guyat et al., 1993; Haas, 1999), because it must be restricted to the illness experience that the patient has (Haas, 1999), for it being able to depict the patient reported outcome of medical care (I. Wilson & Cleary, 1995), or in order to establish the impact of disease in daily life (Nanda & Andresen, 1998). This means that it is a measurement from the patient's perspective, and it is currently becoming a **therapeutic aim** (Alonso, 2000). Furthermore, there is also a paradigm shift in health perception: the original WHO definition for health is currently labeled as insufficient, evidencing the need for further focus on peoples's capabilities (Huber et al., 2011; Pietersma, van den Akker-van Marle, & de Vries, 2013).

Figure 14. Quality of life and HRQoL, adapted from Ware & Dewey (2000).



Thus, HRQoL focuses on subjective evaluation. The context of this assessment is circumscribed to the health status' influence, overall healthcare, and activities regarding prevention and promotion of wellbeing (Lizán-Tudela, 2009). The definition of HRQoL relies on the **three** fundamental dimensions of wellbeing, that is, **physical**, **psychological-cognitive**, and **social**. The physical aspects entail function deterioration, symptoms and pain resulting from the illness and/or its treatment; the psychological dimension includes a broad range of different emotional states, such as anxiety and depression, and intellectual and cognitive functions, such as attention and memory; the social aspects are related to isolation and self-esteem, linked to the social role in chronic disease (Ruta, Garrant, Leng, Rusell, & MacDonald, 1994).

Research in the last decades has focused on HRQoL due to some interesting findings, such as its independent association to mortality (Benyamini & Idler, 1999; Heidrich, Liese, Löwel, & Keil, 2002) and functional limitation (Idler, Russell, & Davis, 2000), and for its ability to predict healthcare resources independently from other variables (Lam, Fong, & Lauder, 2002; Pappa & Niakas, 2006). Findings indicated that self-rated health, measured through a question about general health, was associated independently from clinical diagnoses and other risk factors to mortality from 6 to 9 years (Idler & Angel, 1990; Kaplan & Camacho, 1983; Mossey & Shapiro, 1982). Also, HRQoL scarcely correlates with other biomedical or pathological illness variables, which suggests it doesn't quite overlap as much as it complements, and therefore offers a further global approach of the intended objective (Alonso, 2000).

Chronic pain disorders are often linked with impairment of physical and social functions, sleep alterations, and anxiety and depressive disorders, overall deeply affecting the patient's total life situation. However, these associated symptoms have a further negative

impact and might affect as much as the pain per se. Therefore, when evaluating treatment modalities and rehabilitation programs for chronic pain patients, it is essential to calculate their impact on the individual's function in daily life, and not only to strive for pain reduction. Hence, HRQoL **questionnaires** are frequently used as a complement to **morbidity** data (Brorsson, Bernstein, Brook, & Werko, 2002).

Models and theories for HRQoL

In biomedical literature, there are several models that aim to integrate global notions such as general health, perceived quality of life, and health-related quality of life value within the framework of what is relevant in healthcare services. HRQoL is the value appointed to the quality of life as affected by impairments, perceptions, functional status, and social opportunities, and as influenced by disease, injury, policy, or treatment (Patrick & Erickson, 1993). This is reflected in the fact that, until recently, the most common model was the adaptation of the WHO (Wood, 1980), which defines **four** differentiated levels of health problems assessment. The first level belongs to **pathology**, susceptible of being observed, through molecules, cells, and tissue. The second level is that of **deficiency**, or evident abnormalities in functions or structures of any organ. The third level corresponds to **impairment**, or functional limitation to carry out expected or normal social or family functions. Lastly, the fourth level is represented by the concept of **disability**, which manifests in a relational level, and is intimately associated with HRQoL (Pope & Tarlov, 1991). This model suggests a bidirectional cause-effect link amongst these levels, and contemplates no existing universal threshold that would determine the change from one level to the next (Alonso, 1998).

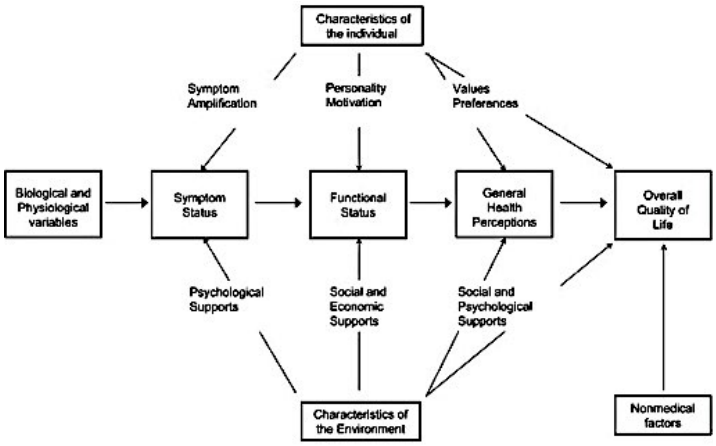
However, many authors have struggled with the theoretical basis of this term. What may possibly be adding to this conceptual confusion is the fact that health status and quality of life are intrinsically related, thus giving rise to the conception of HRQoL, or the acknowledgement of quality of life within the clinical care and research setting (Fayers & Machin, 2007). It has been suggested that the measures of this term should include indicators of pathology, functional status (physical, psychological, and social spheres), and of health perceptions (Patrick & Erickson, 1993); as well as, ideally, integrating information about death and about health opportunity (Patrick & Bergner, 1990). In spite of this, the **WHO-**

QOL group (WHOQOL Group, 1995) depicted quality of life as subjective, multidimensional, and encompassing **positive functioning and negative dimensions;** and the consequent myriad of applied studies and scarce analytical research has impeded acquiring consistency in the theoretical framework. From this perspective, quality of life was defined as the individual view of the position in life within the cultural and value system framework, and the link between this vital stance and goals, expectations, standards, and interests. The group then proposed six dimensions to this model, composed by different subdomains: physical, psychological, independence level, social relationships, environmental factors, and spiritual, religious and personal beliefs. Thus, quality of life could be generally defined as the perceived and self-reported degree of wellbeing yielding of the individual's assessment of their objective and subjective elements (e. g. having economic resources as an objective condition and being satisfied with those resources as subjective) in different life domains (Urzúa & Caqueo-Urízar, 2012). Since this model, which strictly speaking is a HRQoL model, did not reach a consensus amongst researchers, other models arose that were fundamentally linked to specific diseases. Arguably, the models presented below in this section are the most commonly used HRQoL models (Bakas et al., 2012).

Wilson and Cleary model

Indeed, and stemming from the lack of inclusion of the totality of variables commonly encompassed in the HRQoL evaluation and the failure to specify the associations between different measures, Wilson and Cleary (1995) developed a model. In it, they blended two different types of **comprehensive** approaches for health: the first kind from a **clinical perspective**, focusing on etiological agents, pathological processes and physiological findings; and the second from a **social sciences paradigm**, emphasizing functioning and general wellbeing dimensions (Figure 15).

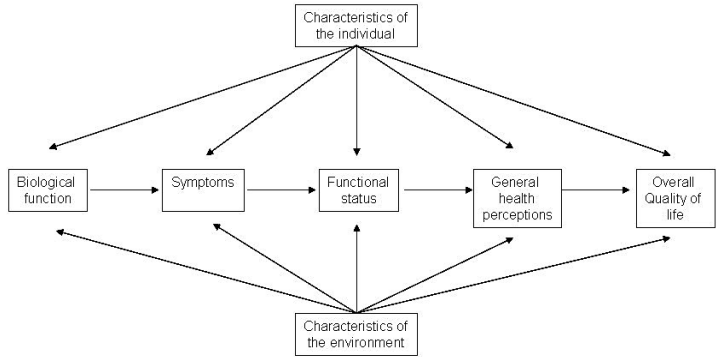
Figure 15. Wilson and Cleary's causal pathway model, from Wilson, I. and Cleary, 1995. The horizontal arrows show the main, albeit not exclusive, direction of causality.



Ferrans et al. model

A revision of Wilson and Cleary's model was published by Ferrans, Zerwic, Wilbur, and Larson (Ferrans, Zerwic, Wilbur, & Larson, 2005). Although the **five major domains** of the original remained, Ferrans and colleagues explicitly **defined individual and environmental characteristics**, and simplified the depiction by **removing non-medical factors and labels** on the relationship-portraying arrows (Figure 16). Further, they contributed **additional theoretical background** regarding the main concepts (Ferrans & Powers, 1992) and provided examples of instruments to optimize measurement. Following these authors' explanations, the model describes dominant causal relationships, albeit reciprocal links are implied. The revised conceptual model could be utilized in any healthcare discipline, as it helps explain the associations of clinical variables that relate to quality of life. The model suggests causal bonds between five different types of patient outcome measurements. In the end, quality of life is the patient's overall satisfaction with life.

Figure 16. Revised Wilson and Cleary Model by Ferrans et al., from Ferrans, Zerwic, Wilbur, & Larson, 2005.

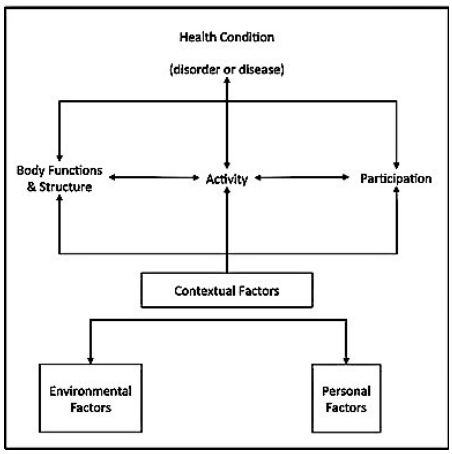


World Health Organization International Classification of Functioning Disability and Health

The **World Health Organization International Classification of Functioning, Disability, and Health (WHO ICF)** is a model created to depict health and health states, while providing a **standard and universal language** across disciplines and cultures (World Health Organization, 1980, 2001, 2007). The WHO ICF has shifted its conception from highlighting the "consequences of disease" in 1980 −as in the aforementioned model− to "components of health" in 2001 (World Health Organization, 1980, 2001). The more recent model covers toddlers, children, and adolescents (World Health Organization, 2007). The WHO has defined HRQoL as a person's perception of their health and health-related aspects of wellbeing (Cieza & Stucki, 2008; World Health Organization, 2001, 2007). Also, the model has further described health and health-related domains in terms of functioning and disability (Figure 17). The WHO ICF encompasses elements within two main parts: **Part 1** is centered on functioning and disability (body functioning and structures, activities, and participation), while **Part 2** focuses on contextual factors (environmental as well as personal).

However, and contrarily to the models by Wilson and Cleary (I. Wilson & Cleary, 1995) and Ferrans and colleagues (Ferrans et al., 2005), the WHO ICF is **not HRQoL-exclusive** and it explicitly indicates the causal and reciprocal relationships amongst concepts. Also, whereas the first two models were mainly intended for individual application, the WHO ICF model can explain the health of individuals, families, communities, cultures, and populations. Cieza and Stuki (Cieza & Stucki, 2008) argue that the WHO ICF categories included in functioning can serve to ultimately operationalize HRQoL, but are not the only potential application. The WHO ICF resembles more a mapping and classification structure than a guide for hypothesis formulation in the HRQoL domain.

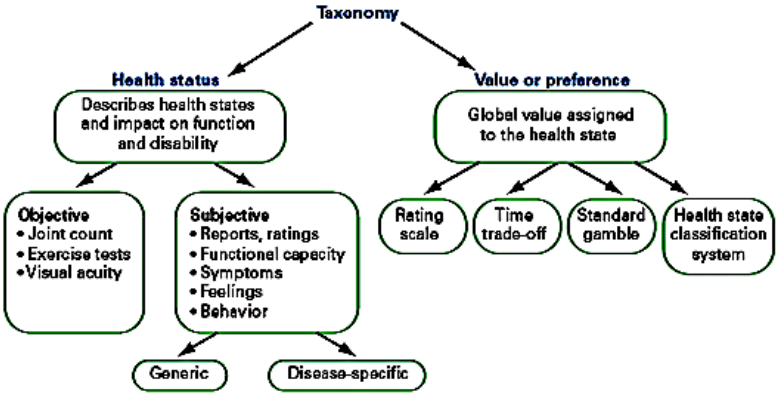
Figure 17. WHO ICF, from World Health Organization. International Classification of Functioning, Disability, and Health: ICF. 2001.



Evaluation and assessment instruments

In this section, different means of evaluating HRQoL will be discussed according mainly to the scope and aims of the present research. These instruments evaluate patients' reported perceptions of daily functioning, as well as physical, social, and psychological wellbeing. These numerous measures can be categorized into **three** basic types: **generic**, **condition-specific**, and **value** or preference-based (McHorney, 1999).

Figure 18. HRQoL taxonomy, adapted from Tsevat et al. (1994).



Generic and condition-specific health instruments are essentially created through traditional psychometric testing principles such as validity, reliability, and responsiveness (Hays, Anderson, & Revicki, 1993). In contrast, preference-based health instruments are

constructed according to perceived utility or value and the resulting rater's preference for a particular health state (Naughton & Shumaker, 2003). Preference-based health measures are a crucial part of the application of decision and cost-utility analysis in healthcare research and functioning, including in chronic pain medicine (Drummond, Sculpher, Torrance, O'Brien, & Stoddart, 2005; Korthals-de Bos, van Tulder, van Dieten, & Bouter, 2004).

Many HRQoL assessment tools are designed for general populations and disease-specific groups. Generic HRQoL questionnaires are intended for assessment in different patient groups as well as in healthy populations. They provide a comprehensive valuation of the complex continuum between wellbeing, disability, and death (McHorney, 1999); and contrarily to disease-specific measures, generic measures focus on breadth rather than sensitivity by prioritizing the common elements of health that transcend any particular disease (Maciejewski, 2006). On the other hand, disease-specific measures are used to evaluate the effectiveness of the treatment methods and outcomes of the patients in specific illnesses (Martinez, Ferraz, Sato, & Atra, 1995).

Generic HRQoL instruments

Generic HRQoL measures can be applied in both clinical care and research (Patrick & Erickson, 1993). These tools can enhance the signs and symptoms of illness and other common diagnostic test data (Higginson & Carr, 2001); and they can also strengthen a clinical trial by providing outcomes information on the beneficial and adverse treatment effects from the subjects' perspective (Bottomley et al., 2005). However, **two limitations** of these kind of measures are the potential for a ceiling effect versus a floor effect and the relative lack of sensitivity to change in one health are, such as pain (Atherly, 2006). A maximum ceiling effect occurs when a considerable amount of subjects are rated as being in good health on one generic instrument but not as much on another generic measure. Inversely, a generic instrument may not have the capacity to differ poor health from very poor health, yielding a minimum floor effect (Fayers & Machin, 2007). In other words, a generic measure is **designed to assess overall HRQoL** and therefore may not detect clinically significant change in a specific system or constellation of related symptoms. This is why it is commonly indicated to concurrently use a disease-specific HRQoL tool (Atherly, 2006; Hays, 2005).

Nowadays, the most common measure is in terms of **quality-adjusted life years (QALYs)**, a generic measure of disease burden and cost-effectiveness of healthcare interventions. QALYs merge the quality and quantity of life lived into a one-dimensional outcome (Drummond et al., 2005; Gold, Siegel, Russell, & Weinstein, 1996) in an attempt to estimate the value of health outcomes. Data collection is performed through use of surveys such as EQ-5D. These instruments provide valuations for different levels of predefined domains, such as pain and mobility, thus highlighting quality of life dimensions susceptible of influence by healthcare interventions. Hence, in essence, they are HRQoL measures and are often criticized for not capturing all aspects relevant to quality of life (Dolan, 2009; Pietersma et al., 2013).

Table 11. Most common adult generic HRQoL measurement instruments

Measurement instrument	Health dimensions or domains assessed	Completion time and method
36-Item Short-Form Health Survey (SF-36)	Physical Component Score • Bodily pain • Physical functioning • Role limitations due to physical problems • General health perceptions Mental Component Score • Vitality • Social functioning • Role limitations due to emotional problems • Mental health • Health transitions	7-10 min. (self-administered)
Dartmouth Primary Care Cooperative Information Charts (COOP Charts)	• Pain • Physical fitness • Daily activities • Social activities • Quality of life • Overall health • Change in health • Emotional status • Social support	<5 min. (self-administered)
Nottingham Health Profile (NHP)	• Pain • Energy level • Emotional reactions • Physical mobility • Social isolation • Sleep	10-15 min. (self-administered)

Measurement instrument	Health dimensions or domains assessed	Completion time and method
Sickness Impact Profile (SIP)	Physical dimension • Somatic autonomy • Mobility control Psychological dimension • Psychological autonomy and Communication • Emotional stability Social dimension • Mobility range • Social behavior	15-30 min.
World Health Organization Quality of Life with 100 Questions (WHOQOL-100) and WHOQOL-BREF	The WHOQOL-100 included 24 quality of life facets grouped into 6 domains. The WHOQOL-BREF is the result of finding a four domain solution more appropriate: Physical health • Activities of daily living • Dependence on medicinal substances and medical aids • Energy and fatigue • Mobility • Pain and discomfort • Sleep and rest • Work capacity Psychological • Bodily image and appearance • Negative feelings • Positive feelings • Self-esteem • Spirituality / Religion / Personal beliefs • Thinking, learning, memory, and concentration Social relationships • Personal relationships • Social support • Sexual activity Environment • Financial resources • Freedom, physical safety and security • Health and social care: accessibility and quality • Home environment • Opportunities for acquiring new information and skills • Participation in and opportunities for recreation / leisure activities • Physical environment (pollution / noise / traffic / climate) • Transport	10-15 min.
Euroqol five-item questionnaire (EQ-5D)	A global rating of current health using a visual analog scale (VAS) and five health dimensions: • Mobility • Self-care • Usual activities • Pain/discomfort • Anxiety/depression	≤ 5 min.

Measurement instrument	Health dimensions or domains assessed	Completion time and method
Health Utilities Index (HUI)	Rating scale, or standardized classification system using preference-based scoring. HUI-2: • Sensation • Mobility • Emotion • Cognition • Self-care • Pain • Fertility (not currently used) HUI-3: • Vision • Hearing • Speech • Ambulation • Dexterity • Emotion • Cognition • Pain	5-10 min.
Quality of Well-Being Index (QWB)	• Physical activities • Social activities • Mobility • Symptom/problem complexes	20 min.

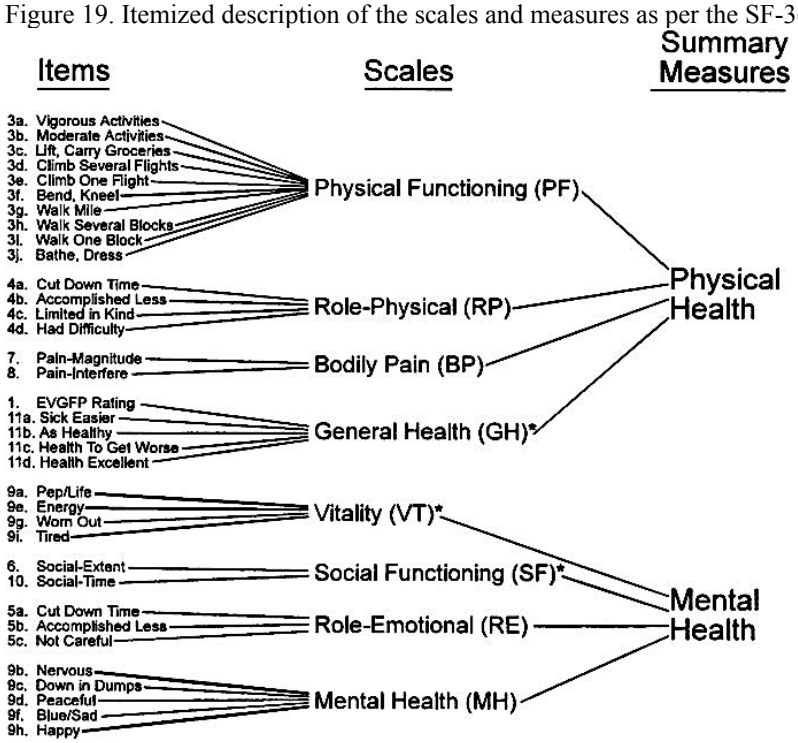
- *SF-36*

Further mention must be made on this questionnaire. It was developed in the early 90's in the United States in order to be applied in the **Medical Outcomes Study (MOS)** (J. E. Ware, Jr. & Sherbourne, 1992). It is a useful **generic HRQoL** that provides with a health status profile and can be used in general population and in specific subgroups, contrast the burden of diverse conditions and within many physical health disorders (Aaronson et al., 1998), detect health benefits yielding of a wide range of different treatments, and assess the health status of individual patients (J. E. Ware, Jr., 2000). The first Spanish adaptation of this instrument was also published in the 90's (Alonso, Prieto, & Anto, 1995) and its reduced version of 12 items, the SF-12, has also become a very commonly used tool in outcomes evaluation within the medical setting.

It is composed by 36 items that evaluate positive and negative health states. It was elaborated from an extensive questionnaire set used in the MOS, encompassing 40 health-related concepts. The minimum number of necessary concepts were selected in order to preserve the validity and operative characteristics of the initial test, which finally included 8 scales (see Figure 19): Physical Functioning (PF), Role-Physical (RP), Bodily Pain (BP),

General Health (GH), Vitality (VT), Social Functioning (SF), Role-Emotional (RE), and Mental Health (MH). Scores on these subscales can be combined to produce two summary scores: the **Physical Component Summary (PCS)** and the **Mental Component Summary (MCS)**. The PCS is obtained by positively weighting the 4 subscales in the physical domain (PF, RP, BP, and GH) and the remaining psychological domain subscales negatively; whereas the MCS is calculated by positively weighting the 4 mental dimension subscales (MH, V, SF, and RE) and negatively weighting the rest of the physical dimension. Additionally, the SF-36 includes a transition item to ask about the change in general health status in comparison to the previous year, which isn't used for any scale computation but provides useful information (Vilagut et al., 2005). Scores for all subscales are expressed ranging from 0 to 100, with a higher score representing a better state of health. It has good validity and reliability in both clinical and healthy community-based samples.

Figure 19. Itemized description of the scales and measures as per the SF-36, from Ware, J. E., Jr., 2000.



In 1996, version 2.0 was created from the original SF-36 (J. E. Ware, Jr., Kosinski, & Dewey, 2000) with the aim of improving the psychometric characteristics of both the PCS and the MCS.

The SF-36 is currently the **most commonly used** generic HRQoL measure in the world (Coons & Shaw, 2005). The Initiative on Methods, Measurement, and Pain Assessment

in Clinical Trials (IMMPACT) has proposed that this survey be included as a generic measure of physical functioning, essentially due to the large amount of data that allows for comparisons among different **chronic pain conditions** and applied treatments (Dworkin et al., 2005). However, despite it being a generic measure, it has been found to be a valid and reliable assessment tool in **RA** as well (Linde, Sørensen, Østergaard, Hørslev-Petersen, & Hetland, 2008; Matcham et al., 2014), correlating well with illness-specific measures such as the Health Assessment Questionnaire (HAQ) (Fries, Spitz, Kraines, & Holman, 1980) and the Arthritis Impact Measurement Scale (AIMS) (Meenan, Gertman, & Mason, 1980).

Disease-specific HRQoL instruments

Despite generic health status measures being applicable in a vast range of types and severities of disease, medical treatments or health interventions, and demographic and cultural groups (Patrick & Deyo, 1989), their limitations must not be disregarded. If applied inadequately, a generic tool may fail to isolate what is of greatest clinical or research relevance (Atherly, 2006). To palliate this deficiency, more that 200 disease-specific HRQoL measures have been created, including for many pain-related conditions (Bonomi, Shikiar, & Legro, 2000; McHorney, 1999). Condition-specific instruments are designed to evaluate specific diagnostic groups or patient populations and to recognize even small baseline differences and incremental changes in the specific and often essential domains or dimensions of a particular illness (Atherly, 2006). Consequently, these kind of measures are particularly appealing to subspecialty care clinicians and health outcomes investigators striving to identify a very concrete interventional benefit (Atherly, 2006).

However, such depth also brings with it at least **two potential drawbacks**. Firstly, a condition-specific instrument may fail to detect the impact of a disease on general function and wellbeing (Coons & Shaw, 2005). Secondly, with such a wide array of disease-specific measures, it is often complicated or even impossible to compare treatment effects among studies of the same or different illnesses. Thus, generic measures have tended to be seen in an overarching theme as common metric for comparisons for treatments, conditions, or patient groups, whereas disease-specific measures make for better complements rather than replacements for generic measures (Atherly, 2006).

Figure 20. The most commonly used generic and specific measures in FM patients, from Gusi, Olivares, Adsuar, Paice, & Tomas-Carus (2010). Quality of life measures in fibromyalgia. Springer New York. In the instance of RA patients, the Rheumatoid Arthritis Quality of Life Questionnaire (RAQoL) should be added to the diagram.

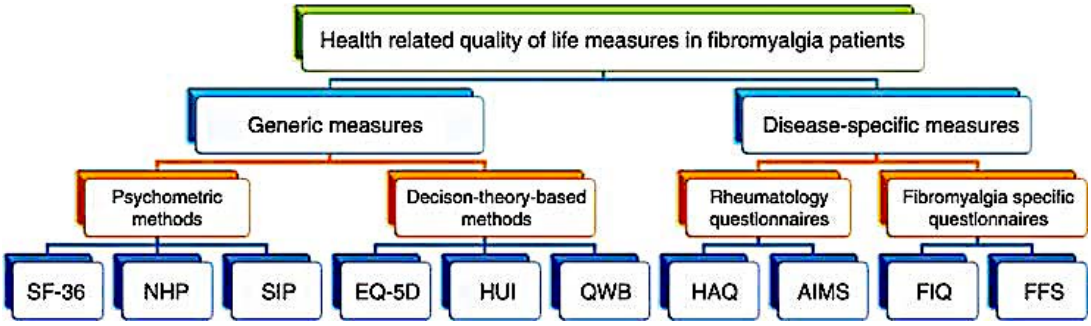


Figure 20 shows the main measures used in FM patients, albeit it applies to RA patients just as well, by adding into consideration the Rheumatoid Arthritis Quality of Life Questionnaire (RAQoL). Furthermore, in RA there are pooled indices of multiple measures (Goldsmith, Smythe, & Helewa, 1993) that have been created, such as the American College of Rheumatology (ACR) core data set (Felson et al., 1993; Tugwell & Boers, 1993; van Riel, 1992) and disease activity score (DAS) (Prevoo et al., 1995; van der Heijde, van't Hof, van Riel, & van de Putte, 1993). Table 12 displays the most common disease-specific HRQoL measures, with a special focus on rheumatic conditions.

Table 12. Most common adult specific HRQoL measurement instruments, focusing on chronic pain and rheumatic conditions

Measurement instrument	Health dimensions or domains assessed	Completion time and method
Roland-Morris Back Pain Questionnaire (RMQ)	Created by Culling. 24 pain and function yes/no items from the SIP relevant to chronic low back pain. Total score ranging from 0 (least disability) to 24 (greatest disability).	≤5 min. (self-administered)
Western Ontario and McMaster University Osteoarthritis Index (WOMAC)	For patients with osteoarthritis of the hip or knee. 24 questions with a five-point Likert or a 100 mm VAS format. • Pain • Disability • Joint stiffness	12 min. (self-administered)

Measurement instrument	Health dimensions or domains assessed	Completion time and method
Stanford Health Assessment Questionnaire (HAQ) and Multidimensional Health Assessment Questionnaire (MDHAQ)	The HAQ and MDHAQ are disease-specific as per RA patients, but may function as well for all rheumatic diseases. HAQ: • Physical function: 4-point Likert scale for functional disability (20 daily living activities in 8 categories of 2 or 3 activities). 8 categories: dressing, arising, eating, walking, bathing, reaching, gripping, and performing errands. • VAS to assess pain and patient global estimate of status. MDHAQ: • Physical function: 10 daily living activities. • VAS for pain and patient global estimate, but unlike the HAQ's 10-cm line requiring a ruler to score, in a numbered-circle format. • Patient self-report RA disease activity index (RADAI) joint count. • 3 non-formally scored psychological items concerning sleep, anxiety, and depression. • Review of systems. • Medical history. • Fatigue VAS. • Questions about change in status, morning stiffness, and exercise. • Demographic data. • Scoring templates for routine assessment of patient index data (RAPID) scores.	5-10 min. (self-administered)
Arthritis Impact Measurement Scales (AIMS)	Disease-specific instrument of physical, emotional, and social wellbeing as an outcome measure in arthritis (RA and osteoarthritis) patients. There is a shortened version, an expanded one (AIMS2), a short-form of the AIMS2 (AIMS2-SF), a children version, and a version for the elderly (Geri-AIMS). 9 scales: • Mobility • Physical activity (walking, bending, lifting) • Dexterity • Household activity (management of money and medication, housekeeping) • Social activities • Activities of daily living • Pain • Depression • Anxiety • Also in AIMS2: arm function, social support, and work	AIMS: 15 min. Shortened AIMS: 6-8 min. AIMS2: 20-30 min. AIMS2-SF: 10 min. Self-administered.
Fibromyalgia Impact Questionnaire (FIQ)	Measure of current health status in FM in clinical and research settings. • Physical function • Work status (missed days and job difficulty) • Depression • Anxiety • Morning tiredness • Pain • Stiffness • Fatigue • Wellbeing over the past week	Approximately 5 min. (self-administered)

Measurement instrument	Health dimensions or domains assessed	Completion time and method
FibroFatigue Scale (FFS)	Observer rating scale destined to assess the severity of symptoms in FM and chronic fatigue syndrome patients. 12 items evaluating: • Pain • Muscular tension • Fatigue • Concentration difficulties • Failing memory • Irritability • Sadness • Sleep disturbances • Autonomic disturbances • Irritable bowel • Headache • Subjective experience of infection	10-15 min. Observer-rated and therefore administered by a trained individual.

- *HAQ and MDHAQ*

In 1978, the **Health Assessment Questionnaire (HAQ)** was developed at Stanford University by James F. Fries, MD, and colleagues. It was one of the first self-report functional status −or disability− measures and is preeminent in many disease areas, such as arthritis. As a popular worldwide tool, it has become a mandated outcome measure in RA clinical trials, as well as in a wide variety of rheumatic diseases, including osteoarthritis, juvenile RA, lupus, scleroderma, ankylosing spondylitis, FM, and psoriatic arthritis. Additionally, it has been applied to HIV/AIDS patients and in normal aging studies. In this sense, and though a disease-specific questionnaire, the HAQ (and MDHAQ, by extension) may also be of value in all rheumatic and non-rheumatic diseases, potentially functioning as a generic questionnaire (Bruce & Fries, 2005). Furthermore, it emphasizes self-reported patient-oriented outcome measures, rather than process measures.

The HAQ includes a scale evaluating functional disability and consists of 20 activities of daily living categorized into eight domains of two or three activities, itemized on a four-point Likert scale ranging from 0 (no difficulty) to 3 (unable to do). The eight categories consist of dressing, arising, eating, walking, bathing, reaching, gripping, and performing errands. Points are added to the score if the patient depends on aids or devices for that category (Fries et al., 1980). The HAQ also adds two 10-cm VAS to evaluate pain and patient global estimate of status, the other two patient reported outcome (PRO) measures in the RA

Core Data Set (Felson et al., 1993; Fries et al., 1980; Tugwell & Boers, 1993; van Riel, 1992).

The HAQ has been modified for its use in standard care as a **multidimensional HAQ (MDHAQ)** (Pincus, Sokka, & Kautiainen, 2005; Pincus, Swearingen, & Wolfe, 1999), in order to save time for the rheumatologist and to improve the quality of patient visits (Pincus & Stein, 1997; Pincus & Wolfe, 2000, 2005; Wolfe & Pincus, 1999). Differences regarding the HAQ include: adding two more complex activities as activities of daily living; not asking queries related to aids, devices, or help from others, which complicates scoring and may not contribute relevantly, as well as potentially increasing scores in an artificial manner; both VAS are in a 21-numbered-circle format instead of a 10-cm line needing of a ruler to score; adding a patient self-report RA disease activity index (RADAI) joint count; including scoring templates and boxes to record scores; and adding three psychological items associated to sleep, anxiety, and depression, not formally scored, a 60-symptom checklist review of systems, medical history, fatigue VAS, items about change in health status, morning stiffness and exercise, and demographic data. Also, scoring templates are available for routine assessment of patient index data (RAPID) scores, encompassing RAPID 3 for physical function, pain, and global estimate as the three PRO measures in the ACR Core Data Set; RAPID 4, which adds self-report joint count; and RAPID 5, including a physician global estimate as well. RAPID 3 has been found to distinguish active from control treatment in RA clinical trials involving leflunomide (Pincus, Amara, & Koch, 2005; Pincus, Strand, et al., 2003), methotrexate (Pincus, Amara, et al., 2005; Pincus, Strand, et al., 2003), adalimumab (Pincus, Chung, & Segurado, 2006), and abatacept (G. Wells, Li, Maxwell, Maclean, & Tugwell, 2005) at levels comparable to ACR or DAS-28 criteria, and significantly correlates with DAS-28 in these trials as well as in standard clinical care (Wolfe, Michaud, & Pincus, 2005). As for the symptom checklist review of systems (ROS), obtaining more than 20 positives on the list suggests FM, while more than 30 is virtually pathognomonic for FM (Pincus, Yazici, & Bergman, 2007).

Indeed, **RAPID 3** is highly significantly correlated with DAS28 and CDAI (Pincus et al., 2010), making all of them **disease-specific indices for RA** (Tugwell & Boers, 1993; van Riel, 1992). Additionally, RAPID3 is useful in **chronic rheumatic diseases**, submitting "vital" information (Pincus, 2008). Thus, the MDHAQ might function as a **"generic"**

questionnaire, as all **rheumatic diseases** include limitations of functional status, pain, and poor global status (Fries & Ramey, 1997). The Health Assessment Questionnaire (HAQ), from which the MDHAQ derived, has been widely used to score disability and health-related quality of life in rheumatic diseases (Birrell, Hassell, Jones, & Dawes, 2000; Kvien, Kaasa, & Smedstad, 1998). RAPID3 could be used in all rheumatic diseases as well (and/or other conditions causing pain and functional impairment), allowing comparative studies of its values in different settings (Pincus & Sokka, 2007). RAPID3 has been mainly studied in RA, to complete physician's assessment of disease activity/severity (Berthelot, 2014).

Moreover, the MDHAQ pain VAS is more sensitive than the WOMAC to discern diclofenac/misoprotol from acetaminophen (Pincus et al., 2001) or celecoxib from acetaminophen (Pincus et al., 2004). In FM, it is possible to distinguish these patients from RA patients as effectively as ESR through the use of ratios of pain or fatigue to physical function scores, as well as the number reported on the review-of-systems symptom checklist (Callahan & Pincus, 1990; DeWalt, Reed, & Pincus, 2004). Additionally, the SF-36, which has been proved valid for use in RA patients, has results that correlate well with patient and physician global assessment and with HAQ scores (Talamo, Frater, Gallivan, & Young, 1997; Tuttleman et al., 1997), findings that can therefore be partially extended to the MDHAQ.

- *FIQ*

The **Fibromyalgia Impact Questionnaire (FIQ)** (Burckhardt, Clark, & Bennett, 1991) was created at the Oregon Health and Science University with the aim of reporting the total spectrum of FM-related problems and subsequent responses to therapy. Since its publication in 1991, it has been widely used to measure therapeutic efficacy and as a FM-specific HRQoL questionnaire. The FIQ is composed of 10 four-point Likert scale questions, the first one including 11 items associated with the ability to perform large muscle tasks in daily activities such as housework and shopping. Items 2 and 3 ask about days of wellbeing and missed work and housework days due to FM symptoms. Items 4 through 10 are horizontal linear scales marked in 10 increments for the patient to rate work difficulty, fatigue, pain, morning tiredness, stiffness, anxiety and depression.

The FIQ is scored so that a higher score indicates a greater impact of the syndrome on the patient's life, the maximum possible score being 100. The average FM patient obtains a

score around 50 and severely afflicted patients commonly score 70 or higher (R. Bennett, 2005).

It has become widely popular and counts notable advantages, such as its short format, easy use, and sensitivity to change in the condition.

Quality of life and chronic pain

Musculoskeletal disorders are among the most common causes for hospital applications: they lead to pain, functional impairment, work disability, and alter quality of life. With the aim of improving HRQoL of musculoskeletal patients, the WHO announced the decade 2000-2010 as the Bone and Joint Decade (A. D. Woolf, 2000). Disorders such as FM and RA are an important group of chronic diseases that can result in quality of life deterioration (G. J. Walker & Littlejohn, 2007), as both of these conditions are the most common reasons of musculoskeletal pain and disability. These two rheumatic conditions share a range of related outcomes: pain and fatigue, but also hardships with daily living activities, going from basic and intermediate functions to more complex tasks such as social roles and paid employment (Reisine, Goodenow, & Grady, 1987; Verbrugge & Juarez, 2006). In effect, patients with daily pain are more likely impaired in daily living activities and less likely to get involved in activities; these associations remained even after adjusting for the potential confounders of age, gender, race, cognitive functioning, and disabling conditions such as arthritis, stroke, congestive heart failure, and Parkinson's disease (Katz, 2002).

Chronic pain patients frequently undergo extensive examination procedures, and may feel misjudged by healthcare professionals when no clear pathology that would explain the pain is found. The lack of a clear-cut diagnosis can be a serious obstacle in finding treatment in the social care system, and due to reduced functional ability and possible loss of income, these patients often feel like a burden to their families (Allcock, Elkan, & Williams, 2007; Karoly & Ruehlman, 2006; Kowal, Wilson, McWilliams, Péloquin, & Duong, 2012). The **invisible nature of a pain condition** contributes to the difficulties of being properly understood by healthcare professionals, the social care system, and the patient's social network of family and friends (Allcock et al., 2007).

Chronic pain patients have been found to report a HRQoL as poor as that of palliative cancer patients (Fedoroff, Blackwell, & Speed, 2014; Fredheim et al., 2008). Additionally,

musculoskeletal disorders are the most common reasons for using healthcare resources (Allaire, Wolfe, Niu, Lavalley, & Michaud, 2005; Verbrugge & Juarez, 2006). Chronic musculoskeletal pain relates to impaired HRQoL, disability indices, and health risk behaviors (Strine, Hootman, Chapman, Okoro, & Balluz, 2005). Further, a study found that in over 1000 patients, participants with back pain and subjects with multiple pain localizations showed the poorest quality of life, and pain catastrophizing was linked the strongest with decreased quality of life, superseding pain intensity (Lamé, Peters, Vlaeyen, Kleef, & Patijn, 2005). Moreover, in rheumatoid conditions such as RA and osteoarthritis, pain has a negative impact and reduces quality of life, as well as gender and age, which have been reported to influence quality of life. Among women with RA, pain may be the strongest predictor for decreased wellbeing, but social support of rheumatoid patients has been found to have a potential moderating effect (Jakobsson & Hallberg, 2002).

Findings show that, almost without exception, individuals with **RA and FM** have **decreased or compromised quality of life** in comparison to healthy age-matched controls (G. J. Walker & Littlejohn, 2007). Both conditions similarly affect psychological and physical status and social roles as other chronic illnesses that contribute to the overall burden of disease, such as heart disease, chronic obstructive pulmonary disease, and diabetes (Australian Bureau of Statistics, 1998; Birtane et al., 2007; Verbrugge & Patrick, 1994). In fact, previous approaches for outcomes in rheumatic conditions placed a strong biomedical emphasis on disease processes, pathophysiology, and/or structural damage, with many models of functional status or quality of life implicitly assuming a linear correlation between disease processes and disability (G. J. Walker & Littlejohn, 2007). However, attempting to apply such models has evident shortcomings in common instances such as FM, with an unclear disease process (Chamie, 1995). Indeed, one obtains a limited account on quality of life without considering the individual's everyday tasks and roles (Maly, Costigan, & Olney, 2006)

More recently, models strive for an exploration of disability that acknowledges the buffering and exacerbating roles of demographic, physiological, psychological, social, and environmental factors regarding poor outcomes for chronic physical conditions (Verbrugge & Juarez, 2006). To this effect, the **WHO** promoted the **International Classification of Functioning (ICF)**, integrating a more inclusive **biopsychosocial framework** of disability,

health, and health-related states (World Health Organization, 2002). Thus, the ICF takes a neutral stance concerning etiology and allows for researchers to reach causal inferences through appropriate scientific methods, attempting to uproot assumptions that organic dysfunction, classified as impairment, is the essential perspective of disability and quality of life (Cieza et al., 2004; Weigl et al., 2004; World Health Organization, 2002). That is, an existing disability entails a cause, but the cause may not be enough to understand the resultant quality of life, since both RA and FM quality of life is not contingent on etiology (Odding et al., 1995; Vlieland et al., 1994; World Health Organization, 2002).

Potential differences yielding from a cross-cultural comparison

Traditionally, work incapacity deriving from musculoskeletal disorder has chiefly stemmed from the physical demands of employment: it constitutes a general assumption that symptoms and impairment arise from tissue injury and can be prevented by improvement of the tasks' ergonomic design in order to decrease mechanical loading. This biophysical paradigm adequately suits some types of musculoskeletal condition, but it has become increasingly obvious that the perspective is of restricted application to the main musculoskeletal causes of work disability, that is, common painful illnesses of the back, neck, and upper limb (Vargas-Prada & Coggon, 2015).

Moreover, the majority of disabling pain of the back and upper limb has a non-specific etiology and no evidence of underlying tissue injury, and even in the presence of clear pathologic findings, it often does not explain the symptom (Endean, Palmer, & Coggon, 2011). Further, the prevalence of musculoskeletal disease and disability has suffered notable temporal changes that are not sufficiently explained by varying physical exposures. For instance, in Britain, long-term work incapacity due to back pain grew more than eight times in the period comprised by 1950 and the early 1990s (Clinical Standards Advisory Group, 1994), coinciding with a reduction of physically demanding work given the greater use of technology and a shift from manufacturing toward service industries.

In light of the above, it is clear that other variables different from mechanical loading have a much greater effect on common disabling musculoskeletal disorders, and that they can greatly evolve over time. Recent research indicates that the drivers of the observed **tendencies** are of **psychosocial** nature and findings support the relevance of psychosocial factors as **predictors** of pain and functioning. Contemporary models of chronic pain posit a key role for psychosocial factors in the adaptation to chronic pain (Evers, Kraaimaat, Geenen, Jacobs, & Bijlsma, 2003; López-Martínez, Esteve-Zarazaga, & Ramírez-Maestre, 2008; Osborne, Jensen, Ehde, Hanley, & Kraft, 2007; Schütze, Rees, Preece, & Schütze, 2010; Tan, Nguyen, Cardin, & Jensen, 2006; Van Damme, Crombez, & Eccleston, 2008). Therefore, it is

a logical understanding that at least a part of them might be **influenced by culture and society**, as well.

Indeed, the **generalizability** of pain coping research yielding from studies with **English-speaking patients**, in particular from the USA since it represents the majority of these studies (Jensen, Keefe, Lefebvre, Romano, & Turner, 2003; McCraken, Vowles, & Gauntlett-Gilbert, 2007; J. Miró et al., 2009; Osborne et al., 2007; Romano, Jensen, & Turner, 2003; Tan et al., 2006; Tan, Teo, Anderson, & Jensen, 2011; Woby, Roach, Urmston, & Watson, 2007), to other cultures, is yet to be elucidated (Ferreira-Valente, Pais-Ribeiro, Jensen, & Almeida, 2011).

The current chapter considers the evidence in this sense and within the scope of **cultural comparison**. Hence, it involves psychological and psychosocial influences and their potential to account for the phenomena observed in the present research, focusing particularly on the psychological correlates of FM and RA.

Adult attachment in Spanish population

Chronic pain is a complex biopsychosocial condition affected by a myriad of psychosocial factors, such as beliefs about pain, fear concerning pain, self-efficacy, catastrophizing, anxiety, depression, work-related complications, lack of social support, and compensation status (Tunks, Crook, & Weir, 2008; Turk & Okifuji, 2002). Thus, to palliate the negative effects of chronic pain on quality of life, it is essential to acknowledge the psychosocial factors linked to the development and maintenance of chronic pain.

Potential risk factors for chronic pain encompass adverse experience during childhood. Indeed, research has found a positive association between childhood physical or sexual abuse and diverse chronic pain-related conditions in adults (Goodwin, Hoven, Murison, & Hotopf, 2003; Linton, 2002; Sachs-Ericsson, Cromer, Hernandez, & Kendall-Tackett, 2009). Also, it has been demonstrated that other adverse childhood experiences different from abuse can predict development of chronic pain. For instance, one longitudinal study showed that negative physical and psychological events experienced in childhood, such as being hospitalized due to a traffic accidents, receiving institutional care, and the passing of the mother, augmented the likelihood of suffering from chronic widespread pain as an adult (Jones, Power, & Macfarlane, 2009). Further, these relationships were not explained by

comorbid psychological distress or social class in adulthood. Hence, although it seems evident that exceptionally negative childhood experiences are associated to developing chronic pain later in life, the link to childhood-related factors in daily life are yet mostly unknown.

Bowlby (1977) suggested that the incipient bond between the parent and child (i.e., attachment) plays a paramount role in normal childhood development and long-term functioning. If caregivers fail to meet the child's needs, normal development is hindered, causing maladaptive personality characteristics and mental health problems such as depression, often found in chronic pain patients. Increasingly, empirical evidence proposes that adult attachment styles are linked to pain-related variables. Thus, in individuals not suffering from pain, insecure attachment has been found to be related to a lower pain threshold and higher pain distress (McWilliams & Asmundson, 2007; Meredith et al., 2006b); while in samples of chronic pain patients, those insecurely attached reported significantly higher levels of pain intensity, disability, and pain suffering, as well as lower pain self-efficacy (MacDonald & Kingsbury, 2006; L. A. McWilliams, B. Cox, & M. Enns, 2000; Meredith et al., 2006a).

In light of the findings regarding how **attachment style** is associated, amongst others, to coping with **pain** and a vulnerability to chronic pain conditions, researchers have been aiming to develop new assessment tools. Many studies have been conducted with different adult attachment evaluation instruments and have highlighted difficulties when comparing measures (Crowell, Fraley, & Shaver, 1999; P. R. Shaver, J. Belsky, & K. A. Brennan, 2000; Stein et al., 2002), reaching the conclusion that convergence among different measures does not go beyond moderate correspondence. In fact, Fraley, Waller, and Brennan (2000) analyzed the most popular adult attachment assessment tools and found two underlying independent dimensions to the evaluated categories, that is, avoidance and anxiety. This work yielded the Experience in Close Relationships (ECR-R), the original version of which (K. A. Brennan et al., 1998) has recently been validated and adapted to Spanish population by Alonso-Arbiol, Balluerca, and Shaver (2007). Another relevant instrument due to its dimensional evaluation is the Attachment Style Questionnaire (ASQ, Feeney et al., 1994). However, in a previous validation study in Spanish population, the original structure found in

an Australian sample was not confirmed and the exploratory factor analysis had interpretation problems (Lafuente, Cantero, & Melero, 1999).

Consequently, researchers Melero and Cantero concluded there was an evident need to create an **adult attachment assessment questionnaire** that reflected the particularities in the **Spanish general population** (Melero & Cantero, 2008). In order to achieve this, they used the **Adult Attachment Questionnaire** (AAQ, or "Cuestionario de Apego Adulto" in Spanish, Melero & Cantero, 2008), which they validated and created, and the Relationship Questionnaire (RQ, Bartholomew & Horowitz, 1991). The first version of the AAQ included 75 6-point Likert items, ranging from 1 (completely disagree) to 6 (completely agree). The questionnaire was completed by selecting all theoretical constructs that attachment research had previously identified as qualitatively differentiating for relationship styles. The categories were self-concept, trust in others, need for approval, dependence/autonomy/self-sufficiency, regarding relationships as unimportant, emotional expression, lack of comfort with intimacy, conflict resolution strategies, relationship dissatisfaction, success orientation vs. personal orientation, fear of relationships, and interpersonal problems. On the other hand, Bartholomew and Horowitz's RQ (1991) allows diagnosis of the individual's attachment style via four general descriptions of affective bond: secure, fearful, preoccupied, and avoidant. In the questionnaire's first part, the person is forced to choose the description most fitting to the way of handling interpersonal relationships; in the second part, questions follow a 7-point Likert scale to show the degree in which each description matches the way of relating to others, from "not at all like me" to "very much like me".

The authors obtained 40 items grouped in four factors that explained a 40% of the total variance. The first factor was termed "**Low self-esteem, need of approval and fear of rejection**", explaining the 14% of variance and encompassing items evaluating low self-esteem, fear of rejection, dependency, preoccupation for relationships, and behavioral and emotional inhibition problems. The second factor assessed anger tendency, rancor, hostility, and possessiveness, with an explained variance of 10%, and was named "**Hostile resolution of conflict, rancor and possessiveness**". The third factor, or "**Emotional expressiveness and comfortableness with intimacy**", explained a 9.4% of variance and evaluated sociability, comfort with expressing feelings, bilateral strategies of conflict resolution, and confidence in explaining problems to others. The fourth factor was named "**Emotional self-**

sufficiency and discomfort with intimacy" and explained 6.6% of variance, assessing a high need of individuality, priority of autonomy over relationship ties, overestimation of personal independence, and emotional commitment avoidance. Theoretically, the first, second, and fourth factors are linked to affective insecurity, while the third factor is associated with security. The reliability for these factors can be seen on Table 13.

Table 13. Reliability indices for the questionnaire's factors

Factor	Reliability (Cronbach's alpha)
Low self-esteem, need of approval and fear of rejection	0.86
Hostile resolution of conflict, rancor and possessiveness	0.80
Emotional expressiveness and comfortableness with intimacy	0.77
Emotional self-sufficiency and discomfort with intimacy	0.68

208 participants, or 46.74% of the sample, were classified as insecure; while 237 subjects, constituting the other 53.26%, were found to be securely attached. More specifically, 29.66% of subjects were **dismissing**, 28.54% were **secure**, 26.07% were **preoccupied**, and 15.73% were **hostile fearful**. This distribution is shown in Table 14, as well as how each scale or conglomerate is composed by a clear **dimensional profile** of different scores. The test's psychometric properties were satisfactory with regards to the reliability analysis and the construct validity, and the total variance explained was 40%.

Table 14. Types of adult attachment according to the four conglomerate analysis

	Hostile fearful	Preoccupied	Secure	Dismissing
Scale 1: Low self-esteem, need of approval and fear of rejection	Very high	High	Very low	Low/ moderate
Scale 2: Hostile resolution of conflict, rancor and possessiveness	Very high	Moderate	Low	Moderate
Scale 3: Emotional expressiveness and comfortableness with intimacy	Low	Moderate/ High	High	Low/ Moderate
Scale 4: Emotional self-sufficiency and discomfort with intimacy	Moderate/ High	Moderate	Low	High
Cases	70 (15.73%)	116 (26.07%)	127 (28.54%)	132 (29.66%)

The four obtained factors are dimensions on which subjects can be represented within a continuum, thus offering a more adequate notion of attachment than the categorical classification, as argued in previous research (Crowell et al., 1999; Fraley & Waller, 1998; P. R. Shaver et al., 2000). However, **categories** can be highly effectual in matters of research and group comparisons, for instance. When the authors performed cluster analysis to this end, they found that their results mainly coincided with respects to the secure, preoccupied, and dismissing styles, but showed certain differences in the fearful pattern. The secure individual appeared sociable, with a tendency to express feelings, and bilateral conflict resolution strategies. The preoccupied style was defined by low self-esteem, need of approval, and fear of rejection, emotional expressiveness, and comfort in relationships. The dismissing pattern is depicted by the aforementioned attributes, but not presenting with problems of self-esteem or lack of confidence. The hostile fearful style encompasses anger, hostility, rancor, and possessiveness, which could theoretically be explained by suppressed anger and non-resolved conflicts towards the attachment figures. Also, this style is depicted by low self-esteem, need of approval, fear of rejection, and high emotional self-sufficiency. Thus, it was described as an individual showing typical behavior of the preoccupied and dismissing styles.

Indeed, **hostility** may be considered as a trait in elevated anxiety attachments, given what Birnbaum, Orr, Mikulincer, and Florian (1997) posited, that these individuals assume emotion-centered conflict resolution strategies, which further exacerbates their anxious state. Spanish authors Melero and Cantero hypothesized that the appearance in their sample of a factor of hostility, rancor, and jealousy and an affective profile of this type might be due to a greater acceptance of **emotional expression** in **Hispanic** population. They noted that when attachment has been evaluated in other cultures, hostility and jealousy were not so notably present, perhaps due to greater peer and social pressure towards emotional self-control (Melero & Cantero, 2008). Furthermore, they added that the high percentage of **dismissing** attachment might be explained by the **current socialization models**, where individuality and autonomy are valued over intimate relationships, and that many secure subjects might be confounded by dismissing individuals due to a high self-sufficiency score.

Regardless of the debate surrounding the construct of adult attachment, the empirical and theoretical body is vast in the Anglo-Saxon world (e.g. S. Bennett & Nelson, 2010; Cassidy & Shaver, 2008; Clulow, 2001; Mikulincer & Shaver, 2007a; Obegi & Berant, 2009;

Pfäfflin & Adshead, 2004; Rholes & Simpson, 2004; Stosny, 1995). However, in Spanish research it is a research area yet to be developed, notwithstanding some efforts (Alonso-Arbiol et al., 2007; Alonso-Arbiol, Shaver, & Yarnoz, 2002; Gómez-Zapiain, 2009; Gómez-Zapiain, Ortiz, & Gómez-Lope, 2011; Melero & Cantero, 2008; Yárnoz, Alonso-Arbiol, Plazaola, & Sainz-de Murieta, 2001). This quantitative and cultural contrast in attachment research is not surprising, as the **universality of attachment** has been a much contested topic causing a **division of stances**: the **first** point of view alleges that attachment is an innate biological mechanism and that each particular culture conditions how this biological heritage manifests in specific behaviors (Cassidy & Shaver, 2008; Main, 1990; van IJzendoorn & Sagi, 1999). The **second** perspective postulates a critique to this universal notion with a reminder of the Western-culture values from which the attachment theory emerged, with scarce to no application to the rest of cultures (Pearson & Child, 2007; Rothbaum, Weisz, Pott, Miyake, & Morelli, 2000a, 2000b).

In spite of the meager literature on transcultural research of adult attachment, a few studies show that levels of anxiety and avoidance differ amongst countries and cultures. For instance, Schmitt et al. (2004) used the RQ in 62 countries and found that the secure and fearful items negatively correlated in 63% of cultures, while the preoccupied and dismissing items proved a negative correlation in only a 25% of cultures. An analysis of the main components showed that the two-dimensional structure of model of self and model of others underlying the four attachment patterns was not the same across cultures. More specifically, while in North America the findings were coherent with Bartholomew's framework, in other regions, such as South America, Western Europe, and the Middle East, the insecure styles clustered together into a single group and contrasted with the secure pattern. Moreover, Alonso-Arbiol et al. (2007; 2008) used the Experiences in Close Relationships with an American and a Spanish sample, finding that the Spanish anxiety mean was higher than the American one, as well as the Spanish avoidance mean was slightly lower than the American one.

Chronic pain and psychosocial factors

Increasingly, empirical evidence is suggesting that adult attachment styles are related to pain-related variables, as has been previously mentioned. For instance, in individuals

without pain, insecure attachment is related to a lower pain threshold and higher pain associated distress (McWilliams & Asmundson, 2007; Meredith et al., 2006b). Further, in samples of chronic pain patients, those insecurely attached reported significantly higher levels of pain intensity, disability, and pain-related suffering and less pain self-efficacy (MacDonald & Kingsbury, 2006; L. A. McWilliams et al., 2000; Meredith et al., 2006a). Indeed, attachment anxiety has been associated to lower somatic pain thresholds, reduced perceptions of control over pain, and a negative concept of one's pain coping ability (Rowe et al., 2012; M. Sullivan, Thorn, et al., 2001). Individuals with anxious or avoidant attachment, with negative models of self and others, have been found to report greater levels of negative pain beliefs (McWilliams & Asmundson, 2007). Also, pain catastrophizing, which is featured in difficult attachment as well, significantly correlates with pain intensity and maladaptive coping (Kratz, Davis, & Zautra, 2011; Martínez et al., 2012; Meredith et al., 2006b; Tremblay & Sullivan, 2010). These findings highlight the link between attachment style and the pain experience and a chronic pain vulnerability (Anno et al., 2015).

Research has consistently shown that, when comparing patients of different countries and cultures (Asenlöf & Söderlund, 2010; Esteve, Ramírez-Maestre, & López-Martínez, 2007; Ferreira-Valente, Pais-Ribeiro, & Jensen, 2014; Karsdorp & Vlaeyen, 2009; López-Martínez et al., 2008; Osborne et al., 2007; Rodero et al., 2011; Sardá, Nicholas, Asghari, & Pimenta, 2009; Tan, Jensen, Thornby, & Anderson, 2005; Woby et al., 2007) **psychosocial factors** proved to be significantly associated with pain intensity and interference and both physical and psychological functioning. For instance, the Cultural and Psychosocial Influences on Disability (CUPID), a large international study contrasting relationships among risk factors for different musculoskeletal pain patterns, found that in comparison to no pain, pain affecting 6 or more anatomical sites proved a stronger association with somatizing tendency than pain involving less than 4 anatomical sites (Coggon, Ntani, Palmer, Felli, Harari, Barrero, Felknor, Gimeno, Cattrell, Vargas-Prada, et al., 2013). This relationship is not restricted to awareness and reporting of musculoskeletal symptoms, but also extends to disability for daily activities deriving from musculoskeletal pain (Carugno et al., 2012; Coggon, Ntani, Palmer, Felli, Harari, Barrero, Felknor, Gimeno, Cattrell, Serra, et al., 2013). Further, this international study also found that a belief that musculoskeletal symptoms are chiefly provoked by work showed stronger and more consistent associations with pain

affecting 6 or more anatomical sites than pain in less than 4 sites (Coggon, Ntani, Palmer, Felli, Harari, Barrero, Felknor, Gimeno, Cattrell, Vargas-Prada, et al., 2013). Overall, it is a consolidated finding that low mood, somatizing tendency, and adverse health beliefs notably condition non-specific musculoskeletal pain, in particular its chronicity and related disability; moreover, differences in societal beliefs may have also intervened in major variation in the prevalence of disabling musculoskeletal pain, between countries and within countries over time (Vargas-Prada & Coggon, 2015).

From a biopsychosocial perspective, it is widely accepted that the way of understanding, expressing, and controlling **pain** is one of many learned behaviors, whose **manifestation** is **culture-specific** (Good, Brodwin, Good, & Kleinman, 1992). In other words, while the stimulation of pain fibers communicating a certain experience to the brain is common to all human beings, the perceptions and control of pain differ amongst societies (Free, 2002). As complex and multifacetic as the pain experience can be, pain perception and behaviors are influenced by the sociocultural setting of individuals suffering pain (Bates, 1987; Montes-Sandoval, 2000; Rollman, 1998; Streltzer, 1997). Furthermore, it has been noted that how and whether people communicate their pain to healthcare professionals is determined by **social and cultural variables** and differences may come from both parties: the expression of pain by patients and the interpretation of pain reporting and behaviors by the professional (Nayak, Shiflett, Eshun, & Levine, 2000). For instance, the effects of ethnicity and level of acculturation on pain perception in Hispanic, Caucasian, and African American FM patients was evaluated (Caldwell, 2001), finding no statistically significant differences in total pain perception across all three ethnic groups. However, it was stated that FM is often misdiagnosed or not diagnosed at all among Hispanics and African Americans in primary care clinics, connoting certain needs in the delivery of culturally competent care (Callister, 2003). Also, in African American and Caucasian RA female patients, differences in coping mechanisms and pain control beliefs have been identified (Jordan, Lumley, & Leisen, 1998).

In the aforementioned large international study conducted in 18 countries (the CUPID), while comparing the prevalence of disabling low back pain and disabling wrist/hand pain among groups of workers with similar physical activities in different cultural environments, the authors purposely focused in decreasing the potential for misinterpretation

and bias by defining pain as making everyday activities difficult or impossible and even using specific diagrams illustrating the anatomical sites of interest. Yet, in some cases, large differences in prevalence were found even among occupational groups from the same country questioned in the same language, or from different countries sharing the same language (Coggon, Ntani, Palmer, Felli, Harari, Barrero, Felknor, Gimeno, Cattrell, Serra, et al., 2013). It has been posited that differences in societal beliefs might begin to explain notable contrasts in the prevalence of musculoskeletal complaints and associated disability reported among workers with similar jobs but dissimilar cultural circumstances (Coggon, Ntani, Palmer, Felli, Harari, Barrero, Felknor, Gimeno, Cattrell, Serra, et al., 2013; Madan, Reading, Palmer, & Coggon, 2008).

Rationale

Attachment theory (Bowlby, 1982) postulates that early experiences with primary caregivers generate models of self and others that persevere in adulthood, guiding behavior and expectations in further relationships, such as with peers and romantic partners. The clinical implications' relevance of these phenomena, particularly the influence of attachment insecurity on medical situations, validates the interest in garnering and producing literature on the subject, such as the vulnerability for emotional distress and psychiatric disorders (Mikulincer & Shaver, 2012). Notably because it is known that insecurely attached individuals have a hindered emotional regulation, which could yield more frequent or intense physical symptoms as a reaction to stressors (Maunder & Hunter, 2001). It has also been posited that people with insecure attachment report physical symptoms because they learned via reinforcement patterns with early carers that explicit communication of emotional distress would likely not merit as much attention and support as expression of physical distress (Stuart & Noyes, 1999).

Consequently, positive relationships between **attachment insecurity** and physical symptom reporting have been found in university students (Feeney & Ryan, 1994; Kidd & Sheffield, 2005; Wearden, Lamberton, Crook, & Walsh, 2005), general population (Liu, Cohen, Schulz, & Waldinger, 2011; Waldinger, Schulz, Barsky, & Ahern, 2006), female primary care health maintenance organization patients (Ciechanowski, Walker, et al., 2002), and somatoform disorder outpatients (Neumann, Sattel, Gündel, Henningsen, & Kruse, 2015). Additionally, attachment insecurity has been positively associated with chronic widespread pain (Davies et al., 2009) and pain ailments, such as headaches (McWilliams & Bailey, 2010). Mention must be made as well to the finding that people suffering from medically unexplained pain are more prone to be more negatively assessed by others than patients with a pain that can be medically explained, or a pain with a known cause (De Ruddere, Bosmans, Crombez, & Goubert, 2016). This is key to the current study, as the term medically unexplained pain/physical symptoms is often used as a conceptual umbrella for syndromes with a contested etiology, such as FM (Richardson & Engel, 2004).

Indeed, **chronic pain** already demands attention and intrudes into every domain of a person's life when it is diagnosed and medically explained, as is the case of **RA**. As it has been noted, it is a rheumatic condition of a mainly organic, autoimmune etiology, in which patients show a chronic, deteriorating, and painful course. Thus, RA constitutes a useful

comparison group and with regards to FM, by highlighting the emotional and psychological aspects of a pain medical condition similar to **FM** (Rodríguez de la Serna et al., 2004; E. Walker et al., 1997b). In fact, there are **precedents in literature** of FM vs. RA assessments, albeit not always exclusive, due to the joined regard of other chronic pain conditions (Borg et al., 2014; Capraro et al., 2012; Kilic et al., 2013; McInnis, Matheson, & Anisman, 2014; Parrish, Zautra, & Davis, 2008; Saperia & Swartzman, 2012; Schleicher et al., 2005). However, the comparison in these examples serves the stressing of psychological features in FM as secondary. That is, as the controversial 1990 ACR diagnostic criteria are used, the focus lies on the characteristic pain expression and experience, insofar neglecting other cognitive and emotional symptoms. However, it is obvious to any healthcare professional that the change from normal to sick is a matter of degree or quantity, which involves both primary (biologic) and secondary (psychosocial and cultural) variables acting as risk factors (Crofford, 2015a). Hence, studying adult attachment styles as well as various psychosocial variables in these two groups would ideally accomplish a deeper and more mindful view of FM in its complexity as a broad syndrome, of psychosomatic nuances. Complementarily, it would also shed light into some of the psychological correlates of a widely accepted organic condition, such as RA, in the same manner as and parallel to FM.

Viewing chronic pain from a **broader perspective** is paramount in current times, in which the American Psychiatric Association in the DSM-5 has replaced the previous categories of somatoform disorder, hypochondriasis, pain disorder, and undifferentiated somatoform disorder with the SSD ("SSD with predominantly somatic complaints" and SSD with pain features" (American Psychiatric Association, 2013). Notably, the previous requirement of medically unexplained symptoms is removed and psychological symptoms associated to the somatic symptoms have been added. This ultimately leads to the inclusion of the inherent **subjectivity** of the symptoms and the placement of the clinician into a complex judging situation that encompasses far more than a biologic or physiological explanation. Consequently, **musculoskeletal pain research** nowadays generally advocates for transcending and superseding monist arguments, tending to view pain as a **fundamentally unitary phenomenon**, albeit with different causes (Merskey, 1984).

Whereas adult attachment has been profusely studied in its relation to psychopathology, much less has been researched on the link between **adult attachment and**

physical illness. In order to successfully guide this budding and etiological research, there is an evident need for a rationale that distinguishes individuals who are highly susceptible to stress, both physiologically and behaviorally, from those with a more appropriate response to stress. Using the attachment paradigm serves this purpose, as it can be used in the manner of a **biopsychosocial model** of disease. Further, in light of all the considerations regarding the distinctive traits in the fearful group of insecure attachment within **Spanish population**, research following cross-cultural guidelines could potentially lead to new data on links with health, and in particular, FM and its high influence of psychosocial factors (McBeth, Macfarlane, Benjamin, & Silman, 2001; Meredith et al., 2008). Conducting a study with the proper sample size, with a comparison group, and within a prospective study framework could pave the road for improving efforts in the understanding of the disease and, hence, the therapeutic field of FM, an illness with yet many unknowns.

Material & Methods

This chapter develops the methodological aspects in the study, with a first section on study design that focuses on the selection criteria that were used and how the sample was recruited, as well as a brief description of the final sample obtained. Next, the procedures for contacting patients are presented, followed by the measures used and, finally, the data analysis plan purported to answer the aims of this dissertation.

Study design and participants

Selection criteria and sample recruitment

The target population in this study were patients with FM and RA according to ACR 2010 diagnostic criteria, all of which were healthcare users from New York and Barcelona, where the research was conducted. In New York, the same inclusion criteria were used to obtain the sample, following the corresponding diagnostic codes in the electronic medical records.

Thus, the sampling frame in this study consisted of a hospital in Barcelona, and a hospital and a rheumatology private office in New York. Study participants were 168 patients that were currently being treated by their rheumatologists. The study followed a survey method and consisted of a cross-sectional design with two groups: one with FM patients and a RA comparison group. All subjects filled out different psychological questionnaires, as well as complied a series of demographic and clinical history forms. Figures 21 and 22 show both hospital samples.

Figure 21. Barcelona hospital sample.

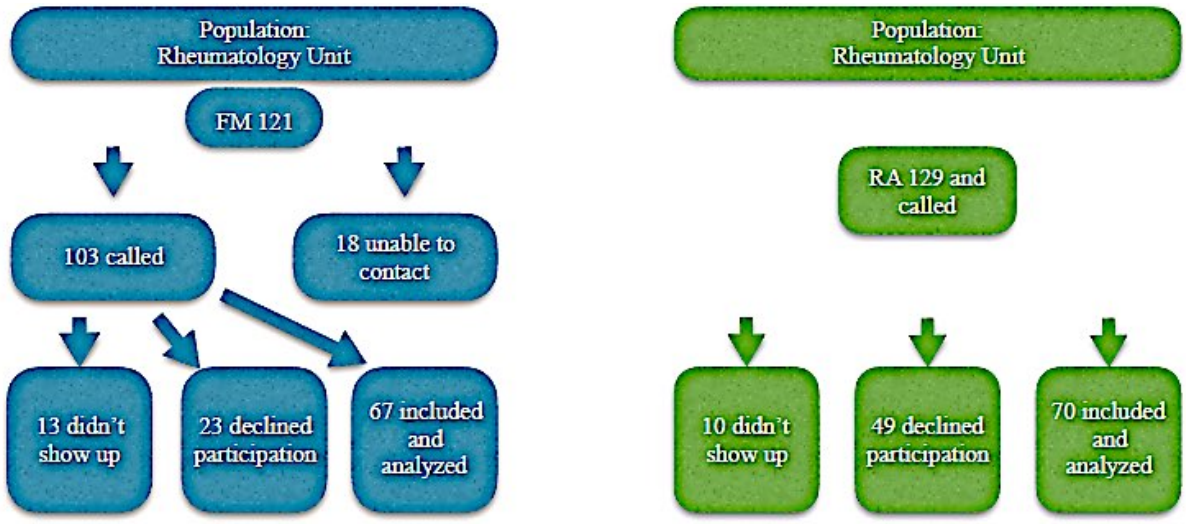
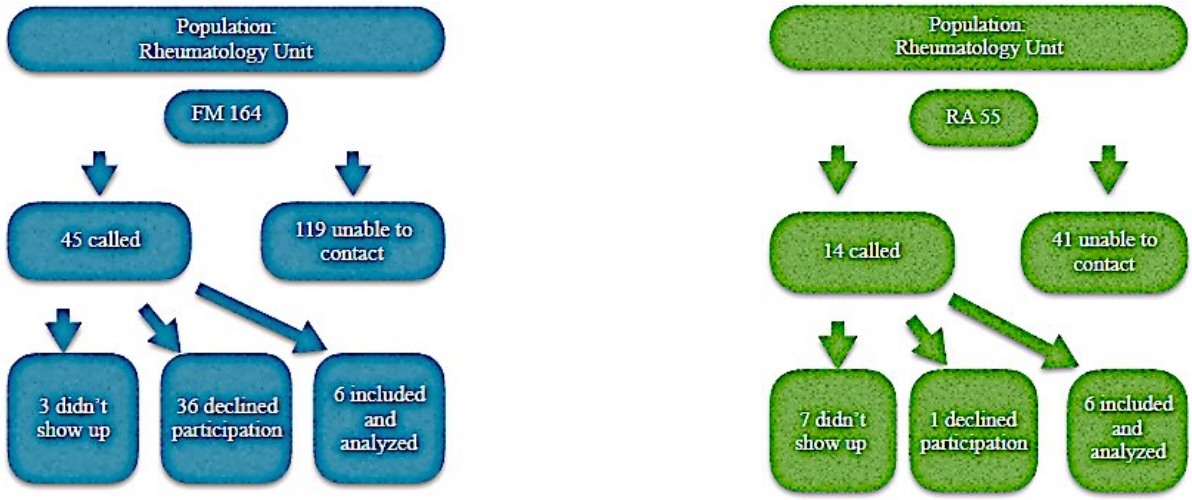


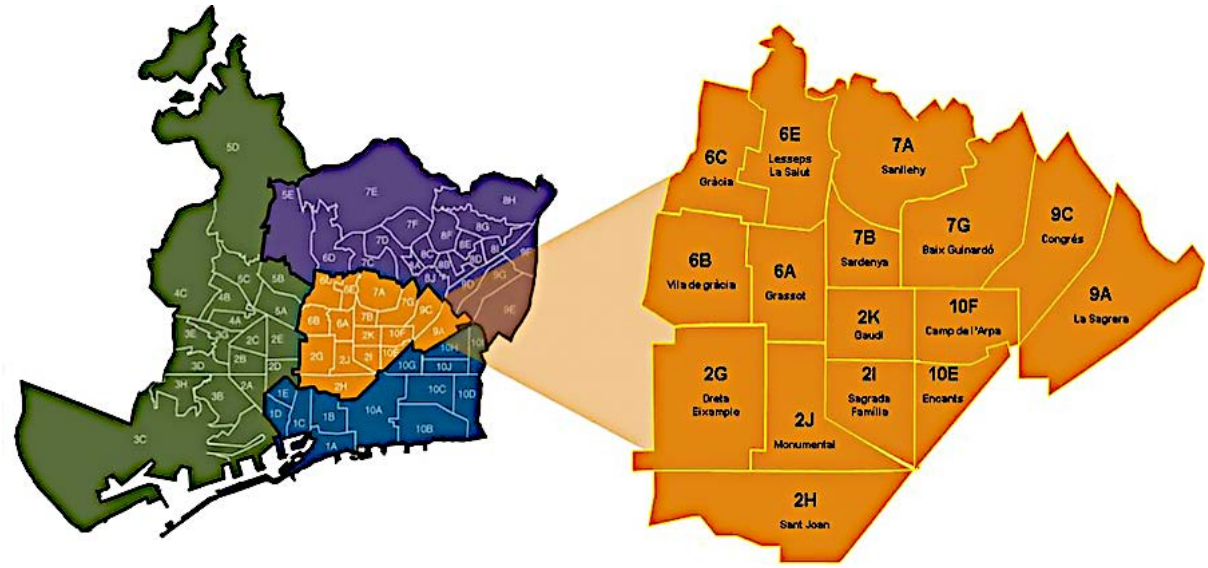
Figure 22. New York hospital sample.



Barcelona

Patients recruited in the first stage of the study were from the *Hospital de la Santa Creu i Sant Pau*, from the Rheumatology Unit.

Figure 23. *Àrea d'Atenció Integral de Salut (AIS) Barcelona Dreta*. The highlighted zone in orange from the city map is the area corresponding to the *Hospital de la Santa Creu i Sant Pau.*



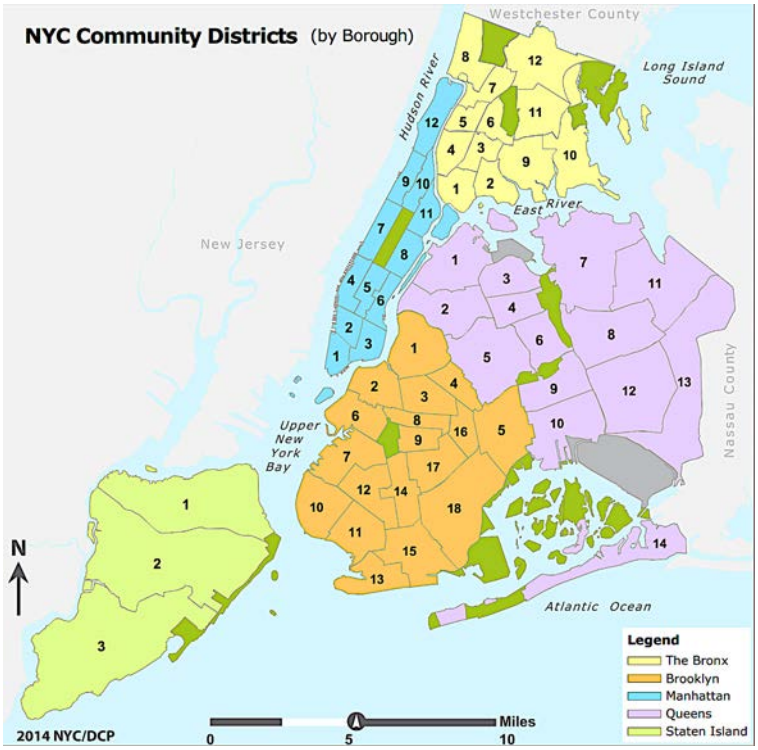
The inclusion criteria were the aforementioned ACR 2010 diagnostic criteria and to be over 18 years of age, as in New York, as well as being Spanish and/or having ties to a

Spanish-speaking country. The unit's rheumatologists provided the RA patient list, and the FM patients were included by the unit's rheumatologists and clinical psychologist.

New York

Patients were mainly recruited at Wyckoff Heights Medical Center, where the rheumatologist in charge tutored the study during this phase and also provided patients from his private office due to shortage deriving from inclusion criteria. Specifically, patients in this stage of the study could not be included if there were indirect or direct ties to a Spanish-speaking country, thus challenging patient selection considerably in a hospital service area (Brooklyn Community District 4) where 65.4% of the community is Hispanic (NYC Census 2010- NYC Community Data Portal). The rheumatologist's private office was also in Brooklyn (Community District 18), with a more varied population.

Figure 24. NYC Community Districts.



Exclusion criteria in both countries were to have the other rheumatic condition (i.e., having both FM and RA) and comorbidity with disabling and severe illnesses, as well as the subject's ethnicity so as to not interfere with the cross-cultural report between samples. That

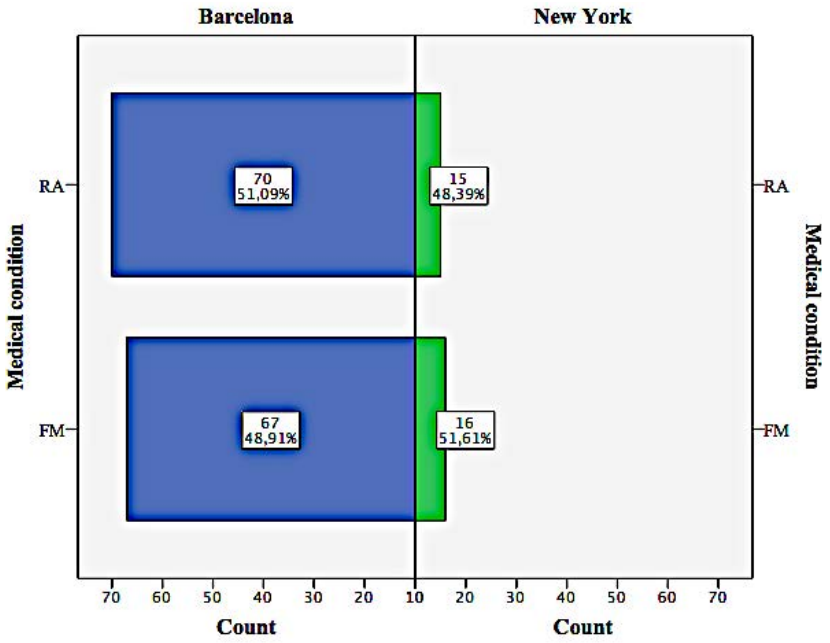
is, for the scientific comparison purposes of this study, in which Spanish particularities of adult attachment were being observed, patients in New York were included on the principle that they didn't have direct family and/or family relations with a Hispanic culture or a country where Spanish is the first language. Therefore, non-probability, purposive sampling was also performed in this second stage of the study, as patients diagnosed and screened for nationality were contacted and asked if they would like to volunteer.

The initial aim was to include a minimum of 70 patients per group and 31 in the American FM and RA groups, but logistical and time-sensible issues hindered the recruitment in New York. Criteria used to establish sample size was based on statistical premises, that is, in the type of tests required to contrast the corresponding hypotheses. Namely, and following the multivariate approach, this sample calculation was performed taking into account Freeman's recommendation for linear regression (Ortega Calvo & Cayuela Domínguez, 2002), which suggests that the number of subjects should be superior to 10 * (k+1), where k expresses the number of covariables. In this instance, the sample size had to be 10 times the number of parameters to be estimated plus one, and the questionnaires used for the present study were considered parameters. Also, a sample size and power calculator (Institut Municipal d'Investigació Mèdica (IMIM), 2012) was used to ponder, from scientific literature (Pincus et al., 2010), that accepting an alpha risk of 0.05 and a beta risk of 0.2 in a two-sided test, 31 subjects were necessary in both groups to recognize as statistically significant a difference greater than or equal to 5 units, assuming the common standard deviation was 7 and anticipating a drop-out rate of 0%. This second calculation was undertaken to accommodate the necessary parametric tests aiming at comparing two independent samples.

Description of the study sample

From the overall 168 study participants, in Barcelona, 70 were RA patients and 67 were FM patients. In New York, the RA sample was constituted by 15 subjects, and the FM sample by 16. As previously mentioned, while the Barcelona samples were entirely recruited at the same hospital Rheumatology Unit, the New York samples were patients being treated by the same rheumatologist, but from the Rheumatology Unit in a hospital and in a private office.

Figure 25. Sample distribution.



Procedures

Patients were initially recruited via telephone, when they were briefly informed and engaged in participation. At this first verbal contact, an explanation was given about the study doctors involved, the main objective of the research, that the questionnaire-based interview would have a maximum duration of one hour, and that there was no intervention or medication involved, as well as no follow-up. Then, at their appointment and prior to the interview, they submitted an informed signed consent, as well as clinical history information and demographic data, regarding number of years since diagnosis, somatic comorbidities, current medication, and other characteristics (age, gender, marital and work status, nationality, and education level). Patients would then comply all the corresponding questionnaires and were thus able to solve any questions or doubts that might have otherwise interfered.

Both the Barcelona hospital's ethics committee and the Institutional Review Board of the hospital in New York approved all procedures and participants gave written informed consent, in accordance to the Declaration of Helsinki.

Barcelona

After obtaining protocol approval by the hospital's ethics committee, recruitment and interviews were carried out from October, 2014 through May, 2015. The study initiated with the diagnosis of ACR2010 FM, firstly executed by the rheumatologist and then further confirmed by the unit's clinical psychologist. In the case of RA patients, as the ACR2010 criteria comprise serologic categories, the diagnosis and patient list was produced by the rheumatologist in the unit. This produced a list of 121 eligible patients; telephone contact was established with 103 of them, 67 agreed to participate and fit the inclusion criteria, and another 13 agreed to come but never showed up. As for the RA patients, 129 were called, and 70 were finally recruited and attended the interview, while 10 agreed to participate but never showed up.

New York

The ethics committee of the hospital approved the protocol prior to study initiation. Recruitment and interviews started on September, 2015, and were carried out until November, 2015. The clinical history information was confirmed with the electronic and medical records, as well as the rheumatologist, and two medical residents aided screening for inclusion criteria and designed a schedule for patient interviews at the hospital. In the case of one patient, the questionnaires were submitted via telephone interview, after all proper signed consents were filled out in person. In another patient's case, due to limited mobility, the interview was carried out during a house call.

From the 15 RA participants, 6 patients were recruited at the hospital. As for the 16 FM patients, 5 were selected at the hospital. The hospital's electronic records listed 55 RA patients (since 1/1/2010) meeting inclusion criteria, 14 of which were contacted, 7 of which agreed to come and never showed up; and 164 eligible FM patients (since 1/1/2013), 45 of which were called, 3 of which failed to show up.

Measures

The instruments and evaluation measures used in the study will be described here and are included in the Appendix section, excepting the copyrighted material. In addition to characterizing the questionnaires, the reliability estimate will be presented through Cronbach's α internal consistency coefficient obtained in this sample, according to the American Educational Research Association (AERA), the American Psychological Association (APA), and the National Council on Measurement in Education (NCME) standards (AERA, APA, & NCME, 2002).

Demographic and clinical information

Subjects were asked to report their age at the moment of the study, nationality, gender, marital and work status, and education level computed in years since primary school. They also confirmed medical information, regarding number of years since diagnosis, somatic comorbidities, and current medication intake. An observation was made when patients were on special diets or taking homeopathic medicine.

Somatic comorbidities were accounted for with the Rheumatic Disease Comorbidity Index (Michaud & Wolfe, 2007), which ranges from 0-9. The formula for the RDCI is as follows: 2 x lung disease + [2 x (heart attack, other cardiovascular, OR stroke) OR 1 x hypertension] + fracture + depression + diabetes + cancer + (ulcer or stomach problem). The RDCI has been demonstrated to be a robust predictor of mortality in the context of rheumatic diseases, and performs well with self-report data (England, Sayles, Mikuls, Johnson, & Michaud, 2015).

Adult attachment

The adult attachment style in Barcelona was assessed via the Adult Attachment Questionnaire ("Cuestionario de Apego Adulto", or CAA) developed by Melero and Cantero for the Spanish population (Melero & Cantero, 2008). It consists of 40 six-point Likert questions that can be grouped into four scales. These four factors, or scales, represent dimensions where the subject can be located in a continuum, offering a more

adequate perspective of attachment than the categorical classification (Bartholomew, 1997; Crowell et al., 1999; Fraley & Waller, 1998; P. Shaver, J. Belsky, & K. A. Brennan, 2000). The first scale provides information about "Low self-esteem, need of approval and fear of rejection" and consists of 13 items, the second assesses "Hostile resolution of conflict, rancor and possessiveness" and groups 11 items, the third evaluates "Emotional expressiveness and comfortableness with intimacy" and is composed of 9 items, and lastly there is the scale "Emotional self-sufficiency and discomfort with intimacy" with 7 items. Grouping these factors into categories, however, yielded attachment patterns that coincided with the secure, preoccupied and dismissing styles, but presented specific features regarding the fearful pattern. Hence, the hostile fearful style presents itself with very high scores in the first and second scales, low in the third and moderate/high in the fourth. The preoccupied style defines the subject when obtaining a high score in scale 1, a moderate score in scale 2, a moderate/high score in scale 3 and a moderate one in scale 4. The scores that characterize the secure style are very low for scale 1, low for scale 2, high for scale 3, and low for scale 4. Finally, the dismissing type has low/moderate scores in the first scale, moderate scores in the second scale, low/moderate scores in the third scale and high scores in the fourth. Participants were placed into one of the four categorical profiles based on the best fit of the scores of these four dimensional scales. Reliability for this questionnaire was assessed using Cronbach's alpha coefficient, which in this sample yielded $\propto = .82$ for all 40 items.

To explore adult attachment patterns in the non-Hispanic population, two questionnaires were used: the Relationship Questionnaire (RQ; Bartholomew, Horowitz, 1991) and the Relationship Scales Questionnaire (RSQ; Griffin, Bartholomew, 1994). Bartholomew and Horowitz (Bartholomew & Horowitz, 1991) developed the RQ according to their four-category two-dimensional model. It is a forced-choice instrument in which the four patterns of attachment are described in brief paragraphs, and respondents rate the degree to which they resemble each prototype style on a 7-point Likert scale. Whereas the primary aim of the RQ is to yield continuous ratings of each of the four attachment styles, the 30-item, 5-point Likert scale RSQ also measures dimensions related to positive or negative models of self and others, and it was

developed by Griffin and Bartholomew (Griffin & Bartholomew, 1994). In both, the individual rates themselves first in the secure scale (5 items; ∝ = .33), second in the fearful scale (4 items; ∝ = .73), third the preoccupied (4 items; ∝ = .37), and fourth in the dismissing scale (5 items; ∝ = .61), providing a profile of the attachment feelings and behavior. This combination of instruments was chosen due to the fact that the CAA derived from the RQ, as well as the recognized benefits of using dimensional self-report attachment instruments in psychosomatic research (Ravitz et al., 2010), and ultimately to increase the internal consistency of attachment style scores. Both tests were administered and then combined to obtain a composite measure of adult attachment, by standardizing and then averaging the scores on each. An example of this procedure can be seen in a publication by Ognibene and Collins (Ognibene & Collins, 1998).

Depression

The Beck Depression Inventory II (BDI-II; Beck et al., 1961) and its Spanish version (Sanz et al., 2013) were used, consisting of a 21-item self-report evaluating cognitive, affective, and behavioral components of depressive symptoms (∝ = .90). It is a 1996 revision of the BDI, developed in response to the American Psychiatric Association's publication of the DSM IV, which changed many of the diagnostic criteria for Major Depressive Disorder. Each answer is scored on a scale value of 0 to 3. Subjects were also classified into groups of minimal, mild, moderate or severe depression according to the score thresholds (0 to 13, 14 to 18, 19 to 27, and 28 to 63, respectively) for the clinical Spanish population (Sanz et al., 2013). Thresholds slightly differed in the original version in reference to the American sample, having the minimal, mild, moderate and severe groups correspond to ranges 14 to 19, 20 to 28, and 29 to 63.

Quality of life

FM life quality and condition severity will be evaluated using the Fibromyalgia Impact Questionnaire (FIQ; Burckhardt et al., 1991) and its Spanish version, the Spanish Fibromyalgia Impact Questionnaire (SFIQ; Rivera, González, 2004) . It is composed of 10 subscales: physical impairment, overall wellbeing, work missed, job difficulty, pain, fatigue,

morning tiredness, stiffness, anxiety, and depression. The score of each subscale was standardized from 0 to 10, and adding the scores of all the items produced a total score ranging from 0 (no severity) to 100 (maximum severity). The usual classification was taken into account, wherein patients are categorized into three groups based on the total score as ≥50 and ≥70, and therefore using the thresholds corresponding to the mild, moderate or severe FM classifications, respectively (Burckhardt, 2003; Monterde & et al., 2004). The SFIQ consists of 19 items ($\propto$ = .79), and the FIQ includes 20 items ($\propto$ = .83).

The Short Form 36 (SF-36; Ware, Sherbourne, 1992) was implemented in the case of RA patients. The Spanish version was also used (Alonso et al., 1995). It is a short quality of life questionnaire with 36 items ($\propto$ = .34), measuring eight multi-item variables: physical functioning, social functioning, role limitations due to emotional problems, mental health, energy and vitality, pain, and general perception of health. Each scale generates a score from 0 to 100, with a high score indicating better health. However, the questionnaire may be converted to two summary scales, that is, the physical and mental components (PCS and MCS).

Pain intensity and its daily life interference

The Brief Pain Inventory was used in its original (Cleeland, 2009) and Spanish versions (Badia Llach et al., 2003). It consists of a 15-item, self-evaluated questionnaire designed to explore pain location and qualitative aspects of pain, as well as measure pain in two dimensions: pain intensity and interference with daily activities. Pain intensity, provided by the Pain Severity Score (PSS, or the mean of 4 pain intensity items), has a maximum value of 10 and a minimum value of 0. The PSS was found to be highly reliable ($\propto$ = .94). The Pain Interference Scale is obtained through the mean of 7 interference items ($\propto$ = .94) that produce a Pain Interference Score, also ranging from 0 to 10 (Cleeland, 2009).

Functional status, disease activity, and health-related quality of life

The Multidimensional Health Assessment Questionnaire (Pincus et al., 1999), and its Spanish version, were used to collect quantitative information on patient status. It includes

scales for Rheumatoid Arthritis Disease Activity Index (RADAI), self-report joint count, psychological distress, a review of systems, change in status, exercise status, morning stiffness, fatigue, recent medical history, and a template to score RAPID3 (Routine Assessment of Patient Index Data 3). RAPID3 comprises the three patient reported outcome measures in the ACR Core Data Set (physical function, pain, and global estimate of status), scoring on a 0-30 scale and being highly significantly correlated with DAS28 and CDAI (Pincus et al., 2010), making all of them disease-specific indices for RA (Tugwell & Boers, 1993; van Riel, 1992). Additionally, RAPID3 is useful in chronic rheumatic diseases, submitting "vital" information (Pincus, 2008). Indeed, the MDHAQ might function as a "generic" questionnaire, as all rheumatic diseases include limitations of functional status, pain, and poor global status (Fries & Ramey, 1997). The Health Assessment Questionnaire (HAQ), from which the MDHAQ derived, has been widely used to score disability and health-related quality of life in rheumatic diseases (Birrell et al., 2000; Kvien et al., 1998). RAPID3 could be used in all rheumatic diseases as well (and/or other conditions causing pain and functional impairment), allowing comparative studies of its values in different settings (Pincus & Sokka, 2007). RAPID3 has been mainly studied in RA, to complete physician's assessment of disease activity/severity (Berthelot, 2014). Cutoff points have been defined, indicating low (<6/30) or high levels (>12/30) of RA activity; more specifically, severity categories have been set for RA in ≤3 for remission, 3.1-6.0 for low, 6.1-12.0 for moderate, and >12 for high severity (Pincus et al., 2008; Pincus, Yazici, Bergman, Swearingen, & Harrington, 2006). As for the symptom checklist review of systems (ROS), obtaining more than 20 positives on the list suggests FM, while more than 30 is virtually pathognomonic for FM (Pincus et al., 2007). In FM, ratios of pain or fatigue to physical function scores, as well as the number of symptoms reported on the ROS, distinguish these patients from those with RA as effectively as ESR (Callahan & Pincus, 1990; DeWalt et al., 2004).

As per reliability, RAPID3 was analyzed first for its physical function items (10; $\propto$ = .89), and also for the entire three patient reported outcome measures (12 items; $\propto$ = .85). Since all 60 items composing the ROS are binary, split-half reliability was performed and therefore the Spearman-Brown coefficient was used, obtaining high reliability (r = .91).

Depression and anxiety

To measure current anxiety and depressive symptomatology in non-psychiatric hospital patients, the Hospital Anxiety and Depression Scale (HADS; Zigmund, Snaith, 1983) and its Spanish version (HAD; Quintana et al., 2003) were used. It was originally developed by Zsigmond and Snaith (1983) as a 14-item self-report scale that excludes somatic symptoms, therefore avoiding their potential confounding in a medical practice setting (Snaith & Zigmond, 1994). Thus, it sheds light into anxiety and depression, in a screening manner, in people with physical health problems. It is composed by two subscales of seven items each corresponding to anxiety and depression and is rated on a 4-point Likert scale. Snaith and Zigmund (Snaith & Zigmond, 1994) proposed the following cut-off points: both for depression and anxiety, scores of 8 or higher show *possible* levels of clinical relevance, and *probable* levels of clinically relevant anxiety were set at 11 or higher.

In this study, the anxiety subscale (7 items; $\propto$ = .87) and the depression subscale (7 items; $\propto$ = .84) were both highly reliable, as well as all 14 items of the questionnaire ($\propto$ = .91).

Figure 6. Questionnaires used in the study

BARCELONA		NEW YORK	
FM	RA	FM	RA
CAA	CAA	RQ/RSQ	RQ/RSQ
BDI-II	BDI-II	BDI-II	BDI-II
SFIQ	SF-36	FIQ	SF-36
BPI	BPI	BPI	BPI
MDHAQ	MDHAQ	MDHAQ	MDHAQ
HAD	HAD	HADS	HADS

CAA: Cuestionario de Apego Adulto. RQ: Relationship Questionnaire. RSQ: Relationship Scales Questionnaire. BDI-II:Beck Depression Inventory II. SFIQ: Spanish Fibromyalgia Impact Questionnaire. SF-36: Short Form 36. FIQ: Fibromyalgia Impact Questionnaire. BPI: Brief Pain Inventory. MDHAQ: Multidimensional Health Assessment Questionnaire. HAD(S): Hospital Anxiety and Depression (Scale).

Data analysis plan

This section offers a description of how data has been treated according to the objectives of the study; that is, analysis and imputation of missing data and treatment of outliers, the different statistical tests that were run, and if the appropriate assumptions were met. Furthermore, the statistical techniques that were implemented will be presented in order by univariate, bivariate, and multivariate, all of which allowed the research questions to be answered.

Data examination and statistical decisions

The key variables of the study consist of patient-reported outcomes through the questionnaires. There were no missing data, as all questionnaires were complied within a single session of approximately an hour under supervision by one of the researchers. Therefore, and prior to analysis, the data were examined using frequencies and descriptive statistics.

Overall, variables were classified into categorical and continuous. The categorical ones included gender, nationality (Spanish/ American), education level (primary/ secondary/ university education), medical condition (FM /RA), marital status (divorced/ domestic partnership/ married/ separated/ single/ widow(er)), work status (employed/ permanent or Social Security disability/ sick leave/ unemployed/ retired), attachment style (secure/ insecure), Spanish and American specific attachment styles (from questionnaire scales, as mentioned above), RDCI for comorbidities (0-1, 1-2, ≥2), and Spanish and American BDI-measured depression levels (established by the aforementioned differentiated thresholds). The continuous variables were patient age, education measured in years, years since diagnosis, BDI scores, SFIQ scores, SF-36's MCS and PCS scores, BPI scores in both scales (PSS and PIS), MDHAQ ROS and RAPID3 scores and its three health-related subscales of physical function, pain, and patient global estimate of status, HADS' anxiety and depression scale scores, and all adult attachment questionnaires' standardized scores.

In the instance of univariate analyses, kurtosis and asymmetry were observed in quantitative variables to check for the assumption of normality. For the bivariate techniques,

normality was confirmed through the Kolmogorov-Smirnov test, as well as homoscedasticity was verified through Levene's test. The group size was also considered, checking the frequency of *n* larger than 30 in order to choose parametric or non-parametric tests. In the multivariate analyses, normality was checked through a normal probability plot of standardized residuals, homoscedasticity was checked by visual examination of a plot of the standardized residuals by the regression standardized predicted value, linearity was tested through a scatterplot of standardized residuals for pairs of variables, independence was checked with the Durbin-Watson test, and absence of multicollinearity was tested by measuring tolerance and the variance inflation factor.

Additionally, in order to quantify differences, the effect size was measured with Cramer's V test in the case of chi-square tests, and Cohen's d for one-way ANOVAs.

All analyses were conducted using version 22 of the SPSS statistical software (IBM Corp., 2013). Unless otherwise indicated, the level of significance was set at $\alpha = 0.05$, two-tailed.

Univariate tests

Clinical and demographic variables of both samples were described through the corresponding measures of central tendency and dispersion. For some of the variables, visual aid was provided through graphs.

Bivariate tests

Pearson correlation coefficients were used to assess the relationship amongst the main study variables. In categorical variables, contingency tables were used and independence was analyzed with chi-square tests. To detect statistical differences between group means, one-way ANOVAs and parametric tests, such as Student's T-tests, were used. Cases that didn't meet the conditions for parametric tests were analyzed with Mann-Whitney U tests and Kruskal-Wallis H tests.

Thus, a one-way ANOVA was carried out to determine whether quality of life differed based on being insecurely or securely attached, nationality, and gender. An independent samples T-test was conducted to detect statistically significant differences between both medical conditions with regards to education level, age, comorbidities, BDI-evaluated

depression, HADS anxiety and depression levels, pain behavior, functional status, and symptom checklist. Contingency tables were used by comparing the categorical variables of gender, having secure or insecure attachment, nationality group, and medical condition with chi-square tests for independence. Preliminary analyses were executed to ensure that the assumptions for multiple linear regression were met.

Moreover, since the American sample size didn't allow for a thorough cross-cultural comparison, interest was taken in checking for any compelling differences between groups before conducting linear regression models. Consequently, several analyses were completed in order to explore any possible specific differences between medical conditions as expressed by patients in both countries. A one-way ANOVA was used to determine significant differences in education level, age, comorbidities and quality of life amongst the four groups contained in both medical conditions from both countries. This was repeated taking into account BDI-measured depression, HADS depression and anxiety levels, pain-related outcomes, functional status, and symptom checklist, and then conducting a Scheffe *post-hoc* test to highlight which specific groups differed. A chi-square test for independence was repeated under the same conditions as the previous contingency tables, but with the different nationalities.

Following the multivariate tests, in order to identify any possible significant differences amongst the specific attachment patterns in BDI depression level, HADS anxiety and depression, pain-related variables, functional status and symptom checklist, a one-way ANOVA and a non-parametric test were performed. Also, an independent samples T-test and Pearson correlations were used to explore the statistical significance of each attachment questionnaire scale in relation to other main variables of the study.

Multivariate tests

Multiple regression analyses were performed in order to identify potential predictors of various dependent variables, which were scores corresponding to MCS and PCS, PSS and PIS, and RAPID3 and ROS. Only the Spanish sample results were taken into account, as the American samples did not offer enough observations per covariate estimated. Prior to including the independent variables in the multiple regression model for analysis, scores were converted into z-scores in order to put data from different sources onto the same scale. Then,

predictors were entered using a simultaneous method, through which each predictor is assessed as though it were entered after all others and by what it uniquely offers, different to other entered variables, to the prediction of the dependent variables. This entry method was chosen due to the fact that there was a small set of predictors (attachment style, anxiety, and depression) and it was unclear which independent variables would create the best prediction equation (J. Cohen, Cohen, West, & Aiken, 2003). In order to establish the overall fit, or variance explained of the model, as well as the relative contribution of each of the independent variables to the total variance explained (Hair, Anderson, Tatham, & Black, 1998), the coefficient of determination (R^2) was firstly taken into account. This coefficient provides information on the predictive precision or proportion of variance in the dependent variable that can be explained by the independent variables.

With regards to the independent variables, the signification of the standardized coefficients was analyzed to determine which predictors are relevantly contributing to the explanation of the criterion variable. Additionally, the β coefficients show the effect of each independent variable on the dependent variable, or how much the dependent variable is altered when each independent variable is altered one unit while all other variables remain constant. Therefore, they were also taken into account in order to compare the strength of the effect of each individual independent variable to the dependent variable.

Aims & Hypotheses

This section presents the aims and hypotheses guiding the present research within its theoretical framework. The general and specific aims will be stated first and the corresponding hypotheses will follow.

Aims

The study goals can be classified into 5 general aims encompassing 14 specific ones (Table 15). The first general aim focuses on the psychological and sociodemographic features of FM and RA patients, specifically on the description of their sociodemographic characteristics. Also, their pain, depression, and anxiety levels are explored, as well as their quality of life and functional status. To this end, validated questionnaires are used.

Table 15. Dissertation general and specific aims

General	Specific
I. To determine the psychological and sociodemographic characteristics of FM and RA patients.	1. To explore the sociodemographic traits. 2. To describe the clinical realities of pain, depression, and anxiety. 3. To analyze the psychosocial dimensions of quality of life and functional status.
II. To compare the psychological profiles of FM and RA.	4. To contrast pain, depression, and anxiety as clinical variables in FM and RA. 5. To ascertain possible similarities and differences between FM and RA regarding quality of life and functional status.
III. To establish adult attachment in FM and RA patients according to the secure, fearful, preoccupied, and dismissing styles.	6. To find and depict the particularities regarding attachment styles in Spanish and American population. 7. To determine similarities and differences among attachment styles in FM and RA.
IV. To analyze and contrast the cultural factor jointly in both medical conditions.	8. To explore any disparities in relation to depression and anxiety between the samples from both countries. 9. To distinguish similarities and differences between sample origins regarding psychosocial dimensions and pain interference with daily activities.
V. To explore how clinical and attachment characteristics are related and influence the patients' psychosocial dimensions.	10. To describe relationships among the analyzed dimensions in both profiles. 11. To study what variables the medical condition's functional status depends on. 12. To analyze what variables quality of life depends on. 13. To verify if depression influences the perception of quality of life and functional status. 14. To detect if attachment subtypes have a potential effect on the illness.

The second general aim consists in comparing both profiles corresponding to these medical conditions. This involved contrasting contrastingly exploring pain, depression, and anxiety; and finding possible similarities and differences in FM and RA with relation to quality of life and functional status. Mainly, statistical analyses are performed in order to obtain this information.

This dissertation's third general aim is centered on the classification into one of the four prototypical adult attachment styles within the FM and RA samples. Further, both medical conditions are contrasted in their similarities and differences regarding attachment patterns, as well as both countries are compared to find and describe possible specificities in attachment styles.

The fourth general aim focuses in the analysis and cross-cultural comparison of both illnesses. In particular, differences concerning anxiety and depression in the Barcelona and New York samples are explored.

This study's fifth and last general aim addresses the examination of potential effects and relationships amongst clinical and attachment traits and with regards to the patients' psychosocial dimensions. Namely, all analyzed variables are described in their relationships in both profiles; also, any variables underlying functional status are determined, as well as with quality of life. Additionally, depression is explored in its possible influence on the perception of quality of life and functional status. Lastly, possible differences among attachment subtypes in relation to the medical condition are ascertained.

Hypotheses

The diverse research hypotheses can be posited according to the research questions, aims, and general goals. These hypotheses also account for the conceptual and theoretical framework and empirical evidence that has been previously presented in the Introduction chapter of this dissertation. The research hypotheses that have guided this study are listed below, following the order of the aforementioned general aims:

General aim I. To determine the psychological and sociodemographic characteristics of FM and RA patients.

H1. Both samples will be predominantly female, especially FM patients.

H2. The demographic, clinical, and psychosocial profile of all patients will suggest a deteriorated health status due to the impact of chronic and/or recurrent pain.

General aim II. To compare the psychological profiles of FM and RA.

H3. Comorbidities and somatic symptoms, expressed in the RDCI and the symptom checklist review of systems respectively, will be higher in the FM sample than in the RA group.

H4. Depression levels will be higher and within clinical categories in FM.

H5. The expression of pain will be more pronounced in the FM sample than in the RA sample.

H6. Functional status and quality of life scores will support the existence of more impairment or deterioration in FM.

General aim III. To establish adult attachment in FM and RA patients according to the secure, fearful, preoccupied, and dismissing styles.

H7. There will be a predominance of insecure attachment in FM in comparison to RA patients, which will mainly be securely attached.

H8. There will be more specific associations between severe FM and RA on one hand, and the different insecure attachment patterns on the other hand.

H9. FM patients will show a more notable prevalence of the hostile fearful/fearful and the dismissing subtypes in contrast to RA patients.

H10. Patients in New York will show a higher prevalence of dismissing attachment in relation to the Spanish samples.

General aim IV. To analyze and contrast the cultural factor jointly in both medical conditions.

H11. Depression and anxiety will be reported differently according to the sample geographical origin.

H12. Psychosocial dimensions, and pain interference with daily activities due to its intimate relationship with quality of life, will be expressed as more deteriorated in Barcelona than in New York due to cultural influence.

General aim V. To explore how clinical and attachment characteristics are related and influence the patients' psychosocial dimensions.

H13. Pain intensity and interference will be highly associated with depression, anxiety, and psychosocial dimensions.

H14. Overall, insecure attachment will have a negative impact on health status, as reported through the clinical and psychosocial variables.

H15. FM severity, according to quality of life and functional status levels, will be more related to the insecure subtypes than in the case of RA.

H16. Depression will explain pain and illness severity, accounted for with quality of life and functional status scores.

Results

In this section, the results obtained in this study are presented. The order followed is the aims presented previously, so as to try to account for the research's main objectives in a coherent manner. Therefore, five subsections will compose this chapter, as shown in the correspondence between sections and specific aims in the table below.

Table 16. Results' organization

Title	Specific research aims
Psychological and sociodemographic features of the samples	1. To explore the sociodemographic traits. 2. To describe the clinical realities of pain, depression, and anxiety. 3. To analyze the psychosocial dimensions of quality of life and functional status.
Comparison of psychological profiles of FM and RA	4. To contrast pain, depression, and anxiety as clinical variables in FM and RA. 5. To ascertain possible similarities and differences between FM and RA regarding quality of life and functional status.
Adult attachment styles in FM and RA patients	6. To find and depict the particularities regarding attachment styles in Spanish and American population. 7. To determine similarities and differences among attachment styles in FM and RA.
Cross-cultural comparison of FM and RA	8. To explore any disparities in relation to depression and anxiety between the samples from both countries. 9. To distinguish similarities and differences between sample origins regarding psychosocial dimensions and pain interference with daily activities.
Relationships among clinical and attachment characteristics and influence on psychosocial dimensions	10. To describe relationships among the analyzed dimensions in both profiles. 11. To study what variables the medical condition's functional status depends on. 12. To analyze what variables quality of life depends on. 13. To verify if depression influences the perception of quality of life and functional status. 14. To detect if attachment subtypes have a potential effect on the illness.

Psychological and sociodemographic features of the samples

The study in Barcelona included 67 FM patients and 70 RA patients, while in New York 16 FM patients and 15 RA patients were recruited. Table 17 shows the variability on the main variables of the study.

Table 17. Measures of central tendency and dispersion

	Demographic variables				Clinical variables						Psychosocial variables						
	Education	Years since diagnosis	Comorbidities (RDCI)	Age	BDI	PSS	PIS	ROS	ANS	DEP	RAPID 3	(S)FIQ	MCS	PCS	FN	PN	PTGL
n	168	168	168	168	168	168	168	168	168	168	168	83	85	85	168	168	168
Mean	12.76	12.81	1.39	57.77	18.07	4.96	5.03	20.44	7.92	6.42	14.70	70.67	49.43	36.08	3.19	6.12	5.39
Median	12.00	10.00	1.00	59.50	14.50	5.50	5.57	20.00	8.00	6.00	16.25	73.32	51.10	34.70	3.00	7.00	6.00
SD	4.98	9.68	1.41	11.56	13.95	2.49	2.89	11.82	5.08	4.44	6.99	16.91	12.10	11.35	1.98	3.00	2.87
Min. - max.	0 - 31	0 - 50	0 - 6	23 - 82	0 - 76	0 - 10	0 - 10	0 - 51	0 - 21	0 - 19	0 - 28	8 - 98.52	9.90 - 72.40	9.50 - 56.30	0 - 9	0 - 10	0 - 10

RDCI: Rheumatic Disease Comorbidity Index. BDI: Beck Depression Inventory. (S)FIQ: (Spanish) Fibromyalgia Impact Questionnaire. MCS: SF-36's Mental Component Summary. PCS: SF-36's Physical Component Summary. PSS: BPI's Pain Severity Score. PIS: BPI's Pain Interference Scale. RAPID3: functional status. ROS: symptom checklist review of systems. ANS: HADS anxiety subscale score. DEP: HADS depression subscale score. FN: RAPID3'S Physical Function component. PN: RAPID3's Pain component. PTGL: RAPID3's Patient's Global Estimate of Status component. SD: standard deviation. Min.: minimum. Max.: maximum.

Table 18. Demographic variables

		n	Age (mean ± standard deviation)	Sex		Nationality	
				Female	Male	Spanish	American
Barcelona	**FM**	67	57.72 ± 10.55	67 (100%)	0 (0%)	67 (100%)	0 (0%)
	RA	70	57.24 ± 12.26	51 (72.9%)	19 (27.1%)	70 (100%)	0 (0%)
New York	**FM**	16	58.69 ± 12.93	16 (100%)	0 (0%)	0 (0%)	16 (100%)
	RA	15	59.53 ± 11.99	13 (86.7%)	2 (13.3%)	0 (0%)	15 (100%)

As may be observed in Table 18, in both countries, the FM sample was exclusively female and the RA sample was primarily composed of women.

Table 19. Marital status variable

		Marital status					
		Divorced	Domestic partnership	Married	Separated	Single	Widow/er
Barcelona	**FM**	6 (9%)	5 (7.5%)	44 (65.7%)	4 (6%)	2 (3%)	6 (9%)
	RA	7 (10%)	2 (2.9%)	47 (67.1%)	2 (2.9%)	7 (10%)	5 (7.1%)
New York	**FM**	3 (18.8%)	0 (0%)	7 (43.8%)	0 (0%)	5 (31.3%)	1 (6.3%)
	RA	0 (0%)	0 (0%)	7 (46.7%)	1 (6.7%)	5 (33.3%)	2 (13.3%)

The Spanish sample was mostly married, while the American one was more evenly distributed into the non-married categories (Table 19).

Table 20. Work status variable

		Work status				
		Employed	Permanent/SS disability	Sick leave	Unemployed	Retired
Barcelona	FM	18 (26.9%)	10 (14.9%)	2 (3%)	16 (23.9%)	21 (31.3%)
	RA	31 (44.3%)	5 (7.1%)	3 (4.3%)	8 (11.4%)	23 (32.9%)
New York	FM	3 (18.8%)	6 (37.5%)	1 (6.3%)	1 (6.3%)	5 (31.3%)
	RA	4 (26.7%)	7 (46.7%)	0 (0%)	1 (6.7%)	3 (20%)

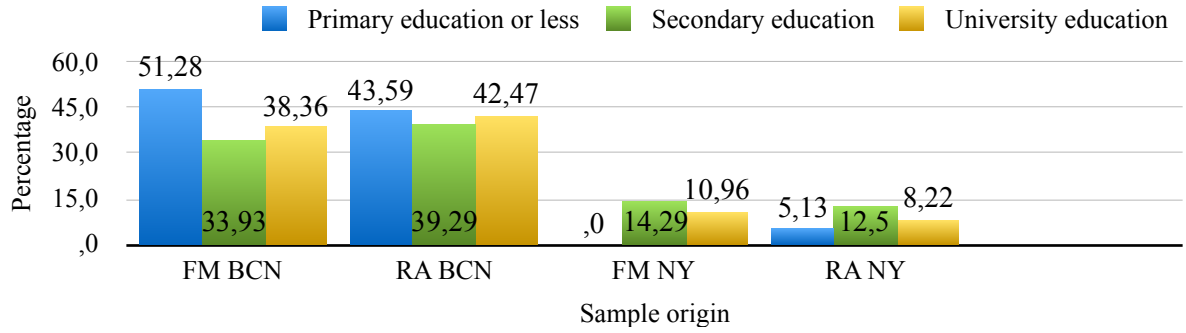
As per the work status characteristics of the sample (Table 20), there was also a considerable amount of American patients with a Social Security disability, compared to their Spanish counterparts. Further, while in New York there were more RA patients with a Social Security disability than FM patients, in Barcelona it was FM patients who had more permanent disabilities than in RA.

Table 21. Education level

		Education level (mean ± standard deviation)
Barcelona	FM	12.17 ± 5.23
	RA	13.18 ± 5.37
New York	FM	13.94 ± 2.74
	RA	12.20 ± 3.45

Table 21 shows the education level variable as a continuous variable, offering scarce possibilities of contrasting different profiles. It is calculated as number of years of education received after the first grade of the primary education level, or Elementary School.

Figure 26. Education level.



As can be seen in Figure 26, the education level measured in years was also converted into an ordinal variable to show this particular graphic distribution through sample origins. In this case, two differentiated profiles can be observed. For instance, a larger percentage of primary educated patients may be observed in both Barcelona samples with respect to the New York ones. However, proportionately there were also larger percentages of university educated patients in both Barcelona samples than in New York.

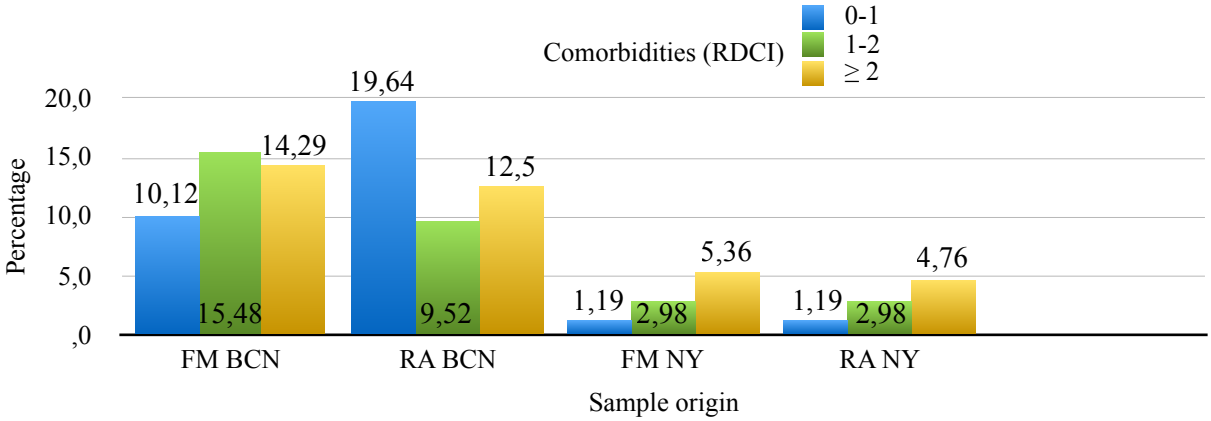
Table 22. Demographic variables and comorbidities

		Years since diagnosis (mean ± standard deviation)	RDCI (range 0-9) (mean ± standard deviation)
Barcelona	**FM**	12.76 ± 8.40	1.42 ± 1.35
	RA	14.01 ± 11.18	1.14 ± 1.44
New York	**FM**	6.81 ± 6.24	2.06 ± 1.65
	RA	13.87 ± 8.81	1.67 ± 1.11

RDCI: Rheumatic Disease Comorbidity Index.

Table 22 shows descriptive data, without conducting bivariate tests. When looking strictly at the year count, that FM patients in New York had been diagnosed substantially earlier than in Barcelona. Moreover, in New York there were higher comorbidity counts, although in both countries the FM samples had a higher RDCI mean compared to RA patients.

Figure 27. Comorbidities in both samples.



As per the Rheumatic Disease Comorbidity Index (RDCI), Figure 27 shows its percentage distribution as an ordinal variable in all samples. When comparing the highest level for comorbidities, both FM samples had higher percentages than the RA samples. However, the New York RA sample had the largest percentage of comorbidities concentrated in the highest possible level of RDCI, emulating the FM phenomenon of a more severe RDCI, while in Barcelona the highest percentage of comorbidity count in the RA sample proved that these patients had less severe comorbidities.

Table 23 shows the different medications used by patients in both countries as reported in the interview. It may also be observed that in the U.S., there is a use of topical ointments and creams indicated for pain management that weren't prescribed in the Barcelona sample.

Table 23. Medication used at the moment of the study

	Barcelona		New York	
	FM Median (P25-P75)	RA Median (P25-P75)	FM Median (P25-P75)	RA Median (P25-P75)
NSAIDs - Paracetamol	1 (1-1)	1 (1-1)	1 (1-1.5)	1 (1-1)
Antidepressants	1 (1-2)	1 (1-1)	1 (1-1)	1 (1-1)
Anticonvulsants	1 (1-1)	1 (1-1)	1 (1-1)	1 (1-1)
Opioids	1 (1-1)	1 (1-1)	1 (1-1)	1.5 (1.25-1.75)
Corticoids	1 (1-1)	1 (1-1)	None	1 (1-1)
DMARDs	None	1 (1-1)	None	1 (1-1)
Biologic DMARDs	1 (1-1)	1 (1-1)	None	1 (1-1)
Anxiolytics/ Benzodiacepines	1 (1-2)	1 (1-1)	1 (1-1)	None
Antipsychotics	1 (1-1)	None	1 (1-1)	None
Muscle relaxants	1 (1-1)	1 (1-1)	1 (1-1.25)	1 (1-1)
Antimalarials	1 (1-1)	1 (1-1)	1 (1-1)	1 (1-1)
Intestinal anti-inflammatory agents	None	1 (1-1)	None	None
Sodium oxybate (Xyrem®)	1 (1-1)	None	None	None
Vitamins and mineral supplements (including folic acid)	1 (1-1)	1 (1-1)	3 (2-3.5)	1 (1-2.25)
Ophtalmological solutions	1 (1-1)	1.5 (1-2.75)	1 (1-1)	None
Homeopathic medicine	1 (1-1)	1.5 (1.25-1.75)	1 (1-1)	None
Special diets (including melatonin)	1 (1-1)	1 (1-1)	1 (1-1)	1 (1-1)
Pain compound 1	N/A	N/A	1 (1-1)	None
Pain compound 2	N/A	N/A	1 (1-1)	1 (1-1)
Topical numbing creme	N/A	N/A	1 (1-1)	1 (1-1)
Admitted marihuana	None	None	None	1 (1-1)
Lidoderm lidocaine patch 5%	N/A	N/A	1 (1-1)	None

P25-P75: Percentile 25-Percentile 75. NSAIDs: Nonsteroidal Anti-Inflammatory Drugs. DMARDs: Disease-Modifying Antirheumatic Drugs (methotrexate, sulfasalazine, hydroxychloroquine, leflunomide, gold salts, azathioprine, cyclosporine). Biologic DMARDs: etanercept, adalimumab, infliximab, certolizumab pegol, golimumab, anakinra, abatacept, rituximab, tocilizumab, tofacitinib. Pain compound 1: gabapentin, diclofenac, amitryptiline, EMLA. Pain compound 2: ketoprofen, baclofen, cyclobenzaprine, bupivacaine, lidocaine, mometasone.

Figure 28. Depression in the American sample.

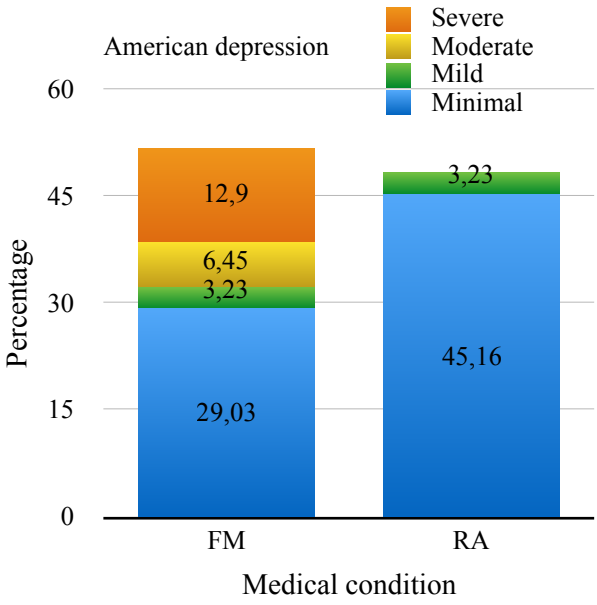


Figure 29. Depression in the Spanish sample.

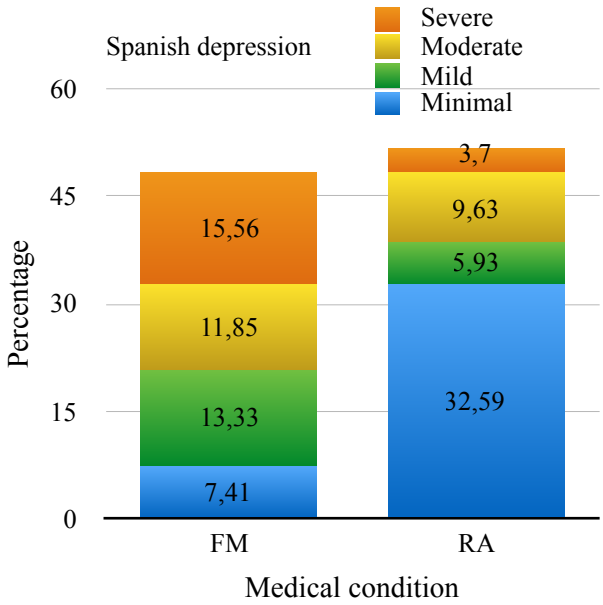


Figure 28 shows the distribution of BDI-measured depression in both samples from New York. The presence of moderate and severe depression can be observed only in FM patients, therefore proving it to be the more depressed sample. RA patients were predominantly not depressed.

Next to it, Figure 29 shows that when depression was set on the Spanish thresholds for the BDI, the FM sample had larger percentages of mild, moderate, and severe depression than RA patients. Once more, most RA patients didn't have clinical depression.

Figure 30. American attachment styles.

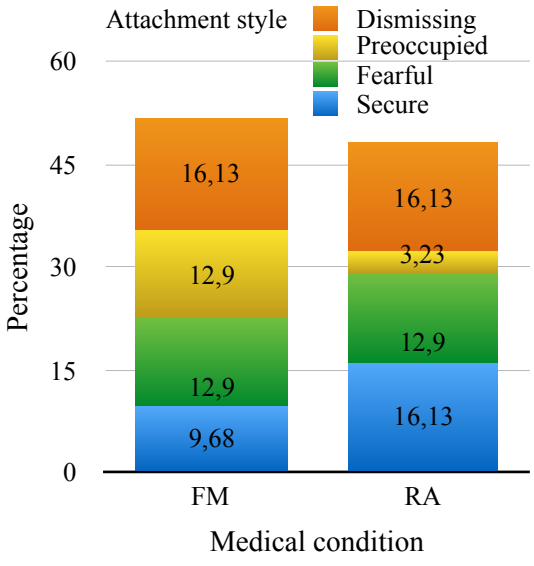


Figure 31. Spanish attachment styles.

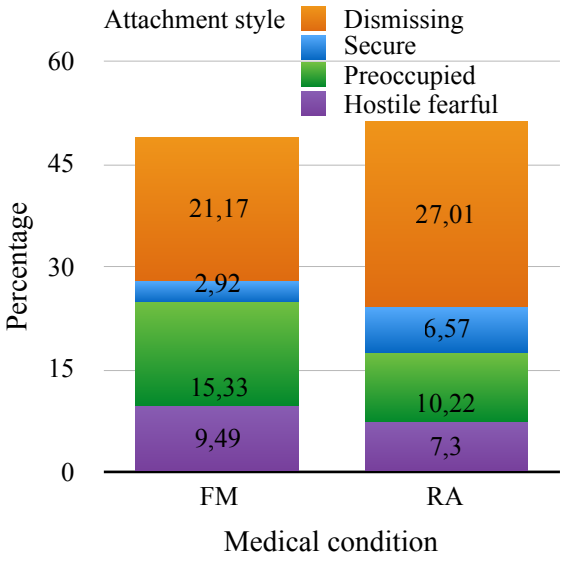


Figure 30 shows the attachment style distribution in the American sample, in which the dismissing style is predominant in both medical conditions. Additionally, the RA sample had the same number of dismissing and securely attached patients, stressing the two most prevalent attachment styles. In contrast, Figure 31 illustrates the percentage distribution of attachment styles in the Spanish sample, showing a larger dismissing percent in both FM and RA, followed by the preoccupied group, the second most prevalent attachment style in this instance. Overall, the Spanish samples appear more evenly distributed, with bigger differences among attachment styles than in the American samples, with closer and harder to distinguish percent groups within and between medical conditions.

Figure 32. Attachment in the American sample.

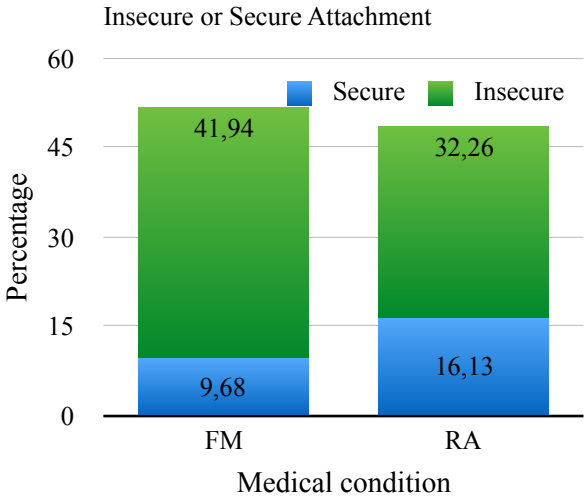
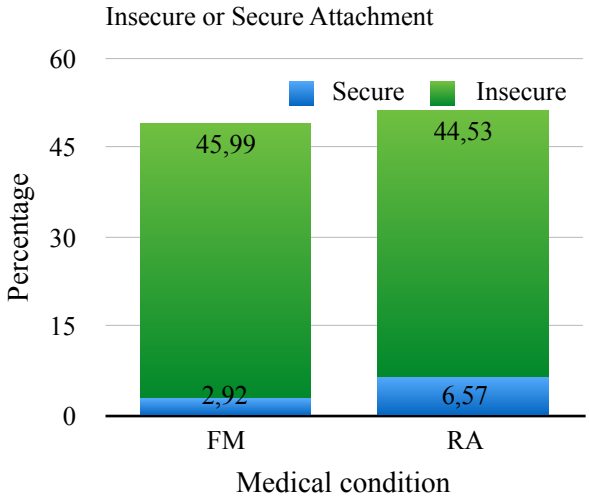


Figure 33. Attachment in the Spanish sample.



Figures 32 and 33 illustrate the proportion of insecure and secure attachment in New York and Barcelona through percentages. In American FM, the count was 3 subjects for secure attachment and 13 for insecure. There were 5 securely attached RA patients and 10 insecurely attached. On the other hand, Spanish FM had 4 secure patients and 63 insecure ones, while in RA there were 9 securely attached patients and 61 insecure ones. In both cases, the insecure attachment styles are clearly preeminent, with a slightly larger RA secure group. The percents appear to show larger disparities among secure and insecure groups in the Spanish samples, in comparison to the American ones.

Figure 34. Functional status in all samples.

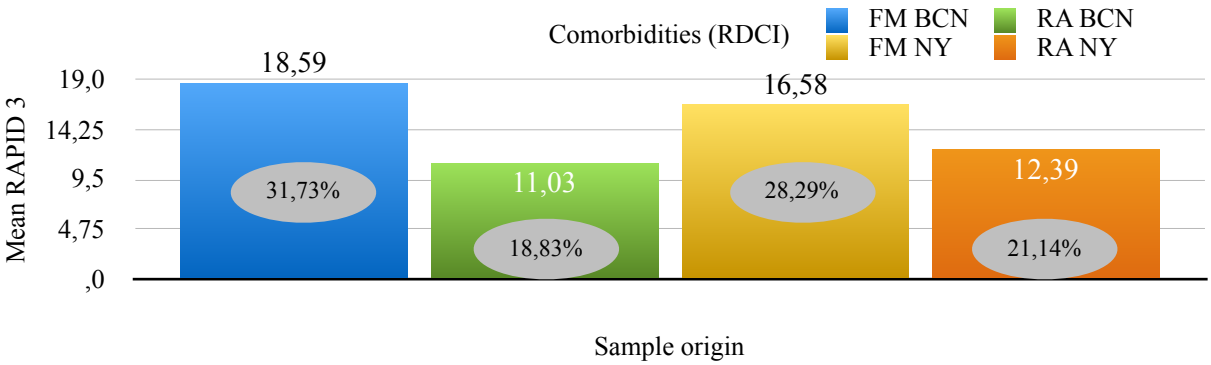


Figure 34 shows a distinctly differentiated profile when comparing both medical conditions with regards to functional status: in both countries, the FM sample had a poorer functional status than the RA sample, albeit the range between means was seemingly wider in Barcelona. Additionally, this suggests worse functional status in Barcelona FM than in NY FM, and worse NY RA functional status than Barcelona RA functional status.

Figure 35. Quality of life in all samples.

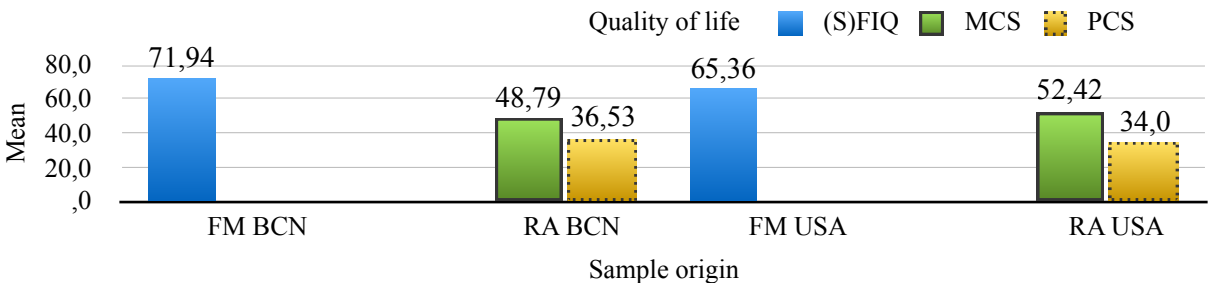


Figure 35 presents the quality of life in RA and FM patients from both countries. In the case of FM, a mean of 50 is expected in these patients, and a mean of 70 indicates a severe form of disease or poorer quality of life. As can be observed, FM patients in Barcelona reached this latter, severe SFIQ mean. Inversely, a higher SF-36 score in RA patients denotes better quality of life. Barcelona RA patients had a lower MCS mean but slightly higher PCS than RA patients in New York.

Comparison of psychological profiles of FM and RA

To begin assessing the relationships between the key variables of the study, Pearson correlation coefficients were obtained among outcomes of the questionnaires (Table 24a and 24b). Statistically significant correlations were found between education level and SF-36's PCS, patient's age and the years since the diagnosis was established, quality of life in FM and all other variables excepting education level and age, quality of life and years since diagnosis, comorbidities and all other variables except for both SF-36 component summaries and patient's global estimate of status, years since diagnosis and physical function, and the rest of variables amongst each other except with age. Inverse relationships with a statistical significance were also found between both SF-36 component summaries and BDI, both pain-related variables, RAPID3 and its three quality of life components, symptom checklist review of systems, HADS' anxiety and depression, and quality of life; and between education level and years since diagnosis, both dimensions of pain from the BPI, RAPID3 and its three quality of life components, symptom checklist review of systems, and age.

Table 24a. Pearson correlations amongst key variables

	Ed.	Years since diag.	Comor.	BDI	PSS	PIS	RAPID 3	ROS	ANS	DEP	Age
Ed.	-	-.158*	-.013	-.064	-.247**	-.176*	-.250**	-.190*	-.101	-.047	-.277***
Years since diag.		-	.051	.049	-.021	.071	.065	-.006	.071	-.044	.235**
Como.			-	.277***	.176*	.215**	.219**	.313***	.226**	.301***	.272***
BDI				-	.477***	.620***	.549***	.609***	.663***	.752***	-.004
PSS					-	.857***	.868***	.636***	.540***	.539***	-.007
PIS						-	.848***	.680***	.624***	.677***	-.046
RAPID 3							-	.700***	.606***	.605***	.009
ROS								-	.611***	.600***	.080
ANS									-	.708***	-.060
DEP										-	-.054
Age											-

* $p < .05$, ** $p < .01$, *** $p < .001$. Ed.: Education. Years since diag.: Years since diagnosis. Comor.: RDCI Comorbidities. BDI: Beck Depression Inventory II. PSS: BPI's Pain Severity Score. PIS: BPI's Pain Interference Scale. RAPID3: functional status. ROS: symptom checklist review of systems. ANS: HADS anxiety subscale score. DEP: HADS depression subscale score. (S)FIQ: (Spanish) Fibromyalgia Impact Questionnaire. MCS: SF-36 Mental Component Summary. PCS: SF-36 Physical Component Summary. FN: RAPID3'S Physical Function component. PN: RAPID3's Pain component. PTGL: RAPID3's Patient's Global Estimate of Status component.

Table 24b. Pearson correlations amongst key variables

	(S)FIQ	MCS	PCS	FN	PN	PTGL
Ed.	-.129	-.059	.319**	-.195*	-.242**	-.222**
Years since diag.	.335**	.013	-.102	.172*	-.008	.049
Como.	.227*	-.182	-.196	.256**	.198*	.150
BDI	.528***	-.481***	-.418***	.517***	.460***	.502***
PSS	.772***	-.244*	-.766***	.639***	.877***	.758***
PIS	.845***	-.467***	-.680***	.677***	.828***	.736***
RAPID 3	.856***	-.414***	-.827***	.833***	.903***	.919***
ROS	.572***	-.473***	-.539***	.623***	.657***	.590***
ANS	.544***	-.621***	-.248*	.495***	.509***	.604***
DEP	.534***	-.566***	-.480***	.550***	.493***	.580***
Age	-.048	.096	-.098	.079	-.005	-.027
(S)FIQ	-	D. A.	D. A.	.697***	.750***	.748***
MCS		-	.145	-.337**	-.311**	-.438***
PCS			-	-.760***	-.727***	-.700***
FN				-	.619***	.693***
PN					-	.729***
PTGL						-

* $p < .05$, ** $p < .01$, *** $p < .001$. Ed.: Education. Years since diag.: Years since diagnosis. Comor.: RDCI Comorbidities. BDI: Beck Depression Inventory II. PSS: BPI's Pain Severity Score. PIS: BPI's Pain Interference Scale. RAPID3: functional status. ROS: symptom checklist review of systems. ANS: HADS anxiety subscale score. DEP: HADS depression subscale score. (S)FIQ: (Spanish) Fibromyalgia Impact Questionnaire. MCS: SF-36 Mental Component Summary. PCS: SF-36 Physical Component Summary. FN: RAPID3'S Physical Function component. PN: RAPID3's Pain component. PTGL: RAPID3's Patient's Global Estimate of Status component.

Before performing a multiple linear regression model, bivariate analyses were carried out to ascertain any potential specific associations between functional status in all patients and other categorical variables that applied to both medical conditions. Therefore, a one-way ANOVA determined there was a statistically significant difference between the insecurely and the securely attached groups with regards to RAPID3 functional status ($F(1, 166) = 6.729$, $p = .010$), RAPID3's pain component ($F(1, 166) = 5.601$, $p = .019$), and RAPID3's patient global estimate of status ($F(1, 166) = 7.574$, $p = .007$), but didn't yield significant differences when looking into the relationship with RAPID3's physical function ($p = .119$). It also found statistically significant differences between gender and the same variables (functional status, ($F(1, 166) = 9.983$, $p = .002$); physical function, ($F(1, 166) = 5.744$, $p = .018$); pain, ($F(1, 166) = 9.691$, $p = .002$); and patient global estimate of status, ($F(1, 166) = 7.574$, $p = .007$)), but produced no significant differences between nationality and these

variables (p = .495 for physical function, p = .606 for pain, .700 for patient global estimate of status, and p = .897 for functional status). Calculating the effect size using Cohen's d for the statistically significant differences produced the results shown in Table 25, showing a medium to high effect size. More specifically, the effect size was medium in the case of attachment and functional status, pain, and patient global estimate of status. In the instance of sex, Cohen's d showed a medium effect as well for functional status, physical function, pain, and patient global estimate of status. Finally, regarding nationality, the effect size was not significant for any of these variables.

Table 25. Measure of effect size in differences amongst categorical variables and health-related quality of life/ disability

	RAPID3	FN	PN	PTGL
Insecure/Secure attachment	0.605	N. S.	0.552	0.642
Sex	-0.737	-0.559	-0.726	-0.642
Nationality	N. S.	N. S.	N. S.	N. S.

RAPID3: functional status. FN: RAPID3'S Physical Function component. PN: RAPID3's Pain component. PTGL: RAPID3's Patient's Global Estimate of Status component. N. S.: Not Significant.

In order to test differences between both groups, an independent samples T-test was conducted and produced the results in Table 26. Statistically significant differences may be observed in depression as measured by BDI, HADS levels of anxiety and depression, both pain-related outcomes, functional status, symptom checklist review of systems, physical function, pain, and patient global estimate of status.

Table 26. Results of comparing both medical conditions with main numerical variables.

Numerical variables (Mean ± standard deviation)	FM (*n*=83)	RA (*n*=85)	*p*
Education	12.51 ± 4.89	13.01 ± 5.08	.517
Age	57.90 ± 10.97	57.65 ± 12.17	.886
Comorbidities	1.54 ± 1.43	1.24 ± 1.39	.160
BDI	24.14 ± 14.46	12.14 ± 10.53	>.001
ANS	10.35 ± 4.76	5.55 ± 4.20	>.001
DEP	8.42 ± 4.41	4.46 ± 3.52	>.001
PSS	6.32 ± 1.85	3.63 ± 2.31	>.001
PIS	6.45 ± 2.20	3.64 ± 2.81	>.001
RAPID3	18.20 ± 5.26	11.27 ± 6.79	>.001
ROS	27.31 ± 10.08	13.73 ± 9.30	>.001
FN	3.99 ± 1.88	2.41 ± 1.75	>.001
PN	7.57 ± 1.91	4.70 ± 3.19	>.001
PTGL	6.64 ± 2.35	4.16 ± 2.81	>.001

BDI: Beck Depression Inventory II. ANS: HADS anxiety subscale score. DEP: HADS depression subscale score. PSS: BPI's Pain Severity Score. PIS: BPI's Pain Interference Scale. RAPID3: functional status. ROS: symptom checklist review of systems. FN: RAPID3'S Physical Function component. PN: RAPID3's Pain component. PTGL: RAPID3's Patient's Global Estimate of Status component.

The FM sample scored higher in BDI depression than the RA sample, $t(149) = 6.138$, $p > .001$; as well as in HADS anxiety and depression, $t(166) = 6.930$, $p > .001$ and $t(156) = 6.426$, $p > .001$, respectively. FM patients also scored higher in both pain-related outcomes, pain severity, $t(159) = 8.357$, $p > .001$, and pain interference, $t(158) = 7.245$, $p > .001$. Additionally, FM patients had a poorer functional status than RA patients, $t(157) = 7.404$, $p > .001$; and they also had more somatic symptoms (symptom checklist), $t(166) = 9.083$, $p > .001$. Lastly, FM patients had higher scores in all of RAPID3's quality of life components, stressing the lower quality of life in comparison to RA patients: in physical function, $t(166) = 5.621$, $p > .001$; pain, $t(137) = 7.099$, $p > .001$; and patient's global estimate of status, $t(162) = 6.213$, $p > .001$.

When comparing the categorical variables of both samples, the only statistically significant difference was found between medical condition and gender (Table 27), despite

the notable differences concerning the control group and insecure attachment. Also, the categorical variable of nationality henceforward was only considered in two groups for multivariate analyses purposes: the sample from Barcelona and the one in New York.

Table 27. Results of comparing both medical conditions with main categorical variables.

Categorical variables *n* (%)		FM (*n*=83)	RA (*n*=85)	*p*
Sex	Female	83 (100%)	64 (75.3%)	>.001
	Male	0 (0%)	21 (24.7%)	
Attachment	Secure	7 (8.4%)	14 (16.5%)	.115
	Insecure	76 (91.6%)	71 (83.5%)	
Nationality	Hispanic	67 (80.7%)	70 (82.4%)	.785
	American	16 (19.3%)	15 (17.6%)	

The statistically significant difference between gender and medical condition, $\chi(1) = 23.345$, p >.001, had Phi and Cramer's V test values of 0.373, and therefore showed a medium effect size.

Adult attachment styles in FM and RA patients

A one-way ANOVA was performed to observe possible differences amongst the specific attachment patterns with regards to BDI depression level, HADS anxiety and depression, pain-related outcomes, functional status, and symptom checklist review of systems. In the Barcelona sample, there were statistically significant differences between groups found in BDI depression ($F(3,133) = 10.716$, $p > .001$), pain interference ($F(3,133) = 4.103$, $p = .008$), symptom checklist review of systems ($F(3,133) = 4.043$, $p = .009$), and HADS anxiety ($F(3,133) = 8.826$, $p > .001$) and depression ($F(3,133) = 7.074$, $p > .001$). The means and standard deviations are provided in Table 28.

Table 28. Results of comparing Spanish attachment styles with main numerical variables

Numerical variables (Mean ± standard deviation)	Hostile Fearful (*n*=23)	Preoccupied (*n*=35)	Secure (*n*=13)	Dismissing (*n*=66)	*p*
BDI	30.13 ± 17.41	22.97 ± 15.69	11.00 ± 6.70	15.09 ± 9.67	>.001
ANS	10.65 ± 4.81	9.80 ± 4.70	4.15 ± 2.70	7.08 ± 4.43	>.001
DEP	8.47 ± 3.99	7.80 ± 4.50	3.08 ± 2.50	5.65 ± 4.06	>.001
PSS	5.47 ± 1.93	5.44 ± 2.43	4.12 ± 2.40	4.59 ± 2.48	.139
PIS	6.14 ± 2.44	5.92 ± 2.79	3.79 ± 2.91	4.57 ± 2.68	.008
RAPID3	16.00 ± 5.46	16.77 ± 6.55	12.10 ± 6.96	13.73 ± 7.04	.059
ROS	24.65 ± 11.74	24.37 ± 12.35	17.31 ± 11.74	17.59 ± 10.96	.009
FN	3.87 ± 2.11	3.55 ± 2.05	2.95 ± 1.52	2.91 ± 1.77	.123
PN	6.48 ± 2.09	7.16 ± 2.74	5.08 ± 3.17	5.53 ± 3.10	.028
PTGL	5.65 ± 2.45	6.06 ± 2.89	4.08 ± 2.49	5.29 ± 2.70	.143

BDI: Beck Depression Inventory II. ANS: HADS anxiety subscale score. DEP: HADS depression subscale score. PSS: BPI's Pain Severity Score. PIS: BPI's Pain Interference Scale. RAPID3: functional status. ROS: symptom checklist review of systems. FN: RAPID3'S Physical Function component. PN: RAPID3's Pain component. PTGL: RAPID3's Patient's Global Estimate of Status component.

Table 29. Measure of effect size in differences amongst Spanish attachment styles

Groups compared in Scheffe's test	BDI	ANS	DEP
Hostile fearful Secure	1.652	2.000	2.000
Hostile fearful Dismissing	1.154	0.750	0.857
Preoccupied Secure	1.048	1.667	1.333
Preoccupied Dismissing	0.583	0.500	N. S.

BDI: Beck Depression Inventory II. ANS: HADS anxiety subscale score. DEP: HADS depression subscale score.

However, when the *post hoc* Scheffe's test was conducted, it only detected the statistically significant differences shown in Table 29, through the presented Cohen's *d*. All of the effect sizes were large, except for the Cohen's *d* 0.583 and 0.5, both of which suggested moderate practical significance.

In the case of the New York sample, a Kruskal-Wallis H test showed that there were no statistically significant differences between attachment style groups (Table 30).

Table 30. Kruskal-Wallis mean ranks from comparing American attachment styles with main numerical variables

Numerical variables (Mean ranks)	Secure (*n*=8)	Fearful (*n*=8)	Preoccupied (*n*=5)	Dismissing (*n*=10)	*p*
BDI	10.56	18.88	20.70	15.70	.169
ANS	9.19	16.31	22.60	17.90	.054
DEP	9.31	17.69	18.70	18.65	.115
PSS	10.94	17.63	20.40	16.55	.267
PIS	11.44	18.69	19.50	15.75	.325
RAPID3	10.06	19.31	18.50	16.85	.176
ROS	9.13	19.00	18.10	18.05	.101
FN	12.63	18.81	16.20	16.35	.594
PN	10.19	18.81	19.70	16.55	.171
PTGL	11.13	18.75	18.40	16.50	.326

BDI: Beck Depression Inventory II. ANS: HADS anxiety subscale score. DEP: HADS depression subscale score. PSS: BPI's Pain Severity Score. PIS: BPI's Pain Interference Scale. RAPID3: functional status. ROS: symptom checklist review of systems. FN: RAPID3'S Physical Function component. PN: RAPID3's Pain component. PTGL: RAPID3's Patient's Global Estimate of Status component.

Nevertheless, due to small sample size in the case of American patients, another nonparametric test was performed but only with the secure and insecure attachment groups. This time, it yielded that there were statistically significant differences between the insecure and secure patterns in the cases of BDI scores, $\chi2(3) = 3.882$, $p = .049$; functional status, $\chi2(3) = 4.600$, $p = .032$; symptom checklist, $\chi2(3) = 6.175$, $p = .013$; HADS-assessed anxiety, $\chi2(3) = 6.110$, $p = .013$, and depression, $\chi2(3) = 5.880$, $p = .015$; and pain, $\chi2(3) = 4.508$, $p = .034$. The mean rank scores of each group are shown in Table 31.

Table 31. Kruskal-Wallis mean ranks from comparing American secure and insecure attachment with main numerical variables

Numerical variables (Mean ranks)	Secure (*n*=8)	Insecure (*n*=23)	*p*
BDI	10.56	17.89	.049
ANS	9.19	18.37	.013
DEP	9.31	18.33	.015
PSS	10.94	17.76	.067
PIS	11.44	17.59	.099
RAPID3	10.06	18.07	.032
ROS	9.13	18.39	.013
FN	12.63	17.17	.221
PN	10.19	18.02	.034
PTGL	11.13	17.70	.076

BDI: Beck Depression Inventory II. ANS: HADS anxiety subscale score. DEP: HADS depression subscale score. PSS: BPI's Pain Severity Score. PIS: BPI's Pain Interference Scale. RAPID3: functional status. ROS: symptom checklist review of systems. FN: RAPID3'S Physical Function component. PN: RAPID3's Pain component. PTGL: RAPID3's Patient's Global Estimate of Status component.

To further explore the attachment styles, and due to the fact that the Spanish questionnaire contemplated different scales than the American ones, an independent samples T-test was run to reveal significant differences in the questionnaire scales in relation to other relevant variables. Scale scores were previously converted into z-scores, as may be seen in Table 32. The test found that RA patients had statistically significantly lower scores in Scale 1 compared to the FM group ($t(135) = 2.565$, $p = .011$).

Table 32. Results of comparing both medical conditions with Spanish attachment questionnaire scales

Attachment styles (Mean ± standard deviation)	FM (*n*=67)	RA (*n*=70)	*p*
Scale 1	0.22 ± 0.97	-0.21 ± 0.99	.011
Scale 2	-0.02 ± 0.92	0.02 ± 1.08	.830
Scale 3	-0.13 ± 1.03	0.12 ± 0.96	.138
Scale 4	0.09 ± 1.04	-0.09 ± 0.96	.302

Scale 1: Low self-esteem, need of approval and fear of rejection. Scale 2: Hostile resolution of conflict, rancor and possessiveness. Scale 3: Emotional expressiveness and comfortableness with intimacy. Scale 4: Emotional self-sufficiency and discomfort with intimacy.

When looking into the two American adult attachment questionnaires, scores were used to create a composite measure, which then was transformed into z-scores (Table 33). In this instance, a Mann-Whitney U test found no statistically significant differences between both medical conditions regarding questionnaire scales.

Table 33. Results of comparing both medical conditions with American attachment questionnaires scales

Attachment styles	Median	*U*	*p*
Scale 1	0.0248	115.500	.859
Scale 2	-0.0500	101.500	.464
Scale 3	-0.1719	95.000	.322
Scale 4	0.1296	99.000	.406

Scale 1: Secure. Scale 2: Fearful. Scale 3: Preoccupied. Scale 4: Dismissing.

Pearson correlation coefficients were then obtained to further analyze the relationships between these scales and other variables, as shown in Table 34. Statistical significance was found in the correlations in both samples between Scale 1 and BDI depression, both pain dimensions, functional status, symptom checklist review of systems, HADS-measured anxiety and depression, physical function, pain, and patient global estimate of status. They were also observed in the Barcelona sample between Scale 2 and BDI

depression, as well as HADS anxiety and depression, Scale 3, corresponding to the secure style, had no significant correlations with any of the variables. Scale 4 showed significant correlations with BDI depression, pain interference, functional status, symptom checklist review of systems, HADS anxiety and depression, and patient global estimate of status. In the New York sample, they were confirmed between Scale 2 and pain interference and pain, and Scale 3 and symptom checklist review of systems and pain. Significant inverse correlations were also found between Scale 1 and BDI depression, both pain-related outcomes, functional status, HADS anxiety and depression, pain, and patient global estimate of status. This latter inverse relationship could be explained by the fact that Scale 1 corresponds to the secure attachment style, thus not enhancing the anxiety and depression levels, the pain-related outcomes, or the functional status. Scale 4, the dismissing style, showed no significant correlations with any variables.

Table 34. Pearson correlations

		BDI	PSS	PIS	RAPID 3	ROS	ANS	DEP	FN	PN	PTGL
BCN	Scale 1	.543***	.258**	.392***	.308***	.352***	.527***	.504***	.277**	.277**	.276**
	Scale 2	.256**	.026	.111	.053	.135	.253**	.193*	.083	.051	.019
	Scale 3	.007	.020	-.014	.046	.058	.012	-.100	.120	-.001	.032
	Scale 4	.328***	.131	.225**	.170*	.178*	.313***	.327***	.139	.136	.179*
New York	Scale 1	-.427*	-.360*	-.359*	-.476**	-.151	-.478**	-.463**	-.241	-.420*	-.532**
	Scale 2	.319	.347	.407*	.344	.264	.332	.275	.170	.416*	.281
	Scale 3	.200	.313	.355	.310	.359*	.323	.154	.248	.356*	.207
	Scale 4	.134	.143	.119	.100	.211	.248	.291	.039	.173	.040

* $p < .05$, ** $p < .01$, *** $p < .001$. BDI: Beck Depression Inventory II. PSS: BPI's Pain Severity Score. PIS: BPI's Pain Interference Scale. RAPID3: functional status. ROS: symptom checklist review of systems. ANS: HADS anxiety subscale score. DEP: HADS depression subscale score. FN: RAPID3'S Physical Function component. PN: RAPID3's Pain component. PTGL: RAPID3's Patient's Global Estimate of Status component. Barcelona (BCN)— Scale 1: Low self-esteem, need of approval and fear of rejection; Scale 2: Hostile resolution of conflict, rancor and possessiveness; Scale 3: Emotional expressiveness and comfortableness with intimacy; Scale 4: Emotional self-sufficiency and discomfort with intimacy. New York— Scale 1: Secure; Scale 2: Fearful; Scale 3: Preoccupied; Scale 4: Dismissing.

Cross-cultural comparison of FM and RA

A one-way ANOVA was conducted to determine differences amongst medical conditions across nationalities, and yielded virtually identical results than without the cross-cultural comparison with some exceptions. No statistically significant differences were observed when comparing education level (p = .473), age (p = .899), and RDCI-assessed comorbidities (p = .094). The same aforementioned significant differences (Table 26) were found between the FM and the comparison group in relation to HADS anxiety and depression levels, pain-related outcomes, functional status, RAPID3's three components, and the symptom checklist ($F(3, 164) = 16.774$, p >.001 for HADS anxiety; $F(3, 164) = 13.746$, p >.001 for HADS depression; $F(3, 164) = 23.064$, p >.001 for pain severity; $F(3, 164) = 17.710$, p >.001 for pain interference; $F(3, 164) = 18.843$, p >.001 for functional status; $F(3, 164) = 27.632$, p >.001 for symptom checklist; $F(3, 164) = 12.283$, p >.001 for physical function; $F(3, 164) = 16.927$, p >.001 for pain; and $F(3, 164) = 13.085$, p >.001 for patient's global estimate of status). However, the *post-hoc* analysis with Scheffe's test showed that there were unexpected differences in the BDI and RAPID3 groups: it didn't reveal statistically significant differences (p = .409) between the BDI depression levels of the Barcelona RA (13.44 ± 10.89) and the New York FM (19.31 ± 13.68) samples, nor in the RAPID3 functional status (p = .303) of the New York FM (16.58 ± 7.30) and New York RA (12.39 ± 8.33) samples. It also revealed similar results for the RAPID3 quality of life components: there were no statistically significant differences (p = .057) between the physical function scores of the Barcelona FM (4.18 ± 1.74) and the New York RA (2.75 ± 2.38) samples; (p = .71) nor in the patient global estimate of status scores between the Barcelona RA (4.14 ± 2.67) and the New York FM (6.06 ± 3.40) samples.

Table 35. Measure of effect size in differences amongst medical conditions and nationalities

Groups compared in Scheffe's test	BDI	ANS	DEP	PSS	PIS	RAPID 3	ROS	FN	PN	PTGL
FM BCN RA BCN	0.934	1.061	0.998	1.434	1.259	1.362	1.490	1.104	1.227	1.116
FM BCN RA NY	1.908	1.412	1.172	1.198	1.401	0.957	1.320	0.683	0.814	0.871
FM NY RA BCN	N. S.	0.839	0.864	1.076	0.693	0.805	1.180	0.426	0.951	0.630
FM NY RA NY	1.371	1.150	1.005	0.907	N. S.	N. S.	1.028	0.179	0.628	0.512

BDI: Beck Depression Inventory II. ANS: HADS anxiety subscale score. DEP: HADS depression subscale score. PSS: BPI's Pain Severity Score. PIS: BPI's Pain Interference Scale. RAPID3: functional status. ROS: symptom checklist review of systems. FN: RAPID3'S Physical Function component. PN: RAPID3's Pain component. PTGL: RAPID3's Patient's Global Estimate of Status component. N. S.: Not Significant.

Table 35 shows that all of the effect sizes were large, except for the Cohen's *d* referring to the difference of PIS between the NY FM sample and the BCN RA sample, which suggested moderate to high practical significance.

Table 36. Results of comparing both medical conditions with main categorical variables.

Categorical variables n (%)		FM		RA		*p*
		Barcelona (*n*=67)	New York (*n*=16)	Barcelona (*n*=70)	New York (*n*=15)	
Sex	Female	67 (100%)	16 (100%)	51 (72.9%)	13 (86.7%)	>.001
	Male	0 (0%)	0 (0%)	19 (27.1%)	2 (13.3%)	
Attachment	Secure	4 (6%)	3 (18.8%)	9 (12.9%)	5 (33.3%)	.027
	Insecure	63 (94%)	13 (81.3%)	61 (87.1%)	10 (66.7%)	
Nationality	Hispanic	67 (100%)	0 (0%)	70 (100%)	0 (0%)	>.001
	American	0 (0%)	16 (100%)	0 (0%)	15 (100%)	

When the chi-square test for independence was repeated by comparing the pathologies in both countries, this time the results showed a statistically significant difference

concerning the insecure attachment, $\chi(3) = 9.144$, $p = .027$ (Table 36). The Phi and Cramer's V test values were 0.233, thus showing a small effect size, or strength of association, in this instance.

Relationships among clinical and attachment characteristics and influence on psychosocial dimensions

This section purports to explore the general aim of how clinical and attachment characteristics are related and influence the patients' psychosocial dimensions. Therefore, it consists of the multiple linear regression models performed in order to analyze the research data that fulfilled the conditions for multivariate tests. This also means that both American samples were not considered due to insufficient sample size; hence, these analyses could not encompass the cross-cultural factor of the study as initially intended.

Analyses were performed firstly with the predictor variable of attachment style and the FM-related criterion variables. Next, models were analyzed with attachment style and the RA-related dependent variables, followed by analyses of HADS' anxiety and depression as predictors of FM dependent variables first and then with RA outcome variables.

FM and attachment style

First, correlation and multiple regression analyses were conducted to examine the relationship between FM quality of life, measured through the SFIQ scores, and attachment style/scale as a potential predictor. The final model showed that the results were not significant and the adjusted R^2 was -.014 (see Table 37).

Table 37. Summary of the final model for SFIQ and CAA scale

	Model	R square	Adjusted R square	F change	Durbin-Watson
FM quality of life	1	.049	-.014	.778	1.995

*$p < .05$, **$p < .01$, ***$p < .001$

As Table 38 shows, FM quality of life was not significant in the model.

Table 38. Coefficients of the multiple linear regression analysis with FM quality of life as the dependent variable

	FM quality of life				
	R^2 = -.01	*B*	β	*t*	*p*
Step 1. Main effects	ΔR^2 = .049				
Constant		71.34		36.62	>.001
Low self-esteem, need of approval, and fear of rejection		2.79	.18	1.182	.242
Hostile resolution of conflict, rancor and possessiveness		-3.29	-.20	-1.397	.168
Emotional expressiveness and comfortableness with intimacy		-.44	-.03	-.234	.816
Emotional self-sufficiency and discomfort with intimacy		-1.44	-.10	-.697	.489

** p <.05, ** p <.01, *** p <.001*

The multiple regression model with the predictor produced significant results, as can be seen in Table 39, yielding an adjusted R^2 of .13. This constitutes a moderate adjusted coefficient of determination.

Table 39. Summary of the final model for PSS and CAA scale

	Model	R square	Adjusted R square	F change	Durbin-Watson
Pain intensity	1	.190	.125	2.934*	2.049

** p <.05, ** p <.01, *** p <.001*

Some of the Spanish attachment scales were significant, as shown in Table 40. Attachment styles explained 13% of the variance in pain intensity. The main effects increased the explanation of the model in a 19%. The dimensions with a specific significant weight were Emotional expressiveness and comfortableness with intimacy; and marginally as well Low self-esteem, need of approval, and fear of rejection and Emotional self-sufficiency and discomfort with intimacy. According to the β scores of these three attachment scales, when they increased, the pain intensity score also augmented. However, as can be observed, the

marginal significance indicates that these results must be understood with caution; thus, only Emotional expressiveness and comfortableness with intimacy must be understood as truly significant in this model.

Table 40. Coefficients of the multiple linear regression analysis with pain intensity as the dependent variable

	Pain intensity				
	$R^2 = .13$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 =$ 0.19*				
Constant		6.56		45.043	>.001
Low self-esteem, need of approval, and fear of rejection		.31	.26	1.717	.092
Hostile resolution of conflict, rancor and possessiveness		-.22	-.18	-1.163	.250
Emotional expressiveness and comfortableness with intimacy		.36	.30	2.349	.023
Emotional self-sufficiency and discomfort with intimacy		-.28	-.27	-1.856	.069

** p <.05, ** p <.01, *** p <.001*

As for pain interference, the final model summary produced significant results, shown in Table 41. The adjusted coefficient of determination produced was small, of .07.

Table 41. Summary of the final model for PIS and CAA scale

	Model	R square	Adjusted R square	F change	Durbin-Watson
Pain interference	1	.136	.074	2.201*	1.836

** p <.05, ** p <.01, *** p <.001*

Table 42 shows that one attachment style was significant, explaining 7% of the variance in pain interference. The main effects increased the explanation of the model in a 14%. The dimension with a specific significant weight was Low self-esteem, need of

approval, and fear of rejection. According to its β score, when it increased, the pain interference score also augmented.

Table 42. Coefficients of the multiple linear regression analysis with pain interference as the dependent variable

	Pain interference				
	$R^2 = .07$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 = 0.14$*				
Constant		6.77		34.954	>.001
Low self-esteem, need of approval, and fear of rejection		.65	.43	2.816	.007
Hostile resolution of conflict, rancor and possessiveness		-.22	-.13	-.910	.367
Emotional expressiveness and comfortableness with intimacy		.01	.01	.061	.952
Emotional self-sufficiency and discomfort with intimacy		-.29	-.20	-1.334	.187

*$p < .05$, **$p < .01$, ***$p < .001$

Regarding functional status, the final model summary did not offer significant results. The adjusted R^2 was -.04 (see Table 43).

Table 43. Summary of the final model for RAPID3 and CAA scale

	Model	R square	Adjusted R square	F change	Durbin-Watson
Functional status	1	.029	-.040	.419	2.075

*$p < .05$, **$p < .01$, ***$p < .001$

As Table 44 shows, functional status was not significant in the model and therefore none of the main effects contributed any notable changes, $F(4, 57) = .419$, $p = .794$.

Table 44. Coefficients of the multiple linear regression analysis with functional status as the dependent variable

	Functional status				
	R^2 = -.04	*B*	β	*t*	*p*
Step 1. Main effects	ΔR^2 = 0.03				
Constant		18.75		32.085	>.001
Low self-esteem, need of approval, and fear of rejection		.79	.18	1.070	.289
Hostile resolution of conflict, rancor and possessiveness		-.64	-.13	-.862	.392
Emotional expressiveness and comfortableness with intimacy		-.29	-.07	-.480	.633
Emotional self-sufficiency and discomfort with intimacy		-.37	-.09	-.598	.552

** p <.05, ** p <.01, *** p <.001*

When symptom checklist review of systems was analyzed, the final model summary did not produce significant results. The adjusted R^2 was .1 (see Table 45). As can be observed, the Durbin-Watson *d* is below the critical value of 1.5, therefore indicating that there might be first order linear auto-correlation of the residuals in the multiple linear regression data.

Table 45. Summary of the final model for ROS and CAA scale

	Model	R square	Adjusted R square	F change	Durbin-Watson
Symptom checklist review of systems	1	.071	.006	1.097	1.325

** p <.05, ** p <.01, *** p <.001*

Table 46 shows that symptom checklist review of systems was not significant in the model and none of the main effects added any notable changes, $F(4, 57) = 1.097$, $p = .367$.

Table 46. Coefficients of the multiple linear regression analysis with symptom checklist review of systems as the dependent variable

	Symptom checklist review of systems				
	$R^2 = .01$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 = 0.07$				
Constant		27.43		21.733	>.001
Low self-esteem, need of approval, and fear of rejection		2.94	.29	1.835	.072
Hostile resolution of conflict, rancor and possessiveness		-1.53	-.15	-.943	.350
Emotional expressiveness and comfortableness with intimacy		.97	.10	.774	.442
Emotional self-sufficiency and discomfort with intimacy		-.83	-.09	-.621	.537

** p <.05, ** p <.01, *** p <.001*

RA and attachment style

First, correlation and multiple regression analyses were performed to examine the relationship between RA quality of life, measured through the SF-36 scores, and attachment style/scale as a potential predictor. When examining the mental component of RA quality of life first, the final model showed that the results were significant and the adjusted R^2 was .21 (see Table 47).

Table 47. Summary of the final model for MCS and CAA scale

	Model	R square	Adjusted R square	F change	Durbin-Watson
Mental component of RA quality of life	1	.263	.205	4.541**	2.058

** p <.05, ** p <.01, *** p <.001*

Table 48 displays that there was one Spanish attachment scale that was significant, explaining 21% of the variance in the mental component of RA quality of life. The main effects increased the explanation of the model in a 26%. The variable with a specific significant weight was Low self-esteem, need of approval, and fear of rejection. According to

its β score, when this attachment scale increased, the mental component of RA quality of life decreased.

Table 48. Coefficients of the multiple linear regression analysis with the mental component of RA quality of life as the dependent variable

	Mental component of RA quality of life				
	$R^2 = .21$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 =$ 0.26**				
Constant		47.10		35.861	>.001
Low self-esteem, need of approval, and fear of rejection		-4.62	-.39	-3.022	.004
Hostile resolution of conflict, rancor and possessiveness		-1.18	-.10	-.710	.481
Emotional expressiveness and comfortableness with intimacy		-.61	-.06	-.483	.631
Emotional self-sufficiency and discomfort with intimacy		-2.36	-.18	-1.374	.176

** p <.05, ** p <.01, *** p <.001*

As per the physical component of the RA quality of life, the final model summary yielded significant results, shown in Table 49. The adjusted coefficient of determination was small, of .11.

Table 49. Summary of the final model for PCS and CAA scale

	Model	R square	Adjusted R square	F change	Durbin-Watson
Physical component of RA quality of life	1	.167	.106	2.749*	1.882

** p <.05, ** p <.01, *** p <.001*

Table 50 shows that there was one Spanish attachment style that was significant in the model, explaining 11% of the variance in the physical component of RA quality of life. The main effects increased the explanation of the model in a 17%. The dimension with a specific

significant weight was Emotional expressiveness and comfortableness with intimacy. According to its β score, when this attachment scale increased, the physical component of RA quality of life decreased.

Table 50. Coefficients of the multiple linear regression analysis with the physical component of RA quality of life as the dependent variable

	Physical component of RA quality of life				
	$R^2 = .11$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 = 0.17$*				
Constant		36.57		25.846	>.001
Low self-esteem, need of approval, and fear of rejection		-2.40	-.21	-1.375	.175
Hostile resolution of conflict, rancor and possessiveness		-.21	-.02	-.132	.896
Emotional expressiveness and comfortableness with intimacy		-3.55	-.30	-2.424	.019
Emotional self-sufficiency and discomfort with intimacy		-1.76	-.14	-.962	.340

** p <.05, ** p <.01, *** p <.001*

The multiple regression model with the predictor produced significant results in the case of pain intensity, as can be seen in Table 51, resulting in an adjusted R^2 of .08. This constitutes a small adjusted coefficient of determination.

Table 51. Summary of the final model for PSS and CAA scale

	Model	R square	Adjusted R square	F change	Durbin-Watson
Pain intensity	1	.143	.079	2.215*	2.258

** p <.05, ** p <.01, *** p <.001*

Table 52 shows that there was a tendency towards statistical significance in one attachment style, explaining 8% of the variance in pain intensity. The main effects increased the explanation of the model in a 14%, and the dimension with a marginally significant weight was Emotional expressiveness and comfortableness with intimacy. According to its β

score, when it increased, the pain intensity score also augmented. Again, this result must be interpreted cautiously, due to a tendency instead of real significance.

Table 52. Coefficients of the multiple linear regression analysis with pain intensity as the dependent variable

	Pain intensity				
	$R^2 = .08$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 = 0.14$*				
Constant		3.50		11.249	>.001
Low self-esteem, need of approval, and fear of rejection		.38	.15	1.005	.319
Hostile resolution of conflict, rancor and possessiveness		.27	.11	.736	.465
Emotional expressiveness and comfortableness with intimacy		.61	.25	1.956	.056
Emotional self-sufficiency and discomfort with intimacy		.50	.16	1.186	.241

** p <.05, ** p <.01, *** p <.001*

As for pain interference, the final model summary also produced significant results, as shown in Table 53. The adjusted coefficient of determination was of .22, which encompasses a moderate magnitude.

Table 53. Summary of the final model for PIS and CAA scale

	Model	R square	Adjusted R square	F change	Durbin-Watson
Pain interference	1	.277	.222	5.074**	2.177

** p <.05, ** p <.01, *** p <.001*

Table 54 displays that, once more, only one Spanish attachment scale was significant in the model, explaining 22% of the variance in pain interference. The main effects increased the explanation of the model in a 28%, and the variable with a specific significant weight was Low self-esteem, need of approval, and fear of rejection. Its β score denotes that when it increased, pain interference also rose.

Table 54. Coefficients of the multiple linear regression analysis with pain interference as the dependent variable

	Pain interference				
	$R^2 = .22$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 =$ 0.28**				
Constant		3.81		10.991	>.001
Low self-esteem, need of approval, and fear of rejection		1.14	.36	2.741	.008
Hostile resolution of conflict, rancor and possessiveness		.34	.12	.835	.408
Emotional expressiveness and comfortableness with intimacy		.43	.15	1.235	.222
Emotional self-sufficiency and discomfort with intimacy		.72	.20	1.548	.128

** p <.05, ** p <.01, *** p <.001*

When functional status in RA was analyzed, the final model summary also produced significant results, as seen in Table 55. The adjusted R^2 was of .33, which constitutes a large adjusted coefficient of determination.

Table 55. Summary of the final model for RAPID3 and CAA scale

	Model	R square	Adjusted R square	F change	Durbin-Watson
Functional status	1	.378	.330	7.892***	2.041

** p <.05, ** p <.01, *** p <.001*

Table 56 shows that three Spanish attachment styles were significant in the model and explained 33% of the variance in functional status. The scales were Low self-esteem, need of approval, and fear of rejection; Emotional expressiveness and comfortableness with intimacy; and Emotional self-sufficiency and discomfort with intimacy. The main effects increased the explanation of the model a 38%. The β scores of the three dimensions with a specific significant weight all imply that when increased, functional status also augmented.

Table 56. Coefficients of the multiple linear regression analysis with functional status as the dependent variable

	Functional status				
	$R^2 = .33$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 =$ 0.38***				
Constant		11.98		15.176	>.001
Low self-esteem, need of approval, and fear of rejection		2.89	.38	3.103	.003
Hostile resolution of conflict, rancor and possessiveness		.52	.08	.580	.564
Emotional expressiveness and comfortableness with intimacy		2.56	.36	3.278	.002
Emotional self-sufficiency and discomfort with intimacy		2.45	.27	2.302	.025

** p <.05, ** p <.01, *** p <.001*

The final model summary for symptom checklist review of systems, in Table 57, also produced significant results. The adjusted coefficient of determination was of 24%, which is of moderate magnitude.

Table 57. Summary of the final model for ROS and CAA scale

	Model	R square	Adjusted R square	F change	Durbin-Watson
Symptom checklist review of systems	1	.293	.238	5.292***	2.492

** p <.05, ** p <.01, *** p <.001*

Table 58 displays that two Spanish attachment scales were significant in the model, explaining 24% of the variance in symptom checklist review of systems. The main effects increased a 29% the explanation of the model, and the variables with a specific significant weight were Emotional expressiveness and comfortableness with intimacy, and Emotional self-sufficiency and discomfort with intimacy. Both β scores denote that when these dimensions increased, symptom checklist review of systems also increased.

Table 58. Coefficients of the multiple linear regression analysis with symptom checklist review of systems as the dependent variable

	Symptom checklist review of systems				
	$R^2 = .24$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 = 0.29$***				
Constant		12.40		13.479	>.001
Low self-esteem, need of approval, and fear of rejection		1.00	.12	.926	.359
Hostile resolution of conflict, rancor and possessiveness		1.58	.19	1.370	.177
Emotional expressiveness and comfortableness with intimacy		2.75	.37	3.074	.003
Emotional self-sufficiency and discomfort with intimacy		2.70	.29	2.223	.031

** p <.05, ** p <.01, *** p <.001*

FM and HADS-measured anxiety and depression

First, correlation and multiple regression analyses were conducted to examine the relationship between FM quality of life, measured through the SFIQ scores, and anxiety and depression as potential predictors. When analyzing FM quality of life first, the final model showed that the results were significant and the adjusted R^2 was .42, and therefore of a large magnitude (see Table 59).

Table 59. Summary of the final model for SFIQ and HADS scales

	Model	R square	Adjusted R square	F change	Durbin-Watson
FM quality of life	1	.443	.423	23.034***	1.760

** p <.05, ** p <.01, *** p <.001*

Both HADS scales of anxiety and depression were significant in the model (see Table 60), explaining 42% of the variance in FM quality of life. The main effects increased the explanation of the model in a 44%. Both variables of anxiety and depression significantly

weighed, and their β scores show that when these dimensions increased, FM quality of life also augmented.

Table 60. Coefficients of the multiple linear regression analysis with FM quality of life as the dependent variable

	FM quality of life				
	$R^2 = .42$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 =$ 0.44***				
Constant		69.20		48.350	>.001
Anxiety		5.54	.39	3.191	.002
Depression		4.90	.35	2.836	.006

** p <.05, ** p <.01, *** p <.001*

The final model summary for pain intensity produced significant results, as shown in Table 61. The adjusted coefficient of determination was moderate, of .18.

Table 61. Summary of the final model for PSS and HADS scales

	Model	R square	Adjusted R square	F change	Durbin-Watson
Pain intensity	1	.202	.182	10.110***	2.234

** p <.05, ** p <.01, *** p <.001*

Table 62 displays that only anxiety was significant in the model, explaining 18% of the variance in pain intensity. The main effects increased the explanation by 20%, and the dimension with a specific significant weight was anxiety. Its positive β score implies that when anxiety increased, pain intensity did as well.

Table 62. Coefficients of the multiple linear regression analysis with pain intensity as the dependent variable

	Pain intensity				
	$R^2 = .18$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 = 0.20$***				
Constant		5.88		28.205	>.001
Anxiety		.67	.34	2.494	.015
Depression		.27	.15	1.084	.281

** p <.05, ** p <.01, *** p <.001*

As per pain interference, the final model also yielded significant results (see Table 63). The adjusted coefficient of determination was large, of .33.

Table 63. Summary of the final model for PIS and HADS scales

	Model	R square	Adjusted R square	F change	Durbin-Watson
Pain interference	1	.346	.329	21.130***	1.839

** p <.05, ** p <.01, *** p <.001*

Table 64 below shows that both scales were significant in the model, explaining 33% of the variance in pain interference. The main effects increased the explanation of the model in a 35% and the dimensions with a specific significant weight were anxiety and depression. Both β scores were positive, entailing that when anxiety and depression increased, pain interference did as well.

Table 64. Coefficients of the multiple linear regression analysis with pain interference as the dependent variable

	Pain interference				
	$R^2 = .33$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 = 0.35$***				
Constant		5.78		25.731	>.001
Anxiety		.70	.30	2.421	.018
Depression		.77	.35	2.827	.006

** p <.05, ** p <.01, *** p <.001*

When looking into the relationship with functional status, the final model summary also yielded significant results, with a large adjusted R^2 of .28 (see Table 65).

Table 65. Summary of the final model for RAPID3 and HADS scales

	Model	R square	Adjusted R square	F change	Durbin-Watson
Functional status	1	.302	.284	17.270***	1.824

** p <.05, ** p <.01, *** p <.001*

Table 66 shows that only one scale was significant in the model, explaining a 28% of the variance in functional status. The main effects increased the explanation of the model in a 30% and the dimension with a specific significant weight was anxiety. Its β score denoted that when anxiety increased, functional status increased as well.

Table 66. Coefficients of the multiple linear regression analysis with functional status as the dependent variable

	Functional status				
	$R^2 = .28$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 = 0.30$***				
Constant		16.67		30.099	>.001
Anxiety		2.41	.43	3.393	.001
Depression		.84	.16	1.260	.211

** p <.05, ** p <.01, *** p <.001*

As for the symptom checklist review of systems, the final model summary produced significant results and a moderate adjusted coefficient of determination of .18, as can be seen in Table 67.

Table 67. Summary of the final model for ROS and HADS scales

	Model	R square	Adjusted R square	F change	Durbin-Watson
Symptom checklist review of systems	1	.196	.176	9.622***	1.746

** p <.05, ** p <.01, *** p <.001*

Table 68 shows that only one scale was significant in the model and it explained 18% of the variance in the symptom checklist review of systems. The main effects increased the explanation in a 20%. The variable that significantly weighed was anxiety, with a β score indicating that when this dimension increased, so did the symptom checklist review of systems.

Table 68. Coefficients of the multiple linear regression analysis with symptom checklist review of systems as the dependent variable

	Symptom checklist review of systems				
	$R^2 = .18$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 = 0.20$***				
Constant		25.40		23.157	>.001
Anxiety		3.04	.30	2.169	.033
Depression		1.80	.19	1.357	.179

** p <.05, ** p <.01, *** p <.001*

RA and HADS-measured anxiety and depression

Lastly, correlation and multiple regression analyses were conducted to examine the relationship between RA quality of life, measured through the SF-36 mental and physical scores, and anxiety and depression as potential predictors. When analyzing the mental component of RA quality of life first, the final model summary produced significant results. The adjusted R^2 was .48, and therefore of a large magnitude (see Table 69).

Table 69. Summary of the final model for MCS and HADS scales

	Model	R square	Adjusted R square	F change	Durbin-Watson
Mental component of RA quality of life	1	.497	.478	26.683***	2.404

** p <.05, ** p <.01, *** p <.001*

Table 70 shows that both HADS scales were significant in the model, explaining 48% of the mental component of RA quality of life. The main effects increased the explanation of the model in a 50%. The dimensions with a specific significant weight were anxiety and depression, with β scores indicating that when anxiety and depression increased, the mental component of RA quality of life decreased.

Table 70. Coefficients of the multiple linear regression analysis with the mental component of RA quality of life as the dependent variable

	Mental component of RA quality of life				
	$R^2 = .48$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 = 0.50$***				
Constant		43.91		33.645	>.001
Anxiety		-6.20	-.42	-3.898	>.001
Depression		-6.05	-.41	-3.866	>.001

*$p < .05$, **$p < .01$, ***$p < .001$

As per the physical component of the RA quality of life, the final model summary yielded significant results as well. The adjusted coefficient of determination was .10, that is, of small magnitude (see Table 71).

Table 71. Summary of the final model for PCS and HADS scales

	Model	R square	Adjusted R square	F change	Durbin-Watson
Physical component of RA quality of life	1	.126	.095	4.109*	1.916

*$p < .05$, **$p < .01$, ***$p < .001$

Table 72 below shows that there was one scale that was significant in the model, explaining 10% of the variance in the physical component of RA quality of life. The main effects increased the explanation of the model by 13%. The dimension that was specifically significant was depression, with a negative β score indicating that when depression increased, the physical component of RA quality of life was reduced.

Table 72. Coefficients of the multiple linear regression analysis with the physical component of RA quality of life as the dependent variable

	Physical component of RA quality of life				
	$R^2 = .10$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 = 0.13$*				
Constant		33.98		19.773	>.001
Anxiety		-.17	-.01	-.087	.931
Depression		-5.43	-.35	-2.422	.019

** p <.05, ** p <.01, *** p <.001*

As for pain intensity, the final model summary also yielded significant results, shown in Table 73. The adjusted coefficient of determination was moderate, of .16.

Table 73. Summary of the final model for PSS and HADS scales

	Model	R square	Adjusted R square	F change	Durbin-Watson
Pain intensity	1	.177	.157	8.631***	2.116

** p <.05, ** p <.01, *** p <.001*

Table 74 shows that one scale was significant in the model, explaining 16% of the variance in pain intensity. The main effects increased the explanation by 18%. The variable with a specific significant weight was depression, with a positive β score, entailing that when this dimension increased, so did pain intensity.

Table 74. Coefficients of the multiple linear regression analysis with pain intensity as the dependent variable

	Pain intensity				
	$R^2 = .16$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 = 0.18$***				
Constant		4.25		14.845	>.001
Anxiety		.37	.13	1.091	.278
Depression		1.032	.34	2.837	.006

** p <.05, ** p <.01, *** p <.001*

When pain interference was analyzed, the final model summary produced significant results as well (Table 75). The adjusted coefficient of determination was large, as it was .45.

Table 75. Summary of the final model for PIS and HADS scales

	Model	R square	Adjusted R square	F change	Durbin-Watson
Pain interference	1	.460	.447	34.968***	2.142

** p <.05, ** p <.01, *** p <.001*

Table 76 shows that both scales were significant in the model, explaining 45% of the variance in pain interference. The main effects increased the explanation by 46%. The dimensions that weighed significantly were anxiety and depression, both with positive β scores, indicating that as they increased, pain interference augmented likewise.

Table 76. Coefficients of the multiple linear regression analysis with pain interference as the dependent variable

	Pain interference				
	$R^2 = .45$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 = 0.46$***				
Constant		4.80		17.873	>.001
Anxiety		.67	.20	2.000	.049
Depression		1.93	.55	5.513	>.001

** p <.05, ** p <.01, *** p <.001*

Examining functional status also yielded significant results, as presented in the final model summary in Table 77. The adjusted R^2 was of .33, showing a large magnitude.

Table 77. Summary of the final model for RAPID3 and HADS scales

	Model	R square	Adjusted R square	F change	Durbin-Watson
Functional status	1	.347	.331	21.012***	1.790

** p <.05, ** p <.01, *** p <.001*

Table 78 displays that both scales were significant in the model, explaining a 33% of the variance in functional status. The main effects increased the explanation in a 35%. The variables with a specific significant weight, anxiety and depression, also had positive β scores, indicating that as they increased, functional status augmented likewise.

Table 78. Coefficients of the multiple linear regression analysis with functional status as the dependent variable

	Functional status				
	$R^2 = .33$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 = 0.35$***				
Constant		13.71		18.604	>.001
Anxiety		1.92	.23	2.181	.032
Depression		3.77	.43	4.013	>.001

*$p < .05$, **$p < .01$, ***$p < .001$

As per the symptom checklist review of systems, the final model summary produced significant results (see Table 79 below). The adjusted coefficient of determination was large, of .44.

Table 79. Summary of the final model for ROS and HADS scales

	Model	R square	Adjusted R square	F change	Durbin-Watson
Symptom checklist review of systems	1	.453	.440	32.774***	2.042

*$p < .05$, **$p < .01$, ***$p < .001$

Table 80 shows that both scales were significant in the model, explaining 44% of the variance in the symptom checklist review of systems. The main effects increased the explanation of the model by 45%. The dimensions that weighed significantly were anxiety and depression, both with positive β scores, denoting that when they increased, the symptom checklist review of systems increased as well.

Table 80. Coefficients of the multiple linear regression analysis with symptom checklist review of systems as the dependent variable

	Symptom checklist review of systems				
	$R^2 = .44$	*B*	β	*t*	*p*
Step 1. Main effects	$\Delta R^2 = 0.45$***				
Constant		17.86		18.886	>.001
Anxiety		3.76	.33	3.327	.001
Depression		5.40	.44	4.485	>.001

** p <.05, ** p <.01, *** p <.001*

Discussion

The results of the present study offer several interesting findings concerning the objectives it pursued. To briefly review, the purpose of this research was to depict the psychological and sociodemographic profiles of FM and RA and contrast them via self-report measures; to highlight how the attachment patterns differed, but also to analyze and contrast the cultural factor jointly in both medical conditions. It aimed to explore how the clinical and attachment characteristics are related and influence the psychosocial dimensions of HRQoL and functional status.

This last chapter presents a discussion of the results, while accounting for the theoretical paradigms and empirical findings argued in the introduction of the dissertation. To this end, the chapter is divided into sections arranged by a brief overview of the findings, an assessment of the extent to which the hypotheses were supported, paired with a corresponding interpretation of the results. Lastly, final conclusions are advanced, with a focus on impact, strengths and limitations, and some implications for furthering practice and research in the area of psychological correlates of FM and RA.

Summary of findings

The FM sample was, as a whole, distinctly more depressed, more anxious, had more pain intensity and interference, worse functional status —as well as its components—, and had more somatic symptoms than the RA control sample. There was also a clear association between the female gender and both medical conditions, and between insecure attachment and being ill, especially in the Barcelona sample. However, being securely or insecurely attached and nationality yielded no relationships with either one of both conditions, as there were no statistically significant differences in these two instances.

Results indicated that the number of years since the diagnosis was established was linked to FM quality of life, and the physical function component of functional status. Additionally, increased comorbidities were associated with nearly all other variables; that is, with increased depression, pain intensity and interference, worse functional status, more somatic symptoms, increased anxiety and depression, older age, and poorer FM quality of life and functional status' physical function and pain. Patient age was not further associated with other outcomes, but all the rest of the studied characteristics in the sample were related amongst each other. Furthermore, a lower educational level was linked to a number of different variables: increased years since diagnosis, higher pain severity and interference and somatic symptoms, worse functional status, and older age. Moreover, and still within the overall sample, insecure attachment was clearly related to functional status and its components of patient global estimate of status and pain; and gender was related as well to functional status and its three components of physical function, pain, and patient global estimate of status.

Exploratory analyses of adult attachment styles showed, in the Spanish sample, that there were clear differences regarding depression, anxiety and depression, pain interference, and somatic symptoms: the hostile fearful pattern had the highest levels, followed by the preoccupied style, then dismissing attachment, and lastly the secure subtype. In the case of functional status' pain, there were also statistical differences, but it was the preoccupied style who had the highest levels, followed by hostile fearful attachment, then dismissing, and lastly the secure style. Functional status displayed a tendency of statistical difference amongst

attachment styles, but as such, was not properly significant. The New York sample also showed no differences in relation to attachment style groups —only a tendency in the case of anxiety. Yet when comparing overall secure and insecure attachment, increased depression, anxiety and depression, somatic symptoms, and functional status, as well as its pain component, were associated to being insecurely attached. Looking specifically into the attachment questionnaire scales highlighted that RA patients scored significantly lower than FM patients in the Barcelona sample in Low self-esteem, need of approval and fear of rejection; however, both American adult attachment questionnaires did not differ between both medical conditions. When questionnaire scales from Barcelona and New York were compared to the rest of the study key variables, in Barcelona most were linked, excepting the Emotional expressiveness and comfortableness with intimacy, which wasn't associated to any pathological variable. In the instance of New York, very scarce relationships were found, apart from the association between scoring low in the secure scale and scoring high in pathological variables.

When comparing both nationalities, there were no differences found between FM and RA with regards to education level, age, and comorbidities. However, both FM samples were generally more anxious and depressed and had the highest pain-related outcomes and somatic symptoms; but the expected differences in BDI depression, on one hand, and functional status and pain interference, on the other, between the Barcelona RA and the New York FM samples, and both New York samples, respectively, were not found. Also, insecure attachment was notably associated with suffering from FM or RA, particularly in Barcelona, as well as the female gender and both medical conditions were related.

As per the explanation in the Barcelona samples of the psychosocial and clinical variables' behavior via attachment and anxiety and depression, attachment was found to predict pain-related outcomes in FM; and RA quality of life, pain-related variables —only a tendency was observed in pain severity—, functional status, and somatic symptoms in RA. Furthermore, in FM, changes in quality of life and pain interference were explained by anxiety and depression; whereas pain intensity, functional status, and somatic symptoms were predicted only by anxiety. In RA, the mental component of quality of life, pain interference, functional status, and somatic symptoms were predicted by both anxiety and depression;

while the physical component of quality of life and pain intensity were predicted only by depression.

Testing of hypotheses

The study was designed to test the following hypotheses:

Hypotheses	General aims
	I. To determine the psychological and sociodemographic characteristics of FM and RA patients.
H1	that both samples would be predominantly female, the FM one in particular
H2	that the demographic, clinical, and psychosocial profile of all patients would suggest a deteriorated health status due to the impact of chronic and/or recurrent pain
	II. To compare the psychological profiles of FM and RA.
H3	comorbidities and somatic symptoms, expressed in the RDCI and the symptom checklist review of systems respectively, would be higher in the FM sample than in the RA group
H4	that depression levels would be increased and within clinical categories in FM
H5	the expression of pain would be more pronounced in the FM sample than in RA
H6	functional status and quality of life scores would support the existence of more impairment or deterioration in FM
	III. To establish adult attachment in FM and RA patients according to the secure, fearful, preoccupied, and dismissing styles.
H7	that there would be a predominance of insecure attachment in FM in comparison to RA patients, which would mainly be securely attached
H8	that there would be more specific associations between severe FM and RA on one hand, and the different insecure attachment patterns on the other hand
H9	FM patients would show a more notable prevalence of the hostile fearful/fearful and the dismissing subtypes in contrast to RA patients
H10	that patients in New York would show a higher prevalence of dismissing attachment in relation to the Spanish samples
	IV. To analyze and contrast the cultural factor jointly in both medical conditions.
H11	depression and anxiety would be reported differently according to the sample geographical origin
H12	psychosocial dimensions, and pain interference with daily activities due to its intimate relationship with quality of life, would be expressed as more deteriorated in Barcelona than in New York due to cultural influence
	V. To explore how clinical and attachment characteristics are related and influence the patients' psychosocial dimensions.
H13	pain intensity and interference would be highly associated with depression, anxiety, and psychosocial dimensions

Hypotheses	General aims
H14	that overall insecure attachment would have a negative impact on health status as reported through the clinical and psychosocial variables
H15	FM severity, according to quality of life and functional status levels, would be more related to the insecure subtypes than in the case of RA
H16	that depression would explain pain and illness severity, accounted for with quality of life and functional status scores

Psychological characteristics of FM and RA patients and comparison of both profiles

One of the aims of the study was to describe the sociodemographic, clinical, and psychosocial traits of FM and RA patients in order to elaborate and then compare the emerging psychological profiles. Sociodemographic characteristics will be developed first, followed by the clinical and then the psychosocial variables, and lastly, a comparison of profiles.

H1. Both samples will be predominantly female, especially FM patients.

H2. The demographic, clinical, and psychosocial profile of all patients will suggest a deteriorated health status due to the impact of chronic and/or recurrent pain.

In general, participants had received a secondary or university level of education, but in Barcelona most patients had a primary education level. Additionally, in Barcelona, more than half of both illness groups were employed or retired, while in New York, only half shared these work statuses. These findings could be stressing the relationship that tends to appear between a disadvantaged educational and employment status and chronic pain (Grol-Prokopczyk, 2017; Gupta et al., 2007). Also, the employment status could be a culturally differential indicator of how chronic pain influences all aspects of life, including work, quality of life, and functional ability (Gureje et al., 1998; Katz, 2002), thereby reducing physical, mental, and social wellbeing (Becker et al., 1997). However, taking into consideration the merely descriptive and cross-sectional nature of these data, it is impossible to know if the dissimilarity in employment status observed in the percentages between

Barcelona and New York might also be expressing the impact of different government policies regarding disability pensions.

Additionally, an ample majority were female, thus confirming the **first research hypothesis** and coinciding with the prevalence and gender ratios in both medical conditions (Carmona et al., 2010; Silman & Pearson, 2002; Vincent et al., 2013). In Barcelona, most patients were in a relationship and co-living, especially FM individuals; whereas in New York, almost half the sample were in the same marital status but the rest were living alone. Despite this observation being limited by its purely descriptive nature, in which only percentages have been considered, it deserves attention for its relationship with HRQoL, or quality of life. That is, quality of life relies on the fundamental wellbeing dimensions of physical, psychological-cognitive, and social. The latter aspects are related to isolation and self-esteem and are paramount due to the social role in chronic disease, which in itself has been found to be deteriorating (Brorsson et al., 2002; Ruta et al., 1994). Further, evidence suggests that patients who receive considerable social support from families and friends, and from their partners in particular, have a better prognosis and less disability (Fitzpatrick, Newman, Archer, & Shipley, 1991; Kraaimaat, Van Dam-Baggen, & Bijlsma, 1995). Certain clinical manifestations, such as pain or fatigue, are more frequent in individuals who do not have social support (Neugebauer & Katz, 2004; Riemsma et al., 1998), and, in general terms, social support has been repeatedly found to be a mediator of illness and is beneficial to a variety of health outcomes (S. Cohen & Wills, 1985; House et al., 1988). Specifically, among women with RA, pain may be the strongest predictor for decreased wellbeing, but social support of rheumatoid patients has been found to have a potential moderating effect (Jakobsson & Hallberg, 2002). In addition, the observation that especially FM patients in Barcelona were living with a couple could be contradicting the research showing that chronic fatigue syndrome and FM patients may have less social support, and perceive more negative social relationships, than the general population (Anderson & Ferrans, 1997; Davis et al., 2001).

Mention must be made to the means of the number of years since diagnosis, which were higher in both RA samples than in the FM ones, without performing statistical tests. In the social care system, the lack of a clear-cut diagnosis can be a serious obstacle in finding treatment, and due to reduced functional ability and possible loss of income, these patients

may often feel like a burden to their families (Allcock et al., 2007; Karoly & Ruehlman, 2006; Kowal et al., 2012). Indeed, the relationship between the years since diagnosis and the previously explained impact of social support on a pain condition lies in the fact that the invisible nature of such an illness contributes to the difficulties of being properly understood by healthcare professionals, the social care system, and the patient's social network (Allcock et al., 2007). However, the difference observed in the number of years passed since the diagnosis between both FM and RA samples could be owing to the particular diagnosis of FM, that has needed to be confirmed overtime with radical changes in its conceptualization, as opposed to RA.

Moreover, clinical variables showed that participants didn't have many comorbidities, as well as had severe FM —or poor HRQoL— and poor RA mental and physical quality of life (R. Bennett, 2005; Vilagut et al., 2005). As per pain-related variables, both means in all study participants were similarly in the equator of the spectrum, ranging from 0 to 10. Regarding BDI depression levels, the American FM sample showed subjects with moderate and severe depression, but the RA sample was predominantly not depressed and had a very low presence of mildly depressed patients. On the other hand, in the Spanish groups, both had all types of clinical and non-clinical depression but in the FM sample there was a much more notable predominance of clinical depression, whereas the RA sample was predominantly not depressed. Overall, study participants had a BDI score mean which indicated mild depression. As for the general HADS anxiety and depression means, anxiety was slightly over the cutoff score, indicating possible levels of clinical relevance, but the depression mean was well within normal and therefore non-clinical limits. Overall, these findings should be considered in its clinical relevance due to the fact that symptoms of depression or anxiety are frequently manifested, particularly at the beginning of the disease, and should not be underestimated (Suurmeijer et al., 2001), as higher mortality has been observed in patients with depression (Ang et al., 2005). Furthermore, patients also showed an increased mean of somatic symptoms and a highly severe functional status mean, all of which supports previous findings that high levels of illness behavior and somatic symptoms have an enhanced risk of developing chronic widespread pain (McBeth et al., 2001). Also, these general findings are consistent with current disability models, which acknowledge the buffering and exacerbating roles of demographic, physiological, psychological, social, and environmental factors

regarding poor outcomes for chronic physical conditions (Verbrugge & Juarez, 2006). The global findings thus far also confirm the study's **second hypothesis**, according to which the demographic, clinical, and psychosocial profile of all patients supports the idea of a generally deteriorated health status, likely due to the impact of chronic and/or recurrent pain.

H3. Comorbidities and somatic symptoms, expressed in the RDCI and the symptom checklist review of systems respectively, will be higher in the FM sample than in the RA group.

H4. Depression levels will be higher and within clinical categories in FM.

H5. The expression of pain will be more pronounced in the FM sample than in the RA sample.

H6. Functional status and quality of life scores will support the existence of more impairment or deterioration in FM.

Prior to comparing these two profiles that have been established, Pearson correlation coefficients were obtained to assess relationships between the study variables in the entire study sample. Education was lowly and inversely correlated to years since diagnosis, pain intensity and interference, functional status, somatic symptoms, age, and all three components of functional status (physical function, pain, and patient's global estimate of status), as well as positively correlated with the physical component of RA quality of life. This indicates a slight protective effect of sorts attributable to having a high educational level, particularly in the highest correlations with clinical variables, such as pain intensity, functional status, and RA physical quality of life. This is consistent with longitudinal epidemiological studies that have shown in chronic pain and other somatic symptoms a history of childhood abuse and traumas, low educational level, social isolation, depression, and anxiety (Nicholl et al., 2009). Additionally, in the pain literature there are many reports of an association between low educational level and longer duration and/or higher recurrence of back pain, and the link between many health-related events and education supports the idea that a low socioeconomic status increases vulnerability or impairs adaptation to illness (Dionne et al., 2001).

Moreover, the number of years that had passed since the diagnosis lowly correlated and was associated positively to FM quality of life and the physical function component of

functional status. Comorbidities were associated to depression, pain intensity and interference, functional status, somatic symptoms, anxiety and depression, age, FM quality of life, and the functional status' physical function and pain. This generalized association with comorbidities could be contemplated from the perspective of the new DSM-5's SSD: it would corroborate the idea that FM is a pain-predominant somatic symptom disorder (Wolfe et al., 2014) and that chronic musculoskeletal pain patients of any etiology are prone to heightened emotional, cognitive, and behavioral responses to chronic pain due to centrally amplified pain and pain-related SSD (Crofford, 2015b).

Indeed, the rest of the correlations were ranging from moderate to high. For instance, depression was moderately associated to pain intensity and functional status' pain, and also inversely to RA physical and mental quality of life, in the sense that when depression increased, RA quality of life was reduced. It was also associated to all three components of functional status; that is, highly to physical function, moderately to pain, and moderately to patient's global estimate of status. Depression was highly linked to pain interference, functional status, somatic symptoms, anxiety and depression, and FM quality of life. It must be noted that the BDI was designed to measure the level of depression in patients with that pre-established diagnosis and its validity of assessing depression in medical conditions has been contested, as several items can be attributed to the medical condition and spuriously increase the sum score of the questionnaire (Knaster et al., 2016).

Further, pain severity was strongly associated to pain interference, functional status, somatic symptoms, FM quality of life, and to all three components of functional status, as well as highly but not as intensely to anxiety and depression. Pain severity also strongly but inversely correlated to RA physical quality of life, meaning when pain severity increased, RA physical quality of life decreased. It lowly and inversely also correlated with RA mental quality of life. On the other hand, pain interference was highly associated to functional status, somatic symptoms, anxiety and depression, FM quality of life, and all three components of functional status. It was also inversely associated with moderation to RA mental quality of life and to a stronger degree with RA physical quality of life. Arguably, the fact that both pain-related outcomes are so intimately related to all other clinical and psychosocial dimensions is due to the fact that chronic pain results from intricate interactions between biological and psychosocial factors, and regardless of if it has an underlying organic cause, it

will pervasively have physiological and psychological consequences (Flor & Hermann, 2004). Following this idea, functional status was also strongly related to somatic symptoms, anxiety and depression, FM quality of life, and particularly to all its three components, It was also inversely and strongly associated to RA physical quality of life and moderately to RA mental quality of life. Additionally, somatic symptoms highly correlated to anxiety and depression, FM quality of life, and all three components of functional status. It inversely correlated to a stronger degree to the physical component of RA quality of life than to the mental one. These findings are consistent with chronic pain research, which suggests a negative link with pain, psychological status, disability, and quality of life that has been found independent from depression (Outcalt et al., 2015).

HADS anxiety showed a high correlation with its own other measurement of depression, FM quality of life, and patient's global estimate of status, and also inversely to RA mental quality of life. It was lowly associated to RA physical quality of life, and moderately to physical function and pain. As for HADS depression, it was highly associated to FM quality of life, physical function, and patient's global estimate of status, as well as inversely to RA mental quality of life. It was moderately associated to pain and inversely as well to RA physical quality of life. These bonds between anxiety, depression, and the other clinical and psychosocial variables highlight the coincidence of neural pathways —such as the adrenergic and the serotonin pathways— essential to chronic widespread pain and mood, which lie in the basis of FM features like autonomic unbalance, altered pain processing and modulation, sleep dysregulation, and anxiety. Further, personality and affective traits such as depression, somatic awareness, and anxiety are linked to genetic changes in the serotonin pathway, albeit they are also related with the risk of chronic pain (Diatchenko et al., 2013). In effect, it is in both the dorsal anterior cingulate cortex and anterior middle cingulate cortex where the integration takes place of negative affect, pain, and cognitive control (Shackman et al., 2011).

As per the psychosocial variables, FM quality of life was intensely associated to all three components of functional status, which proves functional status as a HRQoL measure for rheumatic diseases (Pincus, 2008; Pincus et al., 2008). The same kind of relationship was found, inversely, with RA physical quality of life, but RA mental quality of life only correlated moderately with the three components of functional status. Evidently, all three

components were strongly related amongst them. These correlations support the fact that musculoskeletal disorders are among the most common causes for hospital applications, as they lead to pain, functional impairment, work disability, and alter quality of life. Indeed, patients with daily pain are more likely impaired in daily living activities and less likely to get involved in activities, associations with have remained even after adjusting for the potential confounders of age, gender, race, cognitive functioning, and disabling conditions (Katz, 2002).

In addition, functional status in all patients was also contrasted with regards to having secure or insecure attachment, sex, and nationality. Being securely or insecurely attached showed significant and moderately sized differences in functional status, pain, and patient's global estimate of status. This association contradicts the studies that have found no direct association between insecure attachment and pain intensity (Ciechanowski et al., 2003; Meredith et al., 2006a) or disability (Meredith et al., 2006a) in samples with diverse chronic pain conditions. However, it is consistent with other studies, where in chronic pain patients, general insecure attachment has been associated to catastrophizing (Ciechanowski et al., 2003; McWilliams & Asmundson, 2007), lower self-efficacy (Meredith et al., 2006a), and depression (Ciechanowski et al., 2003; Meredith et al., 2007). It is also partially consistent with a study with chronic widespread pain patients, where general preoccupied attachment was linked with disability and number of pain sites, but not with pain intensity (Davies et al., 2009). Furthermore, gender also showed significant and moderately sized differences in functional status, physical function, pain, and patient's global estimate of status. In spite of the nature of this result, which does not allow for a causal inference to be drawn, it is possible that this finding is confirming the studies on rheumatoid conditions such as RA, where pain has a negative impact and diminishes quality of life, as well as gender and age, which have been reported to affect quality of life. In other words, among women with RA, pain may be the strongest predictor for reduced wellbeing (Jakobsson & Hallberg, 2002).

Subsequently, when the entire sample of FM was compared with the overall sample of RA, there were no differences found in educational level, age, and comorbidities. Thus, the results concerning education and comorbidities definitely establish that the descriptive observations were not sufficiently accurate and there aren't two differentiated profiles. However, statistically significant differences were found with regards to BDI depression,

HADS anxiety and depression, both pain-related outcomes, functional status, somatic symptoms, and all three components of functional status. In all these clinical and psychosocial dimensions, findings indicated that patients with FM had notably higher scores, and therefore poorer health outcomes, than RA patients. This both partially disproves and supports the **—third— hypothesis** that comorbidities and somatic symptoms would be higher in FM than in RA. It also confirms the rest of the **fourth, fifth, and sixth hypotheses** regarding the comparison of profiles, in the sense that FM did indeed have higher depression levels, that the expression of pain would be more pronounced in FM, and that functional status and HRQoL scores would show more impairment or deterioration in FM. In relation to previous research, this reproduces the findings of FM patients having significantly higher lifetime prevalence rates of mood and anxiety disorders, as well as increased numbers of medically unexplained physical symptoms across several organ systems; also, patients of FM have already proved to have equal or greater functional disability and less adaptation to the medical condition than RA individuals (E. Walker et al., 1997a). Further, it corroborates similar findings in Spanish patients as well, where comparing FM and RA patients showed that FM presented more elevated general psychopathology, anxiety and depression levels than in RA, who suffered a mild level of general psychopathology and depression and had no anxiety (Rodríguez de la Serna et al., 2004).

As per comparing the FM sample and the RA sample in the dimensions of sex, attachment, and nationality, the medical conditions were significantly different to a moderate extent only with regards to gender. That is, there was a much more prevailing presence of female patients in FM, with the whole sample being composed of women, than in RA, with a 75.3% of women. In this comparative instance, being securely or insecurely attached was no longer related to having FM or RA, as there were no significant differences. However, this could be because they both equally had a predominance of insecure attachment: in FM, 91.6% of patients had insecure attachment; whereas in RA, 83.5% were insecurely attached. In this case, it would support the idea that attachment behavior is always activated at times of sufficient stress, such as the typical motives of the majority of healthcare interactions —illness, injury, and loss—, which are nuclear triggers of attachment behavior (Hunter & Maunder, 2016). This result would also follow the same idea as recent reports showing that insecure attachment is particularly associated with impaired stress regulation (Flor &

Hermann, 2004), increased symptom reporting (Ciechanowski, Walker, et al., 2002), medically unexplained symptoms (Ciechanowski, Katon, et al., 2002), and somatoform disorders (Waller et al., 2004b). Lastly, attachment insecurity has already been suggested to be related to the development of chronic pain through dysfunctional reactions to episodes of acute pain (Porter et al., 2007); hence, it would reproduce the lack of significant difference between RA and FM in insecure attachment levels, as they are both ultimately medical conditions encompassing chronic pain. In this sense, since attachment-based theoretical approaches emerged in the 1980s and early 1990s, investigators already mainly contended the pain experience as a form of threat activating the attachment system (N. E. Andrews, P. Meredith, J. Strong, & G. F. Donohue, 2014), which would inevitably lead to a cascade of behaviors featuring insecurely attached individuals at greater risk for chronic pain and being less able to cope with established chronic pain (Andreson & Hines, 1994; Kolb, 1982; Mikail et al., 1994).

Adult attachment styles in FM and RA patients

H7. There will be a predominance of insecure attachment in FM in comparison to RA patients, which will mainly be securely attached.

H8. There will be more specific associations between severe FM and RA on one hand, and the different insecure attachment patterns on the other hand.

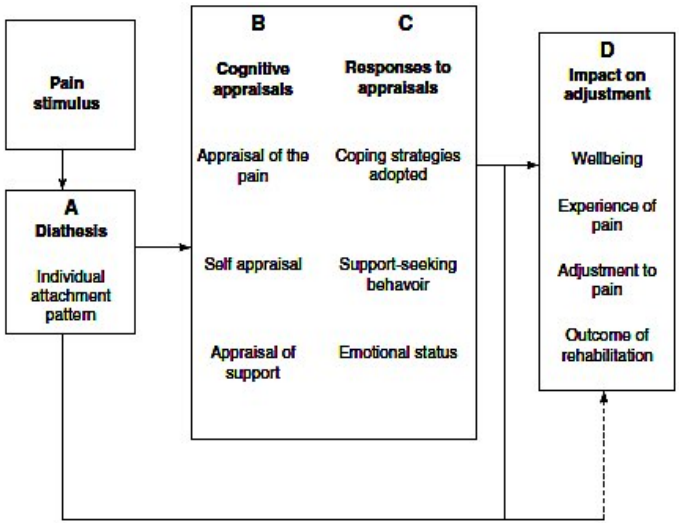
H9. FM patients will show a more notable prevalence of the hostile fearful/fearful and the dismissing subtypes in contrast to RA patients.

H10. Patients in New York will show a higher prevalence of dismissing attachment in relation to the Spanish samples.

Grounding this potent relationship between attachment theory and pain, an attachment-diathesis model of chronic pain was conceptualized (Meredith et al., 2008), focusing on how insecure attachment is both a vulnerability factor for the development of chronic pain as well as for poor outcome of chronic pain. Notably, there is also evidence showing that insecure attachment is more prevalent in medically unexplained pain compared to pain with a clear organic cause, and that poorer outcome in chronic pain is associated to insecure attachment independently of organic pathology (Schroeter et al., 2015). Literature suggested that attachment insecurity, and fearful and dismissing attachment in particular, is

overrepresented in chronic pain populations (Davies et al., 2009; Kowal et al., 2015; Meredith et al., 2005, 2006a). In other words, while there is research finding that approximately 65% of people in normative samples are securely attached and 35% are insecurely attached (Mickelson et al., 1997), in samples with pain patients these numbers are quite likely to be reversed (Kowal et al., 2015; Meredith, 2016).

Figure 36. The Attachment-Diathesis Model of Chronic Pain (from Meredith et al., 2008)



This theorization and evidence was backed when looking into the Spanish attachment styles, as there were significant differences in depression levels, anxiety and depression, pain interference, somatic symptoms, and the pain component of functional status. Functional status also showed a tendency to be significantly different according to attachment pattern. However, comparing the groups with a Scheffe's test detected only the differences, which were for the most part large, regarding mood and anxiety; that is, depression and anxiety and depression. This replicated the studies suggesting an association between depression and general insecure attachment in chronic pain patients (Ciechanowski et al., 2003; Meredith et al., 2007), as if possibly the strength of this association eclipsed the rest of significant differences, since all patients were equally pain-related conditions. Additionally, the most important of the mental health problems that co-occur with insecure attachment is depression, due to its common incidence and its consistent, substantial negative impact on the burden and outcome of physical illness. Depression is not only associated with increased severity of physical symptoms, increased health-care costs, and reduced health-related quality of life

(Evans et al., 2005), but also plays a significant part in increased mortality (Lemogne et al., 2013). In effect, depression is common in those with insecure attachment, especially in the context of medical illness (Ciechanowski et al., 2003; Maunder et al., 2005), and insecure styles of attachment are often linked to deficits in self-esteem and self-efficacy (Mikulincer & Shaver, 2007a). In this study, both the BDI scores and the HADS anxiety and depression scores indicated that the insecure group with the highest levels of depression and anxiety were the hostile fearful, followed by the preoccupied, and finally, the dismissing. In the case of pain interference and somatic symptoms, the same order was reproduced. This is partially consistent with the literature especially linking fearful and dismissing attachment to chronic pain (Davies et al., 2009; Kowal et al., 2015; Meredith et al., 2005, 2006a). However, in the pain component of functional status, the group with the highest expression of pain was the preoccupied style, followed by the hostile fearful, and then the dismissing.

In the New York sample, there were no significant differences among attachment styles —only a slight tendency in anxiety, not truly significant—; but when only secure and insecure attachment was observed, depression, functional status, somatic symptoms, anxiety and depression, and the pain component of functional status showed significant differences. In other words, these results suggest that having American insecure attachment also was linked to more anxiety and depression levels, as well as poorer functional status, increased somatic symptoms, and more HRQoL-related pain. This is consistent with the research finding that romantic anxious —and therefore, insecure— attachment was related to pain intensity and disability in arthritis patients (L. A. McWilliams et al., 2000), and romantic fearful attachment was associated to pain severity in lung cancer patients (Rumble, Keefe, Porter, Miller, Davis, Scipio, Garst, et al., 2006); whereas in individuals with chronic widespread pain, general preoccupied attachment was linked with disability and number of pain sites, but not with pain intensity (Davies et al., 2009), as well as the previously mentioned association between insecure attachment and depression in chronic pain patients (Ciechanowski et al., 2003; Meredith et al., 2007).

When the scales in the attachment questionnaires were analyzed in more detail, significant differences were found in the Spanish attachment questionnaire only. Specifically, RA patients had significantly lower scores in the scale of Low self-esteem, need of approval and fear of rejection in comparison to the FM group, which also means that having FM in the

study sample was associated to having increased Low self-esteem, need of approval and fear of rejection. This finding supports the essential clinical relevance of depression within the classification criteria of FM, due to the fact that this scale measures dimensions, low self-esteem in particular, which is intimately bonded to the symptoms of depression (American Psychiatric Association, 2013; World Health Organization, 1992). In fact, it has been posited that psychological factors, such as anxiety and depression, must be contrasted to some severity and course criteria of FM (Vallejo et al., 2012). Moreover, symptoms of depression are present in 26-71% of FM patients, a rate appearing very high in comparison to RA subjects, who are depressed in 14-23% of cases (Capraro et al., 2012; Murphy et al., 1999; Williams, 2003). In effect, antidepressant treatment used in FM has been found to be effective with both depression and pain (Häuser et al., 2009), which has contributed to bring forth the hypothesis of an etiological link between pain and depression in FM. Nonetheless, there were no significant differences between both medical conditions regarding the American questionnaire scales. Overall, in light of the findings from the previous section as well, the **seventh hypothesis** was partially refuted, as there was no predominance of insecure attachment in FM in comparison to RA patients, except for in the Spanish scale of Low self-esteem, need of approval and fear of rejection, which is notably present in the preoccupied and hostile fearful styles (Melero & Cantero, 2008). Further, the **eighth hypothesis**, according to which there would be more specific associations between severe FM and RA on one hand, and the different insecure attachment patterns on the other, was not confirmed in this study's results, as there were no significantly differentiated attachment profiles between FM and RA.

Thus, the **ninth hypothesis**, which posited that FM patients would show a more pronounced prevalence of the hostile fearful/fearful and dismissing subtypes than RA patients, was also disproved; as well as the **tenth hypothesis**, stating that both New York samples would show a higher prevalence of dismissing attachment in relation to the Spanish samples. In relation to this last **hypothesis**, percentages of attachment styles showed that in New York, both FM and RA had 16.13% of dismissing individuals, while in Barcelona, FM had 21.17% dismissing subjects and RA had 27.01% patients in this insecure substyle. As per the hostile fearful/fearful style, American FM and RA both presented 12.9% of fearful

attachment, while Spanish FM had 9.49% hostile fearful attachment and RA showed 7.3% Therefore, the direct results completely contradicted the **ninth hypothesis**.

Looking into Pearson correlation coefficients allowed to further analyze the relationships between the Spanish and the American attachment scales and clinical and psychosocial dimensions. In Barcelona, Low self-esteem, need of approval and fear of rejection was strongly associated to depression and anxiety and depression, which corroborates the ideas recently developed about depression and this particular scale. It also moderately correlated with pain interference, functional status, and somatic symptoms; and lowly correlated with pain intensity and all three components of functional status. Hostile resolution of conflict, rancor and possessiveness was lowly associated to depression and anxiety and depression. The dismissing style, corresponding to Emotional self-sufficiency and discomfort with intimacy, lowly correlated with pain interference and patient's global estimate of status; and moderately correlated with depression and anxiety and depression. The fact that these two scales show relationships with these dimensions is consistent with the research finding that fearful and dismissing attachment are particularly overrepresented in chronic pain populations (Davies et al., 2009; Kowal et al., 2015; Meredith et al., 2005, 2006a; Schmidt, Nachtigall, et al., 2002), since these two scales are related to the hostile fearful and the dismissing styles, respectively.

On the other hand, in New York, only the secure scale inversely correlated with most dimensions: it was strongly associated with patient's global estimate of status and moderately associated with depression, pain-related dimensions, functional status, anxiety and depression, and pain. In other words, American patients who had secure attachment had a protective effect against depression, pain, disability, and anxiety. Fearful attachment was moderately associated to pain interference and the pain component of functional status, while preoccupied attachment was moderately related to somatic symptoms and the pain component of functional status. Bearing in mind this bond in the present study's American samples between fearful and preoccupied attachment, on one hand, and pain-related variables and reporting of somatic symptoms, on the other hand; it becomes easy to establish similarities with previous findings in research with American patients: romantic fearful attachment has been associated to pain severity in lung cancer patients (Rumble, Keefe, Porter, Miller, Davis, Scipio, Garst, et al., 2006), and in chronic widespread pain patients,

general preoccupied attachment has been linked to with disability and number of pain sites, but not with pain intensity (Davies et al., 2009). Indeed, the protective effect of sorts observed in the study's American secure patients —and the overall effects of the different insecure patterns on the diverse clinical and psychosocial dimensions— could be attesting to how transcendent adult attachment is becoming in psychosomatic research due to its effects on many biopsychosocial phenomena, such as social functioning, stress response and coping, psychological wellbeing, health behavior, and morbidity (Ciechanowski & Katon, 2006; Ditzen et al., 2008; Maunder & Hunter, 2001; Maunder et al., 2005; Maunder, Lancee, et al., 2006; Meredith et al., 2006a; Schmidt, Nachtigall, et al., 2002; Waller et al., 2004b).

Cross-cultural comparison between medical conditions

H11. Depression and anxiety will be reported differently according to the sample geographical origin.

H12. Psychosocial dimensions, and pain interference with daily activities due to its intimate relationship with quality of life, will be expressed as more deteriorated in Barcelona than in New York due to cultural influence.

The two samples were contrasted in both countries and the results were very similar than without the cross-cultural comparison. All four groups did not show differences in educational level, age, or comorbidities. However, FM and RA did differ significantly in anxiety and depression, pain-related outcomes, functional status and its components, and somatic symptoms. Nevertheless, when a Scheffe's test was conducted, it revealed that differences were not significant with regards to BDI depression and functional status: Barcelona RA and New York FM did not have significant differences in BDI depression, and both New York samples' functional status were not different significantly. Also, a similar phenomenon was detected concerning functional status' components, in the sense that physical function was not significantly different in Barcelona FM and New York RA, and patient's global estimate of status did not differ significantly between Barcelona RA and New York FM. Since functional status has been measured in this study with an instrument that can be equally used to obtain HRQoL in rheumatic diseases (Birrell et al., 2000; Fries & Ramey, 1997; Kvien et al., 1998), this could be considered as a lack of significant differences between the HRQoL expressed in New York FM and RA and among both countries. Hence,

the fact that HRQoL was different between FM and RA in Barcelona but not in New York could be due to a cultural explanation. Indeed, the generalizability of pain coping research yielding from studies with English-speaking patients, in particular from the USA since it represents the majority of these studies (Jensen et al., 2003; McCraken et al., 2007; J. Miró et al., 2009; Osborne et al., 2007; Romano et al., 2003; Tan et al., 2006; Tan et al., 2011; Woby et al., 2007), to other cultures, is yet to be elucidated (Ferreira-Valente et al., 2011). Overall, it is a consolidated finding that low mood, somatizing tendency, and adverse health beliefs notably condition non-specific musculoskeletal pain, in particular its chronicity and related disability; moreover, differences in societal beliefs may have also intervened in major variation in the prevalence of disabling musculoskeletal pain, between countries and within countries over time (Vargas-Prada & Coggon, 2015). It is possible that the interplay between BDI depression and the experience of disability and functional status may have been very similar in the present case between countries and medical conditions. Further, it has been posited that differences in societal beliefs might begin to explain notable contrasts in the prevalence of musculoskeletal complaints and associated disability reported among workers with similar jobs but dissimilar cultural circumstances (Coggon, Ntani, Palmer, Felli, Harari, Barrero, Felknor, Gimeno, Cattrell, Serra, et al., 2013; Madan et al., 2008).

These findings answer the **eleventh and twelfth hypotheses**: depression and anxiety, as measured by the HADS, were confirmedly reported different according to the sample geographical origin, since significant differences were found but not through the results obtained with the BDI in depression. However, the **twelfth hypothesis**, according to which psychosocial dimensions and pain interference with daily activities would be expressed as more deteriorated in Barcelona than in New York, was not fully confirmed in the study's results. Significant differences were found in pain interference, yet there were the aforementioned contradictions concerning functional status and quality of life. Additionally, functional status did seem more deteriorated in Barcelona, as there were no significant differences between FM and RA in New York.

Furthermore, when the four groups of pathologies in both countries were compared with regards to sex, attachment, and nationality, significant differences with a small effect size were found as well. The ones related to gender and nationality are obvious and have been mentioned previously; however, the fact that the cross-cultural comparison between FM and

RA did yield differences in attachment was a novel finding, as the comparison between FM and RA from the same country had not produced significant differences between the securely and insecurely attached groups. Once again, it is possible that the effects of insecure attachment on disease were different according to the sample's geographical origin, due to the fact that the prevalence of securely and insecurely attached patients in FM and RA showed different proportions in Barcelona and New York. That is, in Barcelona, 94% of FM patients presented insecure attachment, as well as 87.1% of RA patients; while in New York, 81.3% of FM and 66.7% of RA patients were insecurely attached, which was clearly less than in Barcelona. It is not possible to establish if this difference is due to the sample size disproportion between countries or if it is expressing a real difference of attachment insecurity prevalence and impact.

Relationship amongst clinical and attachment characteristics and influence on psychosocial dimensions

H13. Pain intensity and interference will be highly associated with depression, anxiety, and psychosocial dimensions.

H14. Overall, insecure attachment will have a negative impact on health status, as reported through the clinical and psychosocial variables.

H15. FM severity, according to quality of life and functional status levels, will be more related to the insecure subtypes than in the case of RA.

H16. Depression will explain pain and illness severity, accounted for with quality of life and functional status scores.

This last section is dedicated to the general aim of how clinical and attachment characteristics are related and influence the patients' psychosocial dimensions. To this end, and due to the multiple linear regression models that were required, both American samples were not included in the analyses for their insufficient sample size. Therefore, the cross-cultural factor of the study could not be integrated in this stage as initially intended.

Figure 37. Mechanisms by which insecure patterns of attachment may contribute to disease, from J. J. Hunter and R. G. Maunder, 2016.

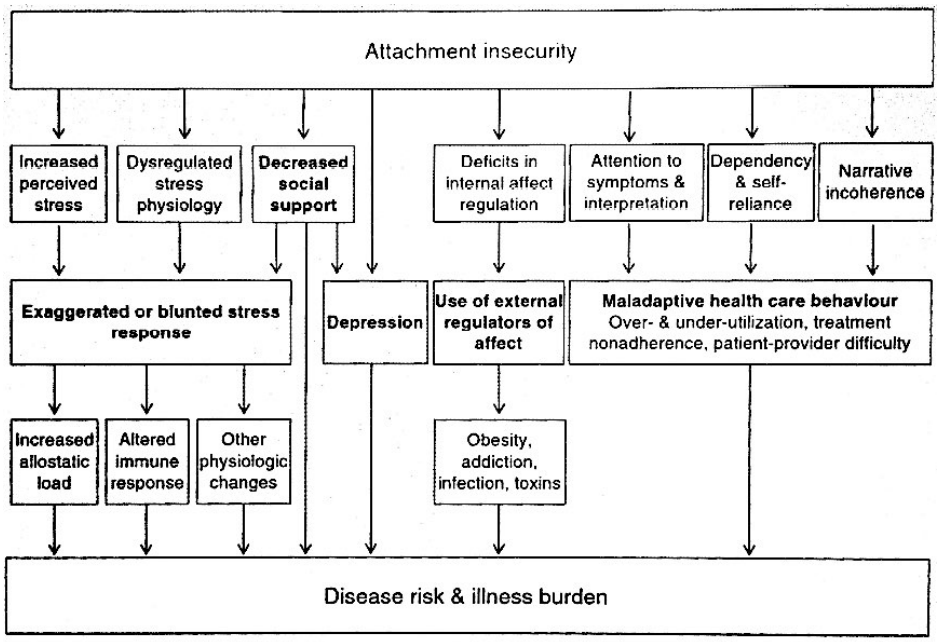
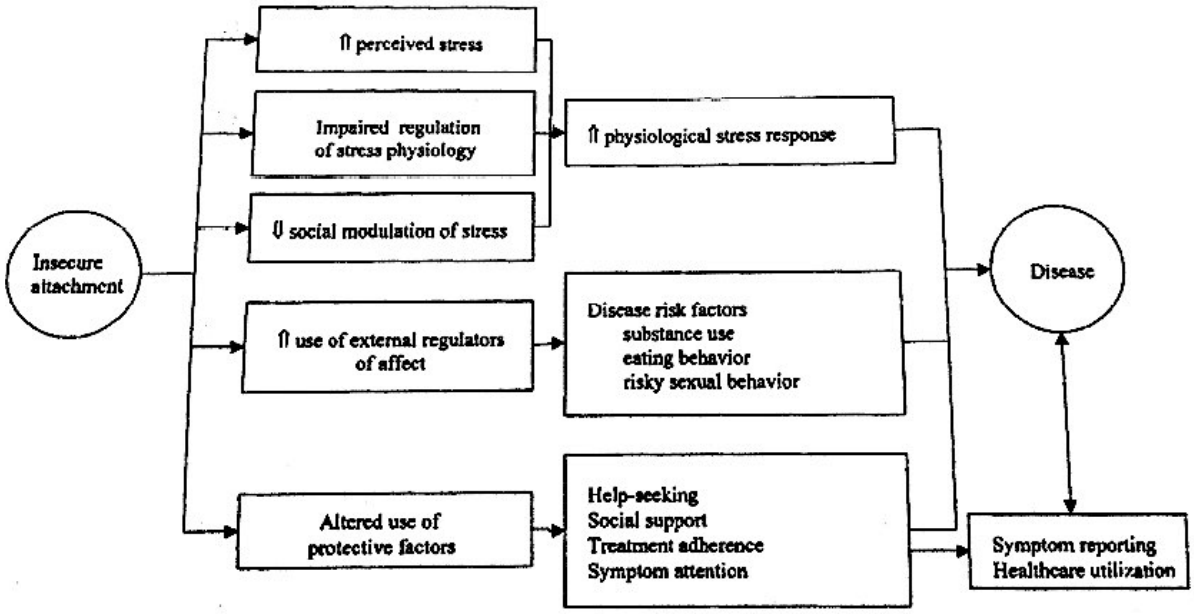


Figure 38. Model of hypothesized mechanisms by which attachment security could contribute to disease, from Maunder, R. G. and Hunter, J. J., 2001.



Figures 37 and 38, discussed in the Introduction chapter of this dissertation, are relevant here due to their explanation of how insecure attachment might contribute to disease. Figure 37 is a more recent and complete version of Figure 38, and shows the effect of

attachment insecurity on different elements over a lifetime, the intricacy of the interrelationships in order to constitute a risk factor for disease and a chronic illness encompassing a considerable burden for the patient. It is chiefly with this theoretical framework in mind that the analyses in this section were performed.

Hence, in a first step, Spanish attachment styles were studied as a predictor in the relationship with FM-related dimensions, then with RA, and lastly, analyses were performed of HADS' anxiety and depression as predictors of FM variables and then with RA outcomes.

When attachment was examined, it was found to significantly predict only pain-related dimensions in FM. In particular, a worse pain intensity was explained by increases in Emotional expressiveness and comfortableness with intimacy, corresponding to the secure style of attachment; whereas a worse pain interference was explained by a higher Low self-esteem, need of approval, and fear of rejection.

On the other hand, in RA, attachment predicted with significance quality of life, pain interference, functional status, and somatic symptoms. More specifically, a poorer RA mental quality of life was explained by an increased Low self-esteem, need of approval, and fear of rejection; while a more deteriorated RA physical quality of life was predicted by a higher Emotional expressiveness and comfortableness with intimacy. A tendency towards significance showed that pain intensity could be predicted, with caution, by Emotional expressiveness and comfortableness with intimacy; whereas a worsened pain interference was explained by an increased Low self-esteem, need of approval, and fear of rejection, identically as the instance of FM. Further, a more deteriorated functional status was significantly explained by a higher Low self-esteem, need of approval, and fear of rejection; higher Emotional expressiveness and comfortableness with intimacy; and increased Emotional self-sufficiency and discomfort with intimacy. Lastly, augmented somatic symptoms were predicted by a higher Emotional expressiveness and comfortableness with intimacy and an increased Emotional self-sufficiency and discomfort with intimacy.

Thus, the **fifteenth hypothesis** of this dissertation, positing that FM severity, according to quality of life and functional status levels, would be more related to the insecure subtypes of attachment than in the case of RA, was not confirmed by the results. What is more, attachment had a more significant and potent impact on RA and its severity than in FM, where it seemingly just affected the pain experience. Also, an interesting result is that,

both in FM and RA, secure attachment was involved as well, in pain severity and in quality of life, particularly in its physical dimension in the case of RA. This, coupled with the fact that functional status and somatic symptoms in RA were affected by both secure and insecure attachment, leads to the assumption that attachment security could be exerting a more enhanced influence on the physical sphere of chronic pain. Moreover, in the model presented earlier (Figure 37), attachment insecurity is suggested to yield disease risk and burden through depression as one of the main pathways. Therefore, it is likely that these study's results are hinting a direct effect of attachment security and an indirect effect of attachment insecurity in RA through depression. The idea of the latter depression effect is supported when looking back at the Pearson correlation coefficients, showing the strongest relationships between depression and anxiety and depression with two insecure subtypes: Low self-esteem, need of approval and fear of rejection and Emotional self-sufficiency and discomfort with intimacy.

This is also consistent with attachment literature, in which researchers have dedicated efforts in understanding how attachment impacts psychosocial functioning, in particular, how attachment is related to depression (e.g. Catanzaro & Wei, 2010; Kobak & Sceery, 1999). Early work regarding the relationship between adult attachment insecurities, of both the anxious and avoidant varieties, and depression consistently found a positive association (e.g. Armsden et al., 1990). However, these first studies providing evidence of a positive direct association lacked an explanation of the mechanisms underlying this relationship (Roberts et al., 1996). In fact, the attachment-psychopathology liaison is moderated by a wide array of biological, psychological, and sociocultural factors, and mental disorders per se can undermine a person's sense of attachment security (Mikulincer & Shaver, 2012). More recently, and according to attachment theory as it has been previously reviewed, research has established that the link between attachment insecurities and psychopathology in general —and depression, in this instance—, is mediated by several pathways, the most important of which are self-representations, emotion regulation, and problems in interpersonal relations. These pathways explore concepts such as lack of self-cohesion, unstable self-esteem, over-dependence on external approval, self-criticism, impairment of coping strategies due to absence of emotionally accessible and responsive others, emotion amplification and exaggeration of worries, and interference with the acquisition of social skills due to recurrent

failure to obtain support from attachment figures (Mikulincer & Shaver, 2012). Furthermore, there are also precedents of an individual's attachment style influencing the relationship between depression and pain (Andersen, 2012; Martínez et al., 2012; Meredith et al., 2006a; Sockalingam et al., 2013; Tremblay & Sullivan, 2010), added to certain interpersonal problems that are correlated with a high prevalence of pain and depression: submissiveness and nonassertiveness, and self-sacrificing and friendly submissive behavior (Adler & Gattaz, 1993; Lackner & Gurtman, 2004).

Evidence corroborates Bowlby's prediction that factors that lead to insecure attachment also augment the risk of depression. A possible hypothesis might be that the developmental experience of attempting to relate to an unavailable parent and being thwarted yields learned helplessness, a state that consistently causes depression (Seligman & Maier, 1967). Another option is that attachment insecurity may increase the risk of depression by increasing vulnerability to the effects of stress. Additionally, insecure styles of attachment are often linked to deficits in self-esteem and self-efficacy (Mikulincer & Shaver, 2007a). For all these reasons, depression is common in those with insecure attachment, especially in the context of medical illness (Ciechanowski et al., 2003; Maunder et al., 2005). Hence the importance of recognition and management of depression, as sometimes it is the most malleable element of a vicious cycle of disease and the consequences of illness (Hunter et al., 2016).

Moreover, this study's findings partially support the diverse research linking adult attachment and certain aspects of the overall pain experience. For instance, insecure attachment has been related to nearly twice the prevalence of chronic widespread pain as secure attachment in a community sample (Davies et al., 2009), as well as with increased reporting and suffering of pain among chronic pain patients (MacDonald & Kingsbury, 2006; L. A. McWilliams et al., 2000; Meredith et al., 2007). In fact, insecurely attached subjects without chronic pain conditions also proved to have increased catastrophizing hypervigilance, decreased pain thresholds and self-efficacy to episodes of acute pain or experimentally induced pain (Martínez et al., 2012; Meredith et al., 2006b; C. L. Wilson & Ruben, 2011). In light of this evidence, it has been suggested that attachment insecurity is related to the development of chronic pain through dysfunctional reactions to episodes of acute pain (Porter et al., 2007). Indeed, depression as well is commonly observed to coexist with chronic pain

and is chiefly associated with higher levels of reported pain and increased functional impairment (Arnow et al., 2006; Bair et al., 2003; Demyttenaere et al., 2006), thus contributing to a challenging diagnosis due to overlapping somatic symptoms. Insomnia, fatigue, and change in activity constitute symptoms that can be related to both pain and depression, albeit according to DSM-5, symptom criteria that are fully attributable to the medical condition should not be included in the diagnosis (American Psychiatric Association, 2013). In other words, the present study's results seem to corroborate the strong impact of adult attachment on disease, regardless of the specific illness or whether it is one chronic pain condition or another. Ultimately, they also support the **fourteenth hypothesis**, since overall, insecure attachment did have a negative impact on health status, as reported through the clinical and psychosocial variables.

Looking into the results linking anxiety and depression with FM and RA further consolidate the previously discussed ideas. In FM, worsened quality of life and pain interference were both significantly explained by incremented anxiety and depression; whereas pain intensity, poor functional status, and increased somatic symptoms were predicted only by higher anxiety. In RA, poorer mental quality of life, pain interference, functional status, and increased somatic symptoms were all predicted by both higher anxiety and depression; while worse physical quality of life and pain intensity were predicted only by higher levels of depression. Thus, these results shed light on the **thirteenth and sixteenth hypotheses** of this dissertation: according to the **thirteenth**, pain intensity and interference would be highly associated with depression, anxiety, and psychosocial dimensions. In FM, pain interference was explained by anxiety and depression conjointly, but pain intensity was only explained by anxiety. In RA, pain interference was also predicted by anxiety and depression, but pain intensity was solely predicted by depression. This proves a higher association of anxiety with FM and of depression with RA, in relation to both dimensions of the pain experience. It also partially confirms the **sixteenth hypothesis**, which posits that depression would explain pain and illness severity, accounted for with quality of life and functional status scores.

These findings are not only a testimonial of the aforementioned hypothesized indirect effect on RA of attachment insecurity via depression, but are also partly supported in literature, as depression is traditionally more linked with FM than RA. Regarding depression

in FM, a high prevalence of comorbidity has been largely documented (Arnold et al., 2006; Hudson et al., 2004; Weir et al., 2006). Evidence on the subject suggests an increase of comorbidity with depressive symptoms and a lifetime prevalence of major depressive disorder. Also, an association between FM and depression has been found in epidemiological studies (Patten et al., 2006). Several investigations corroborate the hypothesis of a predominance of negative rather than positive emotions in FM (Davis et al., 2004; Finnan et al., 2009; Gross & John, 2003; Sayar et al., 2004; Van Middendorp et al., 2008; van Middendorp et al., 2010; Zautra et al., 2005). Symptoms of depression are present in 26-71% of FM patients; a rate that appears very high in comparison to RA subjects, for instance, who are depressed in 14-23% of cases (Capraro et al., 2012; Murphy et al., 1999; Williams, 2003). In fact, antidepressant treatment used in FM has been found to be effective with both depression and pain (Häuser et al., 2009), which has contributed to bring forth the hypothesis of an etiological link between pain and depression in FM. In a functional magnetic resonance imaging (fMRI) study, the neuronal activation pattern of the pain neuromatrix in FM was found to be modulated by comorbid depression, producing an increase of activation in the brain areas involved in affect processing, such as the cingulate cortex, the anterior insula cortex, and the amygdala (Giesecke et al., 2005). Additionally, the alterations in the HPA stress axis in FM are similar to those described in depression (Lund et al., 2006; McBeth et al., 2007; Wingenfeld et al., 2007). One study suggested that depression in FM chiefly determines pain perception, as opposed to RA where pain is rather due to peripheral stimuli (Scheidt et al., 2014); the main hypothesis is that anxiety and depression lower the pain threshold, thus exerting an influence on pain perception (Giesecke et al., 2005). This main hypothesis is chiefly the influence that seems to be stressed in the dissertation's results.

As per arthritis, the Land et al. (van't Land et al., 2010) study provides evidence of the damaging impact of depression on psychosocial functioning without serving as an independent cause for the development of arthritis, noting that disability could be a mechanism through which arthritis may lead to depression (van't Land et al., 2010). Furthermore, depression has been shown to contribute to adverse health outcomes in previously diagnosed arthritis patients, for instance, exacerbating inflammatory processes, interfering with functioning, decreasing medical adherence, and aiding maladaptive health behaviors that create risk for greater disease activity and medical comorbidities (Nicassio,

2010; van't Land et al., 2010). Self-sacrificing tendencies have also been proved as moderators in the relationship between pain and physical symptoms in RA (Bai et al., 2009; Hyphantis et al., 2013), which is linked to depression (Chance et al., 1996). Certain psychological approaches have proven effective in randomized clinical trials in fostering adaptive coping and health behaviors, and palliating pain, disability, and mood disturbance in patients with RA and osteoarthritis (Astin et al., 2002; Dixon et al., 2007). These studies are consistent in a definitive manner with the present dissertation's result of the effect of depression on RA.

Consequently, authors such as Birtane et al. (Birtane et al., 2007) have depicted RA and FM as having a greater incidence of poor physical and psychological functioning in comparison to healthy controls, as well as they have identified higher depression levels in FM patients than in RA. In effect, FM is associated with the worst psychological functioning, given the frequency of depressive symptoms in individuals with FM (Wolfe et al., 2010).

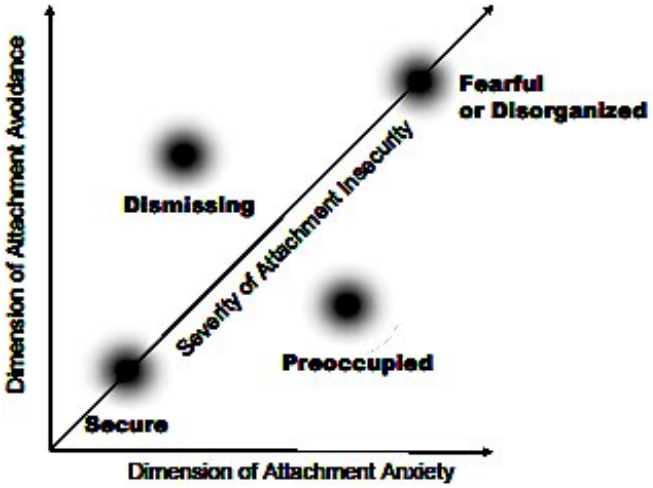
However, a review of the evidence highlights the overarching theme of a bond between overall chronic pain and mood disorders. Indeed, approximately between 30% and 60% of chronic pain patients have comorbid depression (Goesling et al., 2013). The prevalence of a lifetime history of major depression or another mood disorder is even higher; furthermore, these data are muddled by a 50% prevalence of pain in patients with a primary diagnosis of depression (Crofford, 2015b). Depressed patients notably report more unexplained physical symptoms, such as pain and fatigue, and use more health resources than nondepressed patients (Bair et al., 2003). These findings highlight the conception of a bidirectional relationship between the presence and severity of pain and depression: in fact, a large longitudinal study of primary care patients with persistent pain of the back, hip, or knee stated that change in pain was a strong predictor of depression severity, and vice versa (Kroenke et al., 2011). There is extensive evidence of the high conjoint prevalence of mental health conditions with chronic pain (Bair et al., 2003; Banks & Kerns, 1996; Haythornthwaite et al., 1991). Additionally, as has been presented earlier, depression and pain often coexist, respond to similar treatments, aggravate one another, and share biological pathways and neurotransmitters (Blier & Abbott, 2001; Gallagher & Verma, 1999). Depression has a direct effect on the development of pain and some studies have also shown indirect effects, or mediation, of depression on pain, whereas other studies showed depression as an intervening

variable (between another variable and pain) in path analysis. For instance, in the fear-avoidance model (Lethem et al., 1983), depression is posited as a mediator of prospective bonds between the fear-avoidance model and pain variables, achieving a better prediction of model variables (Seekatz et al., 2013). Additionally, in the communal coping model of catastrophizing (Thorn et al., 2003), catastrophizing thought has a direct effect on pain intensity and predicted the affective component of pain through depression, as well.

Further, pain catastrophizing has been found to significantly correlate with pain intensity and maladaptive coping (Kratz et al., 2011; Martínez et al., 2012; Meredith et al., 2006b; Tremblay & Sullivan, 2010). Anxiety and avoidance, as dimensions of adult attachment, have been proved to predict different aspects of daily pain and coping with pain in chronic pain female patients (Kratz et al., 2011). This could explain the association in the study's results between FM and anxiety. More specifically, self-efficacy in pain-coping has been found to predict physical and psychological wellbeing in FM patients, being self-efficacy negatively associated to pain, anxiety, and depression (Culos-Reed & Brawley, 2000; Sánchez, Martínez, Miró, & Medina, 2011). Also, Arnold et al. (2006) studied how FM patients and other forms of central pain amplification are more susceptible to other psychiatric disorders, in particular depression, post-traumatic stress disorder and other anxiety disorders, and bipolar disorder. The highest odds ratio (OR) observed in this study was in obsessive compulsive disorder (OR=14), and the sex and age adjusted co-occurrence OR for any anxiety disorder in FM was of 6.7 in comparison to subjects without FM (Arnold et al., 2006).

Another idea that is highlighted, or rather hinted, in relation to anxiety, is a conceptual notion already presented in the Introduction chapter of this dissertation and illustrated in the following Figure.

Figure 39. Patterns of adult attachment, from Maunder, R. G. and Hunter, J. J., 2012.



It is possible to find a theoretical bond between the anxiety brought forth in particular by the study's FM patients and attachment anxiety *per se* (also just discussed above, in Kratz et al., 2011). That is, looking at the differences between American attachment styles, a tendency towards a significant difference was found in anxiety, particularly elevated in the preoccupied group. Relatedly, in Figure 39, the preoccupied style is one of the most paradigmatic in attachment anxiety. Also, the only significant differences detected among Spanish FM and RA patients were the ones regarding the attachment scale of Low self-esteem, need of approval and fear of rejection, higher in Spanish FM and mainly increased in preoccupied and hostile fearful attachment, both epitomes of attachment anxiety in Figure 39. Lastly, in the Pearson correlation coefficient matrix between attachment questionnaire scales and the main clinical and psychosocial variables, the strongest relationship in the entire matrix if the between anxiety and Low self-esteem, need of approval and fear of rejection, in the instance of Barcelona.

Conclusions: strengths, limitations, and implications for future research

The results of this dissertation have largely supported the theoretical approached and literature presented in the Introduction section. Attachment insecurity and depression share a common ground, particularly in the study of physical diseases (Watson, Haviland, Greer, Davidson, & Bliss, 1999; M. West, Rose, Verhoef, Spreng, & Bobey, 1998; Wulsin, Vaillant, & Wells, 1999), association which has been namely garnered in the clinical and psychosocial reports of FM and RA patients. Further, this study has contributed in the understanding of the intricate interrelationships between attachment, mood disorders, and the burden of two chronic illnesses.

Depression is commonly observed in a dyad with chronic pain. Banks and Kerns (Banks & Kerns, 1996) introduced the diathesis-stress model for pain and depression, putting forth the idea that chronic pain patients who become depressed may suffer from a certain premorbid psychological predisposition toward developing depression. Multiple factors are involved in the depression-pain linkage, such as neurobiological, genetic, and precipitating environmental factors, also counting psychological, social, and cognitive influences (Bekkouche et al., 2013; Covic et al., 2003; Gale et al., 2012; Goesling et al., 2013; Mongini et al., 2009; Pulvers & Hood, 2013). Another model that helps explain the components involved in the association between depression and pain is Engel's biopsychosocial model (Engel, 1977). On the basis of this approach, psychological stress, or the extent to which individuals feel that external demands exceed their ability to cope, has also shown to significantly correlate with pain and depression (Candrian et al., 2007; Kuiper et al., 1986; Menzies et al., 2013; Pizzagalli et al., 2007). Other similar factors that have proved a link to pain include self-efficacy (E. Miró et al., 2011), mastery (Bierman, 2011), mental defeatism (Tang et al., 2010), catastrophizing, hopelessness and helplessness (Fahland et al., 2012), and personal control (Q. Wang et al., 2015). Furthermore, an individual's attachment style also influences the relationship between depression and pain (Andersen, 2012; Martínez et al.,

2012; Meredith et al., 2006a; Sockalingam et al., 2013; Tremblay & Sullivan, 2010), added to certain interpersonal problems that are correlated with a high prevalence of pain and depression: submissiveness and nonassertiveness, and self-sacrificing and friendly submissive behavior (Adler & Gattaz, 1993; Lackner & Gurtman, 2004).

Thus, it is evident that addressing poor psychological status, and depression in particular, in musculoskeletal conditions is critical due to its impact on the condition. Coadjuvant relationships may occur between pain and depression and disability, yielding a cycle of poor mental and physical health (G. J. Walker & Littlejohn, 2007). In fact, a recent systematic review found that depression and self-efficacy are outcome predictors irrespective of intervention in self-management programs for chronic pain patients; therefore, these factors should be targeted at early stages in management programs, in order to prevent transition to chronic pain disability (Miles et al., 2011). Despite the fact that the pathophysiology of RA and FM are inherently different, the pain system still remains the common substrate, which can be functionally triggered by distress, as with the rest of chronic painful and disabling disorders. This is paramount evidence in advancing towards the comprehension of the causal mechanisms for FM, with its outcome of highly significant effect on the quality of life, and therefore also of improving the management of RA and related musculoskeletal conditions (G. J. Walker & Littlejohn, 2007).

In spite of the fact that most research involving attachment insecurity and disease measures and controls for depressive symptoms, it was not possible to do so in this study. Owing to the insufficient sample size, there was an inability to control for depression or to establish a possible moderator effect with multiple linear regression analysis. This is the first and one of the most important limitations of this research: the difficulties in reaching an initially desired large sample size, due to the naturally complicated access to clinical patients in different settings and countries, as well as it being characteristic of cross-sectional designs, in which there is no well-established relationship between the researcher and the respondent prior to the survey administrations and therefore response rates are often limited (Fraenkel & Wallen, 2009; O'Sullivan, Rassel, & Berner, 2003). Additionally, due to the purposive sampling used in patient recruitment, the exclusion criteria further limited the ability to obtain a large sample in both countries, particularly in New York. Thirdly, it also added a selection bias. Fourth and foremost, the cross-sectional nature of the current study does not

allow inferences of causality to be made or in-depth explorations that strive to analyze changes or trends in variables over time (Gay, Mills, & Airasian, 2009; O'Sullivan et al., 2003). Consequently, attachment evaluation reflects individuals' subjective perceptions of their affective bonds, which may be vulnerable to reporting bias, and the presence of mood disorders of the experience of pain may have had an influence on attachment style reporting as well (Ciechanowski, Worley, Russo, & Katon, 2006; MacDonald & Kingsbury, 2006). This also means that in pursuing an examination of these relationships through cross-sectional data would render it impossible to statistically ascertain if depression is a result of suffering FM or if major depression could entail developing FM, which has also been discussed when studying the etiology of FM and chronic pain in general, for that matter (Wolfe et al., 2014). In this respect, FM differs from RA in that it involves diagnosis based on symptom severity, which is what partly motivated the use of an ACR2010 FM diagnosis in the study participants. Fifth and last, the FM sample comprised entirely female patients, thus not allowing to describe gender particularities in FM that have been featured in literature (Aparicio, Ortega, Carbonell-Baeza, & et al., 2012; Miro, Diener, Martinez, & et al, 2012), albeit this was not well within the aims and scope of this research.

Despite these limitations, the strong theoretical foundation of the hypotheses provides a reasonable context for finding interpretation and it justifies broadening research in this matter. The stability of the findings, mainly the overwhelming association between ACR2010 FM and RA and adult attachment and mood disorders, requires replication in further studies. The inclusion of a securely attached comparison group with no chronic pain or rheumatic condition and increasing the samples within a prospective study framework would achieve confirmation and pave the road for improving efforts in the field of RA and particularly FM, an illness with yet many unknowns. Findings hold particular promise taken in the context in order to pursue adapting treatment in the FM population, especially psychological, in the direction of applying research on adult attachment processes and mood disorders. Notably, this research introduces the idea that RA merits this equally tailored psychological intervention, due to the even more overwhelming and initially unexpected association with depression and insecure attachment.

The conclusive needs to develop novel investigation approaches and to foster more effective treatment among FM and RA individuals are not only supported throughout this

dissertation, but also by the growing body of evidence backing the theoretical associations between the quality of patient-provider relationship, healthcare utilization, and other medical outcomes (Ditzen et al., 2008; Dozier et al., 1994; Dozier et al., 2008; Gunnar et al., 1996; Maunder & Hunter, 2016; Maunder, Lancee, et al., 2006; Maunder, Panzer, et al., 2006; Meredith et al., 2006a, 2007; Schmidt, Strauss, et al., 2002; Waller et al., 2004a). It must be noted that internal working models can change due to interpersonal and emotional relevant life circumstances, despite the essential continuity of the attachment system (Bowlby, 1982; Davila & Cobb, 2004). This entails that psychotherapy, for instance, offers a significant emotional experience which may change conflictive working models (Bowlby, 1988). In this regard, a review of studies investigating changes in attachment style over the therapeutic course has found that increases in attachment security or decreases in attachment insecurity are linked with a better outcome (Mikulincer et al., 2013). That is, in an analogous way to how the parent's secure attachment organization provides the internal resources to respond to the infant appropriately and empathetically, so it seems that a clinician's secure attachment organization may provide the necessary resources to respond sensitively to patients (Dozier et al., 1994). Furthermore, health outcomes are understandably linked to the patient-provider relationship since they each are a manifestation of underlying attachment dynamics: at times of health-related threat or distress, individuals engage in attachment attitudes and behaviors (proximity seeking or avoidance, trust or distrust, expression or suppression of distress) with healthcare providers in a similar way than within the context of a romantic relationship (Ciechanowski, Walker, et al., 2002; Maunder & Hunter, 2016). In effect, attachment behavior is always activated at times of sufficient stress, and the typical motives of the majority of healthcare interactions −illness, injury, and loss− are nuclear triggers of attachment behavior (Hunter & Maunder, 2016).

In conclusion, the data presented highlight the importance of overall adult attachment and mood disorders contributing to the burden and subjective experience of both FM and RA. Further investigation is now required in the aforementioned directions in the hopes to improve the quality of life and function of these two medical conditions, which still require of palliative treatment as they yet hold no definitive cure.

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