Effect of Organochlorine Insecticides on Serum PBI Level in Occupationally Exposed People

by DORA WASSERMANN, M. WASSERMANN, M. DJAVAHERIAN
The Hebrew University-Hadassah Medical School, Jerusalem
Department of Occupational Health
and
I. GORIN, M. BARISH, and R. TAVOR
Kupat Holim Health Insurance Institution, Israel
Department of Industrial Medicine, Yehuda

Organochlorine insecticides (OCI) have invaded the environment during the last quarter of a century and consequently become an integral part of the "internal milieu" of the animal body.

This raises the question of the toxicological effects of this man-made state on living beings, and in the broader view, the biological implications of the ubiquity of OCI.

The concentration of OCI in the animal body is higher in tissues having a high content of lipids. The OCI plasma level is a function of the external supply and of the tissue-plasma circuit. Workers occupationally exposed to these insecticides have a higher plasma level of OCI than non-occupationally exposed workers (1,2,3).

The lack of symptoms in people occupationally exposed to OCI (except for acute or subacute casualties) led to the supposition of the innocuity of the presence of these compounds in the human body. Animal experiments and human studies have shown that OCI intervene in the metabolism of some xenobiotics as well as some naturally occurring compounds of importance in body processes. Thus, OCI affects the function of the nervous system (4,5), the detoxication of different compounds, the immunological response to environmental hazards (6,7,8), etc.

As far as the endocrine system is concerned, most attention has been paid to the hypophyso-adrenal system following the early findings of Nelson and Woodard (9). This paper reports on OCI effects on the hypophyso-thyroid system.

Method

Protein Bound Iodine (PBI) was determined in the serum of 42 workers handling OCI pesticides and in a control group of 51 workers from another plant, non-occupationally exposed to pesticides. The Barber Dry-Ash method (10), partly modified, was used for PBI assessment.

Results

The mean serum PBI in the workers non-occupationally exposed to OCI was 6.93 µg%. With occasional exceptions all the values obtained were within the normal limits. The workers occupationally

Bulletin of Environmental Contamination & Toxicology
Vol. 6, No. 1, 1971, published by Springer-Verlag New York Inc.

exposed to these insecticides had a mean of 5.42 µg PBI/ 100 ml with all individual values included in the normal range (4-8 µg PBI/ 100 ml). Serum PBI values in the occupationally exposed workers were significantly lower ($p < 0.01$) (Table 1).

TABLE 1

Serum PBI Levels (µg/ 100ml)

Age Group	A. Non-Occupationally Exposed Workers		B. Workers Occupationally Exposed to OCI		Statistical Evaluation
	Cases	Mean ± S.D.	Cases	Mean ± S.D.	A vs. B
20-34	16	6.70 ± 1.26	26	5.26 ± 0.42	$p < 0.01$
35-44	17	7.28 ± 1.04	8	5.42 ± 0.39	$p < 0.01$
45-64	18	6.82 ± 0.84	8	5.99 ± 0.35	$p < 0.02$
TOTAL	51	6.93 ± 1.12	42	5.42 ± 0.49	$p < 0.01$

Comments

The clinically euthyroid state of people occupationally exposed to pesticides gives the impression that the hypophyso-thyroid system is not influenced by the presence of OCI in the human body.

The findings reported on the effects of DDT and related materials on the nervous system (4,5) suggest an increased activity of the hypophyso-thyroid axis. The thyroids of rats submitted to DDT were found to be enlarged (11) and showed a histological appearance of hyper-function (unpublished data). The rate of loss of ^{131}I from the thyroid gland was significantly faster for DDT treated rats whereas the rate of oxygen consumption was unaffected (11). These findings suggested a compensated hypothyroidism in chronic administration of DDT in rats.

In the present study, the PBI serum level is lower in workers occupationally exposed to OCI than in controls. Danowski, et al (12), and Marshall and Tompkins (13) reported a decrease of PBI serum level in two patients treated with o,p'-DDD for metastatic adrenal carcinoma. These findings were explained by competition between o,p'-DDD and thyroxine for plasma thyroxine-binding globulin owing to the presence of two phenyl groups attached to the same carbon atom in the chemical structure of DDT and related materials, as well as in thyroxine (14) with, as a consequence, a diminished PBI level. Large amounts of free thyroxine are thus available and metabolized in the liver. The resulting need for thyroxine is satisfied by an efficient feed back. The hypophyso-thyroid axis is consequently working at a

higher level and compensates the trend towards a critical level of thyroxine.

According to the findings reported in this paper, it can be assumed that the maintenance of a suitable equilibrium under the effect of OCI requires a sustained effort of the hypophyso-thyroid system.

Conclusions

This paper reports on the level of serum PBI in a group of 42 workers handling OCI as compared to a control group of 51 workers not occupationally exposed to these insecticides.

Practically all the serum PBI values were within the range considered as normal. However, the serum PBI was significantly lower in workers occupationally exposed to OCI when compared to the non-occupationally exposed workers ($p < 0.01$).

It is suggested that the presence of OCI in the human body affects the metabolism of thyroxine. Homeostasis is maintained by a sustained effort of the hypophyso-thyroid system.

References

1. W.F. Edmundson, J.E. Davies, G.A. Nachman and R.L. Roeth, Public Health Reports 84,53 (1969).
2. M. Wassermann, Dora Wassermann, I. Ivriani, Pesticides Symposia 1970, edited by W.B. Deichmann, J.L. Radomski, R.A. Penalver, Halos Fl. p. 311
3. R.C. Perron and B.F. Barrentine, Arch. Environ. Health, 20,368 (1970).
4. D.E. Woolley and B.A. Barron, Toxicol. and Appl. Pharmacol., 12,440 (1968).
5. R.D. O'Brien, Insecticides, Action and Metabolism, Academic Press, 1967.
6. M.Wassermann, Dora Wassermann, Zipora Gershon and L. Zellermayer, Ann. N.Y. Acad. Sci., 160, 393 (1969).
7. J. Gabliks, Arch. Environ. Health., 14,698 (1967).
8. D. Fumarola, Ann. Sclavo. 11,1 (1969).
9. A.A. Nelson and G. Woodard, Arch. Path., 48,387 (1949).
10. S.B. Barker, M.J. Humphrey and M.H. Saley, J.Clin. Invest. 30,55 (1951).
11. M.J. Fregly, I.W.Waters and J.A. Straw, Can. J. Physiol. Pharmacol. 46,59 (1967).
12. T.S. Danowski, Me. E. Sarver, C. Moses and J.V. Bonessi, Amer. J. Med. 37, 235 (1964).
13. J.S. Marshall and Lucy S. Tompkins, Endocr. 28,386 (1968).

14. J.H. Oppenheimer, R. Squef, M.I. Surks and H. Hauer, J. Clin. Invest. 42,1769 (1963).

Supported by Grant 06-004-3 from the U.S.A. Department of Health, Education and Welfare, Public Health Service, Bureau of Occupational Safety and Health,Environmental Control Administration.

Reprints requests to Department of Occupational Health, Hadassah Medical School, The Hebrew University, P.O.B. 1172, Jerusalem, Israel. Dr. D. Wassermann.