Scand J Work Environ Health 1988;14(1):55-56
Issue date: 1988

Aromatic deoxyribonucleic acid adducts in white blood cells of foundry and coke oven workers.

by Hemminki K, Perera FP, Phillips DH, Randerath K, Reddy MV, Santella RM

Affiliation: Institute of Occupational Health, Helsinki, Finland.

This article in PubMed: www.ncbi.nlm.nih.gov/pubmed/3393882

Print ISSN: 0355-3140 Electronic ISSN: 1795-990X

Scand J Work Environ Health 14 (1988): suppl 1, 55—56

Aromatic deoxyribonucleic acid adducts in white blood cells of foundry and coke oven workers

by Kari Hemminki, MD,[1] Frederica P Perera, DPH,[2] David H Phillips, PhD,[3] Kurt Randerath, MD,[4] M Vijayaraj Reddy, PhD,[4] Regina M Santella, PhD[2]

We have undertaken a blind study among foundry workers to establish dose-response relationships using the ^{32}P-postlabeling technique and benzo(a)pyrene (BP)-deoxyribonucleic acid (DNA) antibodies. In this report we summarize the first results of these studies and compare the data obtained by the two methods. Individual studies have been reported in detail elsewhere (3 and unpublished manuscript of Perera et al).

Materials and methods

Blood samples were obtained from healthy volunteers working in a Finnish iron foundry. Industrial hygiene measurements for polycyclic aromatic hydrocarbons were carried out in the foundry in 1978—1980; as the work processes have remained practically unchanged since then, these measurements were used by two industrial hygienists familiar with the foundry to grade the volunteers for daily exposure by job description. BP levels in the workplace atmosphere were used as guidelines to assign the exposure to polycyclic aromatic hydrocarbons as high (>0.2 μg BP/m^3), medium (0.05—0.2 μg/BP/m^3), and low (<0.05 μg BP/m^3). Reference samples were obtained from individuals coming from different parts of Finland to the Institute of Occupational Health for examination. Their job titles did not indicate occupational exposure to polycyclic aromatic hydrocarbons. Information on current smoking habits (cigarettes/d) was obtained for all the subjects.

Coded samples were assayed by the competitive enzyme-linked immunosorbent assay essentially as described previously with fluorescence detection (2). For postlabeling a previously published method was used (1, 4, 5).

Results

The BP-DNA antigenicity of foundry worker DNA samples correlated with the estimated exposure levels as shown in figure 1.

When adjustment was made for cigarette smoking and time since vacation, BP exposure was significantly related to adduct levels ($P = 0.0001$). Each of the three exposure groups had significantly elevated adduct levels in comparison to those of the referents. Among the exposed workers, the low group differed significantly from the high and medium ones. Smoking, age, or sex had no significant effect on aromatic DNA levels.

The foundry workers' samples were assayed by the ^{32}P-postlabeling technique in two laboratories. Laboratory 1 gave the results as the estimated number of adducts/10^8 nucleotides, while laboratory 2 gave an adduct score between zero and three. In laboratory 1 the DNA samples from workers in the high- and medium-exposure categories had an average of 1.8 polycyclic adducts/10^8 nucleotides; DNA from the low-exposure category and from the referents had mean adduct levels of 0.06 and 0.2/10^8 nucleotides, respectively, which was the approximate limit of sensitivity of the assay. A large number of samples was run by laboratory 2. The adduct score was 2.0 for the high-exposure group, 1.8 for the medium-exposure group, 0.8 for the low-exposure group, and 0.2 for the reference group. These values showed a clear correlation with the estimated exposure levels at the workplace.

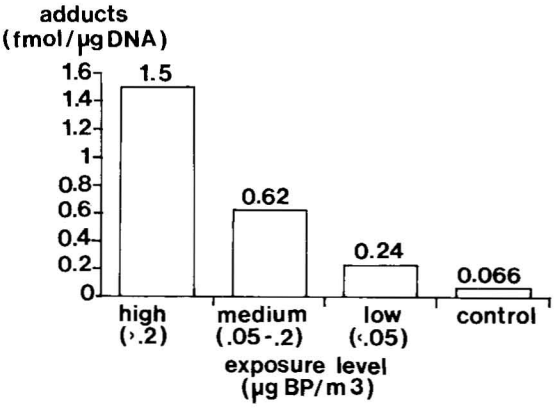

Figure 1. Benzo(a)pyrene (BP) antigenicity of white-blood cell deoxyribonucleic acid (DNA) from foundry workers with high, medium, and low exposure to polycyclic aromatic hydrocarbons and from a reference group (control).

[1] Institute of Occupational Health, Helsinki, Finland.
[2] Columbia University School of Public Health, Division of Environmental Sciences, New York, New York, USA.
[3] Chester Beatty Laboratories, Institute of Cancer Research, London, England.
[4] Department of Pharmacology, Baylor College of Medicine, Houston, Texas, USA.

The correlation coefficients of the results were determined. The numeric results of the antibody assay and the postlabeling assay by laboratory 1 were transformed into logarithmic results; the postlabeling score determined by laboratory 2 was used as such. The antibody assay and the postlabeling score had a relatively high correlation of 0.70, while the correlation to the postlabeling results of laboratory 1 was lower, but still reasonable, ie, 0.46. The correlation between the postlabeling results of laboratories 1 and 2 was intermediate, ie, 0.51.

References

1. Gupta RC, Reddy MV, Randerath K. ^{32}P-post-labeling analysis of non-radioactive aromatic carcinogen-DNA-adducts. Carcinogenesis 3 (1982) 1081—1092.
2. Perera FP, Poirier MC, Yuspa SH, Nakayama J, Jaretzki A, Curren MM, Knowles DM, Weinstein IB. A pilot project in molecular cancer epidemiology: Determination of benzo[a]pyrene-DNA adducts in animal and human tissues by immonassays. Carcinogenesis 3 (1982) 1405—1410.
3. Phillips DH, Hemminki K, Alhonen A, Hewer A, Grover PL. Monitoring occupational exposure to carcinogens: Detection by ^{32}P-postlabelling of aromatic DNA adducts in white blood cells from iron foundry workers. Mutat Res (in press).
4. Phillips DH, Hewer A, Grover PL. Aromatic DNA adducts in human bone marrow and peripheral blood leukocytes. Carcinogenesis 7 (1986) 2071—2075.
5. Reddy MV, Randerath K. Nuclease P1-mediated enhancement of sensitivity of ^{32}P-postlabeling test for structurally diverse DNA adducts. Carcinogenesis 7 (1986) 1543—1551.