TOXICITY OF POLYCYCLIC AROMATIC HYDROCARBONS (PAHs) IN DIESEL EXHAUSTS

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Abstract

The paper presents results of analysis of Polycyclic Aromatic Hydrocarbons (PAHs) concentration and toxicity emitted from 1,9 TDI self-ignition. Because of unstable parameters of self-ignition engine work (pressure and temperature jumps), PAHs were extracted from two phases: gas phase and solid phase (particle matter - PM). A chromatographic method (Capillary Gas Chromatography) of polycyclic aromatic hydrocarbons identification and analysis, because of their low level of concentration in exhaust gases, needed to be supported by sample purification and enrichment stages. Calibration of the chromatograph was made by attested mixture of 16 model samples (according to EPA, USA). Two different methods for toxicity estimation was used in this researches. The authors used relative carcinogenic coefficients (RCC) which was determined by Nisbet and LaGoy for individual polycyclic aromatic hydrocarbons in relation to benzo(a)pirene. Samples consist PAHs was also tested for cytotoxicity in standardized cell-culture system (human cell line A549, mouse fibroblasts line cell L929). Cell growth, cell morphology and cell viability were used as parameters to determine the cytotoxicity of the materials. The measure the lethality effect on cells was determined spectrophotometrically with the use of a mitochondrial enzyme activity assay for mitochondrial succinate dehydrogenase activity by 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT assay). Cells were exposed to the biomaterials for 24, 72 and 120 h. The results of in vitro tests are discussible. A lack of correlation between toxicity measurement methods which was used in these researches was observed.

Keywords: *polycyclic aromatic hydrocarbons, exhaust toxicity, relative carcinogenic coefficient*

1. Introduction

Vehicular emissions are one of the principal anthropogenic sources of PAHs. Thus, the dust that is deported eider side of a road has high potential exposure to PAHs generated from vehicular activities [3]. Furthermore, road dust has become an important pollutant source of PAHs to water and air because of the effect of run-water and wind dispersion [5] . Especially diesel engines are responsible PAHs emission because of specific conditions of fuel combustion.

People are exposed to these compounds in the atmosphere, water, food and smoke. PAHs enter

the environment in flue gas, fly ash, residue and wastewater and exist in the atmosphere, soil and water. PAHs are more prevalent in the gas phase and absorbed onto small particles [2].

The most toxic PAHs have been determined and listed according to their mutagenic and carcinogenic characteristics. There are 16 high priority PAHs listed by EPA: naphthalene (NaP), acenaphthylene (AcPy), acenaphthene (AcP), fluorene (Flu), phenantrene (Phe), anthracene (AnT), fluoranthene (Flu), phenanthrene (Phe), anthracene (AnT), fluoranthene (Fla), pyrene (Pyr), benzo(a)anthracene (BaA), chrysene (Chr), benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene (BaP), benzo(e)pyrene (BeP), dibenzo(a,h)anthracene (DBA), indeno(123-c,d)pyrene (IND), Benzo(g,h,i)perylene (BghiP).

Two PAH formation mechanisms have been suggested, pyrolisis and pyro-synthesis, as most PAH formation is related to the organic radicals released during thermal processes [5].

The firs survey of PAHs in ambient air was performed in London in the 1950s, and since then, routine monitoring has been carried out at several locations, locally as well as around the world [3].

The ability of organic chemicals to cause health effects varies greatly from those that are highly toxic, to those with no known health effect. As with other pollutants, the extent and nature of the health effect will depend on many factors including level of exposure and length of time exposed. Eye and respiratory tract irritation, headaches, dizziness, visual disorders, and memory impairment are among the immediate symptoms that some people have experienced soon after exposure to some organics. Many organic compounds are known to cause cancer in animals; some are suspected of causing, or are known to cause, cancer in humans [6].

In the paper the toxicity estimation of polycyclic aromatic hydrocarbons in diesel engine exhaust is presented on example of researches on an inner catalyst effectiveness.

The inner catalyst is a solution design by the authors based on implementation of active factor in combustion chamber of engine. It is suspected that the active factor e.g. precious metal could influence on combustion process by catalyze of fuel-air combustion reaction (even prior-combustion reactions what is correlated with abbreviation of combustion delay period).

2. Experiment

A modified 1,9 TDI . self-ignition engine (diesel engine) was employed as a research engine. An engine modification was application of platinum-rhodium coating on engine valves. Conventional fuel (commercial diesel oil) was used as engine fuel. 1,9 TDI engine was loaded with AVL α 240 engine break. The most In experiment characteristic points of functional engine work was chosen: rotational speed 1800 r.p.m. and engine loads: idle run and 150 Nm. The engine modification was based on application of platinum-rhodium active coating on surface of the engine valves. Zirconium ceramic was used as a catalyst support (also as a local thermal barrier).

Because of unstable parameters of self-ignition engine work (pressure and temperature jumps), PAHs were extracted from two phases: gas phase and solid phase (particle matter - PM). A chromatographic method (Caplillar Gas Chromatography) of polycyclic aromatic hydrocarbons identification and analysis, because of their low level of concentration in exhaust gases, needed to be supported by sample purification and enrichment stages. Calibration of the chromatograph was made by attested mixture of 16 model samples (according to EPA, USA).

Two different methods for toxicity estimation were used in this researches. The authors used relative carcinogenic coefficients (RCC) which was determined by Nisbet and LaGoy for individual polycyclic aromatic hydrocarbons in relation to benzo(a)pirene [1]:

- naphthalene (NaP) – 0.001,
- acenaphthylene (AcPy) – 0.001,
- acenaphthene (AcP) – 0.001,
- fluorene (Flu) – 0.001,

- phenantrene (Phe) – 0.001,
- anthracene (AnT) – 0.01,
- fluoranthene (Flu) – 0.001,
- phenanthrene (Phe) – 0.001,
- anthracene (AnT) – 0.001,
- fluoranthene (Fla) – 0.001,
- pyrene (Pyr) – 0.001,
- benzo(a)anthracene (BaA) – 0.001,
- chrysene (Chr) – 0.01,
- benzo(b)fluoranthene – 0.001,
- benzo(k)fluoranthene – 0.001,
- benzo(a)pyrene (BaP) – 1.00,
- dibenzo(a,h)anthracene (DBA) – 1.00,
- indeno(123-c,d)pyrene (IND) – 0.10,
- Benzo(g,h,i)perylene (BghiP) – 0.01.

Each, detected in exhaust, PAH concentration was multiply by the RCC of each polyaromatic hydrocarbon and then all factors was sum up to estimate RCC for total PAHs.

Samples consist PAHs was also tested for cytotoxicity in standardized cell-culture system (human cell line A549). Cell growth, cell morphology and cell viability were used as parameters to determine the cytotoxicity of the polyaromatic hydrocarbons from diesel engine exhausts. The measure the lethality effect on cells was determined spectrophotometrically with the use of a mitochondrial enzyme activity assay for mitochondrial succinct dehydrogenase activity by 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT assay). Cells were exposed to the biomaterials for 24, 72 and 120 h.

The cells used in the experiments were the human lung adenocarcinoma cell line A549 (ATCC CCL 165), the mouse fibroblast-like cell line L929 (ATCC CCL-1), and human leukocytes.

The A549 cells were maintained in Dulbecco's modified Eagle's minimum essential medium (DMEM), and the L929 cells were kept in Eagle's supplemented with 10% calf serum (c.s), 2 mM L-glutamine, antibiotics (100 U/mL penicillin and 100 lg/mL streptomycin).

According to Polish standard (PN-EN ISO 10993-5) cytotoxicity was investigated on two cells lines. Measurings in vitro growth of mouse fibroblast L929 and human A549 cell line.

For cytotoxicity test, the cells were seeded in the 24-well plates (Nunc) of 1 ml at density of 2x105 cells/ml, in the culture medium Eagle's or DMEM with 2% calf serum, penicillin and streptomycin was deposited into each well. Samples of the tested samples contain PAHs were added to prepared cells, which were then incubated for 24 h, 48 h and 72 h at 37°C in the atmosphere of 5% CO2 in air.

The cytotoxicity was defined as the highest dilution of test samples that causes 50% or greater destruction of cells.

3. Results and discussion

The results of the research are presented in Tab. 1. Polyaromatic concentration in diesel exhaust was significantly higher when engine was idling (Tab. 1) in both cases – when engine worked with and without catalyst inside of combustion space. The significant decrease of PAHS concentration (even almost tenfold on idle run and twofold when engine was loaded) was observed when active layers was implicated inside of the engine. The catalyst application was particularly effective when engine was idling (Tab. 1).

The Relative Carcinogenic Coefficient (RCC) estimated for total PAHs detected in diesel exhaust was by one order of magnitude smaller after the inner catalyst application. It is connected almost directly with PAHs concentration in combustion gases.

Tab. 1. The results of concentration, Relative Carcinogenic Coefficient (RCC) estimation and cytotoxicity of polycyclic aromatic hydrocarbons (PAHs) in diesel engine exhaust

Parameter	Engine without catalyst		Engine with catalyst	
	Engine load			
	Idle run	150, N·m	Idle run	150, N·m
PAHs concentration, μg/dm^3	0.12912	0.10680	0.01319	0.04349
Relative Carcinogenic Coefficients	0.00013	0.00011	0.00001	0.00004
Cytotoxiciy [1)], %	> 50 [2)]	50	Non-toxic	50

1) The highest dilution of a test samples that causes 50% or greater destruction of cells (the lowest concentration the highest toxicity), 2) The concentration caused a degradation of a part of the cells line.

Analysis of the diesel engine shows that cytotoxicity of examine samples was insignificant (≥ 50%). The samples consist of PAHs from diesel engine with the inner catalyst exhaust (Pt/Rh active ceramic on 1,9 TDI engine glow plugs) was less cytotoxic than without active coating inside of the engine combustion space.

On the Fig. 1 an example of microscope picture of control-sample (a) and a sample contains cells degraded by contact with polycyclic aromatic hydrocarbons from diesel engine exhausts (b) is shown.

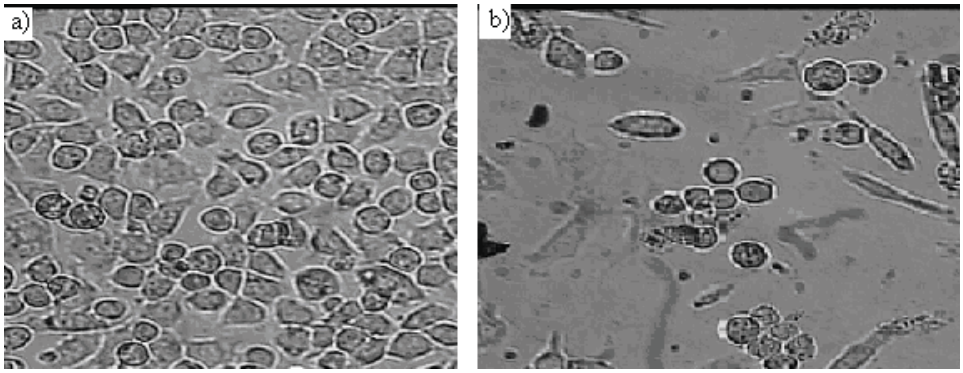

Fig. 1. The examples of microscope pictures presents human lung cells in control-sample (a) and cells degraded by contact with PAHs from diesel engine (b)

The important notice is a fact that lack of correlation between a applied toxicity measurement methods. Because of a serious problem of toxic emissions from diesel engine which is, according to recent publications [1-7]), often determine by emitted hydrocarbons, it is necessary to define and unify the method of toxicity estimation.

4. Conclusions

It has been found that implementation of catalytic coating on engine glow plugs causes decrease in polycyclic aromatic hydrocarbons concentration in exhaust gases, what results in decrease of the sum of Relative Carcinogenic Coefficient (RCC).

The samples consist of PAHs from diesel engine with the inner catalyst exhaust (Pt/Rh active ceramic on 1,9 TDI engine glow plugs) was less cytotoxic than without active coating inside of the engine combustion space.

The results of in vitro tests are discussible. A lack of correlation between toxicity measurement methods which was used in these researches was observed.

Because of a serious problem of toxic emissions from diesel engine it is necessary to define and unify the method of toxicity estimation.

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