Early growth response gene 2 (EGR2)

Air pollution is linked to brain inflammation, which accelerates tumorigenesis and neurodegeneration. The molecular mechanisms that connect air pollution with brain pathology are largely unknown but seem to depend on the chemical composition of airborne particulate matter (PM).

Ljubimova et al., sourced ambient PM from Riverside, California, and selectively exposed rats to coarse (PM2.5-10: 2.5-10 µm), fine (PM<2.5: <2.5 µm), or ultrafine particles (UFPM: <0.15 µm). They characterized each PM type via atomic emission spectroscopy and detected nickel, cobalt and zinc within them. They then exposed rats separately to each PM type for short (2 weeks), intermediate (1-3 months) and long durations (1 year). All three metals accumulated in rat brains during intermediate-length PM exposures. Via RNAseq analysis we then determined that intermediate-length PM2.5-10 exposures triggered the expression of the early growth response gene 2 (EGR2), genes encoding inflammatory cytokine pathways (IL13-Rα1 and IL-16) and the oncogene RAC1. Gene upregulation occurred only in brains of rats exposed to PM2.5-10 and correlated with cerebral nickel accumulation.

They hypothesize that the expression of inflammation and oncogenesis-related genes is triggered by the combinatorial exposure to certain metals and toxins in Los Angeles Basin PM2.5-10 [1)].

1)
Ljubimova JY, Braubach O, Patil R, Chiechi A, Tang J, Galstyan A, Shatalova ES, Kleinman MT, Black KL, Holler E. Coarse particulate matter (PM(2.5-10) in Los Angeles Basin air induces expression of inflammation and cancer biomarkers in rat brains. Sci Rep. 2018 Apr 9;8(1):5708. doi: 10.1038/s41598-018-23885-3. PubMed PMID: 29632393; PubMed Central PMCID: PMC5890281.