FOUNDED 1907

JOURNAL OF THE AIR POLLUTION CONTROL ASSOCIATION

(Formerly Air Repair)

A QUARTERLY JOURNAL DEVOTED TO AIR PURIFICATION

Printed in the United States
Editor: H. C. Ballman
Editorial Asst.: Jane Miller, T. Gra
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AIR POLLUTION CONTROL ASSOCIATION
BOX 93
WILMERDING, PA.

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1957
PLAINTIFF'S EXHIBIT
SA 189

MAY 1957 — CONTENTS — Vol. 7, No. 1

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The Editor welcomes writers interested in air pollution problems and encourages them to use the pages of this journal for the expression of their opinions. But neither he nor the Association assumes responsibility for the validity of the opinions expressed herein.

JOURNAL OF THE AIR POLLUTION CONTROL ASSOCIATION is published quarterly by the Air Pollution Control Association, with torial and executive offices at 609 Station Street, Wilmerding, Pa. Officially renamed Journal of the Air Pollution Control Association in May 1955. me Air Repair eliminated November 1956.

Printed in U. S. A. by Cox Printing Company. Entered as second class matter at the Post Office, Wilmerding, Pa.

5. Some factor in auto exhaust makes it a more potent smog-former than the individual hydrocarbons added.

6. The role of nitrogen oxides in smog formation is a complex one and may be dependent on the type and structure of the organic compound with which it reacts, as well as the concentration ratio.

Future Work

Work done to date on this project has raised many questions that must be answered before a logical solution to the smog problem can be devised. Current work in the facility at Midwest Research Institute is directed toward determining the smog-forming potential of different hydrocarbon types. Work on cycles of auto exhaust other than idle is also under way.

Summary

Hydrocarbons and oxides of nitrogen, in concentrations similar to those found in the Los Angeles atmosphere, were subjected to the influence of sunlight in large glasshouses. Odor, eye irritation, and oxidant concentrations, similar to those of Los Angeles smog, developed in all cases. The sources of hydrocarbons were both auto exhaust and evaporated hydrocarbons.

Eye irritation and oxidant concentration both increased as the concentration of hydrocarbons charged to the chambers was increased. Auto exhaust hydrocarbons were considerably more effective in producing oxidant than were the 2 added hydrocarbons, 3-methylpentane and hexene-1. The effect of oxides of nitrogen concentration was more obscure and complex, but nonetheless important.

It was concluded that automobile exhaust is an important source of smog and must be controlled.

The Chemical and Biological Consideration of Atmospheric Carcinogenic Agents*

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A significant increase in the incidence of cancer of the lung has been reported from various regions of the world during the last 20 to 30 years.[5] This sudden and widespread increase is probably associated with the introduction of carcinogenic agents into the external environment at a time consistent with biological correlation. Of the agents advanced as being causally related to the increase, atmospheric pollution[22] and cigarette smoking[4] have been suggested as the 2 most significant. Since lung cancer in common with all cancers most certainly has multiple factors associated with its initiation and promotion, it appears to us to be an oversimplification to conclude at this time that any single factor is significant to the exclusion of all others. The present report is concerned primarily with the atmosphere as one suggested source of pulmonary carcinogenic agents.

For the sake of clarity, the various known and suspected carcinogenic agents in the atmosphere will at the outset be discussed individually. Any combined or possible inhibitory effects which may possess and their mode of action in relation to the development of cancer will be considered later. A relatively large group of substances strongly suspected as carcinogenic for the lung has already been described. They are being mentioned here for the purpose of completeness as their current significance probably exists only in terms of occupational or localized industrial hazards. Included in this group would be the radioactive dusts and gases[9]; the metal oxide and metal salt dusts and fumes from such elements as chromium[1] and nickel[11]; asbestos[21]; and the organic material, isopropyl oil.[23].

The chemical carcinogens we are primarily concerned with today are those distributed throughout the atmosphere. They fall into 2 groups; polycyclic aromatic hydrocarbons of traditional experimental carcinogenicity and the oxidation products of aliphatic hydrocarbons. The most common source of the former is the exhaust products of internal combustion engines, both gasoline and diesel.[16,17] The latter are derived from unburned gasoline released to the atmosphere incidental to petroleum refining, storage, and distribution as well as from vehicular exhausts.[18] Finally, a group of inorganic dusts will be discussed in relation to a possible role in the pathogenesis of cancer.

A discussion of carcinogenesis should include: 1) consideration of the chemical nature of the suspected or incriminated materials, 2) mechanisms for their deposition on the respiratory epithelium, 3) evaluation of their residence and stability in the respiratory tract and 4) the effect of various agents

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upon the epithelium *per se* as well as upon one another. Incidental mention will be made of the possible contribution of cigarette smoke in the enhancement of the carcinogenic effect of air pollutants.

The aromatic polycyclic hydrocarbons with carcinogenic potency have been the subject of long and intensive study. Their universality in the atmosphere has been reported by investigators in Great Britain[22] as well as by ourselves.[23] The most potent of these compounds is 3,4-benzpyrene. It may be accompanied by other organic compounds, a few of them showing weak activity as, for example, chrysene. Soot is the most ubiquitous carrier of these carcinogenic agents. A strong likelihood exists that other potent aromatic polycyclic carcinogens of the 1,2-benzanthracene type as present in tar fumes or road asphalt dust may be of etiologic significance[2] The experimental carcinogenicity of the polycyclic aromatic compounds has been demonstrated following their recovery from the atmosphere.[3, 19, 20]

Carcinogenicity has also been demonstrated in atmospheric samples either free of aromatic polycyclic hydrocarbons or samples in which their concentration is so low as to be presumably of responsibility for tumor production of these samples.[13] The presence of this second group of compounds belonging to the aliphatic hydrocarbon group has been postulated as being responsible for the carcinogenicity in the polycyclic aromatic-free samples. While this group of compounds is much less well defined chemically, their mode of formation can be outlined. The exposure of gasoline in low concentration to ozone results in the formation of an aerosol with marked oxidizing power. The chemical reactions responsible can be theoretically elucidated. The degree to which the various groups of resulting compounds are formed and to what extent they survive in the atmosphere is not known. Suffice it to say that the reaction occurs exceedingly rapidly and the products contain a variety of oxidants. To a limited degree these have been separated by distillation, chromatography, and standard chemical means. Ozonides, peroxides, peracids, peresters, and end products consisting of aldehydes, ketones, acids, and epoxides probably result from this reaction[12] A complex mixture of these compounds is obtained even following the initiation of the reaction using pure 2-hexene and ozone under laboratory conditions. The complexity of the reaction products is undoubtedly formidably increased when one considers the variety of compounds emitted from vehicular exhausts. This coupled with the instability, explosive nature, and low concentration of the oxidation reaction products makes large scale bioassay for carcinogenicity impractical at present. Consequently, compounds considered to be structurally similar to the reaction products have been chosen for carcinogenic studies. These are more easily obtainable and their purity can be established.

Prior to discussing biological data obtained from carcinogenic studies, it will be profitable to discuss the problems inherent in the testing of compounds or groups of compounds for carcinogenicity. Choice had to be made of the species to be tested, the target tissue, the mode of administration, the dose of the carcinogen, its vehicle, and the duration of experimental time. With current interest centered primarily around lung cancer, the tissue selection appears obvious, that is, the lung. Experimental considerations, however, made it much more expedient to begin with skin painting and subcutaneous injection techniques using mice and rats as the species. This permitted the accumulation of knowledge relative to the toxicity, stability, and the host's reaction to the administered compounds. Following the demonstration of skin carcinogenicity, using extracts of soot from diesel engine exhaust, gasoline engine exhaust, and polluted air, a series of experiments were undertaken using various fractions of these materials for the purpose of identifying specific carcinogens and measuring relative potencies. Only subsequent to these basic experimental endeavors was it expedient to perform inhalation experiments using the lung *per se* as the target tissue. This experimental sequence has been followed through to completion in the instance of aliphatic oxidation products[14] and is in the process of completion utilizing aromatic polycyclic hydrocarbons.

Although data relating to the phenomenon of lung cancer as currently occurring in humans may be only inferentially studied in experimental species, we have by means of experimental methods through data obtained in our laboratory evolved a theoretical mechanism for pulmonary carcinogenicity in man. This necessitates a consideration not only of the carcinogenic agents *per set* but other environmental factors which adversely affect the respiratory epithelium so that carcinogenic activity may result.

The demonstration of carcinogenic materials in the atmosphere required investigation as to their quantitative presence, stability, survival time, and physical state. Representative studies have shown that the polycyclic aromatic hydrocarbon carcinogen, 3, 4-benzpyrene absorbed on soot, is relatively stable when compared to pyrene and other polycyclic hydrocarbons. It is slowly destroyed, however, by polluted air rich in oxidants. The survival time is, however, wholly compatible with respiration and deposition in the lung.[7]

The oxidant material in the atmosphere in addition to the innate carcinogenicity already discussed possesses the property of extracting the aromatic carcinogen from the soot onto which it is normally absorbed. This elutant prop-

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city is of great significance in that it has been previously shown that carcinogenic agents during the state of their desorption on soot are biologically ineffective.[9] At this time it is well to mention that other factors highly capable of being associated with the elution of hydrocarbons from soot are present in our environment. The one in need of great study and worthy of suggestion is cigarette smoke.

Studies by our group and others[12, 14] on the various aspects of air pollution have demonstrated the role of ciliary activity and bronchial mucus secretion as protective factors for the respiratory epithelium. This defense mechanism is operative primarily against inhaled particulate matter. The preponderance of inhaled particulate matter has been shown to be in the particle size range which allows for passage through the nares, the trachea, and the major bronchi with deposition in the smaller bronchi, bronchioles, and alveoli. Particulate matter is then either phagocytized and adsorbed or removed by ciliary action. Studies on human lungs obtained from necropsy reveal that in the instance of soot, approximately 1% of the total amount inhaled during life is retained in the lungs and lymph nodes.[14] Chemical analysis of this soot by ourselves revealed it to be almost entirely free of carcinogenic aromatic polycyclic hydrocarbons. Only minute amounts of pyrene were detectable in all of the lungs studied. This disappearance of the carcinogenic agents suggests that the adsorption bond between them and the soot may have been broken as a result of elutant activity. Whether the liberated carcinogen is harmlessly destroyed or whether it mediates a carcinogenic effect is now under intensive study in our laboratory.

Ciliary movement in association with the mucus barrier serves to remove particulate matter which has been deposited on the lining of the lung. This material is progressively advanced towards the throat region where it is either expectorated or, as is more frequent, swallowed. As mentioned previously, the role of aliphatic oxidation products in the interference with the normal resistance factors of the respiratory epithelium must be regarded in evolving a sequence of lung cancer development. Any interference with these defensive factors reduces ciliary activity and results in extended residence time of particulate matter on the epithelium. This allows for prolonged direct chemical action on the cells *per se*.

Experimentally, this was demonstrated in rabbits that were exposed to synthetic smog of from 1-3 ppm. for periods of from one hr. to several days. Ciliary activity was observed and quantitated, using a dissecting microscope in isolated strips of trachea. Particles used for study were in the size range of 500 mm. Control animals showed a relatively constant rate with particle movement measuring 10 mm./min. In all animals exposed to smog prior to tracheal exteriorization the rate of particle movement was considerably reduced. Smog exposure for 24 hr. completely abolished ciliary activity and particle movement. Measurement of recovery rate as determined by ciliary activity or particle size movement showed a restoration to levels of 35% of normal in 48 hr. after cessation of ciliary activity.

The effect of aliphatic oxidation products on the mucus barrier is equally dramatic. A gradual increase in goblet cell activity and mucus secretion occurs to the point where pools or lakes of mucus form. Following this there is desquamation of the superficial epithelial cells with the resultant exposure of the basal cell layer. This cell layer from which lung cancer presumably originates is then unprotected and carcinogenic agents present and available for biological activity are immediately adjacent to their target. The persistance of the basal cell layer is of further significance in that it allows for restoration of the epithelium following the removal of the irritants. Epithelial replacement is complete with no discernible evidence of prior loss.

Prior to concluding, brief mention of the possible role of inorganic dusts should be made. The stimulating effect of such agents as silica, alumina, and iron oxide on connective tissue proliferation is well known. The recent production of sarcomas by the instillation of these materials in the mesenchyme of experimental species indicates a need for their evaluation as either additive or potentiating agents when associated with other known atmospheric carcinogens.

In conclusion, a review of the factors relating atmospheric pollution to lung cancer strongly suggests that it be regarded as one of the agents etiologically associated with the increase in mortality from lung cancer now being recorded throughout the world. Lung cancer in common with all other neoplasms must certainly have many factors concerned with its initiation and promotion. While the relative significance of these various factors is at present the subject of great controversy, epidemiologic and experimental data obtained from broad programs of lung cancer research strongly support the thesis that air pollution provides a source for agents carcinogenic to the lung. Chemically, carcinogenic agents have been demonstrated in the atmospheric environment, and biologically, a mechanism utilizing laboratory data has been advanced for the pathogenesis of lung cancer. This report certainly does not preclude possible similar roles by other environmental factors.

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