



INTERNATIONAL INFORMATION INSTITUTE

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DEFINITIONS OF DESCRIPTORS AND DESCRIPTOR COMPONENTS USED

(A) the A in parentheses indicates a finding in an experimental animal, as opposed to a human finding for which the descriptor is unmodified

NPB these three letters stand for "no proof but" and are added when the author is expressing an opinion in the admitted absence of real evidence

Causation this term, usually modified by the causative agent such as in "tobacco causation" or "viral causation", indicates a definite expression of opinion that the agent causes or at least helps to cause the disease listed; the term can be further modified by the words "contributing" or "major" in parentheses

Noncausation this is the opposite expression of opinion, i.e., the agent named plays no significant role in the etiology of the disease

Carcinogenicity
Noncarcinogenicity these two words form a similar pair, both referring to the expressed opinion or conclusions of the author

Dose response the direct relationship between the dose administered and the effect obtained, which may be either positive or negative

Etiology this is a much more general term than "causation", and should be used only when the etiology of a disease is discussed in general

Smoking amount this term, often found in conjunction with smoking duration, refers to the number of packs per day

Concession this can be distinguished from a statement of negative opinion in that the author is admitting to a deficiency in a previously expressed positive opinion, i.e., more or less adhering to the positive view of causation or carcinogenicity but admitting that more work is needed, or that the animal findings are not directly applicable to man, etc.

PROJECT IIC

ACCESSION NUMBER 0071

TITLE: PROTEIN DAMAGE BY TOBACCO SMOKE IN VITRO AND ITS INHIBITION BY FILTERS OF PROCESSED WOOL (Eiweisschädigung durch Tabakrauch in vitro und ihre Hemmung durch Filter aus reduzierter Wolle)

AUTHOR(S): Reske, G.

SOURCE: Arzneimittelforschung 13(10): 913-916, October 1963

AFFILIATION: University of Frankfurt, Institute for Physical Biochemistry and Colloid Chemistry, Frankfurt a. M., Germany

ANNOTATION: Photometric studies showed that tobacco smoke inhibits the thermal aggregation of β -lactoglobulin in a buffered aqueous solution. The degree of the effect depends on the conditions used. Although cellulose acetate filters are ineffective, the protein damage induced by tobacco smoke is reduced by about 50% if the smoke is filtered through a material rich in sulfhydryl groups (e. g., wool treated with reducing agents).

ABSTRACT: The findings of Lange on irreversible inactivation of some SH-enzymes by tobacco smoke condensate, and the effect of the latter on the air oxidation of cysteine, reported by Tonge, led the present author to investigate the effect of tobacco smoke on the SH-mediated thermal denaturation of β -lactoglobulin. This seemed of particular interest since "The carcinogenic hydrocarbons demonstrated so far in tobacco tar cannot be considered as the sole cause [of its carcinogenicity] since they are present in such small amount. The degree of protein damage was estimated from the rate of aggregation of the remaining protein, evaluated photometrically. The smoke from a non-filter cigarette which was completely consumed in 1, 4, 8, and 15 puffs produced protein damage of 100, 10-11.5, 33-40, and 51-52%, respectively. In another experiment, the protein damage due to the smoke from either an unfiltered cigarette or one with a cellulose acetate filter which had been consumed in 12 puffs ranged from 50-60%, compared to 25-30% using a cigarette with an SH-filter (reduced wool). Refrigeration or freezing of the system had no effect.

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LG 2018140

80

Primary Descriptors

protein damage
tobacco smoke
sulfhydryl groups
puff frequency
lactoglobulin
cigarette filters
cellulose acetate filters
processed wool filters
tobacco tar carcinogenicity concessions
in vitro studies
tobacco smoke positive dose response

90

Secondary Descriptors

cysteine oxidation
tobacco tar carcinogen content
enzyme inactivation
protein denaturation
protein aggregation

TITLE: REVERSE SMOKING AND ITS ORAL CONSEQUENCES CARIBBEAN AND SOUTH AMERICAN PEOPLES

AUTHOR(S): Quigley, L. F., Jr./Cobb, C. M., Schoenfeld, S., Hunt, E. E., Sr., Williams, P.

SOURCE: J. Am. Dental Assoc. 69:427-442, October, 1964

AFFILIATION: None

ANNOTATION: Examination of 21 habitual reverse smokers for the presence of epidermoid carcinoma of the hard palate revealed varying degrees of leukoplakia on the palate and lips, but no malignancy. The fact of reverse smoking to cause palatal cancer calls into question the presumptive role of tobacco tars and intense radiant heat in carcinogenesis.

ABSTRACT: From a preliminary study in the Netherlands Antilles (South Caribbean) of more than 250 reverse smokers, all of whom but 2 were women, 21 subjects (19 female, 2 male) were selected for complete physical, histological, and oral examinations. These subjects ranged from 38-78 years of age and the majority of them had smoked from 6-20 cigarets per day for 15-63 years. In general, the subjects exhibit heavy tar deposits on the buccal and lingual tooth surfaces; a tough, leathery, and hard palate; reduced saliva flow; and varying degrees of leukoplakia on the palate and lips. No malignant lesions were observed in any of the original 250 or more subjects examined. No explanation can be given for the fact that the general periodontal condition was normal and for some even excellent; further investigation is recommended since, in reverse smoking, "the squamous epithelium is being exposed to the same agents: tobacco tar, nicotine, smoke and heat" as the respiratory tract epithelium, but in the oral case the heat is more direct, resulting in changes typical of inflammatory response to injury. Although several investigators have attributed epidermoid cancer of the hard palate to reverse smoking, this assumption is not suggested by the present data. It is pointed out in the discussion that the incidence of epidermoid cancer of the palate is much higher in Panama, where it occurs primarily in women who practice reverse smoking, than in either the United States or the Netherlands Antilles. It is suggested that the low incidence of palatal epidermoid cancer in the Netherlands Antilles, despite reverse smoking, may result from an ethnic factor, since the population is 70% Negroid.

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PRIMARY DESCRIPTORS

cigarette smoking
reverse smoking
palatal carcinoma tobacco noncausation
tobacco smoke noncarcinogenicity
tobacco tar noncarcinogenicity
epidermoid carcinoma
ethnic factors
tar deposition
oral cancer concessions
respiratory cancer concessions
radiant heat noncarcinogenicity
palatal carcinoma morbidity
sex differences
geographic factors

SECONDARY DESCRIPTORS

smoking
leukoplakia
keratinization
lip lesions
palatal lesions
gingival lesions
Caribbean
Netherlands Antilles
United States
Panama
salivation decrease
Negroes

TITLE: SUMMARY OF THE CONFERENCE
(7th Annual Conference on Research in Emphysema, Aspen 1964)

AUTHOR(S): Wright, G. W.

SOURCE: Medicina Thoracalis 22(3): 420-431, 1965

AFFILIATION: St. Luke's Hospital, Department of Medical Research, Cleveland, Ohio

ANNOTATION: In a critical review, the author points out that although cigarette smoking may be considered an aggravant in chronic lung disease, there is no basis for referring to it as the necessary cause.

ABSTRACT: The author points up the necessity for greater diligence and accuracy in assembling and presenting epidemiological data, with special attention to the use of death certificates and questionnaires. "Death certificates, as originally developed, have very little relevance for our problem" and are "a tool that is entirely unreliable," since the disease listed on the certificate and the conditions found at autopsy show a very poor correlation, particularly for respiratory disease in the United States. After pointing out the difference between a necessary cause of disease, which has to be present in every case, and an aggravant which simply brings a larger number of cases to clinical recognition, he suggests that although cigarette smoke may be an aggravating factor in chronic obstructive bronchopulmonary disease, it is too often referred to as the cause. "Cigarette smoke cannot be thought of as the necessary cause because of the considerable number of individuals who have full-blown chronic bronchitis, . . . but have never smoked a cigarette. . . ." Emphasis is also placed on obtaining a complete smoking history of the individuals who are involved in chronic respiratory disease surveys, and on the need for proper definition of terms such as emphysema and bronchitis. As the influence of smoking on pulmonary function measurements has been shown, it will be desirable to devise a method for measuring smoking intensity, a variable which still seems elusive. The remainder of the discussion ranges over general problems of bronchial infection, proper ventilation/perfusion relationships, including the role of surfactants, anatomical changes in the lung, and blood-gas abnormalities. The problem of viruses is said to be "just so staggering that most of us only speculate about it. . . this is perhaps the area which might give us the greatest step forward in regard to the etiology of both chronic bronchitis and. . . emphysema." Further comment on cigarette smoking is confined to the observation that it can increase the transit time of mucus in the bronchus and consequently increase the chances of infection, since this gives the pathogenic microorganisms more time to multiply.

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Primary Descriptors

tobacco smoking
chronic respiratory disease
chronic bronchitis
pulmonary emphysema
respiratory infection
bronchial mucus
mucus transit time
cigarette smoke
smoking amount
obstructive lung disease
death certificate reliability
questionnaire studies
respiratory disease concessions
respiratory disease etiology
chronic bronchitis tobacco noncausation
bronchitis etiology unknown
emphysema etiology unknown
tobacco smoke aggravant activity
viruses

Secondary Descriptors

pulmonary function
epidemiological studies
lung structure
breathing capacity
cor pulmonale
hypertension
Aspen conference
surfactants
bronchitis definition
emphysema definition

TITLE: EFFECT OF CIGAR SMOKE ON EPINEPHRINE SECRETION IN THE DOG

AUTHOR(S): Westfall, T. C./Watts, D. T.

SOURCE: Proc. Soc. Exp. Biol. Med. 112(4): 843-847, 1963

AFFILIATION: West Virginia Univ. Med. Ctr., Morgantown, W. Va.

ANNOTATION: When 22 anesthetized mongrel dogs inhaled cigarette smoke from a holder inserted into a tracheal cannula, there was a significant increase in epinephrine levels in both the adrenal vein and the peripheral circulation. This increase varied with the smoking rate and is ascribed to the nicotine content of the smoke.

ABSTRACT: The rate of smoking in these studies was regulated by means of a screw clamp on the piece of rubber tubing which served as a bypass for the cigarette, so that the burning time varied between 3.5 and 8 minutes per cigarette (puff frequency of 1 inhalation per 10-25 seconds). Blood pressure was also monitored. When the adrenal vein was cannulated to facilitate sampling, the epinephrine levels found in dogs anesthetized with chloralose were much higher than in those anesthetized with pentobarbital. The increased sensitivity of chloralose-anesthetized animals is ascribed to failure of chloralose to depress reflexes or block sympathetic nerve transmission. In other experiments in chloralose-anesthetized dogs, inhalation of cigarette smoke led to significantly increased levels of epinephrine in the vena cava, and in the femoral artery and vein, as well as to increased blood pressure. Rapid smoking led to markedly higher epinephrine levels (126 $\mu\text{g/liter}$) than slow smoking (25.1 $\mu\text{g/liter}$). In 2 determinations on peripheral artery blood, there was also a marked increase in norepinephrine (from 1.0 to 20.2 $\mu\text{g/liter}$) in response to cigarette smoke inhalation. The smoking of corn silk cigarettes had no effect on epinephrine levels; asphyxia of 3 minutes duration and reduction of the oxygen content of the inspired air had relatively small effects on epinephrine secretion, indicating that the nicotine in the smoke was responsible for the observed effects. The authors remark that "although consideration must be given to species and weight differences, these results indicate that a similar effect can occur in man." (Support from the U. S. Public Health Service, Div. of Gen. Med. Svc., and the National Institute of Mental Health.)

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Primary Descriptors

cigarette smoking
mongrel dogs
blood norepinephrine level increase (A)
blood pressure increase (A)
nicotine pharmacology
blood epinephrine level increase (A)
smoke inhalation
tracheal cannulation
nicotine positive dose response (A)
animal experiments
epinephrine secretion (A)

Secondary Descriptors

animal concessions
adrenal vein blood
venous blood
arterial blood
vena caval blood
chloralose anesthesia
pentobarbital anesthesia
sympathetic nerve transmission
U.S. P.H.S. grant
U.S. N.I.M.H. grant
blood catecholamine level
asphyxia
hypoxia
corn silk cigarettes
cubebs
puff frequency

PROJECT TIC

ACCESSION NUMBER 0086

TITLE: SMOKING AND THE HEALTH OF OLDER MEN: II. SMOKING AND VENTILATORY FUNCTION

AUTHOR(S): Weiss, W./Boucot, K. R., Cooper, D. A., Carnahan, W. J.

SOURCE: Arch. Envir. Health 7(5):538-547, 1963

AFFILIATION: Women's Med. Coll. Pennsylvania, Philadelphia, Pa.

ANNOTATION: In order to "quantitate the degree of ventilatory impairment in smokers of various types, degrees, and durations," pulmonary function was studied in 338 consecutive men reporting for routine check-up to the Philadelphia Pulmonary Neoplasm Research Project and in 104 "normal" men: nonsmokers with negative chest photofluorograms and no symptoms. The quantitative correlation demonstrated between amount smoked and decreased pulmonary function suggests "that cigarette smoking is a factor in impairment of the vital capacity and one-second forced expiratory volume."

ABSTRACT: In various components of the "abnormal" group there were the following incidences of abnormal 1-minute, forced expiratory volumes: nonsmokers 6% (2/36); ex-smokers 15% (10/68); smokers, cigarettes only 20% (22/109); smokers, cigarettes, cigar/pipe 14% (5/37); smokers, cigar/pipe 14% (5/37). There was a direct correlation between the number of men with abnormal forced expiratory volume and cigarette consumption or duration of smoking. When abnormal x-rays, symptoms, and smoking were considered in regard to abnormal forced expiratory volume only 7% of current cigarette smokers had negative x-rays and no symptoms in contrast to 25% with abnormal x-rays and/or symptoms. Cough was "most common in cigarette smokers, especially moderate and heavy cigarette smokers" (0.5- >1.0 packs/day); although ex-smokers exhibited cough as seldom as nonsmokers, they showed as high a percentage with dyspnea as current smokers. This may indicate that cough is partially reversible, when it is due to tobacco smoke, but that dyspnea, if due to emphysema, "is not likely to be reversible even if smoking is stopped." These findings "are consistent with those previously reported for this group of older men with regard to the relationship between chronic cough and smoking." They suggest chronic bronchial obstruction due to irritative bronchitis. The fact that ex-smokers have ventilatory values intermediate between those of nonsmokers and those of current smokers also suggests that such bronchial obstructive disease may be reversible to some extent. "The fact that abnormal ventilatory function occurs essentially in smokers who have symptoms and/or abnormal x-ray findings suggests that smoking does not lead to diminished function until it is associated with clinical evidence of respiratory disease". "There was a clear relationship between photofluorographic evidence of emphysema, generalized fibrosis, and inactive tuberculosis on the one hand and smoking habits on the other hand. Emphysema was five times higher among current cigarette smokers than among men who never smoked." There was an inverse relation, however, between incidence of emphysema and amount smoked, "possibly due to the fact that people with emphysema tend to smoke less because of bronchial irritation." It is also mentioned that the incidence of tuberculosis was higher for smokers than for nonsmokers, and that "ex-smokers tended to be taller than either nonsmokers or current smokers." [Support from the Philadelphia Tuberculosis and Health Association, the American Cancer Society, and the Pennsylvania Thoracic Society.]

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LG-27324

LG 2018148

Primary Descriptors

pulmonary function decrease
cigarette smoking
vital capacity decrease
forced expiratory volume decrease
pipe smoking
cigar smoking
pulmonary emphysema
chronic cough
combined smoking
smoking amount
smoking discontinuation
bronchial obstruction
bronchial irritation
smoking duration
tobacco smoke positive dose response
tobacco smoke negative dose response
bronchitis tobacco causation NPB

Secondary Descriptors

maximal expiratory flow rate
constitutional factors
body height
bronchitis reversibility
Philadelphia Pulmonary Neoplasm Research Project
lung fibrosis
pulmonary tuberculosis
Philadelphia TB & Health Assn. grant
American Cancer Soc. grant
Penna. Thoracic Soc. grant
chest radiology
dyspnea
statistical study

419-27325

PROJECT TIC

ACCESSION NUMBER 0196

TITLE: "TEST FOR NICOTINE"

AUTHOR(S): Hockett, R. C.

SOURCE: J. Amer. Med. Assn., 187(1): 74, Jan. 4, 1964

AFFILIATION:

ANNOTATION: A question is posed as to the nature of the test for nicotine and whether it could be used to determine if nicotine is deposited on the tonsil tissue of a smoker. The conclusion is that such detection would be extremely difficult by any method.

ASSTRACT: The standard method for determining nicotine involves steam distillation of nicotine and related alkaloids from a strongly alkaline solution, collection in dilute acid, and precipitation of nicotine with silicotungstic acid. A colorimetric method, based on the reaction between nicotine and cyanogen bromide with the addition of beta-naphthylamine, has been applied to the determination of microgram quantities in blood. "The detection of nicotine on tonsil tissue by such methods is likely to be difficult. The total nicotine in the main stream smoke of one king size cigarette without filter may approach 2.75 mg and only a small fraction of the total smoke would be expected to deposit on the tonsil. Free nicotine base is very rapidly absorbed through the mucous membranes and would therefore be expected to disappear rapidly from any particulate matter deposited on the tonsil. The action of mucus and saliva would also be expected to remove such deposits from the surface."

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LG - 27337

LG 2018150

Primary Descriptors

nicotine test
tonsil tissue
nicotine deposition
cigarette smoke nicotine content
nicotine absorption

Secondary Descriptors

silicotungstic acid
cyanogen bromide
β-naphthylamine
main-stream smoke
king-size cigarettes

41A - 27338

PROJECT TIC

ACCESSION NUMBER 0310

TITLE: GLUCOSE BLOCKAGE OF THE INCREASE IN STROKE VOLUME PRODUCED BY SMOKING

AUTHOR(S): Moses, D. C./ Powers, D., Soloff, L. A.

SOURCE: Circulation 29, 320-324, June, 1964

AFFILIATION: Temple University Health Sciences Center, Phila., Pa.

ANNOTATION: The increase in cardiac output and stroke volume induced by smoking was shown to be significantly reduced in 7 healthy male smokers by the prior intravenous administration of 10% glucose (15 g).

ABSTRACT: The changes in cardiac output, heart rate, and stroke volume in response to smoking (two cigarettes within a 15-minute period), the intravenous administration of 10% glucose ("less than one-fourth that present in the average American meal"), and smoking preceded by intravenous glucose were investigated in 7 healthy normal males, 19-26 years of age, who habitually smoked at least one package of cigarettes daily. There was a significant increase in all 3 parameters after smoking, corroborating similar results reported after cigarette and pipe smoking or the intravenous administration of nicotine. While pretreatment with glucose prior to smoking did not alter the heart rate response, it did significantly decrease the cardiac output and stroke volume responses brought about by tobacco smoking; the mechanism of this glucose effect is as yet unknown, but it suggests that the cardiac responses to tobacco "may be nutritional responses rather than harmful ones." [Support from the Tobacco Industry Research Committee, the Heart Association of Southeastern Penna., and the Arlene Dickler Research Fund.]

ABSTRACTOR

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21 G - 27340

LG 2018152

Primary Descriptors

cigarette smoking
stroke volume increase
cardiac output increase
glucose administration
heart rate increase
cardiovascular disease concessions

Secondary Descriptors

pipe smoking
nicotine administration
epinephrine secretion
norepinephrine secretion
blood fatty acid levels
T. L. R. C. grant
Heart Assn. SE Penna. grant
Arlene Dickler Res. Fund grant

416-27341

TITLE: SMOKING AND MOUTH ROAT CANCER

AUTHOR(S): Moore, C.

SOURCE: Am. J. Surg. 108: 565-569, October, 1964

AFFILIATION: Univ. of Louisville Sch. of Medicine, Louisville, Ky.

ANNOTATION: A study of 78 previously cured patients showed that second cancers of the mouth and throat occurred at a much higher rate among those who continued to smoke (17 of 49) than among those who quit (1 of 29). It is strongly suggested "that tobacco is a determining factor in mouth-throat cancer, without which relatively few such cancers would develop", and that "a highly worthwhile degree of protection" results from stopping smoking.

ABSTRACT: The appreciable frequency, especially in smokers, of often-fatal, new squamous carcinomas of the mouth-throat region several years after control of the first carcinoma prompted a follow-up study (mean follow-up of 6.7 years) of 78 patients (initially smokers) who were living and well 3 years following treatment for squamous cancer of the mouth or throat. The 78 subjects divided themselves into 2 groups: those who quit smoking following their first cancer (29 subjects), and those who continued smoking (49 subjects). Of the 29 who had quit, one person developed a second "tobacco-area squamous cancer", while 17 second cancers developed in the group who continued smoking. It is mentioned that 7 of an original group of 85 patients with oropharyngeal carcinomas had never smoked, so that "their cancers had causes other than tobacco", that "several" patients developed lower esophageal second cancers, which were not counted in the study, and that "the infrequency of cancer in certain oropharyngeal sites" makes statistical studies difficult. The author points out that in most studies, "the factors of innate susceptibility to cancer.... are not considered or equalized" and suggests that his two groups may have been "more homogeneous than an inbred strain of laboratory animals," while admitting that the patients may have varied widely with respect to race, alcohol intake, diet, weight, previous syphilis, or some other factor which could account for the second cancer distribution. He also suggests "that tobacco may act as a promoter rather than a carcinogen per se," but points out that from a clinical standpoint "this is quibbling if the withholding of an external substance can control the major incidence of the disease." While maintaining that there is probably, in many cases, "a reversible precancerous state in the oral mucosa" which can be reversed by cessation of smoking, he admits that "the numbers of the subgroups are quite small and the number of uncontrolled variables in any human groupings necessarily numerous; therefore, few will consider the causal connection as proved. Furthermore, any serious concept of cancer causation must include a multitude of factors acting together, of which an external chemical agent can only be one."

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Primary Descriptors

oral cancer tobacco causation (contributing)
throat cancer tobacco causation (contributing)
smoking discontinuation
tobacco smoking
squamous carcinoma
cigarette smoking
oral cancer conceptions
throat cancer conceptions
cancer etiology conceptions
statistics conceptions
promoting activity
tobacco carcinogenicity conceptions
constitutional factors conceptions

Secondary Descriptors

larynx carcinoma
buccal floor carcinoma
tongue carcinoma
palate carcinoma
gingiva carcinoma
buccal mucosa carcinoma
tonsil carcinoma
lung carcinoma
pharynx carcinoma
esophagus carcinoma
tobacco chewing
snuff dipping
pipe smoking
sex differences
smoking amount
smoking duration
oral cancer morbidity
throat cancer morbidity
precancer reversibility
mucous membrane

TITLE: EXPERIMENTAL STUDY ON THE ETIOLOGY OF CANCER TYPES SPECIFIC TO INDIA
a) ORAL CANCER; b) KANGRI CANCER

AUTHOR(S): Ranadive, K. J./ Gothoskar, S. V., Khanolkar, V. R.

SOURCE: Acta Unio Int. Contra Canc. 19(3-4): 634-639, 1963.

AFFILIATION: Indian Cancer Research Center, Parel, Bombay, India

ANNOTATION: In a series of mouse skin-painting studies designed to investigate the possible role of tobacco chewing in oral cancer, alkaloid-free tobacco extract showed a "weak carcinogenic effect," although it was capable of acting as a co-carcinogen with 3,4-benzpyrene. Tests of the co-carcinogenic activity of heat, local injury, dietary deficiencies, and spices are proposed.

ABSTRACT: A series of five experiments was carried out to test the carcinogenicity of an alkaloid-free acetone extract of tobacco. "Vaddakan" tobacco of the Meenampalayam variety was powdered and treated with 2% HCl for removal of alkaloids and then extracted with acetone, obtaining extracts partially free of alkaloids (E₉) and completely free of alkaloids (E₁₀). Sixty to 95 weeks of biweekly skin paintings with total tobacco extract along with weekly application of a co-carcinogen, croton-oil, induced papillomas in 50% and frank carcinomas in 28.6% of hybrid mice (Paris albino XVII x C57 Black), but no carcinomas in inbred Swiss. Tobacco extract tested as a co-carcinogen biweekly with one painting of 3:4 benzpyrene produced two frank carcinomas and one doubtfully malignant papilloma in 13 Swiss (Baldy) mice, but no carcinomas among Swiss mice. None of the controls treated with only acetone and croton-oil or 3:4 benzpyrene developed tumors. These tests indicate the weak carcinogenic effect of the tobacco extract, although "tobacco is capable of promoting the cancerous process initiated by other carcinogenic factors." In preliminary test on the etiology of Kangri cancer, "Chinar-tar" (soot) in combination with croton-oil induced papillomas in 61.3% and frank carcinomas in 9.7% of Swiss mice, thus indicating its possible carcinogenicity.

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Primary Descriptors

tobacco chewing
tobacco extracts
oral cancer
Kangri cancer
mice
skin-painting
epidermal carcinoma (A)
Chinar-tar
acetone extracts
solvent extraction
papillomas (A)
Indian tobacco
animal experiments
skin cancer (A)
skin cancer
oral cancer tobacco causation NPB
skin cancer soot causation (contributing)
oral cancer concessions
tobacco extract carcinogenicity
tobacco extract co-carcinogenicity

Secondary Descriptors

dietary factors
tissue damage
soot
thermal factors
basal cell carcinoma
squamous cell carcinoma
3,4-benzpyrene
croton oil
Swiss mice
hybrid mice
betal nut
sex differences
Vaddakan tobacco
Meenampalayam tobacco
genetic factors

TITLE: PULMONARY PHYSIOLOGICAL MEASUREMENTS IN SMOKERS AND NONSMOKERS

AUTHOR(S): Hensler, N. M. / Giron, D. J.

SOURCE: J. Amer. Med. Assn., 156(10): 885-889

AFFILIATION: Scott Air Force Base, Ill.

ANNOTATION: A study of pulmonary function in 163 middle-aged airforce officers revealed significantly lower vital capacity, forced expiratory volume, maximum ventilatory volume, and residual volume in smokers than in non-smokers, but no significant relationship to the amount smoked. The authors ascribe the differences to smoking and conclude that smoking can result in airway obstruction and air trapping or hyperinflation, differing only in degree from those seen in patients with emphysema.

ABSTRACT: All subjects were free of significant heart or lung disease. The nonsmokers were those "who have never smoked or who had smoked only very occasionally in the past...." Pipe, cigar, and ex-cigarette smokers were excluded. Smoking history ranged from 15 to more than 25 years. In response to a simple yes or no type of questionnaire, there was no consistent or significant difference between smokers and non-smokers with respect to asthma, wheezing, or pneumonia. Symptoms of cough and shortness of breath were professed more frequently by smokers than by nonsmokers; this difference fell just short of statistical significance. The pulmonary measurements made included: maximum voluntary ventilation; one-second and three-second vital capacities; and lung volume (determined by the closed circuit helium dilution method, with the use of a gas analyzer-respirometer). The smokers showed significant mean reductions in vital capacity, forced expiratory volumes (1 and 3 seconds), maximum ventilatory volume, residual volume and the residual volume/total lung capacity ratio. There was no significant difference in total lung capacity. There was no statistically significant difference related to increased cigarette consumption. "It is worth noting that the group reported here is a relatively homogeneous population.... The entire adult life of each individual studied has been lived in a similar environment, that of the US Air Force. Since Air Bases are almost universally located at some distance from population and industrial centers, air pollution from factory and automobile exhaust would not be expected to be a significant factor," they say. "The only detectable background difference is that one group smoked and the other did not. The inescapable conclusion is that this fact accounts for the differences in lung function between these two groups." The authors note that chronic bronchitis and emphysema, which they tentatively ascribe to the chronic irritant effect of inhaled tobacco smoke, probably play a greater role in human disability and mortality than lung cancer.

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Primary Descriptors



cigarette smoking
lung function decrease
lung volume decrease
cigarette smoking amount
vital capacity decrease
chronic bronchitis tobacco causation (major)
pulmonary emphysema tobacco causation (major)
airway obstruction
questionnaire studies

Secondary Descriptors

Air Force personnel
chronic cough
lung cancer mortality
bronchitis morbidity
emphysema morbidity
military officers
respirometer
vitalometer
helium dilution method
asthma morbidity
pneumonia morbidity
dyspnea morbidity
atmospheric pressure
flying personnel
maximum voluntary ventilation
forced expiratory volume
wheezing morbidity
age factors
air pollution

TITLE: TOBACCO SMOKING BLOOD GROUP

AUTHOR(S): Higgins, I. T. T./Drummond, R. J., Oldham, P. D., Bevan, B.

SOURCE: Brit. Med. Journal, Nov. 9, 1963, pp. 1167-1169

AFFILIATION: Med. Res. Council, Epidemiological Res. Unit, Cardiff, Wales

ANNOTATION: A study of 1061 persons in South Wales revealed no significant relationship between smoking habits and either blood type or secretory status. However, the frequency of type B among nonsmokers (15.6%) was "slightly above the average for the whole group of smokers" (10.6%), leading the authors to conclude that "the unimpressive association observed in the present series is not in conflict with Cohen and Thomas's data, and when the two sets are combined the significance is quite high," suggesting a genetic element in the adoption of smoking.

ABSTRACT: The authors note initially that "it is surprising that practically no work has been done on smoking and the blood groups," despite the similar smoking habits of identical twins and the differences between smokers and nonsmokers with regard to physique, temperament, and accident proneness. In view of the work of Cohen & Thomas, who found an excess of blood type B in both occasional smokers and non-smokers, the authors feel that the deficiency of blood type B observed by them in both regular and occasional smokers may not have been merely a sampling error, and suggest that an association between blood type B and smoking "may be of the relatively simple form of a contrast between those who have never smoked and those who have." They note, however, that no particular blood group has so far been shown to be associated with either lung cancer or chronic bronchitis.

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Primary Descriptors

smoking habits
genetic factors
tobacco smoking
blood groups
cigarette smoking
blood group B
smoking blood group correlation
smoking amount

Secondary Descriptors

miners
South Wales (United Kingdom)
chronic bronchitis
lung cancer
secretory status
Rhesus factor
cigar smoking
sex differences
racial differences
pipe smoking
Negroes
twin studies

PROJECT 71C

ACCESSION NUMBER 0088

TITLE: EXPERIMENTAL CLINIC FOR PREVENTING CHRONIC BRONCHITIS

AUTHOR(S): Wood, C. H./ Meadows, S. H.

SOURCE: Brit. Med. J., November 2, 1963, pp. 1088-1092

AFFILIATION: London Sch. Hygiene Trop. Med., London, England

ANNOTATION: An experimental clinic for patients with early bronchitis is described in which cessation of smoking is suggested as a therapeutic measure. "Improvement in cough and phlegm occurred more often among those (28/76) who stopped smoking than in those who decreased or made no change."

ABSTRACT: The clinic attempts to "(1) reduce irritation of the respiratory tract by helping patients to stop smoking and teaching them practical steps to reduce the damage done by air pollution; (2) combat infection by the use of influenza vaccine and antibiotics; and (3) teach them to breathe by daily exercises." Among 109 "regular attenders", at time of follow-up, two had increased their smoking and 31 were non-smokers or ex-smokers. In most patients there was little change in forced expiratory volume after attendance at the clinic. By way of introduction, the authors note that chronic bronchitis is a more significant cause of morbidity and mortality than either tuberculosis, pneumonia, or lung cancer, and that "permanent and progressive disability" can result from "both chronic irritation, by air pollution and cigarette smoke, and recurrent or chronic infection." [Support from the North West Metropolitan Regional Hospital Board (England).]

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Primary Descriptors

tobacco smoking
chronic bronchitis
cigarette smoking
chronic cough
phlegm production increase
pulmonary function decrease
smoking discontinuation
bronchitis prevention
respiratory tract irritation
respiratory tract infection
chronic bronchitis tobacco causation (contributing)
chronic bronchitis air pollution causation (contributing)

Secondary Descriptors

smoking habits
air pollution
pipe smoking
Northwest Metropolitan Regional Hospital
Board grant
breathing exercises
lung cancer mortality
chronic bronchitis mortality
forced expiratory volume

