Table 1. ESR parameters and line widths of Cu(II): thiostrepton complex in different solvents.

Complex in the solvent	Liquid nitrogen temperature $g_{\parallel}$	$g_{\perp}$	$A_{\parallel}$ (10^{-4} cm^{-1})	Room temperature g	ΔH_{pp}	A (10^{-4} cm^{-1})
Dimethylformamide	2.2765	2.0646	134	2.1312	235 G	—
Dimethylsulphoxide	—	2.0696	—	2.1434	135 G	—
Pyridine	2.2762	2.0580	154	2.1042	—	67.31
Powder sample	—	—	—	2.1315	170 G	—

Therefore in pyridine, with the solvent entering into co-ordination at the unoccupied co-ordinating positions of the metal ion, the predominant ligand environment around each metal in the complex would be of N atoms. IR and CD studies of the complex have indicated that some of the O atoms of the amide carbonyls and N atoms of azomethine groups of the ligand are involved in co-ordination with the metal ion (unpublished results). In such a case, with the additional N atoms of pyridine entering into coordination with the metal, it appears that the ligand structure around each metal ion of the complex in this solvent is a distorted octahedral symmetry.

Based on these data, the following conclusions are drawn:

i) The observed g values of the complex (Table 1) indicate a well co-ordinated metal ion.

ii) The unresolved ESR spectra of the complex in DMSO and DMF at a very low concentration even at LNT are indicative of a very strong persisting interaction between the Cu(II) ions present well within the complex molecule.

iii) The $g_{\parallel}$ and $A_{\parallel}$ values of the complex in DMF at LNT are characteristic of the stereo structure arising due to either Cu-$(DMF)_4^{2+}$ or Cu-$(DMF)_6^{2+}$ formation[5].

iv) Pyridine is entering into co-ordination with the Cu(II) ion of the complex, while DMSO is working as a non-interacting solvent.

v) Since the four Cu(II) ions present in each molecule of the antibiotic appear to have different ligand environment, it is not possible to conclude from these ESR parameters, a precise ligand structure around the metal ion.

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Residues of dichlorodiphenyltrichloroethane and metabolites in zooplankton from the Arabian Sea

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Studies on concentrations of organochlorine pesticide residues of the dichlorodiphenyltrichloroethane (DDT) group in zooplankton from the Arabian Sea show that these organisms metabolize DDT mainly to dichlorodiphenyldichloroethane (DDD) which is known to be degraded to the excretable dichlorodiphenylacetic acid (DDA) in higher organisms. This might explain the higher values of total DDT residues noted in zooplankton from the Arabian Sea compared to those in fish.

STUDIES have been made earlier on dichlorodiphenyltrichloroethane (DDT) residues in zooplankton from the Arabian Sea[1,2]. In the present study the levels of the metabolites of DDT namely, dichlorodiphenyldichloroethene (DDE) and dichlorodiphenyldichloroethane (DDD) were measured as it was felt that they might play a role in the bioaccumulation of DDT in the higher trophic levels. The zooplankton samples were collected using a neuston net from the Arabian Sea during the 47th cruise of *ORV Sagar Kanya*. Samples from three stations in a transect off Bombay (Figure 1),

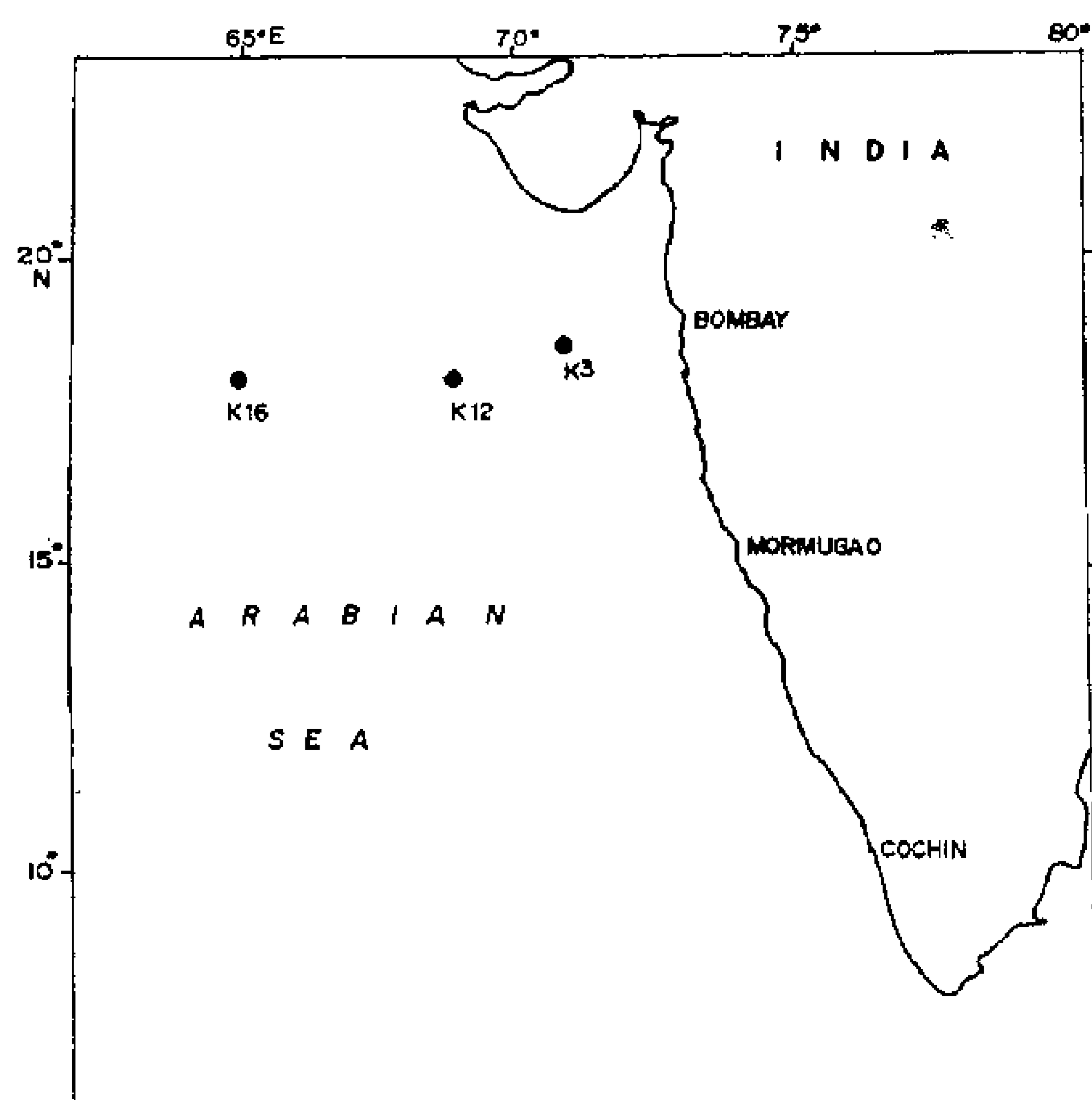

Figure 1. Location of collection sites.

one coastal (K-3;18°42'N 71°16'E) and two open ocean (K-12;18°N 69°E and K-16; 18°N 65°E), were processed for organochlorine pesticide residues according to the method of Harvey *et al.*[3] The residues were analysed using a Perkin Elmer Model 8900 gas chromatograph fitted with a ^{63}Ni electron capture detector (240°) and a glass column (1.83 m × 0.64 cm OD, 3 mm ID) packed with 4% SE-30 + 6% OV-210 on 80/100 Chromosorb W-HP (200°). Carrier gas was argon (+5% methane) at a flow rate of 65 ml^{-1}. The residues were identified and quantitated with reference to standard (EPA) compounds. The detection limit was 0.002 μg g^{-1} (3, $n = 10$) for *p,p'*-DDD. Aldrin included as an internal standard indicated a recovery of 92 to 95% by this method.

DDT and its metabolites, DDD and DDE were detected in all the samples, confirming the presence of DDD reported earlier in zooplankton[2]. The DDE fraction was lower than the DDD fraction at all stations (Table 1), indicating that DDD is the major product of DDT metabolism in zooplankton. While total DDT (t-DDT; DDT+DDE+DDD) residue concentrations decreased in a gradient from nearshore to offshore (Table 1), the concentration of DDD was seen to increase from near the coast to offshore. This could perhaps be attributed to the slower removal, through sedimentation, of pesticides in the open ocean compared to nearshore on account of lower particulate loads, leading to a longer interaction time between DDT residues and zooplankton in the surface waters. Also, differences were noted in the concentrations of metabolites of DDT in samples dominated by different species of zooplankton obtained from two depths. As seen from Table 1, the concentrations of DDD, DDE and DDT in zooplankton consisting mainly of ostracods sampled at the surface at station K-3 were 19.96%, 12.19% and 67.84% of t-DDT, respectively. On the other hand, in the sample dominated by copepods and decapods obtained from 0.5 m at the same station the values were 31.93%, 24.31% and 43.76%, respectively. These differences may reflect varying abilities of the different species to degrade DDT. Laboratory studies have suggested different patterns of degradation of DDT by different species of zooplankton: no significant metabolism of *p,p'*-DDT was observed in copepods[4] but aquatic insects have been found to degrade the same primarily to DDE while crustacea convert it to a variety of other compounds besides DDE and DDD[5]. The quantity of DDD produced was, however, less than 10% whereas in the natural environment we found that the total amount of DDD present in mixed species of zooplankton ranged from about 20% to 45% of the total DDT present. The food chain has been envisioned as the major source of DDT accumulation in fish[6] and zooplankton form an important link in the transfer of organic material from the primary producers to the planktivorous fishes. However, studies on organochlorine pesticide residues in zooplankton from the Arabian Sea[1,2] including the present one have all shown that the concentration of t-DDT in zooplankton is much higher than that in both planktivorous and carnivorous fish from the coastal as well as the open ocean regions of the Arabian Sea[7]. The reason for this could be that DDD, which the present study has shown to be the major metabolite of DDT in zooplankton, can, in the higher trophic orders be metabolised to DDA, a water-soluble hence excretable compound[8]. This is an

Table 1. Levels of DDT and metabolities in the Arabian Sea zooplankton.

Station No.	Sampling depth	Major zooplankton	Residue conc. (ng. g^{-1}. wet. weight)*							Conc. as % t-DDT		
			o,p'-DDD	*p,p'*-DDD	*o,p'*-DDE	*p,p'*-DDE	*o,p'*-DDT	*p,p'*-DDT	t-DDT (μg g^{-1}	DDD	DDE	DDT
K-3	Surface	Ostracods	52.7 ± 0.05	59.8 ± 0.04	27.30 ± 0.02	41.40 ± 0.03	159.20 ± 0.15	223.20 ± 0.19	0.563	19.96	12.19	67.84
	0.5 m	Decapods	8.05 ± 0.002	14.10 ± 0.003	11.90 ± 0.007	8.90 ± 0.002	27.11 ± 0.01	16.20 ± 0.004	0.083	31.93	24.31	43.76
K-12	Surface	Ostracods	29.1 ± 0.02	28.9 ± 0.01	10.40 ± 0.02	16.20 ± 0.006	20.6 ± 0.02	63.9 ± 0.03	0.169	34.29	15.73	49.97
K-16	Surface	Ostracods	30.2 ± 0.05	21.2 ± 0.03	8.6 ± 0.03	13.8 ± 0.02	9.9 ± 0.06	25.7 ± 0.06	0.109	46.98	20.48	32.54

t-DDT: (DDE+DDD+DDT)
*Mean of three replicates.

important point to be noted especially while making impact assessments of DDT input and output in aquatic environments.

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Phosphoglucomutase-I, esterase-D, acid phosphatase, glyoxalase-I and phosphoglucoisomerase in hair sheath of cadavers

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One hundred and three cadavers were typed for five red cell enzymes detected in the hair sheath. Matching probabilities for the five enzymes were highly significant. The complementary discrimination probability, which measures the likelihood that two individuals do not match, was examined. Satisfactory results were obtained for all the enzymes, even when samples stored at room temperature for up to 18 days were used. Hair sheath typing of these enzymes thus constitutes a powerful method of identification of individuals in cases of advanced putrefaction and mass disaster, where hair may be the only clue material.

EXAMINATION of hair for personal identification from the forensic point of view is important in those cases where the only clue material found is hair. The enzymes phosphoglucomutase (PGM), adenosine deaminase (ADA), esterase-D (EsD), phosphohexose isomerase (PHI) and 6-phosphogluconate dehydrogenase (6-PGD) have been found in sufficient concentration to allow typing by starch and agarose–starch electrophoresis, and by isoelectric focusing for some of the enzymes. Although typing of hair sheath from healthy donors for the determination of various enzymes has been reported by many workers[1–6], typing of cadavers has not been reported. In the present study hair sheaths from 103 cadavers from a North Indian population were typed for five red cell enzymes. Samples were also stored at room temperature to determine the stability of enzyme for typing.

Hair samples, with sheaths, were obtained from 103 cadavers sent for autopsy. Blood samples from the cardiac region were also taken. Red blood cells and hair samples of known PGM, EsD, acid phosphatase (AP), glyoxalase-I (GLO-I) and phosphoglucoisomerase (PGI) type were also taken as controls.

Plucked hair, bearing sheath cells, were attached to a glass slide using cellotape in such a way that the roots were completely exposed to air. The hair samples were stored at room temperature for up to 6 weeks.

Hair sheaths from five or six hairs from each case were scraped with a scalpel and then macerated with a few drops of distilled water. Extracts prepared from old samples were treated with a drop of Cleands reagent for 4–5 min before electrophoresis.

Electrophoresis and detection of PGM, AP and GLO-I were carried out according to the *Biological Manual* (Metropolitan Police Forensic Science Laboratory, London) except that the gel was a mixture of agarose and starch, and some of the conditions of electrophoresis were changed: PGM, 1:2 agarose–starch, 16–17 V/cm, 2.5 h; GLO-I, 2:1 agarose–starch, 10–11 V/cm, 105 min; AP, 1:2 agarose–starch, 18 V/cm, 3 h. Samples were typed for EsD according to Harris and Hopkinson except that a mixed gel was prepared (2:1 agarose–starch) and the run was at 15 V/cm for 2 h. PGI isoenzymes were separated by horizontal starch gel electrophorosis as described by Papiha and Chahal[7] using fructose 6-phosphate (disodium salt) in the staining mix.

The results are given in Table 1. The gene frequencies obtained for PGM, GLO-I, EsD, AP and PGI agree with those obtained for northern Indian populations[8–12].

The results of tests of enzyme typability of stored

Table 1. Phenotypes and allele frequencies for five enzymes detected in blood and hair sheath of cadavers from North India.

Enzyme	Phenotype	No. of samples*	Allele frequency
PGM	1–1	53	*PGM1* 0.684
	2–1	35	*PGM2* 0.315
	2–2	15	
EsD	1–1	62	*EsD1* 0.771
	2–1	35	*EsD2* 0.228
	2–2	6	
AP	p^{a+b-}	13	p^a 0.33
	p^{a+b-}	42	p^b 0.669
	p^{a-b+}	48	
GLO-I	1–1	8	*GLO1* 0.266
	2–1	39	*GLO2* 0.733
	2–2	56	
PGI	1–1	101	*PGI1* 98.05
	3–1	2	*PGI3* 1.90

*Total number of samples tested, 103.