UNITED
NATIONS

A

General Assembly

Distr.
GENERAL

A/37/102
25 February 1982

ORIGINAL: ENGLISH

Thirty-seventh session
Item 54 of the preliminary list*

CHEMICAL AND BACTERIOLOGICAL (BIOLOGICAL) WEAPONS

Note verbale dated 24 February 1982 from the Permanent Representative of the United States of America to the United Nations addressed to the Secretary-General

The Permanent Representative of the United States of America presents her compliments to the Secretary-General of the United Nations and has the honour to inform him that the United States has further information to provide pertaining to the use of chemical weapons in the continuing conflicts in Afghanistan, Kampuchea and Laos.

In late 1981, the United States Government received reports that a chemical attack had taken place in Kampuchea during the fall of 1981 which resulted in many deaths. It is now in a position to provide information, based on analyses of blood samples, which is consistent with trichothecene exposure and which tends to support the hypothesis that a trichothecene-based agent was used in the attack. Additionally, these preliminary results seem to indicate that, even weeks after the incident, blood samples from victims of trichothecene attacks may be able to provide important evidence to support charges of chemical/biological warfare use.

Several weeks after the alleged attack, blood samples were drawn by trained medical personnel from nine survivors and from four control individuals of similar age and background. Detailed medical histories were also taken.

The nine survivors reported that exposure had occurred in an attack in which only ground munitions were employed. They had also waded through a contaminated body of water. Symptoms experienced included vomiting of blood, blurred vision, bloody diarrhea, difficult breathing, dry throat, loss of consciousness, frontal headache, tachycardia and facial dema.

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* A/37/50.

Blood smears, as well as heparinized and non-heparinized blood samples, were forwarded to the United States for analysis. A portion of each sample was submitted on a blind basis to Professor Chester Mirocha (University of Minnesota) for analysis to determine whether trichothecenes or trichothecene metabolites were present. Additional portions and the blood smears were submitted to the United States Army Medical Research Institute of Infectious Diseases (USAMRIID), Fort Detrick, Maryland, for a series of tests, including white blood cell counts, haemoglobin, haematocrit, reticulocyte count, mean cell volume, and various liver and kidney function enzyme tests.

Using the selected ion-monitoring gas chromatography/mass spectroscopy analysis technique, Dr. Mirocha tentatively identified a metabolite of T_2 toxin (i.e., HT_2) in the blood of two alleged victims, on the basis of selected ion masses and gas chromatographic retention times.

Since white cell levels are depressed by exposure to trichothecenes, white blood cell counts were taken. The white cell counts of all but one of the nine exposed individuals were depressed below normal (which is approximately 7,400) and two individuals had extremely low counts (i.e., 1,700 and 3,000). White cell counts of two of the control individuals also were slightly depressed (5,100 and 6,500), however, and the sampling size was so limited that there was no real statistical difference between control and exposed groups when Student's T-test was applied. (The results are contained in the annexed table.) Alterations in liver and kidney function enzymes were also observed, but it was impossible to determine whether these effects were due to toxin exposure or to deterioration of the samples in transit.

Experimental studies in animals have shown that T_2 toxin is metabolized by liver microsomal enzymes to its deacetyrated derivative, HT_2. Ellison and Kotsonis 1/ have also shown that homogenates of human liver tissue rapidly deacetylate T_2 toxin to form HT_2 toxin; therefore, it is not surprising that this metabolite of T_2 was tentatively identified rather than T_2 itself. The presence of detectable amounts of HT_2 several weeks after alleged exposure to T_2 is surprising and somewhat disturbing. In animal studies 2/ radiolabelled T_2 and its metabolites were rapidly excreted from the body with approximately 80 per cent of the radioactivity excreted by 48 hours after exposure. On the basis of these studies, it was believed unlikely that metabolites of the trichothecene toxins would be detectable in blood later than 72 to 96 hours after an attack. Analysis for the compounds was performed, however, owing to the remote possibility that some of the compounds may be tightly bound to protein or lipids and would not be rapidly excreted. The tentative identification of HT_2 toxin in the blood of victims several weeks after exposure seems to indicate the presence of a depot or

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1/ Ellison and Kotsonis, Applied Microbiology, vol. 27, 1974, pp. 423 and 424.

2/ See the review by Y. Ueno, "Trichothecenes: Overview Address," in Mycotoxins in Human and Animal Health (J. V. Rodrick, C. W. Hesseltine, M. A. Mehlman, ed.), Pathotox Publishers Inc., Park Forest South, Illinois, 1977.

storage site for trichothecenes within the body. Binding experiments 2/ have shown that trichothecenes bind strongly to certain cellular constituents, notably, ribosomes, polysomes and sulfhydryl enzymes. These studies support the hypothesis that although most of the compounds are rapidly cleared from the body, the binding characteristics of some of the metabolites may result in the storage of small amounts within the body for considerable periods of time. If so, serious consideration must be given to long-term effects of small amounts of the trichothecenes, as well as the acute effects of large doses. Further experimental research is needed to define the extent of this problem.

The tentative identification of HT_2 in the blood of two victims cannot be taken as conclusive scientific proof of toxin exposure since the trace amount of the compound present precluded unequivocal identification and quantitation, and also because many other medical problems in addition to toxin exposure can cause a decrease in white cell counts. It is interesting to note that the individual who showed the lowest white cell count also showed the greatest amount of the compound tentatively identified as HT_2 in his blood and was reported to have received the greatest exposure to the agent. He was exposed to contaminated water for over 30 minutes and was the only victim who fell down in the water and actually swallowed some of it. However, the results of these two independent analyses, coupled with the description by victims of symptoms correlating exactly with those associated with trichothecene poisoning, provide strong circumstantial evidence that trichothecenes were used as chemical agents in yet another chemical attack in South-East Asia.

Trichothecenes have been identified previously in environmental samples taken from several other chemical attacks in Laos and Kampuchea. Analysis of control vegetation, water, soil, corn and rice samples from these areas, as well as reviews of published scientific literature, indicate that the particular toxins that have previously been identified are not known to occur naturally in the combinations found and at the levels detected in South-East Asia. The latest analysis results contribute another piece of evidence to the growing body of data supporting the charge that trichothecenes have been used as weapons in South-East Asia.

Therefore, in accordance with General Assembly resolutions 35/144 C of 12 December 1980 and 36/96 C of 9 December 1981, the Permanent Representative of the United States of America requests that this information be provided to the United Nations Group of Experts to Investigate Reports on the Alleged Use of Chemical Weapons. Additionally, the Permanent Representative again requests that ~~this submission be circulated as an official document of the General Assembly~~ under item 54 of the preliminary list.

As it has done in the past, the United States will continue to co-operate fully with the Secretary-General and the Group of Experts and will do its utmost to provide additional information and evidence as it becomes available and any further appropriate assistance which might facilitate the task of the experts.

ANNEX

Table

Peripheral blood haemograms of Kampuchean victims of chemical attack

Patient No.	RBC[a]	Hg[b]	Hct[c]	WBC[d]	Retic[e]	MCV[f]	MCH[g]	MCHC[h]
1	specimen clotted							
2	4.46	12.6	37	4 700	1.0	84	28.5	34
3	4.90	11.8	40	5 700	0.4	81	26	32
4	4.90	10.3	34	1 700	2.1	70	21	30
5	4.92	15.0	46	5 300	1.2	93	32	34
6	4.04	12.6	37	4 300	0.8	93	31	34
7	4.88	15.6	46	3 000	0.5	94	32	34
8	5.56	17.0	50	8 700	1.5	91	31	34
9	4.88	11.2	35	5 000	1.0	73	23	32
Controls:								
10	6.23	12.5	41	7 200	0.8	66	20	30
11	4.47	11.9	38	8 000	0.9	85	26.5	31
12	4.88	12.9	41	5 100	2.0	85	26.5	32
13	5.16	15.6	46	6 500	1.0	90	30.5	34

Table (continued)

Patient No.	RBC[a]	Hg[b]	Hct[c]	WBC[d]	Retic[e]	MCV[f]	MCH[g]	MCHC[h]
Normal range:								
male	4.5-6.0	14-18	40-54	7 400		80-94	27-32	33-38
female	3.5-5.0	12-16	37-47	+2 000				

	BUN[i]	Creatinine	SGPT[j]	Alkaline Phosphatase
Normal Range M	7-20	0.4-1.7	6-37	24-69
F				23-71
1.	9.0	3.5	48	132
2.	8.5	0.8	36	47
3.	8.0	1.4	12	75
4A.	11	1.3	6	94
4B.	10.5	1.2	6	68
5.	6.0	1.6	12	84
6.	7	1.2	18	115
7.	8.5	1.7	6	69
8.	10	1.5	36	79
9.	12.5	1.4	12	70
10.	10.5	1.8	12	86

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Table (continued)

	BUN[i]	Creatinine	SGPT[j]	Alkaline Phosphatase
11.	12	0.8	24	74
12.	12	1.4	6	76
13.	9.0	1.2	30	102

a/ Red blood cells x 10^{-6} (/cc)

b/ Haemoglobin (gm/100cc)

c/ Haematocrit (per cent)

d/ White blood cells (/cc)

e/ Reticulocytes (/cc)

f/ Mean corpuscular volume (u^3)

g/ Mean corpuscular haemoglobin (uug)

h/ Mean corpuscular haemoglobin concentration (per cent)

i/ Blood urea nitrogen (mg per cent)

j/ Serum glutamic pyruoic transaminase
