Organometals and Organometalloids
Occurrence and Fate in the Environment

F. E. Brinckman, EDITOR
National Bureau of Standards

J. M. Bellama, EDITOR
University of Maryland

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CONTENTS

Dedication ................................................................. xi
Preface ................................................................. xiii

1. Biosynthesis of Organometallic and Organometalloidal Compounds 1
   Frederick Challenger

2. Biotransformations of Sulfur as Evolutionary Prototypes for
   Metabolism of Metals and Metalloids ............................ 23
   George E. Parris

3. Occurrence of Biological Methylation of Elements in the
   Environment ......................................................... 39
   Y. K. Chau and P. T. S. Wong

4. Kinetic and Mechanistic Studies on B12-Dependent Methyl
   Transfer to Certain Toxic Metal Ions ............................ 54

5. Aqueous Chemistry of Organolead and Organothallium
   Compounds in the Presence of Microorganisms ................... 65
   F. Huber, U. Schmidt, and H. Kirchmann

6. Bioorganotin Chemistry: Stereo- and Situselectivity in the
   Monoxygenase Enzyme Reactions of Cyclohexyltin Compounds ... 82
   Richard H. Fish, John E. Casida, and Ella C. Kimmel

7. Anaerobic and Aerobic Alkylation of Arsenic ................... 94
   Barry C. McBride, Heather Merilees, William R. Cullen,
   and Wendy Pickett

8. Arsenic Uptake and Metabolism by the Alga Tetraselmis Chui 116
   N. R. Bottino, E. R. Cox, K. J. Irgolic, S. Maeda,
   W. J. McShane, R. A. Stockton, and R. A. Zingaro

9. The Chemistry of Organometallic Cations in Aqueous Media ...... 130
   R. Stuart Tobias

10. Organosilanes as Aquatic Alkylators of Metal Ions ............. 149
    Richard E. DeSimone

11. Influence of Environmental Parameters on Transmethylation
    between Aquated Metal Ions .................................... 158
    K. L. Jewett, F. E. Brinckman, and J. M. Bellama

12. Demethylation of Methylcobalamin: Some Comparative Rate
    Studies .......................................................... 188
    John S. Thayer

13. Mechanisms for Alkyl Transfers in Organometals ................ 205
    Jay K. Kochi
<table>
<thead>
<tr>
<th>Pathways for Formation of Transition Metal–Carbon Bonds in Protic Media</th>
<th>235</th>
</tr>
</thead>
<tbody>
<tr>
<td>James H. Espenson</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Unstable Organometallic Intermediates in a Protic Medium</th>
<th>247</th>
</tr>
</thead>
<tbody>
<tr>
<td>James H. Weber and Mark W. Witman</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Chloramine Equilibria and the Kinetics of Disproportionation in Aqueous Solution</th>
<th>264</th>
</tr>
</thead>
<tbody>
<tr>
<td>Edward T. Gray, Jr., Dale W. Margerum, and Ronald P. Huffman</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Chlorination and the Formation of N-Chloro Compounds in Water Treatment</th>
<th>278</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dale W. Margerum, Edward T. Gray, Jr., and Ronald P. Huffman</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ozone and Hydroxyl Radical-Initiated Oxidations of Organic and Organometallic Trace Impurities in Water</th>
<th>292</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jürg Hoigné and Heinz Bader</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Partition of Organoelements in Octanol/Water/Air Systems</th>
<th>314</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stanley P. Wasik</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Aspects of Mercury(II) Thiolate Chemistry and the Biological Behavior of Mercury Compounds</th>
<th>327</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allan J. Canty</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mercury, Lead, and Cadmium Complexation by Sulphydryl-Containing Aminoacids. Implications for Heavy-Metal Synthesis, Transport, and Toxicology</th>
<th>339</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arthur J. Carty</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Release Mechanisms of Organotin Toxicants from Coating Surfaces: a Leaching Model for Antifouling Coatings</th>
<th>359</th>
</tr>
</thead>
<tbody>
<tr>
<td>Charles P. Monaghan, Vasant H. Kulkarni, and Mary L. Good</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Metal-Ion Transport Mediated by Humic and Fulvic Acids</th>
<th>372</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gordon K. Pagenkopf</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Organotins in Biology and the Environment</th>
<th>388</th>
</tr>
</thead>
<tbody>
<tr>
<td>J. J. Zuckerman, Robert P. Reisdorf, Harry V. Ellis III, and Ralph R. Wilkinson</td>
<td></td>
</tr>
</tbody>
</table>

Index                                                                                                    425
Aspects of Mercury(II) Thiolate Chemistry and the Biological Behavior of Mercury Compounds

ALLAN J. CANTY
Chemistry Department, University of Tasmania, Hobart, Tasmania, Australia

Complex formation between mercury compounds and thiols, e.g. cysteine, is believed to play a major role in the biological chemistry of mercury(I). 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 BALH₃ 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 BALH₃ greatly increases the amount of mercury in the brain compared with the bodily distribution in the absence of BALH₃ 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. BALH₃ 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 BALH₃ (24).

Isolation of Hg(BALH) (27,28) and evidence for the formation of [Hg(BALH)]₂⁻ (27), (PhHg)₂BALH (28), and (RHg)ₙBALH₃⁻ [n = 1 (29), 2 (28); R = CH₂CH₂(OMe)CH₂R⁻] were reported by several workers soon after the introduction of BALH₃ as an antidote for heavy metal poisoning. Mercuric chloride reacts immediately with BALH₃ in water to form a white solid identified as Hg(BALH) (27,28,30,31).

\[ \text{HgCl}_2 + \text{BALH}_3 \rightarrow \text{Hg}^{(\text{BALH})} + 2\text{HCl} \]

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=But) (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 \( \nu_\text{SHgS} \) 348 and \( \nu_\text{S-S} \) 298 cm⁻¹, similar to that of Hg(SMe)₂ (377 and 297 cm⁻¹) and well removed from tetrahedral mercury in Hg(SBut)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,But,Ph) are monomeric in chloroform. Hg(BALH) is insoluble in water, even at concentrations of ca. 10⁻⁴M (40). An impure form of Hg(BALH) can be isolated by reaction of mercuric acetate with BALH₃ 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, \([\text{Hg(DMPH)}\text{py}_3]_2\), as it crystallizes as Hg(DMPH)py₁,₅ 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.

RS — Hg — SR

Figure 2.

Figure 3.

Figure 4.
and pyridine solubility suggests higher solubility in lipid tissue than more aqueous regions. The neutral complex may be present as a dimer \([Hg(BALH)_{2}]_2\) 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.

\[
\begin{align*}
Hg^{2+} + BALH^{2-} & \rightleftharpoons Hg(BALH) & \log K = 25.74 \pm 0.45 \\
Hg(BALH) + BALH^{2-} & \rightleftharpoons [Hg(BALH)_2]^{2-} & \log K = 8.61 \pm 0.10
\end{align*}
\]

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 \(v(Hg-S)\) 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 \(PhHg(II)\) alkoxydes in benzene (Figure 7) (41).

\(^1H\) NMR spectroscopy is particularly useful for characterization of organomercury compounds. Thus, \((MeHg)_2BALH\) has \(J(^1H-199Hg)\) 169 Hz for the \(MeHg(II)\) group, and \(PhHg(II)\) thiolates have \(J(orthoH-199Hg)\) 144-158 Hz and \(J(orthoH-metaH)\) 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
Figure 5.

\[ \text{R} \quad \text{Hg} \quad \text{SR} \]

Figure 6.

\[ \begin{align*}
\text{R} & \quad \text{Hg} \quad \text{SCH}_2 \\
\text{R} & \quad \text{Hg} \quad \text{SCH} \\
\text{CH}_2\text{OH} &
\end{align*} \]

Figure 7.

\[ \begin{align*}
\text{Ph} & \quad \text{Hg} \\
\text{Hg} & \quad \text{Ph} \\
\text{O} & \quad \text{R} \\
\text{R} & \quad \text{O}
\end{align*} \]
with concentrations in the absence of BALH₃ treatment.

It was found that (PhHg)₂BALH decomposes at ambient temperature in acetone, benzene, and methanol to form Ph₂Hg (30,35) (Table I).

\[(\text{PhHg})_2\text{BALH} \rightarrow \text{Ph}_2\text{Hg} + \text{Hg}\text{(BALH)}\]

Table I
Decomposition of Some Phenylmercury (II) Thiolates

<table>
<thead>
<tr>
<th>Complex</th>
<th>Solvent</th>
<th>Yield of Ph₂Hg(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(PhHg)₂BALH</td>
<td>acetone</td>
<td>96</td>
</tr>
<tr>
<td>(PhHg)₂BALH</td>
<td>benzene</td>
<td>100</td>
</tr>
<tr>
<td>PhHg(H₃cyst)</td>
<td>benzene</td>
<td>55</td>
</tr>
<tr>
<td>PhHg(H₃pen)</td>
<td>benzene</td>
<td>81</td>
</tr>
<tr>
<td>(PhHg)₂(H₂cyst)·H₂O</td>
<td>benzene</td>
<td>44</td>
</tr>
<tr>
<td>(PhHg)₂H₂pen</td>
<td>benzene</td>
<td>43</td>
</tr>
</tbody>
</table>

*From reference 35,36. Suspensions at ambient temperature were stirred magnetically for seven days. Ph₂Hg was isolated as a pure solid from the filtrate.

Yield of Ph₂Hg based on Phᵢ.

CH₃cyst = SCH₂CH(NH₃)CO₂; H₂cyst = SCH₂CH(NH₂)CO₂; similarly for HSCMe₂CH(NH₃)CO₂, DL-penicillamine.

If this reaction occurs in vivo it may also contribute to redistribution of mercury, and to indicate whether Ph₂Hg formation may be a general biological reaction in the absence of BALH₃ 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.

\[2\text{PhHgO}_2\text{CMe} + \text{H}_4\text{cyst} \rightarrow (\text{PhHg})_2(\text{H}_2\text{cyst})\cdot\text{H}_2\text{O} + 2\text{MeCO}_2\text{H}\]

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 Ph₂Hg (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 \( \text{PhHgO}_2\text{CMe} \) and \( \text{BALH}_3 \) are reacted in ethanol immediate precipitation occurs and \( \text{Ph}_2\text{Hg} \) may be obtained from the filtrate on filtration. If \( \text{Ph}_2\text{Hg} \) is formed \textit{in vivo} then the biological behaviour of \( \text{Ph}_2\text{Hg} \) is of interest as phenylmercury compounds, e.g. \( \text{PhHgO}_2\text{CMe} \), are still widely used in agriculture and medicine. It has been reported that \( \text{Ph}_2\text{Hg} \) 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 \( \text{PhHg}(\text{II}) \) 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 \( \text{PhHg}(\text{II}) \) compounds (45, 46, 47) it interacts only weakly with donor molecules (48, 49, 50). Similarly, \( \text{Me}_2\text{Hg} \) does not form complexes (45) but \( \text{MeHg}(\text{II}) \) forms stable complexes, e.g. \([\text{MeHgL}]^+\) 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 \( \text{Ph}_2\text{Hg} \) 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 \( \text{Ph}_2\text{Hg} \) 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 \( \text{Ph}_2\text{Hg} \).

During the first hour after injection mercury from \( \text{Ph}_2\text{Hg} \) 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
Urine and Faecal Excretion of Mercury from Rats within Two Days of Injection

<table>
<thead>
<tr>
<th></th>
<th>HgCl₂</th>
<th>PhHgO₂CMe</th>
<th>Ph₂Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Percentage of dose excreted</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urinary Excretion:</td>
<td>2.5</td>
<td>2.2</td>
<td>4.8</td>
</tr>
<tr>
<td>Faecal Excretion:</td>
<td>5.2</td>
<td>4.5</td>
<td>12.2</td>
</tr>
<tr>
<td>Urinary + Faecal:</td>
<td>7.7</td>
<td>6.7</td>
<td>17.0</td>
</tr>
</tbody>
</table>

*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 Ph₂Hg injection is consistent with distribution of mercury as Ph₂Hg, 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 Rf value of Ph₂Hg contained 5.19 mg of Hg/g of silica gel compared with 0.75 mg/g for silica gel at lower Rf 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

\[
\text{C}_6\text{H}_5\text{Hg}^+ + \text{H}^+ + \text{C}_6\text{H}_6 + \text{Hg}^{2+}
\]

A similar breakdown may occur for Ph₂Hg, 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 Ph₂Hg 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

<table>
<thead>
<tr>
<th>Time</th>
<th>Brain</th>
<th>Fat</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mercuric chloride</td>
<td></td>
</tr>
<tr>
<td>20 min. (2)</td>
<td>0.16 ± 0.02</td>
<td>10.5 ± 2.8</td>
</tr>
<tr>
<td>1 hr. (2)</td>
<td>0.33 ± 0.07</td>
<td>4.8 ± 2.1</td>
</tr>
<tr>
<td>6 hr. (2)</td>
<td>0.16 ± 0.01</td>
<td>3.4 ± 1.3</td>
</tr>
<tr>
<td>1 day (2)</td>
<td>0.24</td>
<td>15.7 ± 5</td>
</tr>
<tr>
<td></td>
<td>phenylmercuric acetate</td>
<td></td>
</tr>
<tr>
<td>20 min. (2)</td>
<td>0.14 ± 0.04</td>
<td>5 ± 2</td>
</tr>
<tr>
<td>1 hr. (2)</td>
<td>0.47 ± 0.03</td>
<td>4.4 ± 1.2</td>
</tr>
<tr>
<td>6 hr. (2)</td>
<td>0.9 ± 0.3</td>
<td>4.7 ± 0.2</td>
</tr>
<tr>
<td>1 day (2)</td>
<td>0.65 ± 0.02</td>
<td>3.5 ± 0.3</td>
</tr>
<tr>
<td></td>
<td>diphenylmercury</td>
<td></td>
</tr>
<tr>
<td>20 min. (2)</td>
<td>0.9 ± 0.2</td>
<td>147 ± 13</td>
</tr>
<tr>
<td>1 hr. (2)</td>
<td>0.7 ± 0.2</td>
<td>10.4 ± 3.2</td>
</tr>
<tr>
<td>6 hr. (2)</td>
<td>0.26 ± 0.01</td>
<td>10.1 ± 3.4</td>
</tr>
<tr>
<td>1 day (2)</td>
<td>0.20 ± 0.03</td>
<td>3.6 ± 0.2</td>
</tr>
</tbody>
</table>

A. Dose of 6 mg. of Hg/kg of rat.

B. Dose of 1.5 mg. of Hg/kg of rat.

<table>
<thead>
<tr>
<th>Time</th>
<th>Brain</th>
<th>Fat</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 min. (1)</td>
<td>0.04</td>
<td>2.34</td>
</tr>
<tr>
<td>20 min. (1)</td>
<td>0.01</td>
<td>0.9</td>
</tr>
<tr>
<td>20 min. (1)</td>
<td>0.3</td>
<td>27.8</td>
</tr>
</tbody>
</table>

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

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

### Acknowledgements

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Literature Cited.


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