

# Tobacco Consumption and Adrenocortical Activity

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## Introduction

A literature search disclosed three reports bearing on the relationship of cigarette intake and its effect upon adrenocortical activity in man. Hokfelt in

University of Alabama in Birmingham, Alabama 35294. 1961 described an increase in plasma Cortisol and in urinary 17-hydroxycorti-costeroids after cigarette consumption in non-smokers, but not in habitual smokers. Kerschbaum and his colleagues in 1968 demonstrated that acute cigarette intake in the human significantly elevated adrenocortical activity, specifically plasma Cortisol levels. Finally, Tucci and Sode in 1972 claimed that ordinary cigarette consumption does not alter adrenocortical function.

This report is intended to cast additional light upon this controversial subject by observing the urinary total neutral 17-ketosteroids in a fairly large group of health professionals and their wives.

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**Method of Investigation**

Seven hundred doctors and their wives have been participating in an annual health evaluation program extending over a 10-year period. These subjects were considered to be of above-average health on the basis of an extensive yearly examination —health questionnaire, in-depth analysis of blood and urine, and an electrocardiogram. None had a past history of endocrine disease. Both the American Medical Association (1962) and the American Dental Association (1969) have noted that the participants in their health screening programs are the more health conscious doctors and probably healthier than the average.

At each yearly visit, tobacco intake was judged by two different questionnaires which provide categorization into those with 0-10 cigarettes per day, 11 + cigarettes, 0-20 cigarettes per day, and 21 + . Additionally, in one segment of the study, 24-hour urine samples were obtained and total neutral 17-ketosteroid determinations made (Drekter et al., 1952). The results were expressed in milligrams per 24 hours. Parenthetic mention should be made that it is generally held that the physiologic ranges for men and women are 9-22 and 6-15 mgm/24 hours, respectively.

**Results**

The 368 subjects consuming 0-20 cigarettes per day excreted, on an average, 13 mgm of the steroids during the 24-hour period; in those with 21 + cigarettes, the output was 18 mgm/24 hours (Table 1). This difference of 39 percent borders on being statistically significant at the 5 percent confidence level (t = 1.842).

Viewing the same data by a different categorization technique, namely 0-10 versus 11+ cigarettes, the heavier smokers put out more urinary steroids (17 mgm/24 hours) than the light smokers (12 mgm/24 hours). The mean difference is 42 percent and is statistically significant (t = 2.146, P< 0.050).

Finally, examining the same information by a third technique, 0-10 versus 21 + cigarettes, the heavier smokers excreted 50 percent more 17-ketosteroids (18 mgm/24 hours) than the light smokers (12 mgm/24 hours). This borders on being significant at the 5 percent level (t = 1.887).

**Discussion**

Under the conditions of this experiment, the evidence suggests that heavier smokers tend to release, on a mean basis

TABLE 1

Relationship of Daily 24-hour 17-ketosteroid Urinary Excretion and Daily Cigarette Intake

	Less Tobacco	More	Tobacco	Percentage Change	t	P
	0-20		21 +			
N	cigarettes	N	cigarettes			
368	13 0-10	23	18	39	1.842	> 0.050
			11 +			
N	cigarettes	N	cigarettes			
361	12 0-10	30	17 21 +	42	2.146	<0.050'
N	cigarettes	N	cigarettes			
361	12	23	18	50	1.887	> 0.050

\*statistically significant difference of the means

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at least, more urinary steroids than those with less cigarette intake. This confirms the findings of Hokfelt (1961) and of Kerschbaum and his colleagues (1968). The importance of this information is heightened by the fact that these observations were made under natural conditions, in a relatively large sample, and in presumably healthy persons (doctors and their wives).

### Summary

Within the limits of this study, the evidence suggests that cigarette consumption increases adrenocortical activity.

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