The Analyst

Platinum concentrations in urban road dust and soil, and in blood and urine in the United Kingdom†

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Increasing Pt concentrations from vehicle catalysts have been reported from a number of countries. Analysis of Pt and Pd in soils and road dusts taken from areas of high and low traffic flows in SE England show concentrations of Pt in the range < 0.30–40.1 ng g^{-1} and Pd in the range < 2.1–57.9 ng g^{-1}. Higher concentrations of Pt are associated with high traffic densities. Samples taken from streets of lower traffic flows were found to contain the lower concentrations of the ranges. Pilot studies of Pt concentrations in blood and urine using ICP-MS have been carried out. Platinum concentrations in whole blood were: precious metal workers, 780–2170, mean 1263 pmol l^{-1} (0.152–0.423, mean 0.246 μg l^{-1}); motorway maintenance workers, 645–810, mean 744 pmol l^{-1} (0.126–0.158, mean 0.145 μg l^{-1}); Imperial College staff, 590–713, mean 660 pmol l^{-1} (0.115–0.139, mean 0.129 μg l^{-1}). Platinum concentrations in urine in pmol Pt per mmol creatinine were: precious metal workers, 122–682, mean 273 [0.21–1.18, mean 0.47 μg Pt (g creatinine)$^{-1}$]; motorway maintenance workers, 13–78, mean 33.7 [0.022–0.135, mean 0.058 μg Pt (g creatinine)$^{-1}$]; Imperial College staff, 28–130, mean 65.6 [0.048–0.224, mean 0.113 μg Pt (g creatinine)$^{-1}$]. Detection limits were 0.03 μg l^{-1} for both blood and urine. The possible health effects of increasing Pt in the environment are discussed. Platinum provides an excellent example of the significance of speciation in metal toxicity. Platinum allergy is confined to a small group of charged compounds that contain reactive ligand systems, the most effective of which are chloride ligand systems. Metallic Pt is considered to be biologically inert and non allergenic and since the emitted Pt is probably in the metallic or oxide form, the sensitising potential is probably very low. Platinum from road dusts, however, can be solubilised, and enter waters, sediments, soils and the food chain. There is at present no evidence for any adverse health effects from Pt in the general environment, particularly allergic reactions.

Keywords: *Platinum; environment; blood; urine*

Vehicle exhaust catalysts became mandatory in the UK at the beginning of 1993 in response to the emission standards introduced in the EU. Modern three way catalysts, which control NO_x emissions as well as CO and HC, typically contain 1 to 3 g of Pt group metal (PGM)[1] supported on a ceramic honeycomb monolith housed in a stainless steel box similar in shape to that of a conventional silencer. Catalytic converters can be considered as mobile sources of Pt and the prime source of Pt found in road dusts and surface sediments;[2–6] Pt emissions from catalytic converters are due to the abrasion of the surface of the catalyst.[5]

A number of papers discuss Pt concentrations in the vicinity of roads in Sweden[2,3] in the USA and in Germany.[4–11] Alt and Messerschmidt[11] have reported that Pt concentrations in motorway soils in Germany ranged from 15–30 ng g^{-1}, which is considerably higher than background concentrations in soils (range 0.09–4 ng g^{-1}); alpine and forest soils were reported to contain 0.09 and 0.1 ng g^{-1}, respectively.

A number of authors have reported that Pt concentrations in the environment increased after the introduction of catalytic converters. A study of roadside dust and stream sediment[2,4] in Sweden demonstrated an increase in Pt concentrations from 1984 to 1991. Helmers *et al.*[6] have attributed the large increase in Pt concentration in sewage sludge in Stuttgart since 1988 to releases from catalytic converters. The levels remained almost constant between 1972 and 1987, when significant increases began with steep rises each year. The value for 1992 (605 ng g^{-1}) showed an almost eight-fold increase over the 1987 concentration (78 ng g^{-1}).

In Sweden both roadside dusts and sediment from gully pots were found to contain Pt, the former containing 39–88% less Pt than the gully pot sediments.[2] Concentrations of Pt in the gulley pot sediments (< 63 μm) ranged from 3.5 to 15 ng g^{-1}. Sequential extraction of Pt in the road surface sediment showed that Pt appeared in the following operationally determined fractions: exchangeable, 15%; carbonate, 10%; Fe/Mn oxide, 32%; organic, 26%; and residue, 26%. In the gulley pot sediment Pt was found only in the organic form and the authors suggested that the higher organic content of gulley pot sediments results in a transformation of Pt species.[2,3] Thus although it is generally accepted that Pt emissions are in the form of Pt metal or oxide particulates,[5] there is evidence that at least part of this is soluble and can undergo transformations in the environment thus providing a mechanism for Pt to enter the food chain. Studies in Australia have suggested that a major pathway of Pt into the human body is *via* the diet with an average dietary intake of 1.44 μg Pt per day.[12] Platinum has been shown to be present in blood and urine samples from populations in both Germany[13] and Australia.[14]

Since the introduction of vehicle catalytic converters, there has been little information concerning Pt metals in the UK environment. We have reported[15] the concentrations of Pt metals in urban road dust and soil in an area in the south of London (Richmond, Kew and Kingston), where the concentration of Pt ranged from < 0.3 to 8 ng g^{-1} in soils and from 0.42 to 29.8 ng g^{-1} in road dusts. The results showed higher

† Presented at The Sixth Nordic Symposium on Trace Elements in Human Health and Disease, Roskilde, Denmark, June 29–July 3, 1997.

concentrations of Pt in areas of high traffic flow, and it was concluded that the potential exposure would be higher for those living in urban environments or along major highways. This pilot study was undertaken with the following objectives: to investigate the concentrations of Pt in body fluids (blood and urine) in the United Kingdom; to correlate blood Pb concentrations with Pt concentrations to test the hypothesis that there is direct exposure to Pt from traffic fumes; and to consider the possible health effects of increasing concentrations of Pt in the environment.

Experimental

Analysis of urban soil and road dust samples

Procedures for the determination of Pt by fire assay–ICP-MS (detection limit 0.1 ng g^{-1}) have been described previously.[15–17]

Biological samples

Protocol for collection of blood and urine specimens

The study population consisted of 10 motorway maintenance workers, seven platinum refinery workers (positive controls) and five Imperial College staff working in the Royal School of Mines. Venous blood samples were collected in EDTA tubes. Duplicate samples of 2 ml were collected by venipuncture. Urine samples were collected as early morning spot samples.

Determination of platinum in urine

Sample preparation. Some of the urine samples contained a precipitate. All samples were thoroughly mixed and duplicate 10.0 ml volumes were digested in quartz conical flasks by adding 2.0 ml of 16 M HNO_3 and heating to dryness at 150 °C. The residues were dissolved by adding 100 μl of 16 M HNO_3 and 1.0 ml of water then transferred to weighed polycarbonate tubes and diluted, by weight, to 10.0 g with water. The digested solutions (10.0 ml) were prepared for analysis by adding 1.0 ml of 100 μg l^{-1} Au solution in 0.12 M HCl as internal standard. Three blank solutions were prepared by treating 10 ml volumes of water in exactly the same way as the urine samples. Calibrating standards were prepared by adding to a series 10.0 ml portions of a digested control urine, 1.0 ml of 100 μg l^{-1} Au solution in 0.12 M HCl and 1.0 ml of Pt standard solutions in 0.12 M HCl with added concentrations of 0, 0.5, 1.0, 2.0 and 4.0 μg l^{-1}.

Urine analysis

The concentrations of Pt in digested urine samples were measured by ICP-MS. The parameters set for data acquisition included measurements at masses 195 (Pt) and 197 (Au). The calibration graph showed much better linearity when Pt data were measured independently of the Au internal standard (the software allows data to be re-processed). All urine results reported were obtained without reference to the added internal standard. Subsequent blood analyses (see below) were carried out using Tl as internal standard, which is now used as routine. Creatinine measurements in urine were measured colorimetrically using an alkaline–picrate reaction and a Beckmann (Fullerton, CA, USA) CX7 automatic analyser.

Determination of platinum in whole blood

An initial measurement of Pt in the whole-blood samples following a 1 + 19 dilution with a diluent containing NH_4OH, $(NH_4)_2H_2EDTA$, Triton X 100 and using Au as internal standard gave a detection limit of 0.1 to 0.3 μg l^{-1} (in blood). This failed to detect Pt in all but two samples. Therefore the analyses were repeated using a digestion procedure which afforded a much lower detection limit of 0.03 μg l^{-1}.

Sample preparation. Duplicate 1.0 ml portions of whole-blood samples were digested in quartz conical flasks by adding 2.0 ml of 16 M HNO_3 and heating to dryness at 150 °C. The residues were dissolved by warming with 100 μl of 16 M HNO_3 and water, transferred quantitatively to weighed polycarbonate tubes and diluted, by weight, to 5.0 g with water. The digested solutions were prepared for analysis by diluting 3.0 ml in polycarbonate tubes, with 3.0 ml of 0.12 M HCl and 100 μl of 0.1% m/v Triton X 100 solution; 600 μl of 10 μg l^{-1} Tl solution in 0.12 M HCl was added as an internal standard. Three blank solutions were prepared by treating 1.0 ml portions of water in exactly the same way as the blood samples. Calibrating standards were prepared in 0.12 M HCl with Pt added at 0, 0.025, 0.05, 0.05, 0.10, 0.20 and 0.40 μg l^{-1}; 3.0 ml portions of these standards were diluted with 3.0 ml of a digested control blood sample, 100 μl of 0.1% m/v Triton X 100 solution and 600 μl of 10 μg l^{-1} Tl solution in 0.12 M HCl. The concentrations of Pt added were equivalent to the range 0–2.0 μg l^{-1} in whole blood.

Whole blood analysis

The concentrations of Pt in digested blood samples were determined by ICP-MS. Measurements were made at 195 (Pt) and 205 (Tl) for the internal standard.

The determinations were controlled by the analysis of the spiked digested samples as internal quality control (IQC) samples within each analytical run at a frequency of one single IQC per not more than four duplicated tests. The mean 'recovery' or proximity to target for these IQC urine and blood samples are given in Table 1. Recovery in the digestion process is under investigation and will be addressed in a full study. The relative standard deviations for urine analyses ranged from 0.133 at 0.04 μg l^{-1} to 0.02 at 0.850 μg l^{-1}. For whole blood the RSDs were: 0.09 at 0.150 μg l^{-1}, 0.02 at 0.500 μg l^{-1}, and 0.015 at 2.00 μg l^{-1}. Detection limits for ICP-MS analyses were 3 *s* of blank measurements. These were 0.030 μg l^{-1} for both blood and urine. Platinum concentrations in water were < LOD of 0.00130 μg l^{-1}, obtained by direct analysis, *i.e.*, no dilution.

Determination of lead in blood was carried out as described previously.[18]

Results

Concentrations of platinum in soil and road dust samples

A summary of the Pt concentrations in soil and road dust samples from Richmond and Kew are shown in Table 2.

Concentrations of platinum in whole blood

Blood Pt concentrations for the three main groups are shown in Table 3. The concentrations of Pt in blood are higher in the precious metal workers than in the other two groups and the concentrations are significantly different between the groups (Kruskall–Wallis non parametric analysis of variance p = 0.00033).

Table 1 Mean recovery or proximity to target for IQC* samples

IQC Matrix	Concentration range of Pt/μg l^{-1}	Recovery (%) Mean	*s*	*n*	Ratio Samples: IQCs
Urine	0.50 to 4.0	99	2	7	3.1
Blood	0.125 to 2.0	95	8	9	2.7

* IQC; spiked digested samples as internal quality control.

Concentrations of platinum in urine

Urine Pt concentrations are shown in Table 4. The urine Pt concentrations are significantly different between the groups (Kruskall–Wallis non parametric analysis of variance $p = 0.00057$), with the precious metal workers again having considerably higher levels than the other two groups. The highest values for Pt in both blood and urine were from one precious metal worker. An analysis of the association between Pt concentrations in whole blood and urine shows that the two are correlated when the three groups are combined (correlation coefficient $r = 0.89$, 95% confidence interval 0.76–0.95). The linear relationship between Pt in blood (pmol l^{-1}) and Pt in urine (pmol $mmol^{-1}$ creatinine) is given by: Pt (blood) = 2.07 Pt (urine) + 647.

Concentrations of lead in whole blood

Lead concentrations in whole blood are shown in Table 5. No statistical differences were found between the three groups (Kruskall–Wallis non parametric analysis of variance $p = 0.334$). There is no significant correlation between blood Pt and blood Pb for the unexposed populations (*i.e.* motorway maintenance workers and Imperial College staff).

Discussion

Concentrations of platinum in soils and road dusts

In Richmond, where samples of both top soils and dusts were analysed, the highest values occur in road dust, especially in those samples collected at major road intersections which occur as nodes on through routes used by London commuter traffic. The road dusts on minor roads, however are generally comparable with values in soils representing the local background which is close to 1 ng g^{-1}. Samples of soils and dusts taken from the roads in Richmond have been classified into four general categories as shown in Table 1. These results indicate that traffic is the source of Pt in dusts and soils near busy roads.

Table 2 Platinum/ng g^{-1} in soil and dust from four road categories in Richmond and Kew (from ref. 15)

Road category	*n*	Range	Mean
Major road intersections	5	11.2–23.7	20.8
Major roads	13	7.17–24.3	12.9
Intermediate roads	3	0.42–4.92	1.93
Minor roads	8	0.35–4.26	2.29

Concentrations of platinum and lead in body fluids

The values presented here show a clear correlation between Pt in urine and Pt in blood for exposed individuals. The exposed precious metal workers showed the highest levels of Pt in both blood and urine. The other groups probably represent background levels. There appears to be no correlation between blood Pb and Pt concentrations in blood and urine. The motorway maintenance workers who might have been expected to be highly exposed to traffic emissions were working on a new motorway in a rural area. The Imperial College staff live and work in London, with a high exposure to traffic fumes. It would appear, therefore, that exposure to traffic as measured by blood Pb levels was not markedly different for the different groups. As Pt concentration in blood is not correlated with blood Pb then it cannot be concluded with certainty that traffic emissions are the primary source of Pt. Since this is a key issue it is necessary that blood Pt concentrations should be measured in a group of individuals whose blood Pb is known to be raised from exposure to traffic emissions. The numbers in this pilot study are small and a more detailed investigation is required. Vaughan and Florence[12] concluded that diet was the main route for human intake in Australia. It may well be that diet is the main route in the UK, but more work must be carried out before firm conclusions can be reached.

From the pilot study it can be concluded that: individuals, both exposed and unexposed have measurable concentrations of Pt in both whole blood and urine; there is a correlation between Pt in blood and in urine, particularly for exposed individuals; and there is no correlation between Pt and Pb in blood, in this pilot study.

Health effects of platinum in the environment

Increasing Pt concentrations from vehicle catalysts have been reported from a number of countries. The question then arises of whether an increase in Pt concentrations in urban dusts and sediments poses a threat to human health[18] through the inhalation of fine dusts. The main hazard of Pt exposure is seen in workers occupationally exposed to halogenated salts.[19] Adverse health effects following occupational exposure in a photographic studio to complex Pt salts were first described[20] in 1911. In 1945 Hunter,[21] in studies on workers in four British Pt refineries, reported 52 out of 91 showed symptoms of sneezing, wheezing and shortness of breath. Latency from first contact with Pt to the development of symptoms varied from a few months to 6 years. The Health & Safety Executive[22] lists

Table 3 Platinum concentrations in whole blood/pmol l^{-1} (μg l^{-1} in parentheses)*

	n	Mean	*s*	Grand mean	Range
Precious metal workers	7	1263 (0.246)	467	1200	780–2170 (0.152–0.423)
Motorway maintenance workers	10	744 (0.145)	46.3	743	645–810 (0.126–0.158)
Imperial College staff	5	660 (0.129)	51	659	590–713 (0.115–0.139)

* 1.95×10^{-4} pmol Pt = μg Pt

Table 4 Platinum concentrations in urine/pmol (mmol creatinine)$^{-1}$; [μg (g creatinine)$^{-1}$ in parentheses]

	n	Mean	*s*	Grand mean	Range
Precious metal workers	7	273 (0.47)	206	224	122–682 (0.21–1.18)
Motorway maintenance workers	10	33.7 (0.058)	21.6	28.5	13–78 (0.022–0.135)
Imperial College staff	5	65.6 (0.113)	38.7	58.0	28–130 (0.048–0.224)

watering of the eyes, sneezing, tightness of the chest, wheezing, breathlessness, cough, eczematous and urticarial skin lesions, signs of mucous membrane inflammation as typical of Pt salt sensitisation. Newman-Taylor[23] has shown that sensitised workers removed immediately from further contact with Pt salts showed no evidence of long term effects.

Platinum provides an excellent example of the significance of speciation in metal toxicity. Metallic Pt appears to be biologically inert and non-allergenic. Platinum allergy is confined to a small group of charged compounds that contain reactive ligand systems the most effective of which are chloride ligands[24,25] The allergic response increases with increasing number of chlorine atoms, the most potent compounds being hexachloroplatinic(IV) acid and its ammonium and potassium salts, and potassium and sodium tetrachloroplatinate(II). Non halogenated complexes and neutral complexes including the anticancer drug cisplatin, $[Pt(Cl)_2(NH_3)_2]$ are not allergenic, perhaps because they do not act as haptens. The antitumour activity of cisplatin is due primarily to its intrastrand DNA cross linking activity. While occupational exposure to Pt compounds has been monitored intensively over recent years, there is little information on general population exposure and health effects.

It has been shown that Pt in road dusts has increased after the introduction of vehicle catalysts and Alt and Messerschmidt[11] have addressed the problem of general health effects of Pt emissions from vehicle exhaust catalysts. There is at present no evidence for any adverse health effects from Pt in the general environment, particularly allergic reactions. An immunological study[26] investigated the allergenic effects of particulate exhaust samples on workers already sensitive to Pt salts. Skin prick tests, in a preliminary study on only three subjects, were all negative up to a concentration of 5 μg ml^{-1} which would normally be sufficient to elicit a response. Metallic Pt is considered non allergenic and since the emitted Pt is probably in the metallic or oxide form, the sensitising potential is probably very low.

Platinum from road dusts, however, can be solubilised, and enter waters, sediments, soils and the food chain. Platinum in material of plant and animal origin appears to be bioavailable.[12] Certain Pt compounds are known to be cytotoxic and have mutagenic and carcinogenic effects, and to have effects on microorganisms at very low concentrations.[24] It is evident that more information concerning exposure of the human population to Pt metals in water, air and foodstuffs and the health effects of such exposure is urgently required.

This project was supported by the Department of the Environment (UK).

Table 5 Lead concentrations in whole blood/μg dl^{-1}

	n	Mean	*s*	Grand mean	Range
Precious metal workers	7	4.03	1.86	3.76	2.7–8.0
Motorway maintenance workers	10	3.02	0.57	2.98	2.5–4.0
Imperial College staff	5	3.28	1.53	2.96	1.8–5.0

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Paper 7/05920E
Received August 8, 1997
Accepted December 4, 1997