HISTONE PTMs & CANCER

PTMs can be miswritten, misinterpreted and miserased, which leads to PTM imbalance → CANCER.

18% of all female tumours MOST COMMON & 2nd MOST LETHAL CANCER

<table>
<thead>
<tr>
<th>Tumour</th>
<th>ER exp.</th>
<th>PR exp.</th>
<th>HER2 exp.</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>luminal a</td>
<td>+/-</td>
<td>+/-</td>
<td>-</td>
<td>Favourable</td>
</tr>
<tr>
<td>luminal b</td>
<td>+/-</td>
<td>+/-</td>
<td>+</td>
<td>Favourable</td>
</tr>
<tr>
<td>triple negative</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Poor</td>
</tr>
<tr>
<td>HER2+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>Poor</td>
</tr>
</tbody>
</table>

Table 1. Breast cancer classifications and prognosis according to gene expression.

ENZYMATIC ACTIVITY

A one to one relation was seen between methylation and HMTs. However, it was different regarding acetylations, although some HATs were seen with a higher prevalence than others, such as p300 and the CREB binding protein (CBP) or NCOAs.

HISTONE PTMs & CANCER

PTM approaches were 1. Histone PTMs as biomarkers for breast cancer? 2. Gene specific pattern of histone PTMs? 3. Use of histone PTMs in anti-cancer therapy?

Histone PTMs have a correlation with cancer and with breast tumours phenotypes and prognosis but further data need to be collected in order for them to be considered biomarkers. Ex: global loss of H4K16ac or H4K20me, more aggressive tumours when cells had a low level of H4K20me3, etc.

No locus-specific alterations in histone PTMs when developing new therapies to fight breast cancer. Due to their reversible nature, enzymes in charge of PTMs were targeted. HDAC inhibitors have provided very promising results in mono-therapies (i.e. scriptaid) and in combinatorial therapies (vorinostat + tamoxifen).

CONCLUSIONS

REFERENCES