

$p$ -(CH<sub>3</sub>)<sub>2</sub>CHC<sub>6</sub>H<sub>4</sub>CH(CH<sub>3</sub>)CH(CH<sub>3</sub>)(CH<sub>2</sub>)<sub>3</sub>CH<sub>3</sub>, 3-methyl-2-(4-isopropylphenyl)heptane.

For naming cyclic hydrocarbons with side chains according to (α), it is advisable in many cases to use the common names of simple aromatic hydrocarbons.

*Examples:* *o*-CH<sub>3</sub>C<sub>6</sub>H<sub>4</sub>C<sub>2</sub>H<sub>5</sub>, 2-ethyltoluene; (CH<sub>3</sub>)<sub>2</sub>C<sub>6</sub>H<sub>4</sub>CH:CH<sub>2</sub>(1,3,2), 2-ethenyl-*m*-xylene; CH<sub>3</sub>C<sub>6</sub>H<sub>3</sub>(C<sub>2</sub>H<sub>5</sub>)CH(CH<sub>3</sub>)<sub>2</sub>(1,2,4), 2-ethyl-*p*-cymene.

- II. When several cyclic hydrocarbon residues are united by an aliphatic chain the name of the compound will be derived from that of the aliphatic hydrocarbon, provided radical names are available for the cyclic hydrocarbon residues.

*Examples:* C<sub>6</sub>H<sub>5</sub>CH<sub>2</sub>C<sub>6</sub>H<sub>5</sub>, diphenylmethane; C<sub>6</sub>H<sub>5</sub>CH<sub>2</sub>CH(C<sub>6</sub>H<sub>5</sub>)(CH<sub>2</sub>)<sub>2</sub>CH<sub>3</sub>, 1,2-diphenylpentane.

If this is not the case, or if the possibility of using a convenient radical name makes it desirable, the name of the compound will be derived from that of one of the cyclic hydrocarbons, on the principle of substitution.

*Examples:* C<sub>14</sub>H<sub>9</sub>CH<sub>2</sub>C<sub>6</sub>H<sub>5</sub>(2), 2-benzylanthracene (better than phenyl-(2-anthryl)methane); C<sub>16</sub>H<sub>9</sub>CH<sub>2</sub>CH<sub>2</sub>C<sub>6</sub>H<sub>5</sub>, (β-phenylethyl)pyrene.

#### RULE 49b

When the cyclic hydrocarbons treated of in rule 49a carry functions which can be expressed only by a prefix, the same possibilities for names exist as those indicated in rule 49a.

*Examples:* C<sub>6</sub>H<sub>5</sub>CHClCH<sub>2</sub>Cl, 1,2-dichloro-1-phenylethane or (α, β-dichloroethyl)benzene; C<sub>6</sub>H<sub>5</sub>CH<sub>2</sub>CH(CH<sub>3</sub>)CH<sub>2</sub>Cl, 3-chloro-2-methyl-1-phenylpropane or (γ-chloroisobutyl)benzene; *p*-ClC<sub>6</sub>H<sub>4</sub>CH<sub>2</sub>CH<sub>2</sub>Cl, 4-chloro-1-(β-chloroethyl)benzene or 2-chloro-1-(4-chlorophenyl)ethane.

For naming derivatives of monocyclic hydrocarbons which have common names, it will be of advantage to employ these names.

*Examples:* *p*-ClC<sub>6</sub>H<sub>4</sub>CH<sub>3</sub>, 4-chlorotoluene (4-chloro-1-methylbenzene); *p*-ClC<sub>6</sub>H<sub>4</sub>CH<sub>2</sub>Cl, 4,ω-dichlorotoluene (4-chloro-1-(chloromethyl)benzene, 4-chlorobenzyl chloride); CH<sub>3</sub>C<sub>6</sub>H<sub>4</sub>(NO<sub>2</sub>)CH(CH<sub>3</sub>)<sub>2</sub>(1,2,4), 2-nitro-*p*-cymene (2-nitro-1-methyl-4-isopropylbenzene).

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## SPECIAL ARTICLES

### TOBACCO SMOKING AND LONGEVITY<sup>1</sup>

In the customary way of life man has long been habituated to the routine usage of various substances and materials that are not physiologically necessary to his continued existence. Tea, coffee, alcohol, tobacco, opium and the betel nut are statistically among the more conspicuous examples of such materials. If all six are included together as a group it is probably safe to say that well over 90 per cent. of all adult human beings habitually make use of one or more of the component materials included in the group. All of them contain substances of considerable pharmacologic potency if exhibited in appropriate dosage. Widespread and long-continued experience, however, has shown that the moderate usage of any of these materials, if measurably deleterious at all, is not so immediately or strikingly harmful physiologically as to weigh seriously against the pleasures felt to be derived from indulgence, in the opinion of vast numbers of human beings. The situation so created is an extremely complex one behavioristically, and not a simple physiological matter, as it is sometimes a little naively thought to be. Purely hedonistic elements in behavior, which are present in lower animals as well as in man, have a real importance. Indeed they frequently override, in their motivational aspects, reason as well as

purely reflex physiological inhibiting factors. There are undoubtedly great numbers of human beings who would continue the habitual use of a particular material they liked, even though it were absolutely and beyond any question or argument proved to be somewhat deleterious to them. Most of them would rationalize this behavior by the balancing type of argument—that the keen pleasure outweighed the relatively (in their view) smaller harm.

The student of longevity is not primarily interested in the behavioristic aspects of the situation under discussion. His concern is to appraise quantitatively, with the greatest attainable accuracy, the effect of each of these habitual usages upon the duration of life. This problem is necessarily statistical in its nature, for in the ordinary way of usage the effect upon longevity of any of the materials mentioned is not sufficiently strong or immediate to be disentangled in the individual from the effects of other and more powerful factors that are involved, such as infections, for example. An approximate evaluation of the statistical effect of these minor and secondary factors influencing longevity can, however, be reached by the application of actuarial methods (life table construction) to groups of individuals. For the maximum effectiveness of this methodology in the premises, the groups to be compared should be each as heterogeneous or random as possible in their compositions relative to all other characteristics *except* the one of degree of habitual usage of the particular material under discussion, and as homogeneous

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as possible relative to that. We shall then have a dispersed and counterbalancing effect within each group of all such factors as economic and social status, occupational and racial differences, etc., the plus variants relative to each such factor offsetting more or less evenly the minus variants; while there will be a concentrated, unidirectional and statistically cumulative effect, if any, of the habitual usage factor under test, since all components of a group will be alike in respect of it.

The purpose of this paper is to report a part of the results of an investigation of the influence of tobacco upon human longevity, planned and carried out along the lines indicated above. The material was drawn from the Family History Records<sup>2</sup> of this laboratory. It is composed of data collected at first hand and *ad hoc*. The accuracy of the data as to the relative degree of habitual usage of tobacco and as to the ages of the living at risk, and of the dead at death can be guaranteed. The figures presented here deal only with white males, and concern only the usage of tobacco by smoking. The material falls into three categories, as follows: *non-users* of tobacco, of whom there were 2,094; *moderate smokers*, of whom there were 2,814; and *heavy smokers*, of whom there were 1,905. In other words, the results presented here are based upon the observation of 6,813 men in total. These men were an unselected lot except as to their tobacco habits. That is to say, they were taken at random, and then all sorted into categories relative to tobacco usage.

Complete life tables have been constructed for the three groups defined above relative to tobacco usage by smoking. The tables start at age 30 and continue to the end of the life span, by yearly intervals. Here only a condensation of the tables can be presented. This is done in Table 1, where the death rate (1000  $q_x$ )

and survivorship ( $l_x$ ) function are given by five-year intervals.

Those not particularly accustomed to life table figures will perhaps get quickly a better grasp of the general import of the results from Fig. 1 than from the table. Fig. 1 shows the survivorship lines plotted from the individual year figures.

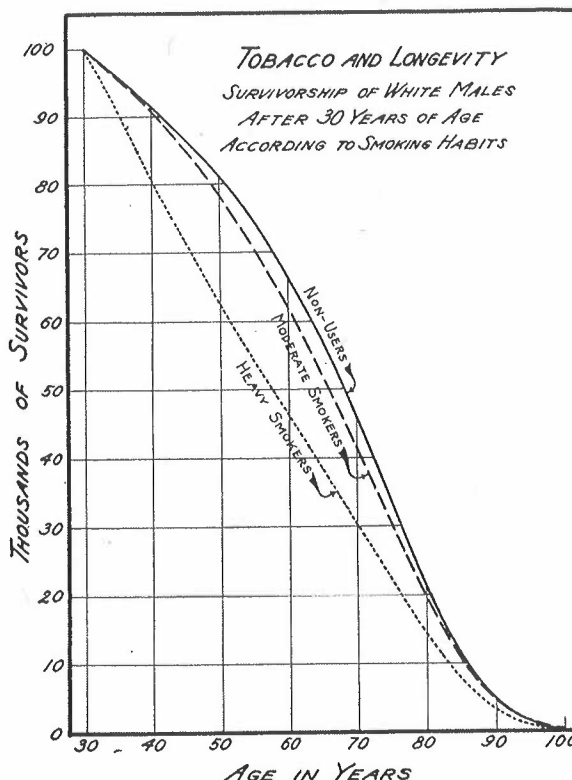


FIG. 1. The survivorship lines of life tables for white males falling into three categories relative to the usage of tobacco. A. Non-users (solid line); B. Moderate smokers (dash line); C. Heavy smokers (dot line).

TABLE 1  
THE DEATH RATE (1,000  $q_x$ ) AND SURVIVORSHIP ( $l_x$ ) FUNCTIONS, AT FIVE-YEAR INTERVALS, STARTING AT AGE 30, OF (a) NON-USERS OF TOBACCO; (b) MODERATE SMOKERS WHO DID NOT CHEW TOBACCO OR TAKE SNUFF; (c) HEAVY SMOKERS WHO DID NOT CHEW TOBACCO OR TAKE SNUFF. WHITE MALES

Age	Non-users		Moderate smokers		Heavy smokers	
	1,000 $q_x$	$l_x$	1,000 $q_x$	$l_x$	1,000 $q_x$	$l_x$
30	8.18	100,000	7.86	100,000	16.89	100,000
35	8.78	95,883	9.63	95,804	21.27	90,943
40	10.01	91,546	11.89	90,883	23.91	81,191
45	12.04	86,730	14.80	85,129	25.69	71,665
50	15.16	81,160	18.61	78,436	27.49	62,699
55	19.32	74,538	23.67	70,712	30.09	54,277
60	26.73	66,564	30.49	61,911	34.29	46,226
65	36.88	57,018	39.83	52,082	41.20	38,328
70	51.69	45,919	52.84	41,431	52.72	30,393
75	73.02	33,767	71.28	30,455	72.33	22,338
80	103.22	21,737	97.95	19,945	100.44	14,494
85	142.78	11,597	136.50	10,987	139.48	7,865
90	197.49	4,753	190.23	4,686	193.68	3,292
95	273.2	1,320	265.1	1,366	268.9	938

<sup>2</sup> For an account of the nature of these Family History Records see R. Pearl, *Biotypologie*, T. 2, pp. 105-122, 1934, and other references there cited.

However envisaged, the net conclusion is clear. In this sizable material the smoking of tobacco was statistically associated with an impairment of life duration, and the amount or degree of this impairment increased as the habitual amount of smoking increased. Here, just as is usually the case in our experience in studies of this sort, the differences between the usage groups in specific mortality rates, as indicated by  $q_x$ , practically disappear from about age 70 on. This is presumably an expression of the residual effect of the heavily selective character of the mortality in the earlier years in the groups damaged by the agent (in this case tobacco). On this view those individuals in the damaged groups who survive to 70 or thereabouts are such tough and resistant specimens that thereafter tobacco does them no further measurable harm as a group.

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