The aim of this master thesis was to investigate the ability of selected individual carcinogenic polycyclic aromatic hydrocarbons (c-PAHs: benzo[a]pyrene, B[a]P; dibenzo[a,l]pyrene, DB[a,l]P), an artificial mixture of c-PAHs (c-PAH mix) and extractable organic matter (EOM) from urban air particulate matter (PM) to induce oxidative damage in vitro. Two cell lines (human hepatoma cells, HepG2, and human diploid lung fibroblasts, HEL) were treated for 24 h and 48 h with various concentrations of compounds or mixtures. The studied oxidative stress markers included 8-oxodeoxyguanosine (8-oxodG) as a marker of oxidative DNA damage, 15-F2t-isoprostane (15-F2t-IsoP) as a marker of lipid peroxidation and protein carbonyl groups as a marker of oxidative damage to proteins. The response of the cell lines to the tested compounds and mixtures differed substantially. In summary the results demonstrate the ability of EOM to induce oxidative damage to DNA and lipids after 24 h of treatment and to proteins after 48 h, in HepG2 cells. The effect of c-PAHs was substantially less. The induction of oxidative damage by c-PAHs and EOM in HEL cells was weak. Since c-PAHs had lower ability to cause oxidative damage that was limited only to longer incubation periods, it is probable that other components of EOM are responsible for increased levels of oxidative markers in HepG2 cells.