Noble Metals: A Toxicological Appraisal of Potential New Environmental Contaminants

by Paul E. Brubaker,* John P. Moran,* Kenneth Bridbord,* and F. Gordon Hueter*

The public health benefits expected by reducing known hazardous emissions from mobile sources should not be compromised by increasing levels of other potentially hazardous unregulated emissions. Catalytic converters are going to be used to meet the statutory requirements on carbon monoxide and hydrocarbon emissions from light duty motor vehicles. Platinum and palladium metals are the catalytic materials to be used in these emission control devices. Preliminary experimental evidence and analysis of the impact of these control devices on the future use and demand for platinum indicates that this metal may appear at detectable levels in the environment by the end of this decade. At the present time, platinum and palladium are not present in the public environment and represent potentially new environmental contaminants as a consequence of use of this new abatement control technology. There is relatively little information available to adequately assess the potential health hazards that may be associated with exposure to these metals and their compounds. Analysis of the environmental problems and concerns associated with possible new environmental contaminants are discussed. Limited estimates are made on community exposure by use of a meteorological dispersion model. Biodegradation potential and attention is also given to the limited toxicological information available.

Introduction

Continuing international progress in commerce and technology has had a profound impact on the entire global ecosystem. Air, water, soil and biota; all compartments of the environment have been intentionally and unintentionally contaminated with a wide variety of toxic substances. The manufacture of many commercial chemicals designed for use in closed systems as well as those intended for direct environmental applica-

tion and regulated by stringent legislative controls have been unexpected pollutants that threaten environmental vitality. Natural resources have been utilized in many industrial and commercial applications that modify, redistribute and concentrate them in a fashion that distorts their equilibrium in the ecosystem. It is now recognized that such distortions can pose a threat to public health and welfare of immediate and future generations (1,2).

The epidemiology of health adversities associated with atmospheric contamination has shown that air pollution alone contributes to increased mortality and morbidity, aggravates and accelerates the pathogenesis

* Special Studies Staff, Office of the Director, National Environmental Research Center, Research Triangle Park, North Carolina 27711.

of chronic respiratory diseases, and may induce a variety of immediate and delayed systemic pathologies (3–8). National concern has provided a mandate in the amended Clean Air Act (1970) and the National Environmental Policy Act of 1970 to establish standards of environmental quality which afford protection of public health and welfare. Establishing and enforcing prudent standards of environmental quality requires development and continual refinement of a broad integrated data base that is sufficiently quantitative to permit adequate risk-hazard assessment and reasonable cost-benefit analysis (9). While standard compliance serves as an impetus for developing new abatement control technologies of immediate economic significance on a national scale, it is important to recognize public health priorities and the nature of pollution-induced health adversities that influence national economic policy on a long-term basis. The National Emissions Standards Act (Title II of the Amended Clean Air Act 1970), recognizes exhaust emissions from motor vehicles threatens public health in many urban areas (10–15). In order to guarantee ambient air quality and assure public health protection, a 90% reduction in carbon monoxide and hydrocarbons from 1971 emission levels of light-duty motor vehicles was scheduled for compliance by 1976. A similar reduction in oxides of nitrogen from these same mobile sources was required by 1977. In the absence of marketable engine designs to achieve these reductions as scheduled, compliance with these standards has lead to the development of an emissions abatement technology that involves oxidation or reduction catalytic conversion of these regulated exhaust emissions (CO, HC, NO_x) to carbon dioxide, nitrogen, oxygen, and water. However, public health benefits achieved by new abatement technology should not be compromised by increasing levels of other emissions presently unregulated or by the introduction of new environmental contaminants (16).

Assessment of public health hazards associated with exposure to existing ambient air pollutants encompasses epidemiology, atmospheric monitoring, meteorological dispersion analysis, and aerometric chemistry with aligned clinical, and experimental toxicology programs. The integration of these desciplines forms the basis of the national Community Health and Environmental Surveillance System (CHESS) (17). However, analysis of new substances presents a modified strategy that requires toxicological analysis prior to their appearance in ambient air.

Classical toxicology routinely involves administration of graded dosages of a given substance by various exposure routes to several species of adult experimental animals. The nature of the injury produced is determined, critical organs are identified and dose-response curves constructed from acute, subacute, chronic and subchronic exposures in order to predict toxicity over a limited dosage range beyond those determined experimentally. Threshold limit values are time-weighted exposure limits used in industrial hygiene to minimize adverse health effects among employees subjected to daily exposures of various chemicals found in occupational atmospheres (18). While they should not be used to distinguish harmful from safe concentrations, they are based upon information derived from previous occupational health observations, experimental clinical programs and animal toxicological investigations. This information, useful for industrial hygiene purposes, is only of limited value for environmental health considerations due in part to the heterogenity in health of the general population and abatement program requirements. Since ambient air pollutants contaminate several compartments of the ecosystem, public health considerations and control programs must also consider their biotransformation potential and risk-hazard analysis of alternate exposure routes. For example, special reference is made to metallic compounds, particularly methylmercury (19, 20). Furthermore, biotransformation also includes metabolic modification of inactive parent compounds that become active upon

absorption. Therefore, toxicological analysis of new ambient air pollutants should not necessarily be confined to inhalation routes if absorption and biotransformation are distinct possibilities.

Adverse health effects may arise from prolonged exposure to low levels of environmental agents, intermittent exposure to high levels, and may also appear as a consequence of multiple environmental factors acting in concert. The health effects arising from low level exposures to hazardous environmental agents encompasses a continuous and progressively complex spectrum of subtle physiological anomalies that have an increasing probability of becoming irreversible/incurable upon continuous exposure (21). In general, these exposure conditions tend to amplify pathology in susceptable subgroups of the population predisposed by age, preexisting illness, and genetic insufficiencies (22–24). The complexity of pollution-induced disease etiology is considerably reduced due to manifestation of discernable response parameters in these individuals. The pollutant–health effect relationship becomes exceedingly complex in the absence of parameters that delineate onset, progression and termination of stress response. Among immediate adverse effects that can be expected in healthy individuals are those arising from interference in normal defense mechanisms such as reduced tolerance resulting from metabolic and immunological deficits and induction of allergic hypersensitives (25,26). Moreover, health adversities associated with chronic low level exposure conditions often appear among both healthy and predisposed individuals following prolonged latent periods. Teratogenic, carcinogenic, and mutagenic effects are terminal endpoints of such exposures that appear following latent periods that range from months to years and to subsequent generations, respectively (27–34).

Ambient air pollutants constitute a broad range of biologically active chemical and physical agents. The inhalation hazards of many chemical compounds associated with automotive exhaust emissions have been described (6,7). While it is important to emphasize accuracy in toxicological analysis, it is of equal importance to stress integration of protocols (35). Selection of appropriate experimental animals, use of protocols that have biological equivalents in epidemiological and clinical studies, use of parameters that serve as exposure indicators as well as those that may predict impending pathology provide an important means of linking experimental observations with environmental surveillance of community health. Quantitative toxicological analysis and abatement program effectiveness depends upon adequate identification of active biological materials. Those chemicals that have a potential for wide use, broad distribution, and can attain hazardous exposure levels should require toxicological analysis for sufficient assessment of population and exposure hazards. Evaluation of new compounds should be completed at an early stage in order to assess their environmental compatability and to determine abatement program feasibility. While the elimination of sulfuric acid aerosols and particulate sulfates, a function of fuel composition, are of immediate concern, the use of catalytic convertors on light-duty motor vehicles may represent a source of new environmental agents of broad distribution potential, the attrition products of platinum-group metals. In the absence of sufficient toxicological information necessary for assessing public health hazards, the purpose of this report is to address the following problem areas: analysis of past national consumption and use of noble metals and evaluation of their potential for future environmental contamination; review of relevant information from reported industrial hygiene experience; estimation of exposure levels under various meteorological conditions; Biomedical aspects of concern, namely, immediate and delayed hypersensitivity, carcinogenicity, and biotransformation (methylation); identification of areas of concern regarding regulation and control.

Table 1. Consumption of platinum group metals and their distribution among major industries in the United States. [a]

	1961	1972	Annual average 1961–1972
World production of platinum-group metals			
G/yr	—	—	84,989,134.43
Oz troy/yr	—	—	2,732,506.01
Tons/yr	—	—	93.62
U. S. consumption of platinum-group metals			
G/yr	—	—	38,862,413.15
Oz troy/yr	—	—	1,249,474.73
Tons/yr	—	—	43.81
U.S. consumption, % of average annual world production	—	—	45.7
U.S. platinum-group metal consumption among major consuming industries, %			
Electrical	54.2	34.4	43.4
Chemical	20.9	37.8	28.5
Petroleum	4.6	8.1	9.1
Medical	8.0	8.1	6.8
Glass	4.6	2.7	5.0
Jewelry	6.5	3.2	5.1
Miscellaneous	1.1	5.6	2.7

[a] Data of Bureau of Mines Mineral Yearbook (36).

Table 2. Platinum and palladium consumption and their distribution among consuming industries in the United States (1961–1972). [a]

	Annual average 1961–1972
Consumption of platinum	
G/yr	14,274,845.38
Oz troy/yr	448,953.97
Tons/yr	15.72
Consumption of palladium	
G/yr	20,837,161.55
Oz troy/yr	669,940.57
Tons/yr	22.95
Platinum consumption, % of total noble metals use	36.7
Palladium consumption, % of total noble metals use	53.61
Noble metal consumption, % as platinum and palladium	90.31

[a] Data from Bureau of Mines Mineral Yearbook (36).

Use of Platinum-Group Metals in the U.S., the Impact of Catalytic Convertors, and Their Future Potential for Environmental Contamination

Platinum, palladium, ruthenium, osmium, rhodium, and iridium are those metals collectively known as the platinum group of the noble metals class. These metals owe their importance to their desirable physical properties, e.g., their great resistance to most corrosive agents. Furthermore, they have been extensively used as catalysts in many industrial operations. The use and distribution of platinum group metals in the United States is presented in Table 1. The average annual U.S. consumption was 45.7% of the average annual world production of platinum group metals for the period 1961 to 1972 (36). During this period, the distribution of these metals arranged according to decreasing consumption among the various industries in the United States are: electrical (43.4%), chemical (28.5%), petroleum (9.1%), medical (6.8%), jewelry (5.1%), glass (5.0%), and miscellaneous (2.6%). (Table 1). Approximately 90% of the platinum group metals consumed were platinum and palladium (Table 2).

The use of catalytic reactors for control of motor vehicle exhaust emissions has been considered for at least a decade (37,38). Since then, a variety of mechanical designs and catalytic materials have been explored in this regard (39–41). However, the use of platinum group metals has certain advantages that preclude the use of other materials, namely: their proven effectiveness as catalysts, low-threshold activation temperatures, fast warm-up response, and apparent

Table 3. Estimated impact of platinum demand required for catalytic exhaust control systems (1975–1990). [a]

	U.S. estimated annual Pt demand
Average of new platinum demand for catalyst-equipped motor vehicles	
G/yr	43,894,108.16
Oz troy/yr	1,411,250.30
Tons/yr	43.35
Average new platinum demand for converter replacement	
G/yr	41,851,668.37
Oz troy/yr	1,345,583.21
Tons/yr	46.10
Total average new platinum demand	
G/yr	85,745,776.73
Oz troy/yr	2,756,832.48
Tons/yr	94.45
Increase of total demand over present consumption of platinum 2,756,832/458,953	6.0
Increase of platinum demand corrected for recycling[b] 1,166,270/458,953	2.5

[a] Data of Bureau of Mines (44).
[b] From Table 5 (90%).

Table 4. Estimated impact of U.S. platinum demand required for catalytic exhaust control systems for light-duty motor vehicles (1975–1990). [a]

	Estimated annual average 1975–1990
Platinum demand, g/yr	43,894,108
U.S. light-duty domestic vehicle production and foreign car imports, units/yr	14,112,500
Platinum requirements of each catalyst-equipped light-duty motor vehicle, g/unit (oz troy/unit)	3.1 (0.1)

[a] Data of Bureau of Mines (44). Assumes one catalyst per car.

ability to withstand thermal stress (39,42). The amount of platinum and palladium used is apparently dependent in part upon mechanical and chemical design as well as performance required to meet emission reduction requirements.

In recent Bureau of Mines reports (36,43), the impact of this catalytic auto exhaust abatement technology has been estimated, and such data are summarized in Table 2.

Accordingly, the reduction of automotive emissions by the use of catalytic convertors will increase the demand for platinum several times the average annual U.S. consumption and is likely to remain the largest single use category during the post-1975 years (Table 3) (43). Furthermore, analysis of the estimated impact on U.S. platinum demands would require doubling the present world production. The amount of noble metals per catalytic unit has varied in several published reports (Table 4) (43,45–47). Analysis of the projected demands for platinum required for catalyst equipped light-duty motor vehicles is apparently based upon a platinum requirement of 0.10 troy oz per motor vehicle (Table 4). The demand for platinum to supply these needs can be achieved by refining new metals from primary sources, recycling of platinum derived from convertor trade-ins and scrappage of convertor-equipped automobiles. The principal source of recovery is by convertor trade-ins (Table 5). It is important to note that these estimates made on future platinum demands all asume that catalyst durability will extend over 50,000 miles of driving and apparently does not consider the impact of new engine designs that may alleviate requirements for these convertors.

A platinum-palladium catalyst is composed essentially of these noble metals thinly layered (100–200, μm deep) on a bed of porous refractory substrate material (39,48,49). Substrates of choice appear to be a ceramic compound of manganesium oxide, aluminum and silicon (41,48). There have been several substrate designs considered, namely, wire-mesh, Raschig ring, honeycombed monolith, and pellets (41,48). Porosity and maximum surface area and the necessity to minimize back pressure and to provide optimum conditions for catalytic activity are important factors influencing design selection. Pelleted beads and a honeycombed monolith structure are the apparent designs of choice for 1975 automobiles (48). The catalytic convertor is located between the engine and muffler and is protected from shock, vibration and

Table 5. Estimated average annual platinum recovered by various percentages of converter trade-ins and scrappage of catalyst-equipped motor vehicles (CEMV) from 1975 to 1990.

	25%	50%	75%	90%
Average annual platinum recovered by scrappage, g/yr	3,265,815	6,574,396	9,861,594	11,836,635
Average annual platinum recovered by convertor trade-ins, g/yr [a]	10,464,428	20,928,856	31,393,284	37,634,630
Estimated total platinum recycled, g/yr	13,730 243	27,503,252	41,254,878	49,471,265
Percentage of estimated annual average of total platinum demand (85,745,776 g/yr), %	16.1	32.1	48.1	57.7

[a] Data of Bureau of Mines (44).

[b] Convertor trade-ins assumes 50,000 mile durability and one convertor per car (44).

other external factors that influence durability by enclosure in a suitable metal housing (50). In late 1972, estimates on the use of platinum and palladium in a 5.2 ratio was stated to be acceptable for use in these exhaust convertors (43); however, this ratio may vary with convertor design. Furthermore, alloys of platinum group metals and base metals have been considered with the platinum group metals being used in low amounts to initiate the oxidation or reduction reaction (41,49). While the presently available oxidation catalyst will employ platinum and palladium, the material for reduction catalysts may consist of platinum, palladium, ruthenium, base metals or base-metal-platinum group metal alloys (41,50). Although the NO_x reduction catalyst is still in developmental stages, the use of platinum group metals or other catalytic materials is likely to produce contaminants in future exhaust emissions unless advances are made in combustion technology and marketable engine designs that would negate the need for such catalytic convertors.

Anticipated platinum losses to the environment depend in part on catalyst durability, economic feasibility of recycling, efficiency of the recovery process, and upon various percentage combinations of vehicle scrappage and converter trade-ins (50). There have been a number of reports regarding inadequate durability of catalysts and the generation of catalytic attrition products from converter-equipped automobiles (15,48,50–55). A variety of factors can contribute to loss of catalytic material from exhaust convertor systems. Some of these include thermal stress that results in sintering resulting from melting substrate material, fuel composition with catalytic "poisons", fluctuations in exhaust water content, pulsating pressure from exhaust gases and mechanical vibrations from driving conditions that can abrade catalytic material (40,48–50). Assuming that each car has 0.1 oz troy platinum, an analysis of the estimated demand (Table 3), and corrections made for a 90% optimal recycling of catalytic material from scrapped automobiles and used convertor trade-ins, yield an estimated loss of 39.67 tons of platinum annually, i.e., 2.55 g per automobile (Tables 5 and 6). Since the reported values of platinum per car range from 0.005 to 0.1 oz/car, this estimate represents the upper range. A more realistic figure is one adjusted for the average of the various convertor platinum values reported. The adjusted loss would then be 23.31 tons of platinum, anually, or 1.49 g/car (Table 6). It is important to note that the amount of platinum loss expressed per light-duty motor vehicle (Table 6) is an estimated emission value and assumes efficient 90% recovery by recycling procedures. While information regarding platinum emissions from catalytic equipped automobiles is generally not available, there is one report that cites an emission level of 20 μg of platinum per vehicle mile (51). Assuming the expected 50,000 mile durability is valid, then the total amount of platinum lost from this catalyst-equipped automobile would be 1 g

Table 6. Estimated platinum emissions from catalyst-equipped light-duty motor vehicles.

	Estimated annual average	
Platinum recovered by 90% scrappage of light-duty motor vehicles and 90% convertor trade-ins (=58% of estimated annual average Pt demand, g/yr	49,732,550	
Platinum loss, g/yr (tons)		
Demand	85,745,776	(94.45)
Recycled	49,732,550	(54.78)
Loss	36,013,226	(39.67)
Loss per light-duty vehicle, g/unit (36,013.226/14,122,500)	2.55	
Correction of estimated loss from reported levels of Pt/converter, g/unit		
0.1 oz troy/converter	2.55	
0.07	1.79	
0.05	1.53	
0.005	0.13	
Average	1.49	
Pt loss by emission		
Pt loss per car, g/unit	1.49	
Catalyst durability, miles	50,000	
Calculated emission (loss/durability), μg/mile	30	
Reported emission, μg/mile	20	

ᵃ Data of Newkirk and Goluba (*51*).

platinum over a driving range of 50,000 miles as compared to the above estimated value of 1.49 g/car. This value is likely to be a high estimate for post-1975 years, depending upon continuing progress in catalyst design and performance that may require less platinum per convertor unit. There is no information available on palladium loss. However, assuming a 5:2 or a 3:1 ratio of platinum to palladium, the loss of palladium could range from 33 to 40% of the estimated platinum loss. No information has been reported regarding chemical identification of the platinum-emissions. Attention should also be given to the alumina and magnesia oxides used in substrate materials which may also represent new components of the catalyst-modified exhaust emission.

In addition to refractory materials associated with catalyst supporting material, other metallic compounds are also found in various automotive fuels and fuel additives

(*55,57*). The use of tetraethyllead to increase the octane level of gasoline is now recognized as a significant threat to environmental quality and to public health. The transport pattern of lead in the atmosphere serves as an appropriate surrogate for other metallic exhaust emissions (*34,58*). Since manganese is being considered as a fuel additive replacement for lead and since lead is apparently incompatable with catalytic convertor performance, manganese is included as an additional metallic component expected in future exhaust emissions (*35*). The compatability of manganese and other fuel additives with convertor performance and their influence upon the nature of exhaust emissions remains to be determined.

Occupational Hazards of Platinum and Palladium

The principal health adversities resulting from exposure to platinum group metals are respiratory and cutaneous hypersensitivities associated with complex salts of platinum and their aqueous solutions (*59–63*). Platinosis is defined as an allergic reaction following exposure to soluble platinum salts which elicits a progressively more pronounced asthmatic response upon continued exposure (*60*). Immediate allergic hypersensitivity to platinum salts apparently results from induced histamine release elicited by exposure to hexachloroplatinate intermediates in platinum refining operations (*62–64*). There have been no industrial hygiene problems associated with palladium.

Classical allergic symptoms are displayed in platinosis with occasional severe anaphylaxes observed among sensitive or sensitized individuals. Individuals that are not initially sensitive may work in the presence of these allergins for years without noticeable adversities, however, the majority become symptomatic upon continued exposure (*60*). The platinosis syndrome consists of sneezing, rhinorrhea, tightness of chest, shortness of breath, cyanosis with contact urticaria, wheezing and cough that resembles a mix-

ture of hay fever and asthma. Once disabling symptoms appear in any one case that individual will never again become asymptomatic in an atmosphere containing soluble salts of platinum unless desensitized by hyposensitization procedures (60). Hyposensitization has been reported in one individual by intradermal injections of ammonium hexachloroplatinate administered several times daily over a 30-day period (65). While this form of treatment was accompanied by a serum sickness-like reaction, it was deemed successful since subsequent exposure to soluble platinum salts proved uneventful.

Progression of the platinosis syndrome is accompanied by varying degrees of irritation of the upper respiratory tract, conjunctivae and mucous membranes with hypertrophy of lymphatic tissues and a mild lymphocytosis (59–63). The mechanisms proposed for the activity of these compounds involves platinum salts presumably acting as a hapten as a result of combining with serum albumin to form an allergenic hapten-protein complex (66). The immediate hypersensitive reaction may be specific for specific platinum ions. *In vivo* and *in vitro* experimental studies have described the influence of hexachloroplatinate on cilia activity in guinea pigs and notes the release of histamine is restricted to platinic ion of coordination number six (64). A differentiation of the disease into a major form that includes asthma, cutaneous eczema, and urticaria and a minor form with rhinopharyngitis, cough, dyspnea, itching, and conjuctival vasodilation has been proposed. However, while variations occur with symptomology that include features from both forms, the immunological-allergic mechanisms of immediate and delayed hypersensitivity underlie both response modes.

Eczematous dermatitis and allergic contact sensitivity notes a delayed hypersensitive response differing in mechanism from immediate allergic response. Dermal manifestation may be acute with urticaria, itching in exposed areas, subacute in which a typical contact dermatitis is observed and chronic

with secondary eczema and persistent contact dermatitis (67). Dermal reactions to platinum can be particularly dramatic, with dermatosis observed beneath clothing of a sensitive individual with a pocket containing a handkerchief contaminated with platinum salts (2). One industrial hygiene survey carried out over a 5-yr period reports that individuals with red hair, moles, freckles and a history of skin irritation are those that appear to be particularly sensitive to soluble salts of platinum (60). It is to be noted that these compounds are largely insoluble in distilled water with solubility improved in normal physiological saline (63). In this regard, absorption is feasible via dermal and pulmonary exposure routes (68). Normal perspiration and fluids present in the lungs may solubilize insoluble compounds and permit absorption into the body (69,70).

There has been only one report in which health adversities were examined, prevalence of platinosis recorded, and industrial atmospheric platinum levels determined (59). Atmospheric measurements in four platinum refineries that involved 114 employees ranged from 0.9 to 1700 μg/m³. The prevalence of symptomatic individuals among the 91 employees examined was 71%, with 57% suffering from asthmatic response and apparently 14% presenting dermatological problems. The number of individuals with both involvements was not reported. The authors note the severity and intensity of response was highest among those individuals employed in crushing platinum salts and exposed to levels of atmospheric platinum ranging from 400 to 1700 μg/m³. However, analysis of the data presented suggests that exposure risk is apparently equivalent in platinum atmospheres associated with both wet and dry refining procedures. Furthermore, there does not appear to be a definite dose–response relationship (Table 7). Data from other refineries in this report are inadequate for comparative purposes since distinction between wet and dry operations are not clearly defined. It is noteworthy that in one refinery equipped with a ventilation

	Exposure	
	Spray aerosols wet operations	Dust dry operations
Refinery A		
Range of atmospheric Pt, $\mu g/m^3$	7 –20	400 –1,700
No. of employees	18	6
Employees with asthma	6	5
Employees with dermatitis	1	—
Refinery B		
Range of atmospheric Pt, $\mu g/m^3$	1.6– 6.9	4.1– 50.2
No. of employees	29	46
Employees with asthma	14	16
Employees with dermatitis	3	5
Total, Refineries A and B		
No. of employees	47	52
Employees with asthma	20	21
Employees with dermatitis	4	5

[a] Data of Hunter et al. (59).

Table 8. Allergic or irritant reaction of selected metals. [a]

Metal[b]	Allergic/irritant reaction[c]	TLV, mg/m^3 [d]
Barium	Dermal, nasal	0.5
Cobalt	Asthma, dermal	0.5
Chromium	Dermal, upper respiratory	0.1
Copper	Dermal	0.1
Gallium	Dermal	—
Mercury	Dermal	0.1
Nickel	Dermal	—
Platinum	Asthma, dermal	0.002
Thallium	Dermal	0.1
Tin	Dermal	0.1
Vanadium	Asthma, dermal	0.5

[a] Data of Stokinger (67).

[b] Each metal class includes metallic compounds with a wide variety of anions and organic analogs.

[c] Reactions were selected only from industrial hygiene observations.

[d] Data of American Conference of Industrial Hygienists (18).

system, the atmospheric platinum levels ranged from 0.9 to 3.2 $\mu g/m^3$. Mild symptoms of running nose and sneezing were reported in five of the seven individuals in this refinery when they were exposed to spray aerosols of platinum salts. While the current threshold limit value is presently 2.0 $\mu g/m^3$ for soluble platinum salts, initiation of response apparently occurs between 0.19 and 3.2 $\mu g/m^3$. In the later 5-yr refinery survey without aerometric measurements, symptomology was reported to be 100% with 60% of the employees affected with the major asthmatic form and 40% with the minor dermatological form of platinosis (60). A third survey reports a 65% prevalence (62). The latency period prior to the appearance of platinosis symptomology ranges from months to a maximum of 10 yr (62). The 5-yr study is perhaps the best index to incidence over a prolonged period and particularly relevant to environmental considerations.

A variety of chemicals have induced allergic reactions and have posed occupational problems, namely; penicilin, streptomycin, chloropromazine, aromatic amines, chlorinated ethylene derivatives, and tolulene diisocyanates (62,71). However, the potency of the sensitizing capacity of soluble salts of platinum is evident among the threshold limit values established for other metals that have an occupational history of allergic response (Table 8). The threshold limit value (TLV) of soluble platinum salts is 100 times lower than those for other metals that have associated asthmatic conditions reported with exposure. It is important to recall the limitations placed on the use of threshold limit values. Appropriate comparisons of the various metals listed with allergenic properties requires careful inspection of the data base used to establish the threshold limit values. Nevertheless, these values do not assure protection for sensitive or sensitized individuals. Therefore, soluble salts of platinum compounds may contribute to levels of existing airborne allergens in the ambient air with potential aggravation of individuals with preexisting asthmatic or cardiorespiratory deficiencies. Approximately 3–5% of the population in the United States are asthmatics, and this represents a rather large susceptable subgroup (6–10 million people) (9). Furthermore, these compounds

Table 9. Estimated short-term exposures to platinum emissions from catalytic equipped vehicles under various meteorological conditions with a dispersion model. [a]

Receptor	Exposure location	Exposure levels, $\mu g/m^3$			
		Normal meteorological conditions [b]		Adverse meteorological conditions [b]	
		Wind directly across highway	Wind at worst angle	Wind directly across highway	Wind at worst angle
Lead levels					
Automobile passengers	Expressway (from lead measurements) [c]	22–28	39	120	690
Pedestrians	Near expressway (from lead measurements) [c]	17–39	28	110	490
Estimated platinum levels, 25% of light-duty motor vehicles equipped with catalytic convertors (1976)					
Automobile passengers	Expressway	0.001	0.003	0.009	0.05
Pedestrians	Near expressway	0.001	0.002	0.008	0.03
Estimated platinum levels, 100% of light-duty motor vehicles equipped with catalytic convertors (1982)					
Automobile passengers	Expressway	0.004	0.012	0.04	0.20
Pedestrians	Near expressway	0.004	0.008	0.03	0.12

[a] Prepared from: Estimated changes in human exposure to suspended sulfate attributable to equipping light duty motor vehicles with oxidation catalyst (unpublished NERC/RTP working document).

[b] Data of D. B. Lurner, and L. Niemeyer, Meteorology Laboratory, National Environmental Research Center, Research Triangle Park, N.C., personal communication.

[c] Data of National Academy of Sciences Report (58).

[d] Computed by multiplying sulfate levels by adjustments for digerences in emissions per vehicle-mile: (0.00002 μg/mile Pt/(0.05 μg/mile sulfate).

may prove to be additive or synergistic with co-exposure to other ambient air pollutants. Warnings regarding potential hypersensitive reactions to platinum compounds used therapeutically as anticancer agents have been published (69). While the amount of platinum used for medical purposes is small compared to its other uses (Table 1), should the therapeutic use of platinum increase, those individuals undergoing treatment would also need to be considered in evaluating exposure risks to allergenic platinum salts in the ambient air.

Exposure Estimates

At the present time, platinosis has been primarily an occupational hazard. The use, distribution, and value of platinum and other noble metals has restricted their appearance as environmental contaminants. Evidence in support of this conclusion is found in the low levels of platinum observed in prelimi-

nary residue analysis of human tissues from autopsy material (72).

Although extensive trace metal analysis of catalytic modified exhausts are not available, estimates of short-term platinum exposure levels for a dispersion model range from 0.001 to 0.1% of the threshold limit value for soluble platinum salts, i.e., 0.002–0.2 $\mu g/m^3$ (Table 9). Lead estimates are made assuming a lead emission of 0.07 grams per vehicle mile. The estimated lead levels (16 to 41 $\mu g/m^3$) are close to actual measurements (4.5 to 71.3 $\mu g/m^3$) made under typical meteorological conditions near the Los Angeles freeway (58). The actual values may be lower than estimated as a consequence of lead settling near the roadside. Conversely, underestimations are possible as a result of actual recirculation of settled emission particulate matter by the traffic stream not considered in the model. Platinum exposure levels were estimated using the reported emission value of 0.0002 g/mile

and assumes that the catalytic attrition products are in emission particulate matter similar to that of lead (14,51,55). It is to be recognized that gaseous platinum oxides may form and the emission level reported may represent a low value. Conversely, the catalyst used may have been faulty and the reported value an upper boundary. Estimates were made by using adjustments for the lower platinum emission level and the percentage of light-duty motor vehicles equipped with catalytic convertors, i.e., lead values $\times$ 0.00002 μg/mile Pt $\div$ 0.07 μg/mile Pb $\times$ 0.25 = lead values $\times$ 7.14 $\times$ 10^{-5}. All of the platinum estimates are 1976 and 1982 projections when 25 and 100% of the national light duty motor vehicle fleet would be equipped with catalytic convertors.

In order to evaluate these estimates of platinum levels in the ambient air, consideration must be given to the nature and potency of the expected allergic response, the threshold limit values (2.0 μg/m³), and refinery range observed that produced mild symptoms under ventilated conditions (0.19–3.1 μg/m³). It is not unlikely that platinum sensitization could occur and sensitive individuals react upon continuous short-term exposures (automobile passengers, pedestrians) to estimated levels that may appear under worst meteorological conditions with 25% of the light-duty vehicles equipped with catalysts. The response probability increases considerably when all light-duty motor vehicles are equipped with catalysts since the estimated ambient air platinum levels are 0.1% of the threshold limit value under worst meteorological conditions. Consideration must also be given to exposure risks of urban residents living near busy arterial freeways and subjected to these levels continuously. Furthermore, average hourly peak levels may reach and exceed the threshold limit value under adverse weather conditions in urban areas. It is important to interpret estimated projections cautiously, since the projections are only as valid as the assumptions made. The major significant conclusion from these estimates is that a new environ-

mental agent can be expected in the ambient air at detectable levels by 1976.

The Genesis Problem

The catalytic attrition material identified to date in exhausts from convertor equipped automobiles has been primarily associated with particulate matter (51). Toxicity depends both upon the chemical and physical properties of the particulate matter. Particle size and mass are fundamental in determining residence time in the atmosphere, pulmonary distribution and dosage (14,73,74). In addition to inorganic material (halides, sulfur, phosphorus), polycyclic organic material and trace metals are associated with automotive particulate emissions, some of which have been linked with the incidence of lung cancer (18,56,75–77). Although the quality and quantity of air pollution may vary from city to city, there appears to be sufficient evidence to confirm an urban factor in etiology of lung cancer. It is important to realize that carcinogenicity and the onset and progression of other chronic degenerative diseases can be modified by physiological predisposition, occupational differences, and social-personal health habits (smoking). However, the etiological role of air pollution in human lung cancer has been convincingly demonstrated by epidemiological methods. In comparing lung cancer rates in migrants to rates in their country of origin and the country to which they emigrated it was concluded that differences in death rates are compatible with the changes in their environment and general pollution level (78). Furthermore, individuals in the U.S. who move from high pollution to low pollution regions display ,reduced lung cancer rates, indicating a definite risk-gradient from rural-low to urban-high air pollution areas (7,79). It is important to realize that correlations between residence and lung cancer persist even following corrections made for cigarette smoking (7).

Urban air and cigarette smoke have carcinogenic chemical agents in common that

are present in the benzene-soluble fraction of airborne particulate matter (6,7,10,14, 33,80). The level of polynuclear aromatic hydrocarbons (PNA) in ambient air is indexed by measuring the benzo[a]pyrene (BaP) content (14). While whole PNA samples and BaP have proven to be carcinogenic by various bioassays, it is important to recognize that not all chemical carcinogens have been identified, and the role of polycyclic aromatic hydrocarbons or BaP as human carcinogens has yet to be confirmed (14,80). Furthermore, other unregulated ambient air pollutants, such as sulfur dioxide, ferric oxide, and chromium, have been shown experimentally to exert a potentiating effect on the carcinogenic properties of polycyclic aromatic hydrocarbons (76,81,82). Sulfur dioxide has not only been shown to potentiate the carcinogenicity of benzo[a]pyrene in rats but the tumors observed are morphologically similar to those observed in man (11). Although arguments are raised regarding the relevance of experimental animal data to man and threshold limits for carcinogenicity, sufficient epidemiological and experimental data exist that document a definite risk associated with exposure to carcinogenic and cocarcinogenic agents in the urban ambient air (7,14,83). It is important to recall that reduction of hydrocarbons in compliance with the statuatory requirements of the Clean Air Act is important in regulating photochemical oxidants and is to be distinguished from control of nonmethane carcinogenic hydrocarbons associated with particulate emissions. Accordingly, reductions in carcinogens (benzo[a]-pyrene) and cocarcinogens (metallic ligands) associated with particulate matter could be achieved by regulating particulate emissions. The question remains as to the potential carcinogenicity and cocarcinogenicity of platinum and palladium.

While trace metals may serve as cocarcinogens, the interaction of platinum and palladium with the reticuloendothial systems and modification of immunodefense mechanisms is of particular interest regarding con-

tributions to the carcinogenic potential of polluted ambient air (14). A lifetime feeding study that involved administering 5.0 ppm of palladium chloride to mice in their drinking water significantly and selectively increased the malignant tumor incidence and increased the longevity of male mice (84). While the tumor incidence may reflect the difference in longevity, most of the tumors observed were of the lymphoma-leukemia types (controls: 2, palladium: 10). Adenocarcinoma or pappilary adenocarcinoma of the lungs were the second most prevalent tumors observed (controls: 1; palladium: 6). Apart from their role as possible cocarcinogens, there is an interesting apparent correlation between the capacity of several trace metals to induce contact dermatitis and their association with human carcinogenicity, specific reference is made to nickel and chromium (67). There is one report that indicates exposure to palladium can induce contact dermatitis (85). As noted previously, exposure to soluble platinum salts involves hypertrophy of lymphoid tissues and lymphocytosis.

The theories pertaining to carcinogenic mechanisms are varied. However, in view of the biological activity of platinum and palladium attention is referred to the role of the immunoresponse system in chemical carcinogenicity (14). Tumor specific antigens are present in most neoplasms with early stages of onset marked by extensive lymphocytic infiltration (86–88). While the significance of tumor antigens in carcinogenesis remains unexplained, the involvement of these noble metals with immediate and delayed hypersensitivity, their capacity to alter lymphatic tissue, to bind with DNA and proteins all serve as a reason for concern regarding interference in immunosurveillance mechanisms involved in the carcinogenic process. Additional concerns regarding the biological activity of platinum and palladium are focused upon the rather short latent period for induction of maligencies arising from disturbances in the reticuloendothelial system and the mutational potential of these noble metals (89).

It is not unlikely that various chemical agents that reach bone-marrow tissues, traverse the placental barrier, and interact with genetic material have a potential to be mutagenic and teratogenic as well as carcinogenic. Evidence of transplacental passage of palladium will be presented at this conference (90). Nearly all malignant and many benign neoplasia are characterized by distorted chromosomal karyotypes (27–29,31, 91,92). The clastogenic properties of several metals and metallic ligands are recognized and it is important to realize that these events often occur in the absence of cellular lethality, reflect interaction with DNA and distortions in those mechanisms responsible for accurate cellular genetic repair (27,93). In accord with central dogma of gene expression (27), the appearance of tumor-specific antigens provides evidence in support of mutational theories of carcinogenicity or epigenetic alterations in mechanisms governing gene expression transferred through cytoplasmic inheritance.

Hereditary disease has become a growing concern and can result in expansion of susceptible subgroups in the population with a resultant increase in medicare costs (2,94). Approximately 250,000 genetically abnormal children are born in this country each year with defects ranging from chromosomal abnormalities (trisomy and mongolism) to subtle inborn errors of metabolism (enzymatic defects) requiring clinical treatment, e.g. Lesch-Nyhan syndrome. There are approximately 1876 medical conditions attributed to genetic effects that account for 25% of all infant fatalities and a similar percentage of hospitalized adults (83,95). It has been estimated that each individual carries between 5 and 10 genetically harmful genes (34,37). Since approximately all human carcinogens, are induced by environmental chemicals, and since most mutagens are likely to be carcinogens, attention should be given to adequate analysis of their mutational potential by evaluating distortions in bioequivalent molecular mechanisms governing these genetic lesions of both somatic and

germinal tissues (96,97). We will learn in subsequent papers to be presented (70,90,98), that platinum and palladium can be absorbed by inhalation, dermal, and oral exposure routes (90,98). In this regard, the chemical identity of platinum-group metals in exhaust emissions and their metabolic ligands are critical to the environmental impact analysis of these expected new contaminants.

Biotransformation

It can be anticipated that noble metals associated with the use of catalytic converters will not only be emitted into the air but will also deposit on the ground. Accumulation of platinum and palladium in streams, rivers, lakes, and their sediments can result from discarded catalytic materials as well as from ground water contamination by exhaust attrition products from rain-washed highways. While effects on microbiological flora and plant life are of ecological concern, microbial biomodification in stream and lake sediments is of specific interest, since this may serve to modify toxicity and promote food chain contamination. The environmental impact and public health consequences arising from mercury contamination in the food chain has been well defined (19, 20, 99). The disasters of Minamata and Niigata, Japan, serve to underscore the toxicological significance of environmental biomodifications. Although a variety of mercury compounds have been involved, the etiological agent identified in Minamata and fetal Minamata disease is methylmercury (19,20,99). The stability of the alkyl–mercury bond in biological systems determines its capacity to traverse the blood-brain barrier with irreversible neurological damage, to cross the placenta and accumulate in fetal tissues with dire teratological consequences, and to contaminate and accumulate with increasing concentrations in the food chain.

The transformation of mercury compounds into methylmercury occurs via a bioequivalent reaction common to microbes, plants and animals and involves methylcorrinoid deriva-

tives of methylcabalamin (vitamin B_{12}) (20). Platinum compounds have recently been shown to interfere with methylation chemistry essential to vitamin B_{12} metabolism (100). Apart from methyl group transfer to the metal involved, this interference can impose disturbances in the oxidation of odd-carbon fatty acids and the biosynthesis of essential amino acids (101). In addition to mercury, there have been recent reports indicating that thallium, gold, and platinum can be methylated by this common methyl-cobalamin (vitamin B_{12}) reaction (20,102). While these reports need to be confirmed, the bioenvironmental significance will depend upon the stability of the methylated platinum compounds and level of environmental contamination of these methylation products. With experimental animal models of Minamata disease, the biological response to methylmercury appears to be the result of a disturbance in protein synthesis associated with the endoplasmic reticulum (103–107). Alterations in serum protein level were noted prior to the appearance of neurological signs of poisoning (105). These experimental observations appear to have been clinically confirmed, since many of the Japanese victims of methylmercury poisoning displayed changes in serum protein levels (108). It is noteworthy, that preliminary results indicate that an alteration in serum protein levels appears to be a significant response observed among experimental animals exposed to catalyst modified exhaust emissions containing an estimated 0.029 $\mu g/m^3$ of platinum (60). Preliminary analysis of platinum and palladium residues among organs of experimental animals treated by various exposure routes (oral, intraperitoneal injections, inhalation) indicates the kidneys and liver have higher levels of these metals than other organs of the body (79,90,98). A similar pattern was observed in methylmercury-treated animals. Neurological signs of intoxication appear as a function of the amount present in the brain (99,103). While pollutant burden analyses are useful exposure indicators, organ redistribution and metabolic transfor-

mation tends to limit their capacity in predicting impending pathology.

In order appropriately to assess potential health hazards associated with the possible biotransformation of platinum and palladium further information is required, particularly regarding the stoichiometry and kinetics of their methylation chemistry as well as evidence that stable methylated compounds are of significant toxicity. In this way quantitative estimates regarding transfer and transport processes *in vivo* and in ecological systems can be approached with a reasonable degree of confidence.

Comment

There is insufficient quantitative information available adequately to assess the environmental toxicology of platinum and palladium. These agents are expected to reach detectable levels in the ambient air by 1976, when 25% of the light-duty motor vehicles will be equipped with catalytic convertors. Continued use of catalytic convertors over an ensueing 6-yr period yields estimates of platinum in the ambient air under adverse weather conditions close to the threshold limit value set for soluble platinum salts. Individuals sensitive or sensitized to platinum may react immediately, depending on the level and chemical nature of soluble platinum salts that may be present in exhaust emissions. Since an average latent period of platinoisis is approximately 5 yr, with due consideration given to available information, it would not be unreasonable to expect an increase in asthma and contact dermatitis with continued use of these emission control devices beyond 1982. In this 8-yr period when all of the light-duty motor vehicles in the United States would be equipped with catalytic convertors, an estimated 164 tons of platinum would have been lost to the environment. This may represent an upper range depending on catalyst durability and recycling efficiency. It is important to realize that a large percentage of the automobiles are located in urban areas. The time in which

significant levels of platinum would appear in drinking water or the amount required for potential microbial methylation cannot be determined at this point.

In the absence of adequate information for impact analysis, the areas of concern have been identified and investigations undertaken. Preliminary results will be presented and include the following highlights. Platinum and palladium can be absorbed into biological tissues when administered to experimental animals by various exposure routes (72,90,98); soluble platinum compounds (Pt^{+4}) are more toxic than compounds of other metals of interest (Mn, Pb) when administered orally; however palladium is more toxic than these metals when administered intravenously (98). Absorbed noble metals can traverse the placental barrier in pregnant experimental animals (90). Preliminary cardiovascular studies suggest that palladium can act as a nonspecific cardiac irritant as well as a peripheral vasoconstrictor in experimental animals (90). At the present time, there is relatively little contamination of platinum in human tissues (72).

It is important to recall the low concentrations required for immunological and allergic response. While sulfuric acid aerosols and particulate sulfates may adversely affect existing asthmatics, they are not considered allergenic in the same context as soluble salts of platinum, i.e., these platinum compounds have the capacity to induce asthma as well as aggravate existing asthmatic conditions. Furthermore, reducing sulfur emission products may be alleviated by reducing the sulfur content in gasoline. Regulation of noble metals attrition products would require discontinued use of catalytic convertors in the concurrent advancements in engine design in order to still comply with statutory standards (CO, HC, NO$_x$), the use of alternate convertor designs with high durability (41), or any future legislative constraints on particulate exhaust emissions.

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