THE COMPARATIVE ESTIMATION OF THE RADIATION AND CHEMICAL CARCINOGENIC RISK INDUCED BY THE ATMOSPHERE CONTAMINATION DUE TO RELEASES FROM COAL-FIRED POWER PLANTS

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ABSTRACT

In experiments with 1000 white mongrel mice which inhaled benzo(a)pyrene (BP) and fly coal ash for a long time, these agents increased significantly lung tumour incidence, with the latent period shortened. BP is found to be 10-1000 fold more carcinogenic then fly coal ash. The BP inhalation at a sanitary standard level (0.1 μg/100 m^3 of air) appeared to be equivalent, in murine risk, to the whole-body exposure to a total gamma dose of about 2 Sv. The coal ash inhalation in a concentration of 0.05 mg/m^3 caused the same risk as a dose of 0.05 Sv.

INTRODUCTION

Up to date, relativ contributions of different carcinogenic agents to lung cancers in the population are not quite clear. Therefore, an attempt was made to obtain experimentally a comparative estimation of lung cancer risks due to atmospheric concentrations, actually occurring in population centres, of BP and fly ash released from coal-fired power plants.

MATERIAL AND METHODS

The study involved 1000 white mongrel female mice. Mongrel mice, like man, are known to develop spontaneous lung tumours.

Chronic administration of ash or BP was carried out in special inhalation chambers. In the BP experiment, the mice spent in the chamber 6 hr a day 5 times a week for 3 months; in the coal ash experiment, the chamber residence time was 7 hr a day during 5 months, a total of 48 times. AMADs of coal ash and BP particles were 4.1 and 5.9 μm, respectively. Experimental animals' lungs retained 25% of fly coal ash particles and 9% of BP dust.

The animals were followed up to their natural death. The dead were subjected to autopsy and post-mortem examination.

In mathematical data processing, the Student parameter criterion was used, and the cumulative probability of lung tumour incidence was estimated by an interval procedure.

RESULTS

The investigational patterns and lung tumour incidence in mice are summarized in Table 1.

Table 1

Lung tumour incidence related to coal ash and BP intakes by ingalation

Item	Animal group			
	1(control)	2	3	4
1. COAL ASH				
Chamber-air concentration, mg/m^3	0	2.5	5.0	10.0
Retained in the lung, mg/g of tissue *)	0	1.0	2.0	4.0
Animals with lung tumours, % **)	28	37	61	78 ***)
Animals with ash-induced tumours, %	0	9	33	50
2. BENZO(A)PYRENE				
Chamber-air concentration, μg/m^3	0	0.2	6.3	78.0
Retained in the lung, μg/g of tissue	0	0.0025	0.8	9.0
Animals with lung tumours, %	14	23	37	40 ***)
Animals with BP-induced tumours, %	0	9	13	26

Notes: *) - The ash or BP dose (amount) retained in the lung was calculated by AMADs of respective aerosols.
**) - Using the interval procedure.
***) - $P < 0.01$ as compared with control.

The evidence from Table 1 suggests a positive carcinogenic effect of BP and fly coal ash in the study.

Table 2 gives average latent periods (ALPs) of tumours, time between the onset of administration and the first tumour appearance, and ALPs for the first 25% and 50% of murine tumours following the coal ash inhalation.

The date from Table 2 show a carcinogenic capacity of fly coal ash, thus supporting the results from Table 1. It is noteworthy that coal ash is considerably less carcinogenic than BP - by one to three orders for different concentrations. The dose-response curves for these agents differ: close to linearity for ash and far from it for BP.

Table 2

Average latent periods (ALP) of lung tumours by coal ash concentrations inhaled

Item	Animal group			
	1(control)	2	3	4
	Ash concentration, mg/m^3			
	0	2.5	5.0	10.0
ALP	615±45	545±47	523±40	457±59 **)
First tumour appearance	488	217	283	221
ALP 25%	490±3	402±47	349±47 **)	228±9 *)
ALP 50%	504±10	458±49	409±36 **)	280±30 *)

Notes: *) - $P < 0.01$ as compared with control;
**) - $P < 0.05$ as compared with control.

Mathematical date processing of both experiments gave the following dose-response equations:

for coal ash $Y = 5.2X + 28$ (1)

for BP $Y = 16.4 (Z)^{0.19}$. (2)

where Y is the percentage of animals with lung tumours;
X is the coal ash dose (mg/g of lung tissue);
Z is the BP dose (μg/g of lung tissue).

DISCUSSION

The findings from Table 1 and 2 should be discussed, above all, with respect to the question: if coal ash exhibits a carcinogenic capacity, why then this capacity has not been revealed in other similar studies, e.g. in a detailed one by S.Persson et al [1]?

An analysis of radionuclides, BP and some metals contained in ash showed that the carcinogenic effect of coal ash might result from chromium, nickel, cadmium, arsenic and other metals. Such metals as chromium, beryllium, etc., accumulated in the lung amounted to 10E17 molecules and more [2]. As for the latter part of the question, in the above mentioned [1] and other studies, BP and coal ash were administered to animals intratracheally in suspension, rather

than by natural inhalation. Very soon, the agents administered were rejected. In our stydy, the agent contact with the lung tissue was much longer, without any serious disturbance of the lung physiology.

Another issue of importance is a comparative risk estimation for BP and ash.

Does a low, as compared with BP, carcinogenic activity of coal ash mean no danger? Apparently not, given millions of tons annually released from solid fuel plants into the atmosphere. Moreover, a direct carcinogenic effect of ash was observed in our experiments at air concentrations very close to those actually encountered in the atmosphere. It should also be noted that the carcinogenic effect of ash, like other carcinogenes, e.g. BP, may have no threshold.

Table 1, equations (1), (2) and dose-response relationships known for ionizing radiation permit rough estimates of the radiobiological carcinogenicity equivalent for BP and fly coal ash. Similar death risks from induced cancer are caused by the whole-body exposure to a total gamma dose of 0.054 Sv and by the fly coal ash intake at a concentration of 0.05 mg/m^3 . For a BP concentration of 0.1 $\mu g/100\ m^3$, the radiobiological carcinogenicity equivalent is 1.98 Sv.

CONCLUSION

Earlier, the authors were the first to demonstrate experimentally the ability of fly coal ash inhaled with the air to induce lung cancer [2, 3]. It is also for the first time that the dose-carcinogenic response relationship has been studied for the coal ash and BP intake by inhalation.

The USSR current standards for coal ash and BP concentrations in the air do not prevent the population from a substantially higher risk than that caused by exposure to radiation within radiation protection limits.

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