Organometals and Organometalloids

Occurrence and Fate in the Environment

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Aspects of Mercury(II) Thiolate Chemistry and the Biological Behavior of Mercury Compounds

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Complex formation between mercury compounds and thiols, e.g. cysteine, is believed to play a major role in the biological chemistry of mercury(1). The greater affinity of Hg(II) and MeHg(II) for thiols than other possible biological donor ligands has been well documented by stability constant studies in aqueous solution (2,3). Our interest in mercury(II) thiolates stems from studies of the chemistry of the antidote British anti-Lewisite which indicated that the structure and reactivity of simple thiolate complexes was little understood. In this review our recent work on the interaction of inorganic and organomercury compounds with British anti-Lewisite, simple thiols and sulphur containing amino acids is discussed, followed by an account of animal studies of the distribution and metabolism of phenylmercury compounds. In discussing the implications of chemical results, e.g. reactivity of thiolates, for the biological behaviour of mercury compounds it is assumed here that chemical studies provide only plausible pathways for biological behaviour.

In recent years other workers have reported studies of mercury thiolates that are related to the work described here, in particular nuclear magnetic resonance studies of the interaction of MeHg(II) with thiols (4-9) and the preparation (10-16) and X-ray structural analysis of key complexes of Hg(II), MeHg(II), and PhHg(II) with sulphur containing amino acids (10-15).

Complexes of British anti-Lewisite and other Thiols

British anti-Lewisite [dimercaprol, 2,3-dimercaptopropanol; abbreviated $BALH_3$ to indicate loss of thiol protons on complex formation, e.g. Hg(BALH)] has been used for the treatment of mercury poisoning in humans (17,18) and has been studied extensively in animal experiments(18-24). Although it may be eventually replaced by a more satisfactory treatment, e.g. hemodialysis (25,26), it is successful for poisoning by

inorganic mercury (17,18) and is the most satisfactory antidote for phenylmercury(II) poisoning [animal experiments only to date (18)], but has no therapeutic effect for methylmercury(II) poisoning in humans or animals (18). For PhHg(II) poisoning $BALH_3$ greatly increases the amount of mercury in the brain compared with the bodily distribution in the absence of $BALH_3$ treatment (18,19,20,21), and for MeHg(II) it merely hastens the distribution of mercury and may increase the amount of mercury in the brain (18). An increased mercury content in the brain is undesirable, as it attacks the central nervous system. $BALH_3$ also increases the amount of mercury in the brain following its administration for inorganic mercury poisoning (22,23,24), but this effect has been explained in terms of the timing and dosage of $BALH_3$ (24).

Isolation of Hg(BALH) (27,28) and evidence for the formation of $[Hg(BALH)_2]^{2-}$ (27), $(PhHg)_2BALH$ (28), and $(RHg)_nBALH_{3-n}$ [n = 1 (29), 2 (28); R = $CH_2CH(OMe)CH_2R'$] were reported by several workers soon after the introduction of $BALH_3$ as an antidote for heavy metal poisoning. Mercuric chloride reacts immediately with $BALH_3$ in water to form a white solid identified as Hg(BALH) (27,28,30,31).

$$HgCl_2 + BALH_3 \rightarrow Hg(BALH) + 2HCl$$

Crystal structures of simple thiolates $Hg(SR)_2$ reveal either linear monomers [R=Me (32), Et (33)] (Figure 1) or a polymeric structure with tetrahedral mercury (R=Bu^t) (34) (Figure 2). Infrared and Raman spectra indicate that highly insoluble Hg(BALH) has a polymeric structure based on linear coordination for mercury (31) (Figure 3), rather than the cyclic structure usually presented (Figure 4). Thus, Hg(BALH) has ν_{as}(SHgS) 348 and ν_s(SHgS) 298 cm^{-1}, similar to that of $Hg(SMe)_2$ (377 and 297 cm^{-1}) and well removed from tetrahedral mercury in $Hg(SBu^t)_2$ (172 and 188 cm^{-1}) (31). Spectroscopic properties appropriate for identification of Hg(II) thiolates, e.g. infrared, Raman, and nuclear magnetic resonance, are presented elsewhere (31,35,36,37,38,39).

The simple thiolates $Hg(SR)_2$ are insoluble in water but soluble in organic solvents, e.g. $Hg(SR)_2$ (R=Et,Bu^t,Ph) are monomeric in chloroform. Hg(BALH) is insoluble in water, even at concentrations of ca. 10^{-4}M (40). An impure form of Hg(BALH) can be isolated by reaction of mercuric acetate with $BALH_3$ in pyridine (35). This solid is soluble in pyridine, and the related complex of 1,3-dimercaptopropanol, Hg(DMPH), can be isolated from water and forms a dimer in pyridine (35). The structure of Hg(DMPH) in pyridine is unknown but presumably involves pyridine coordination, $[Hg(DMPH)py_x]_2$, as it crystallizes as $Hg(DMPH)py_{1.5}$ containing coordinated pyridine. The solubility of impure Hg(BALH) in pyridine is of interest as Hg(BALH) is presumably formed in many "environments" *in vivo*,

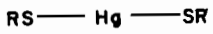
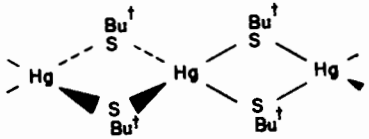


Figure 1.



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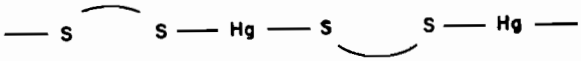

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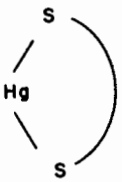


Figure 4.

and pyridine solubility suggests higher solubility in lipid tissue than more aqueous regions. The neutral complex may be present as a dimer $[Hg(BALH)L_x]_2$ related to Hg(DMPH) in pyridine, or possibly as the cyclic complex (Figure 4) with additional ligands coordinated to mercury.

In alkaline solution Hg(BALH) dissolves on addition of excess $BALH_3$ suggesting (27) formation of $[Hg(BALH)_2]^{2-}$, and addition of $BALH_3$ to a solution of impure Hg(BALH) in pyridine results in an increase in conductivity (35). Stability constants for formation of the neutral and ionic complexes in water have recently been determined by potentiometric titration (40), and the very high values contribute to the effectiveness of British anti-Lewisite as an antidote.

$$Hg^{2+} + BALH^{2-} \rightleftharpoons Hg(BALH) \qquad \text{Log } K = 25.74 \pm 0.45$$

$$Hg(BALH) + BALH^{2-} \rightleftharpoons [Hg(BALH)_2]^{2-} \qquad \text{Log } K = 8.61 \pm 0.10$$

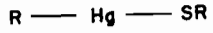
Organomercury derivatives of $BALH_3$ may be obtained by reaction with phenylmercuric acetate in water and methylmercuric acetate in benzene (35).

$$2RHgO_2CMe + BALH_3 \rightarrow (RHg)_2BALH + 2MeCO_2H$$

Infrared and Raman spectra of these complexes and other organomercury thiolates indicate monomeric structures in the solid state as ν(Hg-S) values (326-388 cm^{-1}) are in the region expected for linear coordination for mercury, and coincidence of infrared and Raman values indicate absence of a centre of symmetry at mercury (Figure 5,6) (35), thus excluding dimeric structures similar to that formed by related PhHg(II) alkoxides in benzene (Figure 7) (41).

^1H NMR spectroscopy is particularly useful for characterization of organomercury compounds. Thus, $(MeHg)_2BALH$ has $J(^1H\text{-}^{199}Hg)$ 169 Hz for the MeHg(II) group, and PhHg(II) thiolates have $J(ortho_H\text{-}^{199}Hg)$ 144-158 Hz and $J(ortho_H\text{-}meta_H)$ 6-8 Hz (35).

The complexes $(RHg)_2BALH$ (R=Me,Ph) are insoluble in water but dissolve in pyridine and dimethylsulphoxide, and the related thiolate of lower molecular weight, $PhHgSCH_2CH_2OH$, is soluble and monomeric in chloroform. However, organomercury thiolates formed from naturally occurring thiols *in vivo* are likely to be water soluble, e.g. the L-cysteine complexes $MeHgSCH_2CH(NH_3)CO_2\cdot H_2O$ and $PhHgSCH_2CH(NH_3)CO_2$ contain hydrophilic zwitterionic groups and crystallize from aqueous ethanol (12,36). Thus, displacement of biological thiol ligands with $BALH_3$ is expected to form more lipid soluble complexes, as suggested by Berlin *et al.* (20), and may account for higher concentrations of mercury in brain tissue of animals administered $BALH_3$ after injection of organomercury compounds when compared



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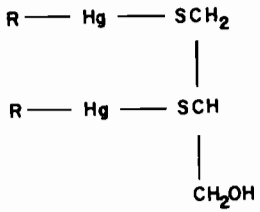

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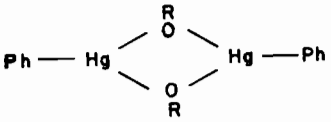

Figure 7.

with concentrations in the absence of $BALH_3$ treatment.

It was found that $(PhHg)_2BALH$ decomposes at ambient temperature in acetone, benzene, and methanol to form Ph_2Hg (30,35) (Table I).

$$(PhHg)_2BALH \rightarrow Ph_2Hg + Hg(BALH)$$

Table I
Decomposition of Some Phenylmercury (II) Thiolates[a]

Complex	Solvent	Yield of Ph_2Hg(%)
$(PhHg)_2BALH$	acetone	96
$(PhHg)_2BALH$	benzene	100
$PhHg(H_3cyst)$	benzene	55
$PhHg(H_3pen)$	benzene	81
$(PhHg)_2(H_2cyst)\cdot H_2O$	benzene	44
$(PhHg)_2H_2pen$	benzene	43

[a]From references 35,36. Suspensions at ambient temperature were stirred magnetically for seven days. Ph_2Hg was isolated as a pure solid from the filtrate.
[b]Yield of Ph_2Hg based on 'Ph'.
[c]$H_3cyst = SCH_2CH(NH_3)CO_2$; $H_2cyst = SCH_2CH(NH_2)CO_2$; similarly for $HSCMe_2CH(NH_3)CO_2$, DL-penicillamine.

If this reaction occurs *in vivo* it may also contribute to redistribution of mercury, and to indicate whether Ph_2Hg formation may be a general biological reaction in the absence of $BALH_3$ a series of PhHg(II) complexes of sulphur-containing amino acids was prepared and their stabilities studied (36). The complexes were synthesized by reaction of phenylmercuric acetate with the amino acids in aqueous ethanol, e.g.

$$2PhHgO_2CMe + H_4cyst \rightarrow (PhHg)_2(H_2cyst)\cdot H_2O + 2MeCO_2H$$

The DL-penicillamine complexes have been prepared by other workers, but the stability of the complexes toward decomposition had not been studied (16).

The amino acid complexes were found to decompose in benzene to form Ph_2Hg (Table I). The importance of these reactions, and decomposition of $(PhHg)_2BALH$, is difficult to assess as they are solvent dependent and rates of decomposition vary, e.g. $(PhHg)_2BALH$ and amino acid complexes may be readily prepared in

aqueous solution, they decompose slowly in benzene, and when $PhHgO_2CMe$ and $BALH_3$ are reacted in ethanol immediate precipitation occurs and Ph_2Hg may be obtained from the filtrate on filtration. If Ph_2Hg is formed in vivo then the biological behaviour of Ph_2Hg is of interest as phenylmercury compounds, e.g. $PhHgO_2CMe$, are still widely used in agriculture and medicine. It has been reported that Ph_2Hg in "scarcely detectable" concentration formed by degradation of phenylmercuric acetate (formerly contained in derelict steel drums), was sufficiently toxic to kill fish within a few hours in the Boone Reservoir, Tennessee Valley (43).

Biological Behaviour of Diphenylmercury

Diphenylmercury has quite different physical and chemical properties than PhHg(II) compounds, e.g. it is a neutral non-polar molecule insoluble in water but soluble in organic solvents and is thus expected to be lipid soluble (44), and in contrast to PhHg(II) compounds (45,46,47) it interacts only weakly with donor molecules (48,49,50). Similarly, Me_2Hg does not form complexes (45) but MeHg(II) forms stable complexes, e.g. $[MeHgL]^+$ with pyridine (51,52), 2,2'-bipyridyl (51,52,53, 54), and 1,10-phenanthroline (52,53).

In distribution and metabolism studies we have injected ethanol solutions of mercuric chloride, phenylmercuric acetate, or Ph_2Hg intraperitoneally into rats (55,56). The rats were sacrificed at intervals ranging from 20 min. to 7 days and samples of blood, brain, liver, kidney, muscle, fat, and spleen were analysed for mercury. In another series of experiments faecal and urinary excretion was monitored for several days after injection.

During the first few days after injection, urinary excretion of mercury was much higher for the diphenylmercury-injected rats than for the phenylmercuric acetate or mercuric chloride-injected rats, with mercuric chloride having the lowest rate of excretion. Faecal excretion was similar for the three compounds, with phenylmercuric acetate being more rapidly excreted (Table II).

Analyses of blood and tissues for total mercury indicated that after initial marked differences in brain and fatty tissue concentrations, the distribution of mercury for Ph_2Hg resembled those of the other compounds after 1 day, but concentrations were generally lower than for the other compounds (55,56). The lower concentrations are explained by the more rapid excretion of mercury from Ph_2Hg.

During the first hour after injection mercury from Ph_2Hg accumulated at a higher concentration in the brain than from the other compounds, but after 6 hours these concentrations had decreased considerably (55,56). The concentration of mercury in fatty tissue was 10-20 times higher for diphenylmercury-

Table II
Urinary and Faecal Excretion of Mercury from Rats within Two Days of Injection[a]

	$HgCl_2$		$PhHgO_2CMe$		Ph_2Hg	
	Percentage of dose excreted					
Urinary Excretion:	2.5	2.2	4.8	8.0	30.5	38.3
Faecal Excretion:	5.2	4.5	12.2	8.4	5.6	3.6
Urinary + Faecal:	7.7	6.7	17.0	16.4	36.1	41.9

[a]From reference 56. Analyses for total mercury, as described elsewhere (55). Two rats were injected intraperitoneally with each compound, dose 248 mg mercury, all rats of weight 160 g.

injected rats at 20 min. after injection, but then rapidly dropped to values similar to the other mercury compounds (Table III). The much higher concentration of mercury in brain and fatty tissue immediately after Ph_2Hg injection is consistent with distribution of mercury as Ph_2Hg, and this was confirmed by thin-layer chromatography. A sample of fatty tissue taken from a diphenylmercury-injected rat 20 min. after injection was blended with benzene using a small Waring blender, and thin-layer chromatography showed the presence of diphenylmercury (ultraviolet irradiation); the silica gel of the plate at the R_f value of Ph_2Hg contained 5.19 mg. of Hg/g of silica gel compared with 0.15 mg/g for silica gel at lower R_f value on the same plate.

It has been established that phenylmercury is degraded to inorganic mercury in a few days in rats (57,58,59,60,61). Daniel et al. (60) represent this breakdown as

$$C_6H_5Hg^+ + H^+ \rightarrow C_6H_6 + Hg^{2+}$$

A similar breakdown may occur for Ph_2Hg, presumably via PhHg(II), as the initial high concentrations of mercury in brain and fatty tissue fall to values similar to that obtained with the other compounds after 6 hr. and 1 hr., respectively. Thus, if Ph_2Hg is formed in vivo its biological effects are difficult to evaluate as it is more rapidly excreted than PhHg(II) and apparently broken down by the body, but has a quite different initial distribution. However, it is of interest to note that although mercury vapour is oxidized to Hg(II) in ca. 30 sec. in blood this is sufficient time for mercury (from vapour) to

Table III
Concentration of Mercury in Brain and Fatty Tissues of Hooded Wistar Rats Injected Intraperitoneally with Mercury Compounds[a]

Time	Brain	Fat
A. Dose of 6 mg. of Hg/kg of rat.		
	mercuric chloride	
20 min. (2)	0.16 ± 0.02	10.5 ± 2.8
1 hr. (2)	0.33 ± 0.07	4.8 ± 2.1
6 hr. (2)	0.16 ± 0.01	3.4 ± 1.3
1 day (2)	0.24	15.7 ± 5
	phenylmercuric acetate	
20 min. (2)	0.14 ± 0.04	5 ± 2
1 hr. (2)	0.47 ± 0.03	4.4 ± 1.2
6 hr. (2)	0.9 ± 0.3	4.7 ± 0.2
1 day (2)	0.65 ± 0.02	3.5 ± 0.3
	diphenylmercury	
20 min. (2)	0.9 ± 0.2	147 ±13
1 hr. (2)	0.7 ± 0.2	10.4 ± 3.2
6 hr. (2)	0.26 ± 0.01	10.1 ± 3.4
1 day (2)	0.20 ± 0.03	3.6 ± 0.2
B. Dose of 1.5 mg. of Hg/kg of rat.		
	mercuric chloride	
20 min. (1)	0.04	2.34
	phenylmercuric acetate	
20 min. (1)	0.01	0.9
	diphenylmercury	
20 min. (1)	0.3	27.8

[a]From reference 56. Recorded as μg of Hg/g tissue, wet weight, and the range of values is indicated. The number of rats in each category is given in parentheses with the time.

achieve an ca. ten-fold higher accumulation in the brain than from inorganic mercury poisoning (27,62) leading to higher toxicity of mercury vapour.

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