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In vitro long-term exposures to PET-NPLs promote carcinogenic hallmarks in BEAS-2B cells

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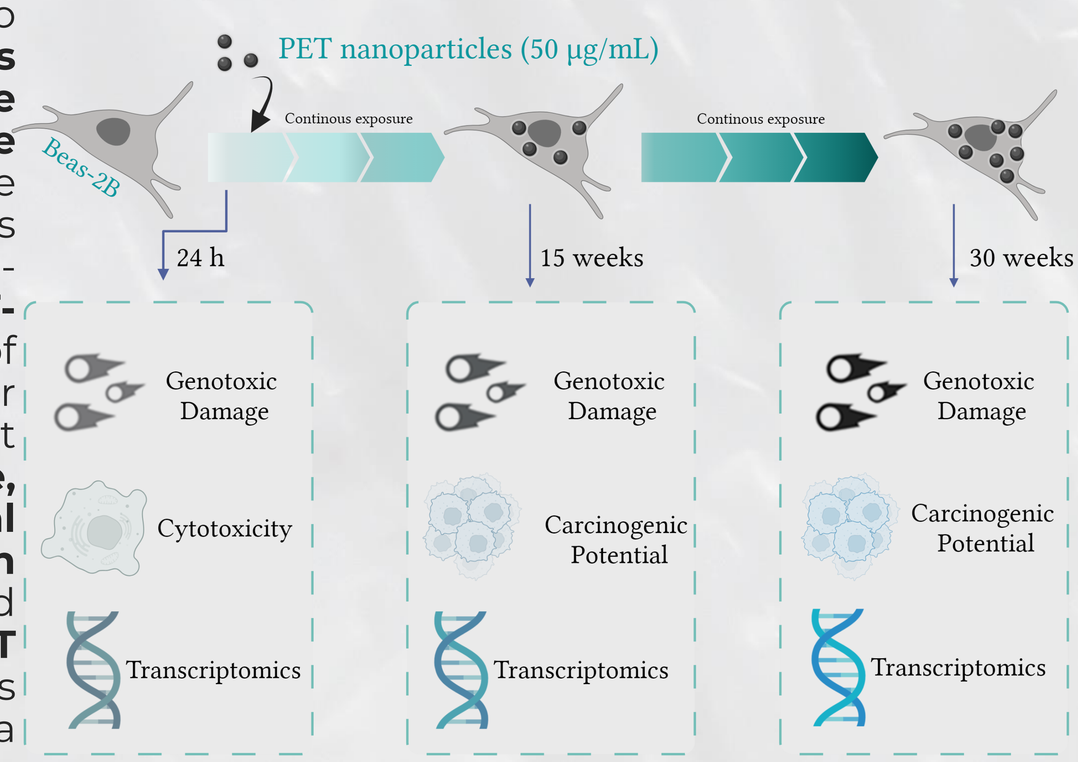
Summary

Nanoplastics (NPLs) are contaminants with the potential to **threat human health. Inhalation is a major route for NPLs exposure,** as evidenced by their detection in human lungs, **where** polyethylene terephthalate (**PET**) has been detected as **one of the most abundant.** However, the potential harmful effects of the accumulation of these particles are still largely unknown, as **chronic effects have not yet been studied**. To study these long-term effects**, BEAS-2B bronchial cell line was exposed to PET-NPLs for 30 weeks.** Genotoxicity, phenotypic hallmarks of carcinogenicity and a panel of genes related with cancer progression were studied and compared between three different time points: 24h, 15 and 30 weeks . **Increased genotoxic damage,** anchorage independent growth ability **and invasive potential was observed after 30 weeks of exposure. Gene expression analysis** results were in concordance with an incremented response in the amount of DEGs over time. GSEA **disclosed EMT and RAS signaling pathways as candidates.** Overall, the results suggest that long-term exposures to PET-NPLs exhibit a carcinogenic potential in BEAS-2B cells.



Results

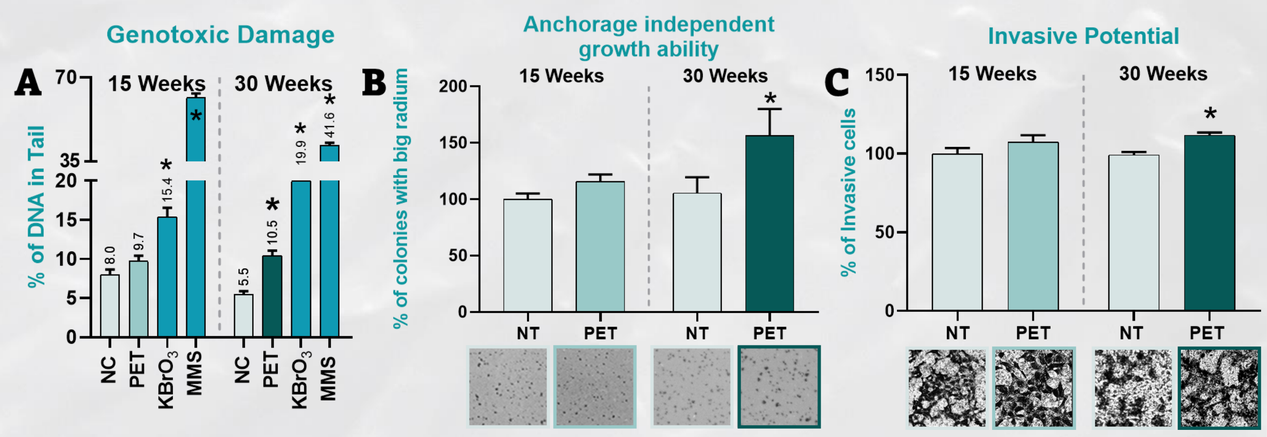


Figure1. *Genotoxic* damage, anchorage independent growth ability and invasive potential of BEAS-2B cells after 15weeks and 30 weeks of exposure.

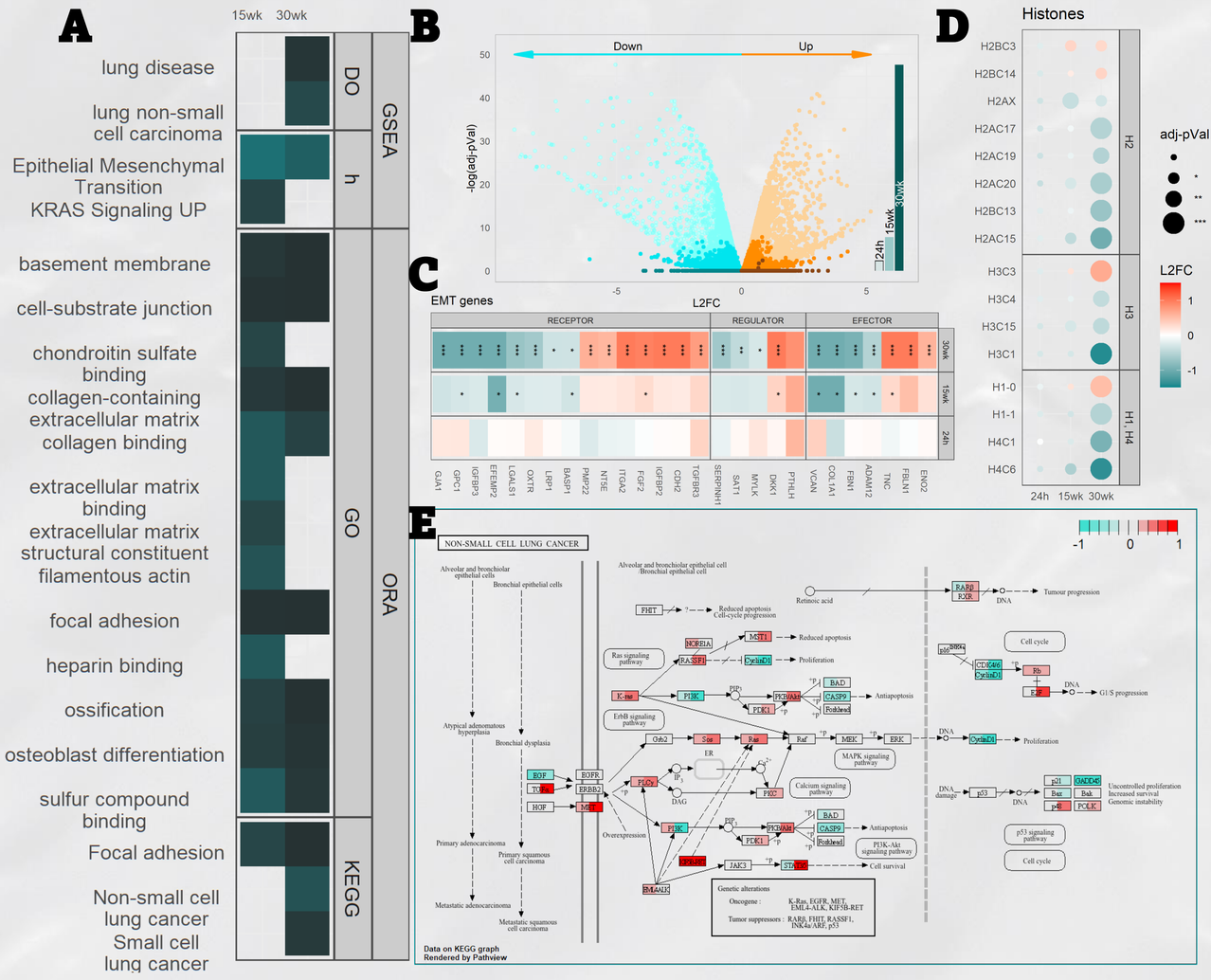


Figure 2. (A) Transcriptomic pathways related with carcinogenic progression, (B) DEGs expression within time of exposure, (C) Gene panels related to EMT pathway, (D) Histone panel, (E)Non-small cell lung cancer data after 15 and 30 weeks of exposure.

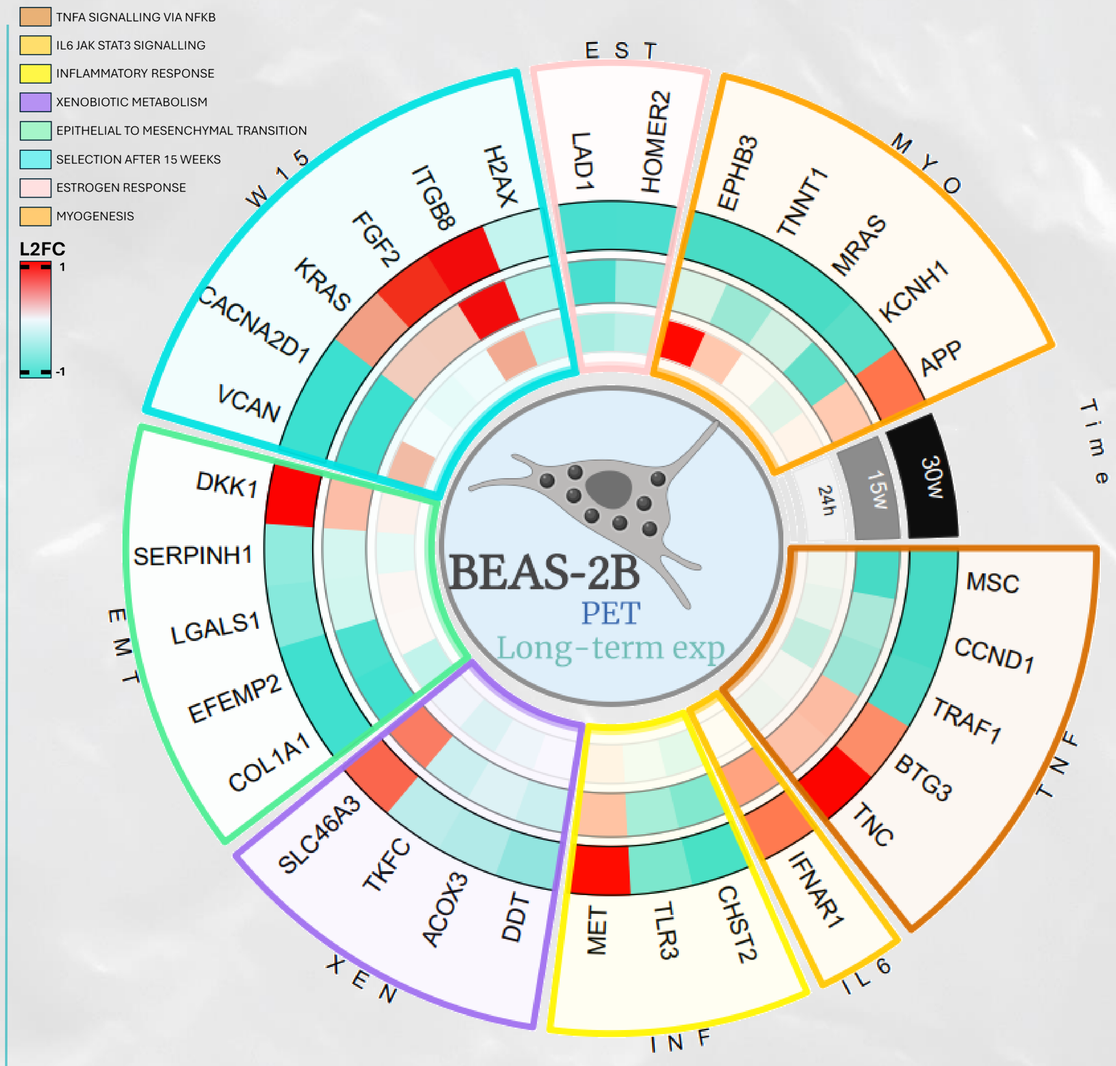


Figure 3. Biomarker candidates on GSEA pathways after 30 weeks of exposure and their progress during time.

Conclusions

After 30 weeks of PET-NPLs exposure, BEAS-2B cells disclose phenotypical carcinogenic hallmarks. Transcriptomics support cancer-related pathway enrichment.

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