Supplementary data 1 Expression of novel common immediate-early genes after WP exposure that are not associated with immune response

Oas3, an enzyme essential for the recognition of invasive viral nucleic acids by RNAse L via the production of the second mesanger 2'- 5'-oligoadenylate,[1] and Cidea, that is a gene with an unrecognized function in lung, however, involved in metabolic regulation in other organs.[2] Oas3 mRNA is increased after HepG2 cells are exposed to tetrachlorodibenzodioxin that is a component of smoke,[3] while Cidea mRNA is dysregulated in lung by exposure of benzo(*a*)pyrene, a polycyclic aromatic hydrocarbon.[4]

The only gene expression that is downregulated in the set of common IEGs is UCP1. UCP1 expression is a marker of thermogenesis in brown adipocytes and is also found in livers.[5] UCP1 protein uncouples the mitochondrial membrane proton gradient in the respiratory chain with the purpose of using the proton motive force as heat generator instead of ATP production.[6] UCP1 is decreased, as are other markers of thermogenesis, under oxidative stress in myocytes.[7] UCP1 is increased by chronic exposure to nicotine in brown adipose tissue of rats[8] and humans.[9] It has been shown that an acute dose of nicotine decreased energy expenditure without changes in respiratory quotient in mice, which suggests that effects of UCP1 on thermogenesis are dependent on the length of exposure.[10]

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