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DATE: 1974

TITLE: Glucocorticoid function of the adrenals in radiowave sickness, in Gordon ZV (ed):
Biological Effects of Radiofrequency Electromagnetic Fields, JPRS 63321.

SOURCE: Arlington, Va, US Joint Publications Research Service, 30 Oct 1974, pp 72-74

MAIN SUBJECT HEADING:

AN	<u>HU</u>	AT	IH	M
ANALYTICS	HUMAN EFFECTS	ANIMAL TOXICITY	WORKPLACE PRACTICES- ENGINEERING CONTROLS	MISCELLANEOUS

SECONDARY SUBJECT HEADINGS: AN HU AT IH M

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Storage/Labeling

UDC 613.62:614.87:537.868.029.64

GLUCOCORTICOID FUNCTION OF THE ADRENALS IN RADIOWAVE SICKNESS

Moscow O BIOLOGICHESKOM DEYSTVII ELEKTROMAGNITNYKH POLEY
RADIOCHASTOT in Russian 1973 pp 55-57

[Article by V.N. Dumkin and S.P. Korenevskaya]

Studies are conducted on corticosteroid excretion in patients with moderate and pronounced manifestations of radiowave sickness by means of their fractionation from urinary extracts by silica gel thin-layer chromatography (TLC). Changes in glucocorticoid metabolism in patients with radiowave sickness are related to damage of the deep structures of the brain which are responsible for regulating the activity of the hypophysis-adrenal cortex system.

It has been recognized that prolonged employment under conditions of intense exposure to SHF radiowaves may lead to the development of a disease which is characterized by a complex of neurological, neurocirculatory, vegetative, and endocrine metabolic dysfunctions [E.A. Drogochina and M.N. Sadchikova, 1964; N.V. Tyagin, 1971; and others].

Detailed biochemical investigations conducted at our clinic on patients with radiowave disease have demonstrated a number of dysfunctions which are obviously the results of changes in the functional state of the mesodiencephalic regions of the brain [I.V. Pavlova, et al., 1970].

These data are in agreement with the results of clinical studies of E.A. Drogochina and M.N. Sadchikova [1964], as well as with the investigations of M.S. Tolgskaya and Z.V. Gordon [1971] who demonstrated significant changes in the cells of the hypothalamic-hypophysial region in animals that had been exposed for long periods of time to the effects of SHF radiowaves.

The present study was conducted to define more precisely the metabolism of glucocorticoid hormones of the adrenal glands, and also the state of the central mechanisms of homeostatic regulation, and the formations which activate the hypophysis-adrenal cortex system in patients with radiowave sickness.

Clinical observations were conducted of 20 patients who at work, especially during the first few years, were subjected to significant intensities of SHF electromagnetic fields in the centimeter range; the patients were from 33-46 years old, and their duration of service ranged from 10-20 years.

The clinical picture of the subjects under investigation was characterized by a complex variety of vegetative vascular dysfunctions and presence of crises, cerebral or coronary vascular insufficiencies, pronounced emotional lability, and asthenic manifestations. Depending on the severity and persistence of these manifestations, the forms of the radiowave disease were classified as moderate or pronounced (M.N. Sadchikova).

In the present study, we separated hydrocortisone (F), cortisone (E), their tetrahydro derivatives (THF, THE), and tetrahydro-17-hydroxy-11-deoxycorticosterone (THS) from urinary extracts by means of silica gel TLC [O. Adamec, et al., 1962]. Control data were obtained on 10 clinically healthy individuals between the ages of 30 and 45 years. The control data obtained in our investigations did not differ significantly from the data in the literature [K.V. Kruzhnina, 1968; and others].

The average results for the background excretion of corticosteroids (mg/day) in the controls and in the patients (I), as well as the excretion in patients on the day of the adrenaline test (II), are presented in Table 1.

It is evident from Table 1 that the levels of these hormones have undergone significant changes including a decrease in the total hormone excretion ($\Sigma(P < 0.01)$) as well as a decrease in tetrahydrocortisone ($P < 0.01$), and cortisone ($P < 0.02$) with an insignificant increase in the THS fraction ($P < 0.01$). Changes in the average value for the F/E ratio indicate disturbances in the mechanism responsible for the transformation of cortisol into cortisone in the patients of this group.

Administration of small doses of adrenalin to healthy people does not result in a significant clinical reaction or activation of the hypophysis-adrenal cortex system which is due to the patency of the central mechanisms responsible for regulating

homeostasis. Administration of small doses of adrenalin to patients with hypothalamic lesions leads to the development of vegetative vascular crises and changes in the excretion of corticosteroids [G.L. Shreyberg, 1962]. In the majority of our patients, the subcutaneous administration of 0.3 mls of 0.1% solution of adrenaline hydrochloride resulted in the development of marked vegetative vascular reactions or crises, usually of the mixed type.

As is evident in Table 1, administration of adrenalin to the patients resulted in statistically significant ($P < 0.01$) increase in Σ and to some extent, an increase in THF, which indicates activation of the hypophysis adrenal-cortex system. However, changes in the levels of the THS, F and E fractions, as well as the F/E ratio, became even more pronounced. This obviously represents persistence of abnormalities in patients with the radiowave sickness, as well as alteration in the transformation of cortisol into cortisone.

It should be mentioned that similar changes in the glucocorticoid function of the adrenal glands have been detected by us in patients with toxic diencephalopathies in chronic intoxication with neurotropic poisons. These considerations led us to the conclusion that the changes in the glucocorticoid function of adrenal glands which we detected in patients with pronounced form of radiowave disease are sequelae of original lesions in the deep structures of the brain which are responsible for the central regulation of corticosteroid synthesis and metabolism.

The resultant data also suggest that the dysfunction in the hypothalamus hypophysis-adrenal cortex system, which appear at a certain stage of development of the pathologic process, may have a pathogenic significance and play a definite role in the formation of the clinical syndrome of radiowave sickness. This provides a basis for designing certain rational approaches in therapy.

Table 1.

	THF	THE	THS	F	E	Σ	$\frac{F}{E}$
Control	0,83±0,7	2,0±0,14	0,01±0,005	0,21±0,02	0,31±0,05	3,42±0,18	0,67
I	0,61±0,08	1,96±0,26	0,07±0,01	0,25±0,06	0,16±0,03	3,07±0,33	1,6
II	0,75±0,09	2,00±0,23	0,08±0,02	0,37±0,08	0,14±0,02	3,33±0,037	2,6