LOU GEHRIG'S DISEASE (ALS)
THE MERCURY CONNECTION!

It cannot yet be said that mercury has been absolutely proven to be a causative factor in the development of ALS (Amyotrophic Lateral Sclerosis; "Lou Gehrig's Disease"); but recently published research definitely points to that probability.

As early as the mid 1950's, it had been established that the clinical features of chronic mercury intoxication at times mimic motor neuron disease. Subsequently, a number of case studies of ALS-like neuropathies caused by various forms of mercury have been documented.

Recent studies from the Departments of Neurology, Chemistry, Toxicology, Pathology and the Sanders-Brown Center on Aging of the University of Kentucky and the Veterans Administration Medical Center of Lexington, Kentucky further implicate mercury in the development of ALS. The following information is extracted from these studies. [It may be noted that some of the authors of these studies are recognizable as having published findings relating mercury to Alzheimer's Disease.]

TRACE ELEMENT IMBALANCES IN AMYOTROPHIC LATERAL SCLEROSIS
Khare, SS; Ehmann, WD; Kasarskis, EJ; Markesbery, WR.
ABSTRACT: Concentrations of 15 elements were determined by instrumental neutron activation analysis in brain, spinal cord, blood cells, serum and nails of Amyotrophic Lateral Sclerosis (ALS) patients and appropriately matched control subjects. Several significant imbalances were detected in trace element levels in ALS samples compared to control samples. Some of these changes are probably secondary to the loss of tissue mass, especially in spinal cord. However the widespread changes observed in Hg and Se levels in ALS tissues deserve special attention. The significance of these alterations in trace element levels in relation to the pathogenesis of ALS is discussed.

The authors stated: "The changes observed in Hg concentration and the interactions of Hg and Se are worthy of special comment and may possibly be relevant to the pathogenesis of ALS. Although an exact mechanism of Hg neurotoxicity has not yet been elucidated, Hg is known to have a high affinity for the sulphydryl groups of proteins and may subsequently inactivate a protein or an enzyme. This could lead to total inhibition of the cellular function and to cell death."

BIO-PROBE COMMENT: The authors also discussed the significance of the selenium depletion, particularly in light of its established
importance in the detoxification of mercury and protection against the adverse effects of mercury. This subject is discussed in detail in the following, more recent and comprehensive publication, which also addressed other important topics such as why ALS may develop in some individuals exposed to harmful agents (such as mercury) but not others.

**TRACE METALS IN HUMAN NEURODEGENERATIVE DISEASES**

**INTRODUCTION**
Several examples of trace metal neurotoxicity causing recognizable classic syndromes have now been established. These have been documented resulting from subacute or sustained chronic exposure to a toxic metal from an identified environmental source or by intentional poisoning.

Implicating toxic metals in the etiology or pathogenesis of chronic neurodegenerative diseases is more challenging for several reasons:

1. **Dating the onset of the human neurodegenerative disease** is uncertain, thereby making the identification of the source of exposure by epidemiologic study difficult. As a further complication of this factor, a significant degree of neuronal loss must occur before clinical dysfunction is apparent. In the case of ALS, it has been shown that 50% of spinal motor neurons will have degenerated before the typical features of the disease are noticed. Therefore, the exposure to a harmful neurotoxin could have occurred many years preceding the clinical onset of the disease.

2. **Neurodegenerative disorders** are caused by the death of select neurons, rather than wholesale destruction of tissue. The neurotoxin could therefore be very specific in its action and effective at a low dose, making systemic toxicity less likely.

3. **Biopsy material** is not usually available until post-mortem, which is at the end-stage of the disease. At this point, trace metal analysis of brain and spinal cord may not accurately reflect the biochemical condition when the disease process was set in motion.

**THE PATHOLOGY OF ALS**
Amyotrophic Lateral Sclerosis is a chronic neurodegenerative disease. It is characterized clinically by progressive atrophy and weakness of skeletal muscle and small local involuntary muscular contractions visible under the skin. Although clinical variants and familial forms of ALS occur, the classical disease is readily identified by physical findings and electrophysiological studies.

Pathologically, ALS is characterized by atrophy and degeneration of selective motor neurons in the ventral spinal cord and the motor cortex.

The etiology and pathogenesis of ALS are unknown. Viral inclusions have not been found, but study of the 5-10% of patients with a familial pattern suggest that a genetic defect may render motor neurons more susceptible to other secondary insults, such as exposure to an exogenous toxin.

**THE INVOLVEMENT OF TOXIC METALS IN ALS**
The toxic trace element theory of the pathogenesis of ALS has received considerable support and derives its attractiveness from three sources:

1. Epidemiologic considerations indicate that long-term exposure to heavy metal is more common among ALS patients compared to controls.

2. An ALS-like syndrome has been linked to chronic intoxication with mercury and lead.

3. Environmental factors have been implicated in the etiology of a related motor neuron disorder, ie, ALS/Parkinson’s/Dementia in Guamanian subjects.

To date, most studies have examined a very basic hypothesis, that ALS may be caused by chronic, low-level exposure to toxic metals. If this hypothesis is true, then one should be able to analyze tissue from ALS patients and demonstrate that the concentration of toxic metals is higher in ALS compared to age-matched controls.

**RESULTS**
We began our studies of ALS in this traditional mode by analyzing several tissues (brain, spinal cord, serum, blood cells, and nails) from patients and controls for 15 elements by instrumental neutron activation analysis (INAA). The most important finding was a significant elevation of
mercury in brain, blood cells, and serum in ALS patients compared to age-matched controls. The elevation of mercury in ALS could reflect a true excess of body burden of mercury, altered turnover, or perhaps binding to unusual intracellular ligands.

The results of our study also indicated that selenium was reduced in the serum and blood cells of ALS patients. The data were more striking when the ratio of mercury:selenium was computed for each sample in order to study both elements concurrently. This approach not only considered the accumulation of a toxic metal, but also evaluated the integrity of potential detoxification mechanisms. The results of our work indicated that mercury was present to excess relative to selenium in ALS blood cells, serum, and brain.

We have considered that mercury accumulation in motor neurons may be a necessary precondition for ALS-type degeneration to occur. This hypothesis predicts that mercury should be enriched in spinal motor neurons of normal spinal cords and that additional factors would impinge on motor neurons to cause their degeneration in ALS. Our formulation is specific in proposing:

1. Mercury accumulation by neurons is a prerequisite for subsequent neurodegenerative changes to ensue.

2. The ALS phenotype develops either by excessive mercury accumulation or inadequate mercury detoxification.

If mercury is, in fact, an etiologic factor in the pathogenesis of ALS, then one would predict the mercury would accumulate in precisely those neurons which ultimately degenerate in ALS. In order to evaluate this hypothesis, the analysis of mercury must be investigated on a cell-by-cell basis.

Because LAMMS (Laser-Activated Microprobe Mass Analysis) did not provide the requisite sensitivity to detect mercury under our conditions, the mercury-specific photoemulsion histochemical (PH) method described by Moller-Madsen and Danscher in 1986 was adapted to human postmortem spinal cord. Mercury was found localized primarily to the nucleus of motor neurons with lesser amounts seen in the cytoplasm. Mercury was also found associated with spinal motor neurons in normal humans. These data, together with the results of the bulk tissue analyses, indicate that spinal motor neurons have an avidity [ED: Strong affinity] for mercury which could possible render them more susceptible to other neurotoxic agents, thereby conferring a selective vulnerability to neuronal degeneration.

METALLOTHIONEIN IN ALS: SOME SPECULATIONS AND DIRECTION FOR FUTURE RESEARCH.

Metal detoxification may be the more critical factor in the pathogenesis of ALS because it appears unlikely that ALS results from a simple, environmental-type exposure based upon population studies.

The metallothionein (MT) family of proteins has not been investigated in ALS. The rationale for studying MT in ALS receives support from the detailed understanding of MT from human and animal studies.

Our preliminary data implies that at least part of the accumulated mercury may be bound to MT in motor neurons. It is premature to seriously speculate on potential mechanisms, although MT could directly detoxify mercury. Alternatively, mercury could conceivably divert MT from its function in copper and zinc homeostasis.

Our findings suggest a potential mechanism to explain the selective death of spinal motor neurons in ALS, namely an imbalance between mercury accumulation and detoxification of mercury. Our hypothesis considers that inadequate mercury detoxification by MT might occur in ALS spinal and cortical motor neurons leading to neuronal death. Impaired detoxification could result from an aberrant MT isoform within spinal motor neurons or altered MT gene expression following mercury exposure.

BIO-PROBE COMMENT: This presentation is dramatic and compelling. The credentials of the investigators, institutions, and publications are impressive. The techniques, investigative protocols and rationale are beyond reproach.

It should be obvious to even the most biased, that continued acceptance of doctrines and rationales that permit human chronic low-level exposure to mercury, are totally without scientific support, and cannot be condoned any longer.

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FLUORIDE WAR ESCALATES!
On 17 August 1993, a press release was issued by a subcommittee of the National Research Council (NRC) of the National Academy of Sciences (NAS), the National Academy of Engineering, and the Institute of Medicine. [A private, non-profit institution that provides science and technology advice under a congressional charter.] The release was entitled:

**DRINKING WATER FLUORIDATION CONCERNS UNWARRANTED**

"Currently allowed fluoride levels in drinking water do not pose a risk of health problems such as cancer, kidney failure, or bone disease, concludes a report released today by the National Research Council. Based on a review of available data on fluoride toxicity, the subcommittee that wrote the report concluded that the Environmental Protection Agency’s (EPA) ceiling of 4 ppm (parts per million) for fluoride in drinking water is appropriate as an interim standard."

"Despite conflicting results from other studies and limited data on possible links between fluoride and the risk of hip or other bone fractures, the subcommittee concluded that there is no basis to recommend that EPA lower the current standard for fluoride in drinking water."

The press release discussed the need for research to evaluate fluoride intake from the numerous sources other than plain drinking water, admitting that this could alter consideration of the existing standard. It also addressed the increasing concern over the occurrence of dental fluorosis (mottled teeth), but opted out of that issue by declaring that "the question of whether to consider this a cosmetic effect or an adverse health effect is one for the regulatory agencies to decide."

The report individually addressed the relationship of fluoride to cancer, kidney disease, stomach and intestinal problems, infertility and birth defects, and genetic mutations. In all cases, the subcommittee decided that fluoride in drinking water presented no health risk to humans. On cancer, the statement was: "Available laboratory data do not demonstrate a carcinogenic effect of fluoride in animals, the subcommittee said. Moreover, the weight of the evidence from more than 50 epidemiologic studies does not support the hypothesis of an association between fluoride exposure and increased cancer risk in humans."

The release stated "the project was supported by the U.S. Environmental Protection Agency" and included a roster of the seven subcommittee members. Two of these are institutional dentists, one is from a university School of Public Health, one from Health and Welfare Canada, one from the National Cancer Institute, and one simply listed as being from Raleigh, North Carolina. The Chairman is Bernard M. Wagner, from the New York University School of Medicine.

**THE FORMAL REBUTTAL**

Rebuttal press releases were immediately issued by the National Federation of Federal Employees (NFFE) and Truth About Fluoride, Inc. (TAF), both of which include scientists who have conducted and published research on fluoride.

The NFFE pointed out that active researchers in the field were excluded from the committee, a number of whom they named, in favor of a list of National Institute of Dental Research (NIDR) grant recipients, noting that the NIDR is a major promoter of water fluoridation. Stating that the NFFE has "fought against the institutional distortions of science that have characterized the federal government's actions on hazards associated with exposure to fluoride ion," they declared that the report "does little to change the Union's opinion about the improper use of science in this matter."

The NFFE represents EPA Headquarters scientists, lawyers and other professionals. Some of these EPA scientists have openly questioned the EPA policy on fluoride in drinking water, one of whom even was fired from EPA for doing so. A Federal Court has ordered the EPA to reinstate him with back pay and damages but, at this date, that has not been done.

The NFFE further questioned "why EPA chose to once again contract out the job of assessing fluoride risks, rather than give the job to sworn-to-duty Civil Service scientists. An honest assessment of risks might lead to publicity that could damage the Public Health Service’s long standing program of trying to convince Americans to fluoridate all public water supplies."

TAF emphasized that "the NAS undermined its own claims of the safety of the current EPA standard by admitting to shocking new evidence that fluoride causes bone fractures in the elderly and bone cancer in young males, that severe dental fluorosis is on the rise in young children, and by
admitting that they are completely baffled about how much fluoride people are exposed to from all sources: their diet, mouthrinses, toothpaste, etc. Instead of calling for an immediate end to the practice of fluoridation and for more research, NRC recommends continuing the massive human experiment of fluoridation, while conducting research to see if the practice is causing harm. This recommendation amounts to human experimentation without informed consent, and should be condemned."

TAF further pointed out: "The EPA, which has jurisdiction over fluoride in drinking water, was required by law to review the levels of fluoride allowed in public water supplies three years after its drastic raising of the maximum contaminant level (MCL) of fluoride in 1985. Instead, they have been delaying their investigation and delegating their duties to others. Hundreds of thousands of taxpayer dollars were given to the NAS to conduct a review of the latest fluoride studies and give their recommendation to EPA."

The TAF release was presented by Dr. Bob Carton, a former research scientist with the EPA. Dr. Carton concluded with the statement: "Cable News Network cameramen were prevented by EPA from filming the public meeting, in an obvious attempt to prevent the public from seeing and hearing for themselves the NRC presentation."

BIO-PROBE COMMENT: There is an obvious escalating difference of opinion over the safety of fluoride being deliberately added to drinking water. So who are we to believe?

There appears to be two keys in the information provided above. The first relates to the "MCL", which is "Maximum CONTAMINANT Level". The EPA formally classifies fluoride as a contaminant, of that there is no doubt.

The second key refers to credibility. On the one hand, published scientific research has clearly established the toxicity of fluoride, even at the levels added to drinking water supplies. The increasing occurrence of "dental fluorosis" is testimony to this. To portray dental fluorosis as merely a cosmetic problem is disgraceful. The condition results in chalky, brittle tooth enamel that is more easily damaged. Dental fluorosis is an adverse health effect, to say nothing of being a possible indicator of systemic damage. The teeth are, after all, a part of the body. In addition, low levels of fluoride have been scientifically demonstrated to have an adverse effect on the immune system.

The potential toxicity of long-term, low-level exposure to fluoride notwithstanding, evidence of the carcinogenicity of fluoride is even more compelling. Food supplements have been totally banned when only one animal study suggests the potential for carcinogenicity.

The bottom line on the issue is documented science vs. committee opinions - which is the more valid and believable? The answer is patently obvious; no amount of unsupportable opinions of select committees can be considered compelling in opposition to scientific documentation. Committees can be structured to provide whatever opinions may be desired. If select committees expect to be believed, they must present scientific documentation to support their conclusions, not merely deny the validity of science demonstrating to the contrary.

If the solution is so clear, than why does the debate continue? The answer to this question also lies in the text provided above. The EPA has a formal responsibility in the fluoride issue. Someone in the EPA is obviously in trouble. The EPA position has been so contrary to the scientific evidence that even EPA scientists have openly objected. SCIENCE vs. POLITICS!

At some point, it will become imperative that the United States Congress step in to protect the interests of its constituency. Considering the growing weight of scientific evidence condemning the deliberate fluoridation of drinking water and the refusal of EPA officials to acknowledge this research, that time appears to be at hand.

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ABSTRACTS

Mercury in the Rat Hypothalamic Arcuate Nucleus and Median Eminence after Mercury Vapor Exposure
Ernst, E; Christensen, MK; Poulsen, EH.
ABSTRACT:

A histochemical technique has been used to reveal mercury deposits in the hypothalamic arcuate nucleus and median eminence of adult male rats. After exposure to long-term, low-level or short-term, high-level mercury vapor, silver-enhanced mercury grains were found in
neurons of the arcuate nucleus. In addition, mercury deposits were found in tanyocytes, ciliated ependymal cells, and in the walls of capillaries. The mechanisms underlying uptake and possible induction of toxic effects are discussed.

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Detection of Mercury in Rat Spinal Cord and Dorsal Root Ganglia after Exposure to Mercury Vapor.
Schionning, JD; Eide, R; Moller-Madsen, B; Ernst, E.
ABSTRACT:
Adult male Wistar rats were exposed to mercury vapor, 50 micrograms Hg/m³, 6 hr/day, 5 days/week, over 1-, 2-, 3-, 4-, 6-, and 8-week periods. Sections from the spinal cord and dorsal root ganglia from spinal levels C1, C5, T6, and L1 were stained with the autoradiographical technique and the distribution of mercury deposits described at light and electron microscopical levels. A quantitative analysis of the amount of mercury in blocks of the spinal cord was performed using cold vapor atomic absorption spectrophotometry. After an exposure period of 2 weeks, silver-enhanced mercury grains could be observed in spinal cord neurons located in Rexed laminae IV-X. Ventral horn motoneurons were heavily stained in all of the spinal cord segments. Ependymal cells and glial cells of both the spinal gray and white matter contained cytoplasmic mercury accumulations in rats exposed to mercury vapor for 4 weeks. In the dorsal root ganglia, only ganglion cells showed a faint mercury staining and the amount of staining was notably less than that seen in the ventral horn motoneurons. At the ultrastructural level, mercury was seen primarily within lysosomes of target cells. The quantitative mercury measurements demonstrated that spinal cords from rats exposed to mercury vapor for 6 or 8 weeks contained a significantly higher concentration of mercury than those from control animals.

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Interactions of Mercury in Rat Brain
Falgona L., et al.
ABSTRACT:
In order to study the metabolism of mercury (Hg), its affinity to metallothionein (MT), and its influence on levels of the essential metals copper and zinc in the brain tissue of rats exposed to elemental mercury (Hg⁰) vapor was investigated. The major findings were:
1. After long-term exposure, about 40% of mercury was found in the brain water-soluble phase (supernatant);
2. In brain supernatant, about 80% of Hg was found in the range of low-molecular-weight proteins, the MT-like protein Hg-Cu-Zn-thionein was isolated and partially characterized;
3. Hg⁰ vapor exposure resulted in increased tissue levels of essential Cu and Zn in addition to exogenous Hg; and
4. Experiments showed that Hg⁰ vapor exposure can induce the stimulation of rat brain MT synthesis.

BIO-PROBE COMMENT: These three studies bear particular significance to the discussion of ALS earlier in this newsletter. The first two demonstrate the presence of mercury in neurons after low-level exposure to mercury vapor, as is encountered from amalgam dental fillings. The second study specifically identifies the mercury in the ventral motor neurons of the spinal cord, which are the specific motor neurons damaged in ALS, even after short-term low-level exposure to mercury vapor. The third study supports the hypothesis advanced by Kasarskis et al., that the relationship of Hg⁰ and MT may in fact be involved in altered or inadequate detoxification processes.

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Daum, JR; Shepherd, DM; Noelle, RJ.
ABSTRACT:
Heavy metals have been shown to exert immunotoxic effects on humoral immunity. To ascertain the mechanisms by which these immunotoxic effects are exerted, the effects of CdCl₂ and HgCl₂ on the biology of murine B-lymphocytes were studied.

It was shown that CdCl₂ and HgCl₂ inhibited B-cell RNA and DNA synthesis. The IC₅₀ (the concentration required to inhibit a specific B-cell function by 50%) for CdCl₂ was 30 microM for RNA synthesis and DNA synthesis. The IC₅₀ for
HgCl₂ was 50 and 120 nanoM for RNA and DNA synthesis, respectively.

Cell cycle analysis revealed that B-cells were arrested throughout the cell cycle with CdCl₂ and HgCl₂. The inhibitory effects exerted by CdCl₂ and HgCl₂ were rapid, inhibiting RNA synthesis within 2 hours of activation.

Differentiation to Ig secretion was inhibited by CdCl₂ and HgCl₂ in culture and there appeared to be selective effects on specific Ig isotypes. IgG₃ production was most sensitive to inhibition by CdCl₂ and HgCl₂ followed by IgG1 and IgG2b and then IgM and IgG2a.

In summary, both CdCl₂ and HgCl₂ exert early, inhibitory effects on B-cell activation. This is manifested by the inhibition of RNA, DNA and antibody synthesis. Selective effects on the production of specific Ig isotypes by these metals may influence the ability of B-cells to mount effective immune responses to pathogens.

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Mercury Contents of Indicators and Target Organs in Rats after Long-Term, Low-Level, Mercury Vapor Exposure.

Eide R. and Wesenberg GR.


ABSTRACT:

Five groups of Wistar rats received graded concentrations of mercury vapor from 10 to 100 ug Hg/m³ 6hr, 5 days a week, from 4 to 11 weeks of age. One group breathing ambient air served as controls. The mercury levels of the indicators blood, hair, molars, and incisors as well as the target organs kidney cortex, cerebrum, cerebellum, liver, lung, spleen, tongue, and femur were measured by cold-vapor atomic absorption spectrophotometry. The mercury vapor had no negative influence on the weight gain of the animals. The results showed that the kidney cortex had the highest concentration of mercury. The mercury contents of all the indicators and all the target organs, with the exception of femur, were positively and significantly correlated with the exposure concentration. The rat molars had the highest correlation coefficient with the kidney mercury values, but no indicator had a significant correlation with all target organs. Rat molars are to some degree comparable to human deciduous teeth regarding time of mineralization and eruption. Based on the results presented in this study, we tentatively suggest that human deciduous teeth can be useful indicators of chronic mercury exposure not only at the exposure concentration level, but also as indicators of the mercury uptake in organs such as kidney and cerebrum.

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Evidence that mercury from silver dental fillings may be an etiological factor in smoking.

Siblerud RL; Kienholz E; Motj J. Toxicol Lett 68(3):307-310, June 1993

ABSTRACT:

The smoking habits of 119 subjects without silver/mercury dental fillings were compared to 115 subjects with amalgams. The amalgam group had 2.5-times more smokers per group than the non-amalgam group, which was highly significant. Because mercury decreases dopamine, serotonin, norepinephrine, and acetylcholine in the brain, and nicotine has just the opposite effect on these neurotransmitters, this may help explain why persons with dental amalgams smoke more than persons without amalgams.

BIO-PROBE COMMENT: Although a study of the relationship between amalgam bearers and non-amalgam bearers and alcoholic beverages has never been done in humans, we feel the same results observed in the Siblerud study would be seen, i.e. amalgam bearers would have a greater desire for and consume more alcoholic beverages than non-amalgam bearers. Animal studies have shown that alcohol suppresses the effects of mercury similar to that observed for nicotine. Of all the deaths being blamed on smoking and alcohol, an interesting statistic might be to determine how many of the deceased were not amalgam bearers. If 80 or 90% had mercury dental fillings, would that be a confounding scientific variable?

FORUM

IAOMT NINTH ANNUAL SCIENCE SYMPOSIUM

DATES: Friday-Sunday, 1-3 October 1993.

30 Sep: 7:00 pm = Welcoming Reception (Cash Bar).

1 Oct: 8:30 am-5:00 pm = Scientific session.

1 Oct: 6:00 pm = Awards Dinner.

2 Oct: 8:30 am-5:00 pm = Scientific session.

3 Oct: 8:30 am = IAOMT 1993 Annual Meeting.
SITE: Hyatt Regency-Oak Brook (Chicago), Illinois. Call (800) 233-1234 for room reservations (specify IAOMT). $78.00/night single or double room.

PROGRAM:

- Murray J. Vimy, D.M.D.: Medical researcher and Assistant Clinical Professor, Department of Physiology, University of Calgary, Canada. Will review his research and present an overview of scientific literature on the biocompatibility of dental amalgam.

- James Masi, Ph.D.: Professor in the Department of Biomedical Engineering, Western New England College, Springfield, Massachusetts. Will discuss the issue of metals in dentistry based on corrosion chemistry and the oral system.

- Boyd Haley, Ph.D.: Professor in medical chemistry at the University of Kentucky. Will discuss published research on Alzheimer's Disease and mercury in the brain tissues.

- Walter Jess Clifford, M.S.: Researcher, clinical laboratorian, author and lecturer. Will present on the adverse influence of dental materials on blood cells and possible reversal of such effects.

- Peter Duesberg, Ph.D.: Professor at the University of California, Berkeley, Department of Molecular and Cell Biology and renowned retrovirologist. Will discuss HIV and the common fallacies regarding the origin and spread of the disease.

- Richard Foulkes, M.D.: Practicing physician and consultant in British Columbia. Author of 46 articles on various aspects of the health care system. Will discuss the safety and efficacy of water fluoridation.


- James Yehi: Of American Environmental Systems. Will present published data and clinical applications on safety from toxins for the staff and patients.

- David C. Kennedy, D.D.S.: Current President of the IAOMT. Will present computer applications for the biocompatible dental practice.


IAOMT members = $245.00. [For this meeting only, courtesy member registration is being extended to members of: The Holistic Dental Assn., The Environmental Dental Assn., TERR, Assn. of Health Practitioners, American Academy of Biological Dentistry, and the Academy of Physiological Dentistry]

Auxiliaries w/Dr. = $25.00 each.
Non-member = $325.00.
Non Health Practitioner = $125.00.
Awards Dinner Tickets = $35.00/person.
* Registrations after 21 September additional late fee = $50.00.

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"DENTISTRY FOR THE 21ST CENTURY" BIOLOGICAL DENTISTRY

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THE ENVIRONMENTAL HEALTH NETWORK, INC. PRESENTS THE FOURTH SCIENTIFIC ASSEMBLY FOR ENVIRONMENTAL HEALTH.

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