Cobalt Concentration in Urine as an Indicator of Occupational Exposure to Low Level Cobalt Oxide

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Abstract: The aim of this study was to verify the relationship between the cobalt oxide concentration in air and the cobalt concentration in urine. In the first study, we measured the cobalt concentration in the urine of 16 workers exposed to cobalt oxide in a Digital Video Cassette manufacturing plant at the beginning and end of their workshift during 2 working cycles. In the second study, we measured the exposure level to cobalt oxide and the cobalt concentration in urine at the end of the workshift of other 16 workers on 3 out of 5 consecutive work days (Monday, Wednesday and Friday). Four workers among the 16 participated twice. In the first study, the cobalt concentration in the urine increased about 1.5-3 fold during each workshift and then tended to decrease rapidly to the control value before the next workshift. In the second study, we examined the relationship between air and urine concentration using 22 samples after excluding data from workers with dust respirators. Comparing the air and urine concentrations for samples under 30 µg/m3 (n=20), a significant correlation was found (r = 0.76). These results indicate that most of the inhaled cobalt oxide was excreted rapidly, and a good relationship was found between air and urine concentrations at the end of the workshift when the exposure level was under 30 µg/m3. Therefore, the cobalt concentration in urine at the end of a workshift can be used as an indicator of one-day exposure to cobalt oxide when the exposure is low level.

Key words: cobalt concentration in urine, cobalt oxide, indicator, relationship.

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Introduction

Cobalt is widely used in industry. For example, it is used as one of the constituents of hard metals [1–4], pigments, dyes, and videotapes. Recently, large amounts of cobalt have been used as a material for rechargeable batteries such as nickel metal hydride and lithium-ion batteries, which are used in hybrid electric vehicles, computers, cell phones, portable tools, audio/visual units, and numerous electronic devices [5]. The demand for cobalt in industry is estimated to be increasing [5].

In the meantime, the widespread application of cobalt has reportedly led to an increase in lung diseases, including diffuse interstitial fibrosis and bronchial asthma, especially among hard metal workers in many countries [6–9]. Cardiomyopathy [10, 11], polycythemia [12], and allergic dermatitis [13] have also been found in these workers. Many hard metals consist of cobalt and other constituents such as tungsten carbide [14]. Recently, hard metal particles have shown a higher genotoxic potential than metal cobalt alone, and epidemiological studies [15,16] have demonstrated a significantly increased mortality from lung cancer among hard metal workers. However, data about the carcinogenicity of the cobalt element alone, with regard to lung cancer, are yet inconclusive [17].

In order to estimate the level of occupational exposure to cobalt of workers, the cobalt concentration in urine has been proposed as a biological indicator of recent exposure [2–4, 18–20]. However, Lison et al. reported that the cobalt concentration in the urine of workers exposed to cobalt oxides poorly reflected the recent exposure level although the workers showed higher urine concentrations than non-exposed subjects [1]. They also reported that when workers were exposed to soluble cobalt compounds (metal, salt, and hard metal), the measurement of the cobalt concentration in urine at the end of the workweek could be recommended for the assessment of recent exposure. Except for this report, there are few reports about biological indicators of exposure to cobalt oxides. The Japan Society for Occupational Health [21] and the American Conference of Governmental Industrial Hygienists (ACGIH) [22] do not yet recommend the urine concentration of cobalt as a biological indicator for recent exposure to cobalt oxides because of a lack of evidence.

A large amount of cobalt oxides are used to make the cathode of lithium-ion batteries. Some processes in cobalt refineries [1] and the cobalt vapor deposition process in Digital Video Cassette (DVC) manufacturing plants have the possibility to generate cobalt oxide dust and to give it off to the ambient air. Therefore, workers in these factories may be exposed to cobalt oxides.

The present study was undertaken to examine the possibility that we can use the cobalt concentration in urine as a biological indicator of occupational exposure to cobalt oxides. For this purpose, we examined the changes in cobalt concentration in the urine of workers exposed to cobalt oxides during 2 working cycles after the summer holidays, as well as the ki-
netics of inhaled cobalt oxides. Second, we examined the relationship between the cobalt oxide concentration in air and the individual urine concentration of cobalt.

**Subjects and Methods**

*Subjects and sample collection*

Sixteen male shift workers involved in the cobalt vapor deposition process in a DVC manufacturing plant were included in the first study.

There are several processes in the manufacturing of DVC. First, in the cobalt vapor deposition process, cobalt materials are heated and cobalt vapor is sprayed and deposited on base films in sealed machines. Second, carbon and lubricants are applied to the films. Third, the films are cut to suitable sizes, checked, and sent to the assembly lines. Finally, the DVC are cased and shipped. Almost all processes are automated and do not expose the workers to cobalt oxide dust. However, some of the workers involved in the cobalt vapor deposition process clean and remove the cobalt oxides that adhere to the inside of the cobalt vapor deposition machines under dry conditions several times during the workshift. In order not to diffuse the cobalt oxide dust into the working area, the cleaning areas are separated from the working area by curtains during cleaning and the dust is removed by several exhaust systems. The workers wear dust respirators during the cleaning. Due to the separation of the areas, hardly any cobalt oxide dust diffuses into the working area. Workers in the working area do not necessarily wear dust respirators except during machine cleaning. We allowed the workers to wear the dust respirators during this study.

The workers in the study were all permanently employed staff and answered a self-reported questionnaire that included age, the duration of cobalt oxide exposure, and current smoking status.

This plant used a three-shift system for this process. The structure of the shift schedule in one working cycle was MMEENNxx (M: morning shift, E: evening shift, N: night shift, x: day off). This rotating shift schedule changed shifts at 08h00, 16h00, and 00h00.

We measured the cobalt concentration in the urine of these workers during the first 2 working cycles after their 10-day summer holidays. During the first working cycle (the first day was M), we measured the cobalt concentration in their urine each day at the beginning and end of the workshift. During the second working cycle, we measured the cobalt concentration in their urine at the beginning of the first workshift and at the end of the last workshift.

Another group of 16 male daytime workers involved in the same process in this plant was included in the second study. In this period, all work was done during the daytime.

All permanently employed workers in this study answered a self-reported questionnaire that included age, the duration of cobalt oxide exposure, subjective symptoms due to work, current smoking status, anamnesis, history of taking multivitamin pills, history of medication for anemia, and history of surgery involving implants. The workers were also asked to write
a list of what work they had done and whether with dust respirators or not for each day during the study.

We sampled the exposure level to cobalt oxides of these workers on 3 days out of 5 consecutive work days (Monday, Wednesday, and Friday) on an individual basis. We also measured the cobalt concentration in the urine of these workers on the same days as the air sampling at the end of the workshift. Four workers among the 16 participated in this study twice; therefore we examined a total of 20 workers' samples. However, the data from workers with dust respirators were excluded because we supposed that the cobalt concentration in the urine of the workers with dust respirators would poorly reflect the exposure level.

In addition, we measured the cobalt concentration in the urine of 25 non-occupationally exposed control male workers. The workers were all permanently employed staff and answered the above-mentioned self-reported questionnaire.

Both the exposed and non-exposed participants agreed to join the studies after being informed of the purpose and significance of the studies.

**Analysis of the particles in the air of the cobalt vapor deposition process**

In order to analyze the particles in the air from the cobalt vapor deposition process, we sampled the particles in a dust container of the exhaust system and analyzed them by an X-ray diffraction method (SHIMADZU, XRD-6100).

**Level of cobalt exposure**

In the second study, personal ambient air samples were collected from each worker equipped with a personal dust sampler (SIBATA, PAS-500) continuously for about 8 h on Monday, Wednesday, and Friday. The air-borne cobalt oxides were collected on a quartz fiber filter of 25 mm diameter (Whatman QM-A). The flow rate of the pump was adjusted to 0.2 l/min.

The total dust collected on the filter was combined with a diluted aqua regia (HNO₃: HCl: H₂O= 1: 1: 15) at 15 ml per sample in order to dissolve the cobalt oxides. The mixture was wet-ashed at 100°C for 30 minutes and left at room temperature, then centrifuged. The cobalt in the supernatant fluid was analyzed with Inductively Coupled Plasma (ICP) Atomic Emission Spectroscopy (Nippon-Jarrell Ash, IRIS-AP). The detection limit for cobalt in air was 1 μg/m³.

**Cobalt concentration in urine**

Samples were collected in an uncontaminated place after the worker had showered and changed into clean clothes. A 500 μl sample was combined with 200 μl of 1% Mg(NO₃)₂ as a modifier and 300 μl of deionized water. The cobalt concentration in urine was analyzed by flameless atomic absorption spectrometry (Perkin-Elmer, SIMMA6000). The detection limit for cobalt in urine was 1 μg/l when determining the cobalt concentration of the exposed
workers, and 0.2 μg/l when determining that of the non-exposed workers. Urinary creatinine concentration was measured according to the Jaffé’s picrate method. The cobalt concentration in urine was represented by μg/g creatinine.

All reagents used in the cobalt analysis were of a special grade (Wako Pure Chem.). Sample containers and glass equipment were used after treatment to remove the metal.

Statistical methods

Data on age were compared among the non-exposed group and the exposed group in the first study and the exposed group in the second study using a one-way fractional ANOVA followed by the Tukey-Kramer test. Data on the duration of exposure between the 2 exposed groups were analyzed by the Student’s t-test. The prevalence of smoking was compared among the 3 groups using the chi-square test. Data on the cobalt concentration in urine were analyzed using the paired t-test in the first study. In order to examine the relationship between the cobalt oxide concentration in air and cobalt concentration in urine in the second study, linear regression analyses were chosen for the testing of differences. Differences were considered statistically significant when P was less than 0.05.

Results

Characteristics of the subjects and cobalt oxide exposure profiles in the study

Table 1 summarizes the characteristics of the subjects and the cobalt oxide exposure profiles found in this study. The mean age of the exposed group in the first study was significantly younger than that of the non-exposed group and exposed group in the second study. The exposed group in the first study was significantly less exposed to cobalt oxides than that in the second study. No significant difference in smoking habits was noticed between the

<table>
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<th>Table 1. Characteristics of the study subjects and cobalt exposure profiles</th>
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<td>Non-exposed group</td>
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<td>Current smokers(%)</td>
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SD: standard deviation, *:P<0.05, **:P<0.01, ns: not significant,
1: results of a 1-way fractional ANOVA followed by the Tukey-Kramer test among 3 groups,
2: results of the Student’s t-test between 2 exposed groups,
3: results of the chi-square test among 3 groups
groups. No workers were taking multivitamin pills, medicines for anemia, or had undergone surgery involving implants.

**Cobalt concentration in the urine of non-exposed workers**

The cobalt concentration in urine from 25 non-occupationally exposed workers had a mean cobalt concentration of 0.5 μg/g creatinine (standard deviation: SD = 0.4). The smokers had almost the same mean cobalt concentration in urine (0.5 μg/g creatinine, SD = 0.4) as the nonsmokers (0.6 μg/g creatinine, SD = 0.5). In the nonsmokers, nine samples were under the detection limit, i.e., 0.2 μg/l. The nonmeasurable concentration was considered to equal the detection limit.

**Analysis of the particles in the air of the cobalt vapor deposition process**

Almost all the peaks of the diffraction pattern of the particles corresponded with those of cobalt oxides (both CoO₃ and CoO). However, the observed peaks of the diffraction pattern differed from that of cobalt metal. This showed that most of the particles that the workers were exposed to were cobalt oxides such as CoO₃ and CoO.

**Change in cobalt concentration in urine (with cobalt oxide exposure) during two working cycles**

In the first study, the cobalt concentration was analyzed at the beginning and end of each workshift in the first working cycle, and at the beginning of the first workshift and at the end of the last workshift in the second working cycle (Fig. 1A). In the first working cycle, the mean cobalt concentration in urine increased about 1.5-3 fold during each workshift, and increased significantly on the second M (P < 0.05), first E (P < 0.01), and first N (P < 0.05) and then tended to decrease to approximately the value of the mean cobalt concentration in urine at the beginning of the first workshift (the control value) during the interval before the next workshift began (Fig. 1B). The intervals of the workshifts between the second M and first E, and the second E and first N were about 24 h, while those between the first M and second M, the first E and second E, and the first N and second N were about 16 h. Therefore, the mean cobalt concentrations in urine at the beginning of the first E and N were slightly lower than those at the beginning of the second M, E, and N, because the longer the duration of non-exposure was, the more inhaled cobalt oxides were excreted.

Regarding the second working cycle, the intervals of the workshifts between the second N in the first working cycle and first M in the second working cycle were about 48 h. The mean cobalt concentration in urine at the beginning of the first M was not higher than the control value, and that at the end of the second N was not higher than that at the end of the second N in the first working cycle (Fig. 1C). This result indicates that the cobalt concentration in urine did not increase during the 2 working cycles.
Biological Monitoring of Cobalt Oxide Exposure

A
First working cycle

Second working cycle

B

C

Fig. 1. A: Experimental schedule. The workers worked in a 3-shift system. The structure of shift schedules in one working cycle was MMEENN (M: morning shift, E: evening shift, N: night shift). The shifts began at 08h00, 16h00, and 00h00. During the first working cycle, we measured the cobalt concentration in urine each day at the beginning and end of the workshift. During the second working cycle, we took measurements at the beginning of the first workshift and at the end of the last workshift.

B: Change in cobalt concentration in urine at the beginning and end of every workshift in the first working cycle. The results are shown as the mean ± standard deviation.

--- : exposure to cobalt oxide, ---- : level of control value, *: P < 0.05, **: P < 0.01, ns: not significant.

C: Change in cobalt concentration in urine at the beginning of the first morning shift and at the end of the second night shift in both the first and second working cycles.

---: first working cycle, second working cycle, M: at the beginning of the first morning shift, N: at the end of the second night shift.
Correlation between the cobalt oxide concentration in air and the cobalt concentration in urine

In the second study, the total number of participating workers was 20 because 4 of the 16 workers participated twice, and all workers were sampled on 3 days. Therefore, we were able to obtain 60 samples. However, we examined the relationship between air and urine concentration in 22 samples because the data from workers with dust respirators were excluded. All personal air samples except for 1 proved to be under 50 μg/m³, which was recommended by the Japan Society for Occupational Health (JSOH) as the Occupational Exposure Limit (OEL) of cobalt dust[21].

When comparing an individual's cobalt oxide exposure level with his cobalt concentration in urine on the same day at the end of a workshift, weak correlations were obtained (r = 0.36). However, when air and urine concentrations were compared for samples under 30 μg/m³ (n = 20), the relationship was linear with a significant correlation (r = 0.76, P < 0.01), as shown in Fig. 2. The relationship between the cobalt oxide concentration in air and cobalt concentration in urine measured at the end of the workshift was expressed by the equation: cobalt in urine (μg/g creatinine) = 1.05 cobalt oxides in air (μg/m³) + 3.02

![Fig. 2.](image-url)
Discussion

Cobalt is present in the everyday environment, and water and food are the main sources of human exposure to cobalt [22]. Several conditions have been reported to increase cobalt excretion in the urine of non-occupationally exposed humans. Alexandersson [4] reported that non-occupationally exposed smokers had a significantly higher mean cobalt concentration in urine (0.6 μg/l) than non-occupationally exposed nonsmokers (0.3 μg/l). However, we think that the degree of this difference hardly interferes with the biological monitoring of occupational exposure. Our results indicate that smokers in the non-exposed group did not have a higher cobalt concentration in urine than the nonsmokers. The cobalt concentration in the urine of persons with surgical implants made from cobalt-containing alloys [23] or those treated with cobalt-containing medication for anemia [24] or those treated with multivitamin pills containing cobalt [25] may be high and may interfere with the biological monitoring of occupational exposure to cobalt [22]. However, there was no worker undergoing these treatments among the participants in these studies. The result from the analysis of the particles from the cobalt vapor deposition process showed that almost all particles were cobalt oxides, not cobalt metal. From these findings, the cobalt concentration in urine was considered to reflect occupational exposure to cobalt oxides in our studies.

Cobalt exposure mainly occurs by inhalation and to a certain extent by mouth. Metallic cobalt and cobalt salt dissolve easily in body fluids, particularly in the presence of oxygen [26]. The carrier protein for cobalt in serum is albumin [27]. Secretion occurs primarily via the kidneys [28]. Lung clearance of cobalt follows a particular pattern involving rapid initial elimination followed by a slow elimination rate [4]. Urinary excretion is characterized by a rapid phase of a few days duration, followed by a second slow phase, which may last two years or more [24].

However, there are few data on the absorption or excretion of various forms of cobalt by the lung and gastrointestinal tract in humans. Lison et al. demonstrated that the cobalt concentration in urine reflected recent exposure to cobalt metal, salts, and hard metal powders (soluble forms) except for cobalt oxides [1]. In their data, the workers exposed to cobalt oxides did not show any significant relationship between the cobalt oxides concentration in air and the cobalt concentration in urine at the end of the workshift, although the cobalt concentration in the urine of workers exposed to cobalt oxides was higher than that of the non-exposed workers. The absorption rates of the various forms are likely to be dependent on their solubility in biological media [1]. Therefore, pulmonary absorption of cobalt oxides in humans is suggested to be lower than that of soluble compounds [1], although significant absorption of cobalt oxides may occur through the dissolution of the cobalt oxide in alveolar macrophage lysosomes [29].

In the first study, the mean cobalt concentration in urine increased about 1.5-3 fold during
each workshift and increased significantly on the second M \((P < 0.05)\), first E \((P < 0.01)\) and first N \((P < 0.05)\). According to this finding, the increases in the cobalt concentration in urine were caused by occupational exposure. Then, the mean cobalt concentration in urine tended to decrease to approximately the control value during the interval before the next workshift began in the first working cycle (Fig. 1B). This result suggested that most of the inhaled cobalt oxides were excreted rapidly during the 16-24 h workshift interval.

Regarding the second working cycle, the mean cobalt concentration in urine at the beginning of the first M was not higher than the control value (Fig. 1C). From this result, we suppose that all cobalt oxides inhaled by the workers in the first working cycle were excreted during the first working cycle, including the two days of no exposure.

The mean value of the interindividual percentage concentration decrease during the 48 h work interruption from the end of the second N in the first working cycle to the beginning of the first M in the second working cycle was 67.6%, which corresponded with the 68% decrease found during the 64 h work interruption from Friday at the end of the shift to Monday morning before the shift in Alexandersson's study in hardmetal-producing industries [4]. From this result, we suppose that the excretion of cobalt oxide was as rapid as that of hard metals under soluble conditions.

The mean cobalt concentration in urine at the end of the second N in the second working cycle was not higher than that at the end of the second N in the first working cycle. This result indicates that the cobalt concentration in urine did not increase during the 2 working cycles.

However, the control value was 5.0 μg/g creatinine (SD=3.7), which was almost ten times higher than that of the non-exposed workers. Past exposure before this study was assumed to have affected the control value. Lison et al. showed that the cobalt concentration in the urine of workers exposed to cobalt oxides was higher than that in the non-exposed workers [1]. They suggested that the finding of an increased cobalt concentration in the urine of workers exposed to cobalt oxides might result from delayed pulmonary absorption of the metal deposited in the lung [1]. Meanwhile, in Alexandersson's study [4], in the exposed study subjects (n=15) who were in hardmetal-producing industries, the cobalt concentration in urine decreased from 60.0 μg/l to 4.9 μg/l, but not to the control group level after a 4-wk vacation without occupational exposure to cobalt. We suppose that this finding also results from delayed pulmonary absorption of the soluble form of cobalt deposited in the lung.

From these results in the first study, although pulmonary absorption of cobalt oxides in humans might be lower than that of soluble compounds, when the exposure is low we suggest that almost all inhaled cobalt oxide is excreted in urine as rapidly as the soluble form of cobalt, and that recent cobalt oxide exposure hardly affects the cobalt concentration in urine during at least 2 working cycles (14 days). However, even if the exposure is low, a small amount of inhaled cobalt oxide deposited in the lung is absorbed by pulmonary alveoli and excreted in urine slowly like the soluble form of cobalt.
Several reports have demonstrated that the cobalt concentration in urine can be used as an indicator of recent exposure[2-4]. Most of these studies have been carried out in hard metal workers, not in workers exposed to cobalt oxides. In the data of Lison et al. [1], the workers exposed to cobalt oxides did not show any significant relationship between the cobalt concentration in urine at the end of workshift and cobalt oxide concentration in air ranging from 23-7772 μg/m³. However, we think that when exposure is high, much of the inhaled cobalt oxide does not dissolve in body fluids and is not excreted in urine because their pulmonary absorption in humans might be lower than that for soluble compounds [1]. This might be the main reason why the mean cobalt concentration in urine was lower in the cobalt oxide group than that in the cobalt metal group, even though the airborne cobalt concentrations were similar between the oxides and metal groups in their report.

In Fig. 2, the results collected from Monday, Wednesday, and Friday were pooled because the results from our first study showed that recent exposure during two working cycles did not affect the cobalt concentration in urine. In our results, when comparing an individual’s exposure level of cobalt oxides with his cobalt concentration in urine on the same day at the end of a workshift, weak correlations were obtained (r = 0.36). However, when the air and urine concentrations were compared for samples under 30 μg/m³ (n = 20), the relationship was linear with a significant correlation (r = 0.76, P <0.01).

We consider that relatively high exposures are not reflected in the cobalt concentration in urine. However, when we consider only low level exposure, a good correlation was found between the cobalt oxide concentration in air and the cobalt concentration in urine.

The relationship between cobalt oxide concentration in air and cobalt concentration in urine measured at the end of the workshift on an individual basis was expressed by the equation: cobalt in urine (μg/g creatinine) = 1.05 cobalt oxides in air (μg/m³) + 3.02. The regression coefficient value from this equation is higher than those of previous reports of hard metal workers, which ranged from 0.67-0.70 [2,3]. One reason for this higher regression coefficient might be that the exposure levels of our samples covered a very low and narrow range compared with these previous reports. Therefore, the degree of cobalt concentration increased by past exposure to cobalt oxides, even if it was small, was likely to affect the regression coefficient. This might also be the reason why the cobalt concentration in urine was 3.02 (μg/g creatinine), which was higher than that of the non-exposure group, without exposure in this equation.

As manufacturing processes have shown good improvement during mechanization, the exposure levels of cobalt oxides are becoming low. Therefore, a biological indicator of low exposure level should be established because cobalt oxides can have an effect on the respiratory passages as a result of sensibilization even if the exposure level is low. Our study findings indicate a high degree of conformity between the exposure levels of cobalt oxides and the cobalt concentration in urine on an individual basis. We suggest that the cobalt concentration in the urine at the end of the workshift can be used as an indicator of one-day exposure...
to cobalt oxide when the exposure level is low, at least below 30 μg/m³. Further studies are needed to demonstrate to what extent the cobalt concentration in urine reflects the cobalt oxide concentration in air.

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Biochem Med 9: 177–183
低濃度酸化コバルト曝露指標としての尿中コバルト濃度の有用性

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要旨：酸化コバルトの経時的変化の検討と、曝露濃度と尿中コバルト濃度の相関関係を検討する目的で、調査1として男性16名を対象に1クールの勤務日作業前後と次クールの初日作業前と最終日作業後の尿中コバルト濃度を測定した。また調査2として別の男性16名を対象に曝露濃度と作業後の尿中コバルト濃度を測定した。調査は5日間勤務の第1、3、5日目に行い保護具着用のない場合のみを検討した結果、調査1での尿中コバルト濃度は作業前に比べ作業後は増加したが、翌日には前値に復した。調査2の22回分のデータがプロットでき、曝露濃度が30μg/m3以下（n=20）ではr=0.76であったことから尿中コバルト濃度は過去の曝露に大きな影響を受けないことと曝露濃度と尿中コバルト濃度の間に強い相関関係があることから、少なくとも低濃度下では当日の曝露指標として作業後の尿中コバルト濃度は有用であると考えられる。

キーワード：酸化コバルト、尿中コバルト濃度、曝露指標、相関関係。

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