Differential effects of PAHs and PAHs-Quinone on oxidative stress damage in A549 cells

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A variety of chemical compounds present in the environment getting more threaten human health, such as cancer, asthma, allergic, and skin disease. Especially, several environmental pollutants can cause adverse health effects through the generation of oxidative stress. Among chemical compounds, polycyclic aromatic hydrocarbons (PAHs), generated through the burning of fossil fuels and incomplete combustion of organic matters, are widespread in the environment and known as carcinogens/endocrine disruptors.

Quinones represent a class of toxicological intermediates which can create a variety of hazardous effects *in vivo*, including acute cytotoxicity, immunotoxicity, and carcinogenesis. Alternatively, quinones are highly redox active molecules which can redox cycle with their semiquinone radicals, leading to formation of reactive oxygen species (ROS). Oxidative stress, caused by ROS, is reported to activate the transcription factor nuclear factor kappa B (NF-κB) in cell line. The redox-sensitive transcription factor NF-κB plays an important role in the expression of a variety of genes participate in regulating the immune response, cell survival, inflammation, and cancer. In addition, activation of the MAPKs, which include ERK, JNK and p38 pathways, contribute to induction of NF-κB activity in response to an array of extracellular stimuli.

This study shows that PAHs and PAHs-quinone have effects through the generation of ROS. To determine whether oxidative damage caused by PAHs-quinone by way of production ROS, we incubated A549 cells with 5 μM of PAHs or PAHs-quinone for 1 hr and measured the generation of ROS and depletion of GSH and transcription factor NF-κB. To examine the intracellular ROS, several PAHs-quinone enhanced generation of ROS significantly. In contrasts, PAHs failed to exert oxidative damage effects rather than PAHs-quinone. Especially, enhanced intracellular ROS level was observed Acenaphthene (ANT) treated cell in the highest. Indeed, oxygenated derivatives PAH, Acenaphthenequinone (AQ), generated ROS higher than ANT. These enhancements showed PAHs-quinone involved an inducing of oxidative damage. A similar degree of generation was observed in Phenanthrene (PA) and 9,10-Phenanthrenequinone (PQ).

Table 1 ROS induction during treatment PAHs and PAHs-Quinone

	ROS level	PAHs (5μM)	ROS level	PAHs-Quinone (5μM)	ROS level
Control	100.00 ± 3.14	Phenanthrene	100.86 ± 0.17	9,10-Phenanthrenequinone	156.68 ± 1.16
H_2O_2	146.73 ± 1.76			Phenanthrene-1,4-quinone	92.95 ± 1.22
NAC	34.54 ± 0.33	Naphthalene	93.31 ± 1.50	1,2-Naphthoquinone	65.98 ± 0.03
		Acenaphthene	120.48 ± 0.74	Acenaphthenequinone	147.64 ± 0.63
		Benz(*a*)anthracene	99.47 ± 2.12	1,2-Benzanthraquinone	96.02 ± 3.17
		Anthracene	96.83 ± 2.11	Anthraquinone	89.85 ± 0.77
				1,4-Anthraquinone	86.44 ± 3.54
		Chrysene	106.96 ± 4.52	1,4-Chrysenequinone	135.38 ± 1.79