

Organochlorine Insecticides in the Blood of Occupationally Exposed People in Sudan

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Organochlorine insecticides were shown to affect the biochemistry of mammalian systems in numerous ways. These include the induction of microsomal drug-metabolising enzymes (HART & FOUTS 1963, 1965), influencing steroid metabolism (KUPFER 1960), increase in cellular growth in vitro (GABLIK & FRIEDMANN 1970) and modification of immunological response to certain toxins (WASSERMANN et. al 1970).

People who are occupationally exposed to these insecticides are more likely to suffer from those and other effects than the general population. In fact, clinical and biochemical studies have already revealed renal abnormalities and aminoacid disturbances in such a group in the U.S.A. (DAVIES et. al 1970). For these reasons it was considered essential to monitor the OC blood levels in some exposed personnel of the Gezira Research Farm, Wad Medani, in order to assess the intensity of exposure and provide information of diagnostic value for future epidemiological work in this region. Despite the long history and continued extensive usage of OC's in the Gezira, no similar work was previously conducted.

Materials and Methods

Blood samples (5 ml) were collected from male subjects by venal punctures and placed in 10 ml glass stoppered tubes containing 0.1 ml of 20 percent aqueous potassium oxalate as an anticoagulant. The tube contents were gently mixed by inversion. Samples were subjected to silica-gel clean-up and extracted with hexane according to a previously described procedure (BROWN et. al 1964). Extracts were concentrated to 1 ml on a warm water bath under a stream of nitrogen.

Aliquots (5 μ l) of extracts were analysed on a Perkin-Elmer F 11 gas chromatograph equipped with a ^{63}Ni electron-capture detector and glass column (3 ft X 3 mm i.d.) packed with 2.5 percent Apiezon L on Chromosorb W AW-DCMS 80-100 mesh. Temperatures of the injection block, oven and detector were 232, 185 and 208°C respectively. Nitrogen at 100 ml per minute was the carrier gas used. The analysis was repeated on a 5 percent QF-1 column, under almost the same conditions as for the Apiezon one. Peak height measurements were used for quantitative determination of residues.

Table 1. Concentrations of Organochlorine Insecticides in Blood of Occupationally Exposed People in Sudan.

Serial No.	Duration of Exposure in Years	Concentration in µg/ml			
		DDE	TDE	p,p' DDT	HEOD
1	2	0.02	-	0.20	0.01
2	3	0.03	-	0.05	-
3	5	0.06	-	0.08	-
4	5	0.03	-	0.07	0.01
5	7	0.04	-	0.07	0.01
6	7	0.09	-	0.13	-
7	9	0.06	-	0.01	-
8	12	0.04	-	0.02	0.01
9	14	0.02	-	0.04	-
10	18	0.03	-	0.03	-
11	18	0.01	-	0.02	-
12	19	0.04	-	0.03	-
13	19	0.12	0.03	0.07	-
14	20	0.03	-	0.07	-
15	20	0.07	-	0.04	-
16	20	0.06	-	0.07	0.05
17	22	0.02	-	0.08	-
18	23	0.08	-	0.08	-
19	23	0.02	-	0.03	-
20	24	0.09	0.02	0.07	-
21	27	0.03	-	0.07	0.01
22	30	0.01	-	1.01	-

Alkali treatment was carried out to confirm the identity of GC peaks in the position of p,p' DDT. Increase in the DDE peaks was considered as an additional evidence for characterising those peaks as p,p' DDT.

Results and Discussion

Residues found in the blood of subjects sampled are shown in Table 1. DDE and p,p' DDT were found in all samples in the concentration range 0.01-0.12 µg/ml for DDE and 0.02-1.01 µg/ml for p,p' DDT. The occurrence of dieldrin (HEOD) was less frequent and at concentrations not exceeding 0.01 µg/ml in most cases. TDE was found in only two cases out of twenty two. An unidentified peak which ran slower than p,p' DDT was observed in the majority of samples. This peak was of considerable size in the case of samples No. 16 and 22.

These subjects are exposed to insecticides in different ways ranging from mixing and spraying to supervision of aerial spraying and insect counting in recently sprayed fields. For this reason their blood residue content was not expected to be correlated with their duration of exposure. Nevertheless some of the highest levels found were in subjects with more than 15 years of exposure.

Plasma levels of p,p' DDT and DDE which are expected to correspond to levels of these chemicals in the brain (DALE et. al 1967) might be as much as twice their levels in whole blood (NACHMANN et. al 1969). Therefore, and in view of the dangers inherent in this type of exposure, efforts should be taken to reduce the residue levels in these and similarly exposed subjects in this region.

References

- BROWN, V.K.H., C.G. HUNTER & A RICHARDSON. Brit. J. Industr. Med. 21, 283 (1964).
DALE, W.E., A. CURLEY & W.J. HAYES, Jr. Ind. Med. Surg. 36, 275 (1967).
DAVIES, J.E., J.B. MANN & P.M. TOCCI. Ann. N.Y. Acad. Sci. 160, 323 (1970).
GABLIK, J & L. FRIEDMANN. Ann. N.Y. Acad. Sci. 160, 254 (1970).
HART, L.G. & J.R. FOUTS. Proc. Soc. Exptl. Biol. Med. 114, 288 (1963).
HART, L.G. & J.R. FOUTS. Biochem. Pharmacol. 14, 263 (1965).
KUPFER, D. Ann. N.Y. Acad. Sci. 160, 244 (1970).
NACHMAN, G.A., J.J. FREAL, A BARQUET & C. MORGADE. Health Lab. Sci. 6, 148 (1969).
WASSERMANN, M., D. WASSERMANN, Z. GERSHON & L. ZELLERMAYER. Ann. N.Y. Acad. Sci. 160, 393 (1970).