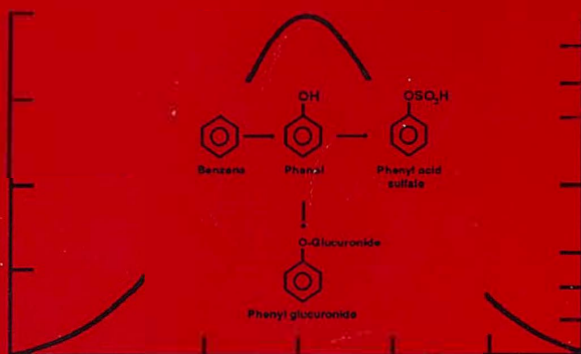


**British Occupational Hygiene Society**  
**Technical Guide No. 9**

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# Biological Monitoring *Reference data*



The BOHS Technology Committee

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H and H Scientific Consultants Ltd, Leeds  
1992 reprinted 1994

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**Technical Guide No. 9**

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# **Biological Monitoring**

## *Reference data*

Working Party on Biological Monitoring of  
The BOHS Technology Committee

E. King, *Consultant*,  
M.K Molyneux, *Shell UK Ltd*,  
R. Wagg, *Health and Safety Executive*.

*Series Editor:* Dr D. Hughes, University of Leeds

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# 1 INTRODUCTION

This *Technical Guide* is complementary to the paper by E. King entitled 'Occupational Hygiene Aspects of Biological Monitoring'.<sup>1</sup> It lists both air and biological limits which are published by various sources for selected chemical substances which have an application in the evaluation of exposure. The format used in this Guide serves to illustrate the complexities both in the ways that different national limits are documented and the factors that are involved.

Biological measurements have been an integral part of occupational health practice for several decades. They apply to materials or their metabolites in blood or the excretory systems (urine, sweat, tears, *etc*) where reversible biochemical or physiological changes are short of diagnosable disease. Air monitoring<sup>2</sup> and biological monitoring<sup>3</sup> are complementary to each other, with one or the other assuming greater importance depending upon the nature of the material concerned and the route of entry.

The values quoted in the tables are attributed by date and name to the sources<sup>4,5,6,7,8,9,10,11,12,17</sup> from which they are derived. Where original documents have not been available, alternative authoritative sources are quoted.<sup>13,14,15,16</sup> The notations given are unique to the sources quoted. In using any of the values quoted, reference must be made back to the source to ensure use according to the criteria of the authority concerned, which may in fact change while retaining the same numerical values. It is left to the reader to ensure that the most relevant and up-to-date information is used.

There are many gaps in the data for the agents listed in this guide and there are many more agents for which there are no apparent biological indices at all. This lack of information may itself encourage the reader to research the whole subject more

## Introduction

deeply and possibly generate original data on which new indices can be based. However, the information given should facilitate the process of prevention of occupational disease by limiting the dose of toxic agent received by the employee to that below which adverse consequences are most unlikely to occur.

## **2 APPLICATION OF BIOLOGICAL MONITORING DATA**

### **2.1 Diagnostic uses**

In clinical diagnosis, particularly in the absence of specific signs or symptoms, such measurements may tilt the balance of probability from an occupational cause to a natural one, or *vice versa*. This use is wholly clinical, a matter of judgement by the doctor concerned, and the relative importance of such measurements may be quite different from that in other applications of the same data.

### **2.2 Health surveillance**

Many workers are under statutory or company medical surveillance, with the twin aims of ensuring that the worker is fit to continue his or her job, and preventing him or her from being harmed by it. As regards the latter, there are in some cases generally agreed criteria for 'suspension' or 'transfer' to prevent the onset of disease. In other cases, it is a matter of clinical judgement.

### **2.3 Pre-employment**

Apart from laboratory or other tests to pick out individuals who may be particularly susceptible to the working environment, it is often of importance to know the individual's baseline level of materials or their metabolites, so as to identify, if not exclude, applicants with a high body burden from previous employment or from other sources.

### **2.4 Research**

The long-term accumulation of biological data, particularly if it can be associated with direct environmental data, may be invaluable for



## **Application of biological monitoring data**

epidemiological purposes, and the setting of 'standards' for both biological and environmental application. Many large companies do this as a matter of policy, with the Health and Safety Executive collecting such data from smaller companies as well as generating its own.

## **3 LIMITATIONS OF AIR SAMPLING**

### **3.1 Variation**

While there may be some circumstances where the contaminant is uniformly distributed throughout the workroom air, these are very rare indeed. In most cases the exposure of the worker is the sum of his presence in the workroom, his nearness to a source of pollutant, and personal exposure at the process, with the last two being the major contributors in most manual work. This leads to gross differences in exposure between workers nominally doing the same job. A group of workers engaged in essentially the same task may have a five-fold or greater range of exposure when measured by personal sampler over a working shift.

### **3.2 Bioavailability**

With many particulate materials, the absorption by the worker depends on the size (and hence site of deposition in the respiratory tract) and solubility in body fluids of the dust to which the individual is exposed. The size frequency of the dust will vary with the distance of the worker from the source of dust. The solubility will depend on both this size frequency distribution and other physical characteristics of the compound.

### **3.3 Routes of intake**

Other confounding factors which may make reliance upon air sampling alone inadequate, are the possibility of absorption through the intact skin, and the possibility of ingestion, directly or *via* contaminated finger nails. Yet another is the possible contamination of pipes or cigarettes, with later intake of fume during smoking. Biological monitoring is particularly useful where there is

## Limitations of air sampling

uncertainty about exposure because of dependence on respiratory protection and protective clothing.

These limitations of air sampling in the evaluation of risk do not in any way invalidate its use. Indeed, for a great many materials, notably fibrogenic dusts, and many carcinogens, we have as yet no basis for prevention other than air monitoring coupled with observation in the workplace (for ingestion and skin contamination). In these cases, the first observable biological indicators are too often irreversible disease.

## 4 MANAGEMENT OF A BIOLOGICAL MONITORING PROGRAMME

When biological monitoring is part of an overall strategy for controlling toxic hazards in the workplace, it is nearer in concept to environmental monitoring than it is to health surveillance. The principal objective is the detection and control of hazardous concentrations of toxic substances before adverse health effects occur. It needs to be emphasised that biological monitoring is designed to *prevent* and not to *detect* adverse changes.

The data from biological monitoring are of direct use to the occupational hygienist giving a valid measure of the absorption of the offending material in individuals, and invaluable when they give a better measure of risk than is possible by air monitoring. The ideal material is one where uptake and clearance are such that infrequency of sampling or delay in analysis do not prevent feedback of the data for environmental control in time to prevent any adverse trend continuing to unacceptable levels. The use of biological monitoring thus requires knowledge and consideration of a different set of factors compared with environmental monitoring. Among these are the following:

### 4.1 Acute responses

Biological monitoring is of little value in preventing the so-called acute effects caused by agents such as CN, CO, N<sub>2</sub>O, and powerful narcotics since, whereas a peak (10 minutes) may well be reflected in blood or excretion, it is impracticable to use such measurements for control. In practice, the prevention of such effects is a matter of safe working practice, with air sampling

## **Management of a monitoring programme**

necessary initially to establish the level of control, and reliance thereafter on factors such as working discipline, pre-entry checks, fail-safe systems and alarms.

### **4.2 Accumulation in body organs**

Many substances accumulate in one or more body organs (liver, kidney, bone, brain, *etc*), with differing half-lives. Thus the measurement of a material in blood or of its excretion may reflect the long-term past exposure (from weeks to years) of individuals as well as recent absorption. In such cases high levels are not necessarily indicative of environmental conditions at the time of sampling. However, there is normally a contribution from recent exposure, and where this can be identified (as in workers without gross past exposure) the data become valid for occupational hygiene practice.

### **4.3 Variations in metabolism**

There is a handful of well documented classes of chemical compound for which there are clear genetic reasons for an individual's differing metabolism. However, for most chemicals there is a range of ability to metabolise, which may be very wide. In addition, illness or diet (including drinking habits) may complicate metabolic processes, to give different rates of excretion, or even perhaps a different metabolic balance. In practice, these last variables are dealt with on an individual basis, and the data interpreted accordingly.<sup>14</sup>

### **4.4 Medical confidentiality**

There are situations where data relating to an individual's blood or other body fluids are a matter of full medical confidentiality. This applies when the data give a direct indication of the individual's state of health, but not so when the data more truly reflect the level of absorption of a harmful material from the working environment. Over the years, a working position has evolved between practitioners whereby the latter data are routinely available, while the former are closely guarded, although they may both originate in the same laboratory. Thus, individual urinary cadmium data are available to the hygienist in confidence, while the protein and sugar tests normally carried out on the same sample of urine are not.

### **4.5 Non-occupational exposure**

Many toxic substances are present in the non-occupational environment, and they or their metabolites may be present in blood or urine because of this. In the past, when occupational exposures were, on the whole, greater than today, the occupationally increased levels were sufficiently high for there to be no real problem in distinguishing them from unexposed levels. This is not always so today, and the difference has been reduced in some cases to a level where statistical analysis of grouped data is necessary to differentiate between exposed and normal populations. Until more sensitive, specific methods can be evolved, this limits the use of biological monitoring in the very case for which is most appropriate, namely, the individual worker.

### **4.6 Timing of sample**

Depending on the half-life of the material or its metabolite in blood and organs, the timing of the sample may influence the significance of the data. For some substances, the timing is immaterial, there being little change in concentration in body fluids over, say, a week, despite the normal fluctuations in exposure. For others, sampling at the end of a peak exposure, or at the end of the full (five-shift) working cycle may be appropriate. The monitoring protocol must take account of this factor, and it must be strictly followed.

### **4.7 Urinary data**

All data from urine samples should be corrected for specific gravity or to creatinine content, urine samples which are outside the normal limits of these factors being discarded. In some cases, dark bottles or preservatives may be required, and their presence or absence should be stated on report forms.

### **4.8 Analysis and quality assurance**

The analysis of biological samples should be carried out only by laboratories experienced in the analyses required. Their procedures and handling facilities should take into account the possibility that these materials may pose an infective risk to the analyst. The laboratories should follow rigid analytical protocols incorporating throughout each batch an appropriate number of calibration and quality control samples. Where external quality control schemes

## **Management of a monitoring programme**

exist for the material concerned, the laboratory should be required to be a member of, and permit disclosure of, its performance data.

### **4.9 Exhaled air**

For many years now the principle of obtaining an estimate of the blood level of some substances by measuring their level in exhaled air has been accepted, but except in the case of ethyl alcohol, the principle has not developed into routine practice. However, attempts are being made to use it in some companies, and where successful this approach may resolve some of the problems of invasive sampling and medical confidentiality. There will no doubt be increasing interest in the use of breath analysis as suitable portable direct reading instruments become available.

### **4.10 Other measures**

There has always been an interest in the measurement of some substances (arsenic for instance) in hair. In addition, some may be excreted and measured in sweat and tears. To date, these are not mainstream measurements in the practice of occupational hygiene.

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**Substance: ANILINE**

**Disease/Effect:** Causes cyanosis which, in severe cases, can result in anoxia and death, delayed for a few hours after exposure.

**Air Standards:**

Skin absorption is relatively more important than inhalation exposure.

HSC UK : Guidance Value 2 ppm TWA (8h). STEL 5 ppm. Sk (1992)  
ACGIH : TLV 2 ppm TWA (8h) Sk (1991-92)  
DFG : 2 ppm TWA (8h). Peak Limit Value Category II. (IIIB) (1989)  
FINLND : 2 ppm TWA (8h). 4 ppm STEL Sk (ILO 1991)  
FRANCE : 2 ppm TWA (8h) Sk (ILO 1991)  
SWEDEN: 1 ppm TWA (8h). 2 ppm STEL Sk (ILO 1991)  
USSR : 1 ppm TWA (8h). 0.1 mg/m<sup>3</sup> STEL Sk (ILO 1991)

**Blood Standards:**

HSC UK :  
ACGIH : BEI methaemoglobin 1.5% haemoglobin, end of shift (1991-92)  
DFG : BAT aniline released from aniline - haemoglobin conjugate 100 µg/l end of shift or after several shifts (1989)  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**ANILINE**

## ANILINE

### *Urine Standards:*

Based on metabolism to p-aminophenol or free aniline.

HSC UK :  
ACGIH : BEI, p-aminophenol 50 mg/g creatinine end of shift (1991-92)  
DFG : BAT free aniline 1 mg/l end of shift or after several shifts (1989)  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

### *Exhaled Air:*

No data given.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

**Substance: ARSENIC (EXCLUDING ARSINE)**

**Disease/Effect:** Acute gastrointestinal effects after massive ingestion. Chronic effects include peripheral neuritis and cardiovascular changes. Carcinogenic to skin and lung.

**Air Standards:**

Site of action and systemic absorption depends on compound and particle size.

HSC UK : MEL 0.1 mg As/m<sup>3</sup> TWA (8h) (1992) (excluding lead arsenate)  
ACGIH : TLV arsenic and soluble compounds 0.2 mg As/m<sup>3</sup> TWA (8h) (1991-92)  
Arsenic trioxide production (A2) (1991-92)  
DFG : TRK oxides, acids and salts, 0.1 mg As/m<sup>3</sup> TWA (8h) III A1 (1989)  
FINLND : Arsenic and compounds 0.01 mg/m<sup>3</sup> TWA (8h) (ILO 1991)  
FRANCE : Arsenic trioxide 0.2 mg/m<sup>3</sup> TWA (8h) (ILO 1991)  
SWEDEN: Arsenic and compounds 0.03 mg/m<sup>3</sup> TWA (8h) (ILO 1991)  
USSR : Arsenic compounds 0.01 mg/m<sup>3</sup> TWA (8h) 0.04 mg/m<sup>3</sup> STEL (ILO 1991)

**Blood Standards:**

Biodynamics suggest no valid use for blood samples because of too rapid loss from circulation.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**ARSENIC (EXCLUDING ARSINE)**

## ARSENIC (EXCLUDING ARSINE)

### *Urine Standards:*

Non occupational (dietary, particularly some seafoods) intake gives urinary levels of less than 50  $\mu\text{g As/l}$ , but occasionally up to 200  $\mu\text{g As/l}$ . Some is excreted unchanged from its original organic compound which may be differentiated from the 'inorganic excretion' by HPLC analysis.

HSC UK : Quotes others - 0.5 mg inorganic As/ $\text{m}^3$  TWA (8h) results in As (total) 300 nmol/mmol creatinine  
ACGIH : BEI Intent to establish arsenic and soluble compounds including arsine 50  $\mu\text{g As/l}$  creatinine, end of work week (B) (1991-92)  
DFG : EKA 330  $\mu\text{g As/l}$  corresponds to 0.1 mg As trioxide/ $\text{m}^3$  TWA (8h) (1989)  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

### *Exhaled Air:*

No data given

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

***Substance:*** BENZENE

***Disease/Effect:*** Causes narcosis and leukaemia. 80% of dose metabolised to phenol and excreted within two days. 12% excreted in exhaled air.

***Air Standards:***

HSC UK : MEL 5 ppm TWA (8h) (1992)  
ACGIH : TLV 10 ppm TWA (8h) A2 (1991-92). Intended change 0.1ppm TWA (8h) Sk (A1)  
DFG : TRK 5 ppm TWA (8h) Peak Limit Value Category III A1 (1989)  
FINLND : 5 ppm TWA (8h). 10 ppm STEL Sk (ILO 1991)  
FRANCE : 5 ppm TWA (8h) (ILO 1991)  
SWEDEN: 1 ppm TWA (8h). 5 ppm STEL Sk (ILO 1991)  
USSR : 10 ppm TWA (8h). 25 ppm STEL Sk (ILO 1991)

***Blood Standards:***

Useful at above 10 ppm air (1987)

HSC UK :  
ACGIH :  
DFG : EKA 3.8 µg benzene/100 mls equivalent to 4 ppm air (1989)  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**BENZENE**

## BENZENE

### *Urine Standards:*

Based on phenol which is dietary dependent and variable, limiting urinary monitoring at low air levels. Recent work on S-phenylmercapturic acid gives high sensitivity, end of shift level of 45 mg S-PMA/g creatinine, equivalent to 1 ppm TWA (8h) (Stommel, P. *et al* 1989).

HSC UK : Phenol not recommended  
ACGIH : BEI 50 mg phenol/g creatinine end of shift (1991-92) (B, Ns)  
DFG : EKA 45 mg phenol/l equivalent to 6 ppm air (1989)  
FINLND :  
FRANCE : 0.12 ppm benzene pre-shift (1979)  
SWEDEN:  
USSR :

### *Exhaled Air:*

HSC UK :  
ACGIH : BEI 0.08 ppm benzene mixed exhaled, 0.12 ppm end exhaled, prior to next shift (1981-92) (Sq)  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**Substance: BERYLLIUM**

**Disease/Effect:** Acute pulmonary effects from oxide fume and soluble compounds. Delayed chronic pulmonary effects from most compounds except ores. Carcinogenic and systemic effects less well documented, but accepted.

**Air Standards:**

HSC UK : OES 0.002 mg Be/m<sup>3</sup> as total inhalable (Guidance Value 1992, to be reviewed)  
ACGIH : TLV 0.002 µg/m<sup>3</sup> TWA (8h) A2. (1991-92)  
DFG : TRK Metal/alloys 0.005 mg Be/m<sup>3</sup> TWA (8h). STEL compounds 0.002 mg Be/m<sup>3</sup> TWA (8h) Peak Limit Value, Category III (A2) (1989)  
FINLND : Beryllium 0.002 mg/m<sup>3</sup> TWA (8h). 0.006 mg/m<sup>3</sup> STEL (ILO 1991)  
FRANCE : Beryllium 0.002 mg/m<sup>3</sup> TWA (8h). (ILO 1991)  
SWEDEN: Beryllium and compounds 0.002 mg/m<sup>3</sup> TWA (8h) SEN (ILO 1991)  
USSR : Beryllium and compounds 0.001 mg/m<sup>3</sup> STEL (ILO 1991)

**Blood Standards:**

No data given.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**BERYLLIUM**



## BERYLLIUM

### *Urine Standards:*

Analyses pre 1970s by AA were too insensitive - cases of disease with no apparent excretion. Inadequate data to date to permit derivation of standards, but levels above 1  $\mu\text{g Be/l}$  accepted as requiring environmental investigation.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR : 2-3  $\mu\text{g Be/l}$  (Ho and Dillon 1987)

### *Exhaled Air:*

No data given

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

## **Substance: CADMIUM**

**Disease/Effect:** Delayed pulmonary oedema from extreme fume exposure. Emphysema from long-term exposure to fume. Renal tubular damage and dysfunction (low molecular weight proteinuria) may occur from long-term exposure giving renal accumulation.

### **Air Standards:**

Site of action depends on particle size, solubility and reactivity of compound (eg fresh fume compared with acid washed pigments).

HSC UK : MEL fume 0.05 mg Cd/m<sup>3</sup> STEL. Sulphide pigments as respirable dust 0.04 mg Cd/m<sup>3</sup> TWA (8h) Fume and other compounds 0.05 mg Cd/m<sup>3</sup> TWA (8h) (1992)

ACGIH : TLV Cadmium dusts and salts (0.05) mg Cd/m<sup>3</sup> TWA (8h) (1991-92)

Cadmium oxide fume (0.05) mg Cd/m<sup>3</sup> (C) TWA (8h) (1991-92)

Cadmium oxide production (0.05) mg Cd/m<sup>3</sup> TWA (8h) (1991-92)

Intended change, cadmium and compounds as Cd, 0.01 mg/m<sup>3</sup> TWA (8h) A2, total dust.

Respirable dust 0.002 mg/m<sup>3</sup> TWA (8h) A2

DFG : No TRKs given but classified as Peak Limit Value, Category III A2 (1989)

FINLND : Cadmium and compounds 0.02 mg/m<sup>3</sup> TWA (8h) CdO fume 0.01 mg/m<sup>3</sup> TWA (8h) (ILO 1991)

FRANCE : Cadmium oxide 0.05 mg/m<sup>3</sup> TWA (8h) CdO fume 0.05 mg/m<sup>3</sup> STEL (ILO 1991)

SWEDEN: Cadmium and compounds inhalable dust 0.05 mg/m<sup>3</sup> TWA (8h). Respirable dust 0.02 mg/m<sup>3</sup> TWA (8h) (ILO 1991)

USSR : Cadmium and compounds 0.01 mg/m<sup>3</sup> TWA (8h). 0.05 mg/m<sup>3</sup> STEL (ILO 1991)

### **Blood Standards:**

Relation of current intake to past (stored) cadmium blurred. Input from smoking (up to 0.3 µg Cd/100mls).

HSC UK : Environmental action at 1.0 µg Cd/100 mls. (Guidance Note EH1 1986)

ACGIH : BEI 10 µg Cd/l (1991-92). Intent to change to 5 µg Cd/l

DFG : BAT 1.5 µg Cd/100 mls (1989)

**CADMIUM**

## CADMIUM

### *Urine Standards:*

As for blood, relationship between current and past intakes blurred. Low molecular weight protein and B2 microglobulin as part of medical surveillance.

HSC UK : Medical intervention above 10  $\mu\text{g}$  Cd/g creatinine. (Guidance Note EH1 1986)

ACGIH : 10  $\mu\text{g/g}$  creatinine (B) (1991-92). Intent to establish 5  $\mu\text{g}$  Cd/g creatinine (B)

DFG : BAT 15  $\mu\text{g/l}$  (1989)

FINLND :

FRANCE :

SWEDEN:

USSR :

### *Exhaled Air:*

No data given

HSC UK :

ACGIH :

DFG :

FINLND :

FRANCE :

SWEDEN:

USSR :

***Substance:* CARBON DISULPHIDE**

***Disease/Effect:*** Systemic and acute poison. Up to 30% excreted in exhaled air. Urinary metabolite TCCA (2-thiothiazolidine-4-carboxylic acid) related to exposure.

***Air Standards:***

Readily absorbed through intact skin.

HSC UK : MEL 10 ppm TWA (8h) Sk. (1992)  
ACGIH : TLV 10 ppm TWA (8h) Sk. (1991-92)  
DFG : MAK 10 ppm TWA (8h) Peak Limit Value Category II. (1989)  
FINLND : 10 ppm TWA (8h). 20 ppm STEL Sk (ILO 1991)  
FRANCE : 10 ppm TWA (8h). 25 ppm STEL (ILO 1991)  
SWEDEN: 5 ppm TWA (8h). 8 ppm STEL Sk (ILO 1991)  
USSR : 10 ppm TWA (8h). 1 mg/m<sup>3</sup> STEL (ILO 1991)

***Blood Standards:***

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**CARBON DISULPHIDE**

## CARBON DISULPHIDE

### *Urine Standards:*

Based on 2-thiothiazolidine-4-carboxylic acid

HSC UK : 4  $\mu$ mol TTCA/mmol creatinine equivalent to 10 ppm air TWA (8h) (1987)  
ACGIH : BEI 5 mg/g creatinine TTCA, end of shift (1991-92)  
DFG : BAT 8 mg/l TTCA end of shift (1989)  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

### *Exhaled Air:*

Measurable, but speed of elimination from blood makes interpretation difficult.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**Substance:** CARBON MONOXIDE

**Disease/Effect:** Forms carboxyhaemoglobin (CoHb) to block oxygen uptake by blood.

**Air Standards:**

Many excessive exposures caused by pockets of gas, requiring control by pre-entry certification based on measurement.

HSC UK : OES 50 ppm TWA (8h). STEL 300 ppm (1992)  
ACGIH : TLV (50 ppm) TWA (8h). STEL (400) ppm (1991-92). Intended change 25 ppm TWA (8h)  
DFG : MAK 30 ppm TWA (8h) Peak Limit Value Category II (1989)  
FINLND : 30 ppm TWA (8h). 75 ppm STEL (ILO 1991)  
FRANCE : 50 ppm TWA (8h) (ILO 1991)  
SWEDEN: 35 ppm TWA (8h). 100 ppm STEL (ILO 1991)  
USSR : 50 ppm TWA (8h). 20 ppm STEL (ILO 1991)

**Blood Standards:**

Smokers have 5-10% CoHb. CoHb has half life of 3-5 h.

HSC UK :  
ACGIH : BEI. CoHb less than 8% haemoglobin, end of shift (1991-92). Notice of intent to establish carboxyhaemoglobin 3.5% of haemoglobin  
DFG : BAT CoHb 5% end exposure or end of shift (1989)  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**CARBON MONOXIDE**

## CARBON MONOXIDE

### *Urine Standards:*

No data given.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

### *Exhaled Air:*

HSC UK :  
ACGIH : BEI CO in end exhaled air, less than 40 ppm, end of shift(1991-92). Notice of intent to establish  
CO in end exhaled air 20 ppm end of shift.  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

**Substance: CHLOROBENZENE**

**Disease/Effect:** Narcotic with possible liver effects

**Air Standards:**

HSC UK : OES 50 ppm TWA (8h) (1992)  
ACGIH : TLV 10 ppm TWA (8h) (1991-92)  
DFG : MAK 50 ppm TWA (8h) Peak Limit Value Category II (1989)  
FINLND : 50 ppm TWA (8h). 75 ppm STEL (ILO 1991)  
FRANCE : 75 ppm TWA (8h). (ILO 1991)  
SWEDEN:  
USSR : 75 ppm TWA (8h). 100 mg/m<sup>3</sup> STEL (ILO 1991)

**Blood Standards:**

No data given.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**CHLOROBENZENE**



## CHLOROBENZENE

### *Urine Standards:*

Based on metabolism to 4-chlorocatechol and p-chlorophenol.

HSC UK :  
ACGIH : BEI Notice of intent to establish, total 4-chlorocatechol 150 mg/g creatinine. Total p-chlorophenol  
25 mg/g creatinine, end of shift (1991-92)  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

### *Exhaled Air:*

No data given.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

***Substance: COBALT***

***Disease/Effect:*** A possible cause of 'hard metal' pneumoconiosis in tungsten carbide manufacture. Allergenic.  
Query carcinogenic.

***Air Standards:***

Particle size selection based on toxicity and fibrogenicity.

HSC UK : 0.1 mg Co/m<sup>3</sup> TWA (8h) (Guidance value 1992, to be reviewed)  
ACGIH : TLV Metal dust and fume 0.05 mg Co/m<sup>3</sup> TWA (8h) (1991-92)  
Cobalt carbonyl and hydrocarbonyl 0.1 mg Co/m<sup>3</sup> TWA (8h) (1991-92)  
DFG : TRK 0.5 mg Co/m<sup>3</sup> TWA (8h) Peak Limit Value, Category III (A2) (1989)  
FINLND : Cobalt dust and fume 0.05 mg/m<sup>3</sup> TWA (8h) Sk (ILO 1991)  
FRANCE :  
SWEDEN: Cobalt and compounds 0.05 mg/m<sup>3</sup> TWA (8h) SEN (ILO 1991)  
USSR : Cobalt dust and fume 0.5 mg/m<sup>3</sup> STEL (ILO 1991)

***Blood Standards:***

HSC UK :  
ACGIH :  
DFG : EKA 2.5 µg/l corresponds to 0.05 mg Co/m<sup>3</sup> (1989)  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**COBALT**

## COBALT

### *Urine Standards:*

Increase during working day: Rapid overnight but incomplete excretion.

HSC UK : 'Normal' less than 3 nmol/mmol creatinine. Excretion 100 times normal. 0.1 mg Co/m<sup>3</sup> TWA (8h) (1987)

ACGIH :

DFG : EKA 300 µg Co/l corresponds to 0.5 mg Co/m<sup>3</sup> TWA (8h) (1989)

FINLND :

FRANCE :

SWEDEN:

USSR :

### *Exhaled Air:*

No data given

HSC UK :

ACGIH :

DFG :

FINLND :

FRANCE :

SWEDEN:

USSR :

**Substance: DIMETHYLFORMAMIDE**

**Disease/Effect:** Irritant, cardiovascular effects and intolerance to alcohol. Metabolised to N monomethyl formamide (half life in hours). Some excreted as mercapturates, suggesting carcinogenic potential.

**Air Standards:**

Rapid intake through intact skin.

HSC UK : OES 10 ppm TWA (8h). STEL 20 ppm Sk (1992)  
ACGIH : TLV 10 ppm TWA (8h) (1991-92)  
DFG : MAK 20 ppm Peak Limit Value Category II (1989)  
FINLND : 10 ppm TWA (8h). 20 ppm STEL Sk (ILO 1991)  
FRANCE : 10 ppm TWA (8h) Sk (ILO 1991)  
SWEDEN: 10 ppm TWA (8h). 15 ppm STEL Sk (ILO 1991)  
USSR : 10 ppm TWA (8h). 10 mg/m<sup>3</sup> STEL Sk (ILO 1991)

**Blood Standards:**

No data.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**DIMETHYLFORMAMIDE**

## DIMETHYLFORMAMIDE

### *Urine Standards:*

Based on metabolism to N methylformamide. Affected by alcohol.

HSC UK :  
ACGIH : BEI 40 mg/g creatinine end of shift, as N methylformamide (1991-92)  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

### *Exhaled Air:*

Detectable in exhaled air (0.1 - 4 ppm) when not detectable in blood. (Ho and Dillon 1987)

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**Substance: ETHYL BENZENE**

**Disease/Effect:** Irritation followed by narcotic effects

**Air Standards:**

HSC UK : OES 100 ppm TWA (8h). STEL 125 ppm (1992)  
ACGIH : TLV 100 ppm TWA (8h). STEL 125 ppm (1991-92)  
DFG : MAK 100 ppm TWA (8h). Peak Limit Value Category 1 (1989)  
FINLND : 100 ppm TWA (8h). 150 ppm STEL (ILO 1991)  
FRANCE : 100 ppm TWA (8h) (ILO 1991)  
SWEDEN: 50 ppm TWA (8h). 100 ppm STEL (ILO 1991)  
USSR : 100 ppm TWA (8h). 50 mg/m<sup>3</sup> STEL (ILO 1991)

**Blood Standards:**

No data given.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**ETHYL BENZENE**

## ETHYL BENZENE

### *Urine Standards:*

Based on metabolism to mandelic acid.

HSC UK :

ACGIH : BEI mandelic acid 1.5 g/g creatinine, end of shift, end of work week (1991-92)

DFG :

FINLND :

FRANCE :

SWEDEN:

USSR :

### *Exhaled Air:*

No data given.

HSC UK :

ACGIH :

DFG :

FINLND :

FRANCE :

SWEDEN:

USSR :

**Substance: FLUORINE AND FLUORIDES**

**Disease/Effect:** Gaseous fluorine gives serious acute effects. Gaseous hydrogen fluoride grossly irritant. All fluorides absorbed in excessive amounts can lead to osteofluorosis.

**Air Standards:**

Need for safe work procedures for gaseous compounds overrides measurement of air levels.

Some compounds readily adsorbed through g.i. tract after direct or post inhalation ingestion.

HSC UK : OES HF 2.5 mg F/m<sup>3</sup> STEL. F 1.5mg F/m<sup>3</sup> STEL. Fluorides 2.5 mg F/m<sup>3</sup> TWA (8h) (1992)  
ACGIH : TLV Fluorine 1 ppm TWA (8h). STEL 2 ppm. (1991-92). Fluoride 2.5 mg F/m<sup>3</sup> TWA (8h) (1991-92)  
DFG : MAK Fluorine 0.1 ppm Peak Limit Value, Category I TWA (8h). Fluorides and HF 2.5 mg F/m<sup>3</sup> TWA (8h) (1989)  
FINLND : Fluorides 2.5 mg/m<sup>3</sup> TWA (8h). Fluorine 2.6 mg/m<sup>3</sup> STEL (ILO 1991)  
FRANCE : Fluorides 2.5 mg/m<sup>3</sup> TWA (8h). Fluorine 2.0 mg/m<sup>3</sup> STEL (ILO 1991)  
SWEDEN: Fluorides 2.0 mg/m<sup>3</sup> TWA (8h). Fluorine 0.2 mg/m<sup>3</sup> TWA (8h). 0.5 mg/m<sup>3</sup> STEL (ILO 1991)  
USSR :

**Blood Standards:**

Very short half life of circulating fluoride limits value. No standards.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :

**FLUORINE AND FLUORIDES**



## FLUORINE AND FLUORIDES

### *Urine Standards:*

'Normal' excretion depends on diet and regional influences. Pre-start of working week data needed for comparison with end shift/end cycle levels.

HSC UK : Quotes NIOSH pre- shift 2.3 mg F/l, post shift 4 mg F/l (1987)  
ACGIH : BEI 13 mg F/g creatinine before shift, 10 mg F/g creatinine after shift (1989/90) (B, Ns)  
DFG : BAT 7 mg F/g creatinine end of exposure, 4 mg F/g creatinine start of next shift (1989)  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

### *Exhaled Air:*

No data given

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

**Substance:** FURFURALDEHYDE

**Disease/Effect:** Irritation with possible neurotoxic effects

**Air Standards:**

HSC UK : OES 2 ppm TWA (8h). STEL 10 ppm. Sk (1992)  
ACGIH : TLV 2 ppm TWA (8h). Sk (1991-92)  
DFG : MAK 5 ppm TWA (8h) (1989)  
FINLND : 5 ppm TWA (8h). 10 ppm STEL Sk (ILO 1991)  
FRANCE : 2 ppm STEL (ILO 1991)  
SWEDEN: 5 ppm TWA (8h). 10 ppm STEL Sk (ILO 1991)  
USSR : 2.5 ppm TWA (8h). 10 mg/m<sup>3</sup> STEL Sk (ILO 1991)

**Blood Standards:**

No data given.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**FURFURALDEHYDE**

## FURFURALDEHYDE

### *Urine Standards:*

Based on metabolism to Furoic acid.

HSC UK :  
ACGIH : BEI furoic acid 200 mg/g creatinine, end of shift (1991-92)  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

### *Exhaled Air:*

No data given.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**Substance: HEXAVALENT CHROMIUM Cr VI**

**Disease/Effect:** Skin and nasal ulceration by mists and dusts. Lung cancer associated with some compounds, particularly those of low solubility. Essential trace element in humans.

**Air Standards:**

Skin contact relevant to percutaneous effects. Large airborne particles trapped in nose cause perforation. Effects depend on solubility. Sometimes mixed with trivalent chromium compounds, requiring differential analysis.

HSC UK : MEL 0.05 mg/m<sup>3</sup> TWA (8h) (1992)

ACGIH : TLV Certain water insoluble compounds 0.05 mg/m<sup>3</sup> TWA (8h) (A1) (1991-92)  
Chromite ore processing (chromate) 0.05 mg/m<sup>3</sup> (A1) (1991-92)

DFG : TRK chromium compounds and fume from arc welding by hand with coated electrodes 0.2 mg/m<sup>3</sup> TWA (8h). Others 0.1 mg/m<sup>3</sup> TWA (8h) Peak Limit Value, Category III (A2) (1989)

FINLND : 0.05 mg/m<sup>3</sup> TWA (8h) (ILO 1991)

FRANCE : Chromium trioxide 0.05 mg/m<sup>3</sup> TWA (8h). 0.01 mg/m<sup>3</sup> SEN (ILO 1991)

SWEDEN: Chromium compounds 0.5 mg/m<sup>3</sup> TWA (8h). Chromic acid 0.02 mg/m<sup>3</sup> TWA (8h). 0.06 mg/m<sup>3</sup> STEL Sk (ILO 1991)

USSR : Chromates 0.01 mg/m<sup>3</sup> STEL (ILO 1991)

**Blood Standards:**

Analyses inaccurate until 1980s, usually too high. Normal blood less than 0.1 µg Cr/100 mls.

DFG : EKA 3.5 µg Cr/100 mls corresponds to 0.1 mg CrO<sub>3</sub>/m<sup>3</sup> TWA (8h) (1989)

OTHER : IARC unexposed, 0-20 mg/100 mls. Chromate workers up to 580 mg/100 mls.  
Chromite workers 0-2 mg/100 mls (1980).

**HEXAVALENT CHROMIUM Cr VI**

## HEXAVALENT CHROMIUM Cr VI

### *Urine Standards:*

Analyses questionable until 1980s. Normal urine less than  $1\mu\text{g Cr/l}$ . With soluble compounds, rise during day, and rapid but incomplete excretion overnight. Probably depends upon solubility in body fluids.

HSC UK : Sample beginning and end of work shift late in week (1987)

ACGIH : BEI Chromium VI, water soluble fume, total Cr in urine  $10\mu\text{g/g creatinine}$  increase during shift,  $30\mu\text{g/g creatinine}$  end of shift end of work week (1991-92) (B)

DFG : EKA  $40\mu\text{g/l}$  corresponds to  $0.1\text{ mg CrO}_3/\text{m}^3$  TWA (8h) (1989)

FINLND :  $30\mu\text{g/l}$ . (Ho and Dillon 1987)

FRANCE :

SWEDEN:

USSR :

OTHER : Italy  $50\mu\text{g Cr/l}$ . (Ho and Dillon 1987). IARC unexposed 0-160 mg Cr/l. Chromate workers 0-780 mg Cr/l. Chromite workers 0 mg Cr/l.  
Literature sources variable and therefore questionable (1980).

### *Exhaled Air:*

No data given

HSC UK :

ACGIH :

DFG :

FINLND :

FRANCE :

SWEDEN:

**Substance: LEAD - INORGANIC**

**Disease/Effect:** Systemic poison causing acute and chronic effects. Symptoms range from constipation (acute), peripheral motor neuropathy and anaemia (chronic).

**Air Standards:**

Absorption *via* the lung and gastrointestinal tract is dependant on particle size and solubility

HSC UK : Air Standard (except organic lead), 0.15 mg/m<sup>3</sup> TWA (8h) (Control of Lead at Work Regs. 1985)  
ACGIH : TLV 0.15 mg/m<sup>3</sup> TWA (8h) (1991-92)  
DFG : MAK 0.1 mg/m<sup>3</sup> TWA (8h) Peak Limit Value Category III (1989)  
FINLND : 0.1 mg/m<sup>3</sup> TWA (8h) (ILO 1991)  
FRANCE : 0.15 mg/m<sup>3</sup> (8h) (ILO 1991)  
SWEDEN: 0.1 mg/m<sup>3</sup> TWA (8h) inhalable; 0.05 mg/m<sup>3</sup> respirable (ILO 1991)  
USSR : 0.005 mg/m<sup>3</sup> TWA (8h), 0.01 mg/m<sup>3</sup> STEL ILO (1991)  
EEC : Limit Value 0.15 mg/m<sup>3</sup> TWA (40h) (1985)

**Blood Standards:**

Based on lead in blood and zinc protoporphyrin

HSC UK : Pb 70 µg/100 mls (male); Pb 40 µg/100 mls (female). (Control of Lead at Work Regs. 1985)  
ACGIH : BEI. Pb 50 µg/100 mls. Zinc protoporphyrin 25 µg/100 mls erythrocytes or 100 mg/100 mls blood (1991-92)  
DFG : BAT Pb 70 µg/100 mls (male) and Pb 30 µg/100 mls (female). No fixed sampling time  
FINLND :  
FRANCE :

**LEAD - INORGANIC**

## LEAD - INORGANIC

### *Urine Standards:*

Based on lead in urine and 2 amino laevulinic acid.

HSC UK :  
ACGIH : BEI 150  $\mu\text{g/g}$  creatinine (1991-92)  
DFG : BAT 2 aminolaevulinic acid 15 mg/l (male) and 6 mg/l (female). No fixed sampling time  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

### *Exhaled Air:*

No data given.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**Substance: LEAD - ORGANIC**

**Disease/Effect:** Affects central nervous system causing insomnia and other psychiatric symptoms

**Air Standards:**

Readily absorbed through the skin but vapour pressure high enough for risk by inhalation

- HSC UK : Air Standard. Tetraethyl lead as Pb 0.1 mg/m<sup>3</sup> TWA (8h) Sk. Tetramethyl lead as Pb 0.15 mg/m<sup>3</sup> TWA (8h) Sk. (Control of Lead at Work Regs. 1985)
- ACGIH : TLV. Tetramethyl lead as Pb 0.1 mg/m<sup>3</sup> TWA (8h). Sk (1991-92). Tetramethyl lead as Pb 0.15 mg/m<sup>3</sup> TWA (8h) Sk (1991-92)
- DFG : MAK tetraethyl and tetramethyl lead as Pb 0.075 mg/m<sup>3</sup> TWA (8h) (1989)
- FINLND : As Pb 0.075 mg/m<sup>3</sup> TWA (8h). 0.23 mg/m<sup>3</sup> STEL. Sk (ILO 1991)
- FRANCE : As Pb 0.1 mg/m<sup>3</sup> STEL. TWA (8h). 0.15 mg/m<sup>3</sup> TML TWA (8h) Sk (ILO 1991)
- SWEDEN: As Pb 0.05 mg/m<sup>3</sup> TWA (8h). 0.2 mg/m<sup>3</sup> STEL Sk (ILO 1991)
- USSR : As Pb 0.005 mg/m<sup>3</sup> STEL Sk (ILO 1991)

**Blood Standards:**

- HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**LEAD - ORGANIC**



## LEAD - ORGANIC

### *Urine Standards:*

Based on lead in urine.

HSC UK : Pb 150  $\mu\text{g/litre}$  (Control of Lead at Work Regs. 1985)

ACGIH :

DFG :

FINLND :

FRANCE :

SWEDEN:

USSR :

### *Exhaled Air:*

No data given.

HSC UK :

ACGIH :

DFG :

FINLND :

FRANCE :

SWEDEN:

USSR :

**Substance: MANGANESE**

**Disease/Effect:** Essential trace element. Long-term high exposure may lead to 'manganism' - similar to Parkinsonism. Metal fume fever from alloying and similar processes.

**Air Standards:**

Very size, solubility, compound dependent, giving variable systemic absorption.

- HSC UK : OES 5 mg Mn/m<sup>3</sup> (TWA (8h) for dusts except for Mn<sub>3</sub>O<sub>4</sub>. OES Mn<sub>3</sub>O<sub>4</sub> 1 mg/m<sup>3</sup> TWA (8h). (1992)  
Fume 1 mg Mn/m<sup>3</sup> TWA (8h), STEL 3 mg/m<sup>3</sup> (1991)
- ACGIH : TLV Dust and inorganic compounds 5 mg/m<sup>3</sup> TWA (8h) (1991-92)  
Fume 1 mg/m<sup>3</sup> TWA (8h). STEL 3 mg/m<sup>3</sup>. (1991-92)
- DFG : MAK 5 mg Mn/m<sup>3</sup> TWA (8h) except for manganous-manganic oxide of 1 mg Mn/m<sup>3</sup> TWA (8h) (1989)
- FINLND : Manganese compounds inorganic 2.5 mg/m<sup>3</sup> TWA (8h). Fume 1 mg/m<sup>3</sup> TWA (8h) (ILO 1991)
- FRANCE : Fume 1 mg/m<sup>3</sup> TWA (8h). Mn<sub>3</sub>O<sub>4</sub> 1 mg/m<sup>3</sup> TWA (8h) (ILO 1991)
- SWEDEN: Manganese compounds 2.5 mg/m<sup>3</sup> TWA (8h). 5 mg/m<sup>3</sup> STEL.  
Fume 1 mg/m<sup>3</sup> TWA (8h). 2.5 mg/m<sup>3</sup> STEL (ILO 1991)
- USSR : Fume 0.2 mg/m<sup>3</sup> STEL (ILO 1991)

**Blood Standards:**

'Normal' up to 1.5 µg Mn/100 mls. No valid standards to date.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :

**MANGANESE**

## MANGANESE

### *Urine Standards:*

'Normal' up to 3  $\mu\text{g}$  Mn/l. As for blood.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

### *Exhaled Air:*

No data given.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

**Substance: MERCURY - INORGANIC COMPOUNDS**

**Disease/Effect:** Affects central nervous system and causes progressive accumulation in liver, kidney, brain and other tissues until equilibrium is reached. Half life in weeks.

**Air Standards:**

Absorption influenced by particle size and solubility. Vapour pressure of metal very temperature dependent above 17°C, to give rapidly changing levels of vapour in air from spillages.

HSC UK : OES 0.05 mg Hg/m<sup>3</sup> TWA (8h). STEL 0.15 mg/m<sup>3</sup> (1992)  
ACGIH : TLV vapour 0.05 mg/m<sup>3</sup> TWA (8h). Compounds 0.1 mg/m<sup>3</sup> TWA (8h) (Sk)  
DFG : MAK 0.1 mg/m<sup>3</sup> TWA (8h) Peak Limit Value Category III (1989)  
FINLND : 0.05 mg/m<sup>3</sup> TWA (8h) (ILO 1991)  
FRANCE : 0.1 mg/m<sup>3</sup> TWA (8h) Sk (ILO 1991)  
SWEDEN: 0.05 mg/m<sup>3</sup> TWA (8h) Sk (ILO 1991)  
USSR : 0.05 mg/m<sup>3</sup> TWA (8h). 0.2 mg/m<sup>3</sup> STEL Sk (ILO 1991)

**Blood Standards:**

End of shift sampling to give recent past exposure. Rapid loss thereafter.

HSC UK : Suggests 150 nmol/l corresponds with 0.05 mg Hg/m<sup>3</sup> TWA (8h) (1987)  
ACGIH : BEI intent to establish 15 µg/l end shift end of work week (1991-92) (B)  
DFG : BAT 5 µg/100 mls (1989)  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**MERCURY - INORGANIC COMPOUNDS**

## MERCURY - INORGANIC COMPOUNDS

### *Urine Standards:*

Normal less than 2  $\mu\text{g}$  Hg/l. Excretion reflects current and past exposure 2 - 4 months

HSC UK : 120 nmol Hg/mmol creatinine as upper limit (1987)

ACGIH : BEI intent to establish 35  $\mu\text{g/g}$  creatinine pre-shift (1991-92) (B)

DFG : BAT 200  $\mu\text{g/l}$  (1989)

FINLND :

FRANCE :

SWEDEN:

USSR :

### *Exhaled Air:*

No data given.

HSC UK :

ACGIH :

DFG :

FINLND :

FRANCE :

SWEDEN:

USSR :

**Substance: MERCURY - ORGANIC COMPOUNDS**

**Disease/Effect:** Effects range from severe permanent disability from, *eg*, methyl mercury chloride, to only slight effects from, *eg*, phenyl mercuric acetate.

**Air Standards:**

Most are lipid soluble, with skin contact presenting often the major source of systemic intake.

HSC UK : OES mercury alkyls 0.01 mg/m<sup>3</sup> TWA (8h) Sk. Other organic compounds 0.05 mg/m<sup>3</sup> TWA (8h) Sk (1992)

ACGIH : TLV mercury alkyl compounds 0.01 mg/m<sup>3</sup> TWA (8h). STEL 0.03 mg/m<sup>3</sup>. Sk (1991-92)  
Aryl compounds 0.1 mg/m<sup>3</sup> TWA (8h) Sk (1991-92)

DFG : MAK 0.01 mg Hg/m<sup>3</sup> TWA (8h) Peak Limit Value Category III. (1989)

FINLND : Alkyl compounds 0.01 mg/m<sup>3</sup> TWA (8h) Sk (ILO 1991)

FRANCE : Alkyl compounds 0.01 mg/m<sup>3</sup> TWA (8h) Sk. Aryl compounds 0.1 mg/m<sup>3</sup> TWA (8h) Sk (ILO 1991)

SWEDEN: Alkyl compounds 0.01 mg/m<sup>3</sup> TWA (8h) Sk (ILO 1991)

USSR :

**Blood Standards:**

It is difficult to conceive a single value for all organic mercury compounds.

HSC UK :

ACGIH :

DFG : BAT 10 µg/100 mls (1989)

FINLND :

**MERCURY - ORGANIC COMPOUNDS**

## MERCURY - ORGANIC COMPOUNDS

### *Urine Standards:*

As with blood, a 'general' level is difficult to define. For many years a limit of 10  $\mu\text{g/l}$  was used for the seed dressing Panogen (methyl mercury dicyandiamide) and this crept into general use. It has no relevance to the seed dressings such as phenyl mercuric acetate.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

### *Exhaled Air:*

No data given

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

**Substance:** METHANOL

**Disease/Effect:** Damage to optic nerve due to metabolism to formic acid and formaldehyde

**Air Standards:**

Absorbed through inhalation, skin and ingestion

HSC UK : OES 200 ppm TWA (8h). STEL 250 ppm. Sk (1992)  
ACGIH : TLV 200 ppm TWA (8h). STEL 250 ppm. Sk (1991-92)  
DFG : MAK 200 ppm TWA (8h). Peak Limit Value Category II (1989)  
FINLND : 200 ppm TWA (8h). 250 ppm STEL Sk (ILO 1991)  
FRANCE : 200 ppm TWA (8h). 1000 ppm STEL (ILO 1991)  
SWEDEN: 200 ppm TWA (8h). 250 ppm STEL Sk (ILO 1991)  
USSR : 200 ppm TWA (8h). 5 mg/m<sup>3</sup> STEL Sk (ILO 1991)  
EEC : Indicative Limit Value 200 ppm TWA (8h) (1991)

**Blood Standards:**

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**METHANOL**



## METHANOL

### *Urine Standards:*

Based on methanol and metabolism to formic acid.

HSC UK :

ACGIH : BEI methanol 15 mg/l end of shift (1991-92). Formic acid 80 mg/g creatinine, before shift at end of work week (1991-92)

DFG : BAT methanol, 130 mg/l end of shift (1989)

FINLND :

FRANCE :

SWEDEN:

USSR :

### *Exhaled Air:*

No data given:

HSC UK :

ACGIH :

DFG :

FINLND :

FRANCE :

SWEDEN:

USSR :

**Substance:** METHYL ETHYL KETONE

**Disease/Effect:** Irritant and central nervous system depressant. Widely distributed *via* blood with very short tissue half life, of the order of 30 mins.

**Air Standards:**

HSC UK : OES 200 ppm TWA (8h). STEL 300 ppm (1992)  
ACGIH : TLV 200 ppm TWA (8h). STEL 300 ppm (1991-92)  
DFG : MAK 200 ppm TWA (8h). Peak Limit Value Category II (1989)  
FINLND : 150 ppm TWA (8h). 190 ppm STEL (ILO 1991)  
FRANCE : 200 ppm TWA (8h) (ILO 1991)  
SWEDEN: 50 ppm TWA (8h). 100 ppm STEL (ILO 1991)  
USSR : 200 ppm TWA (8h). 200 mg/m<sup>3</sup> STEL (ILO 1991)

**Blood Standards:**

Short half life (30 mins) relating to recent exposure only

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :  
Other : 100 ppm TWA (8h) = approx 1.0 µg/ml (Ho and Dillon 1987)

**METHYL ETHYL KETONE**

## METHYL ETHYL KETONE

### *Urine Standards:*

Based on MEK in urine.

HSC UK :  
ACGIH : BEI, MEK 2 mg/l, end of shift (1991-92)  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

### *Exhaled Air:*

Reaches equilibrium in about 2 h.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :  
Other : 1200 ppm TWA (8h) = approx. 4 ppm in exhaled air (Ho and Dillon 1987)

**Substance:** METHYL ISOBUTYL KETONE.

**Disease/Effect:** Upper respiratory tract irritant. Can cause dermatitis. High exposures associated with peripheral neuropathy.

**Air Standards:**

HSC UK : OES 50 ppm TWA (8h). STEL 75 ppm Sk (1992)  
ACGIH : TLV 50 ppm TWA (8h). STEL 75 ppm (1991-92)  
DFG : MAK 100 ppm TWA (8h) Peak Limit Value Category II (1989)  
FINLND : 50 ppm TWA (8h). 75 ppm STEL Sk (ILO 1991)  
FRANCE : 50 ppm TWA (8h) (ILO 1991)  
SWEDEN: 25 ppm TWA (8h). 50 ppm STEL (ILO 1991)  
USSR : 50 ppm TWA (8h). 5 mg/m<sup>3</sup> STEL (ILO 1991)

**Blood Standards:**

No data given.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**METHYL ISOBUTYL KETONE**

## METHYL ISOBUTYL KETONE

### *Urine Standards:*

Based on excretion of MIBK itself.

HSC UK :  
ACGIH : BEI. Notice of intent to establish, MIBK 2 mg/l, end of shift (1991-92)  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

### *Exhaled Air:*

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

**Substance: N HEXANE**

**Disease/Effect:** Peripheral neuropathy possibly due to the metabolite 2.5 hexanedione.

**Air Standards:**

Present as part of 'technical' hexane, and also often found in use with other industrial solvents.

HSC UK : OES 20 ppm TWA (8h) (1992)  
ACGIH : TLV 50 ppm TWA (8h) (1991-92)  
DFG : MAK 50 ppm TWA (8h) Peak Limit Value Category II (1989)  
FINLND : 50 ppm TWA (8h). 150 ppm STEL (ILO 1991)  
FRANCE : 50 ppm TWA (8h) (ILO 1991)  
SWEDEN: 25 ppm TWA (8h). 50 ppm STEL (ILO 1991)  
USSR : 40 ppm TWA (8h). 300 mg/m<sup>3</sup> STEL (ILO 1991)

**Blood Standards:**

No data given.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**N HEXANE**

## N HEXANE

### *Urine Standards:*

Based upon 2,5 hexanedione, with half life of about 10 hours.

HSC UK :  
ACGIH : BEI 5 mg/g creatinine, 2,5 hexanedione, end of shift (1991-92) (Ns)  
DFG : BAT 9 mg/l of 2,5 hexanedione plus 4,5 dihydroxy 2 hexanone, end of shift. 1989)  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

### *Exhaled Air:*

HSC UK :  
ACGIH : BEI 40 ppm n hexane in end exhaled air, end of shift (1989/90) (Sq)  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

**Substance: NICKEL**

**Disease/Effect:** Skin sensitisation and dermatitis. Nasal cancer from nickel carbonyl exposure (now essentially historical). Lung cancer with some compounds.

**Air Standards:**

Intake influenced by particle size and solubility. Particular problems with the compounds found in nickel smelting.

HSC UK : MEL nickel and water insoluble compounds 0.5 mg/m<sup>3</sup> TWA (8h). Soluble compounds 0.1 mg/m<sup>3</sup> TWA (8h).

Carbonyl OES STEL 0.24 mg/m<sup>3</sup> (1992)

ACGIH : TLV nickel, insoluble and soluble compounds, 0.05 mg Ni/m<sup>3</sup> TWA (8h) A1 (1991-92)

Carbonyl 0.12 mg/m<sup>3</sup> TWA (8h) (1991-92). Intended change, delete listing

DFG : Sulphide roasting, fume and dust (1 mg/m<sup>3</sup>) TWA (8h) (1991-92). Intended change, delete listing

TRK, nickel compounds 0.5 mg/m<sup>3</sup> TWA (8h) except for 'inspirable' droplets 0.05 mg/m<sup>3</sup> TWA (8h) Peak Limit Value Category III (A1). Carbonyl 0.7 mg/m<sup>3</sup> TWA (8h) III (A2) (1989)

FINLND : Carbonyl 0.007 mg/m<sup>3</sup> TWA (8h) 0.021 mg/m<sup>3</sup> STEL: soluble and insoluble nickel 0.1 mg/m<sup>3</sup> TWA (8h) Sk (ILO 1991)

FRANCE :

SWEDEN: Carbonyl 0.007 mg/m<sup>3</sup> TWA (8h) Soluble nickel 0.1 mg/m<sup>3</sup> TWA (8h) SEN (ILO 1991)

USSR : Carbonyl 0.001 ppm TWA (8h) 0.0005 mg/m<sup>3</sup> STEL (ILO 1991)

**Blood Standards:**

Accurate analysis only recently (1980s) available. All earlier data (and derived standards) of little value, being grossly too high. Half life of soluble compounds from 17 to 39 hours.

HSC UK :

**NICKEL**



## NICKEL

### *Urine Standards:*

Comments as for blood. Normal values found < 3.9 nmols/mmol creatinine.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR : 0.03 mg/l (Ho and Dillon 1987)

### *Exhaled Air:*

No data given

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

**Substance: NITROBENZENE**

**Disease/Effect:** Depresses central nervous system, leading to coma and death. Blood haemoglobin converted to methaemoglobin, causing anaemia, with spleen and liver involvement

**Air Standards:**

Absorbed through the skin very readily

HSC UK : OES 1 ppm TWA (8h). STEL 2 ppm. Sk (1992)  
ACGIH : TLV 1 ppm TWA (8h). Sk (1991-92)  
DFG : MAK 1 ppm TWA (8h) Peak Limit Value Category II (1989)  
FINLND : 1 ppm TWA (8h). 3 ppm STEL Sk (ILO 1991)  
FRANCE : 1 ppm TWA (8h) (ILO 1991)  
SWEDEN: 1 ppm TWA (8h). 2 ppm STEL Sk (ILO 1991)  
USSR : 1 ppm TWA (8h). 3 mg/m<sup>3</sup> STEL Sk (ILO 1991)  
EEC : Indicative Limit Value 1 ppm TWA (8h) (1991)

**Blood Standards:**

HSC UK :  
ACGIH : BEI methaemoglobin in blood. 1.5% of haemoglobin, end of shift (1991-92)  
DFG : BAT aniline released from aniline-haemoglobin conjugate, 100 µg/l, after several shifts  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**NITROBENZENE**

## NITROBENZENE

### *Urine Standards:*

Based on metabolism to paranitrophenol but not specific to nitrobenzene.

HSC UK :  
ACGIH : BEI paranitrophenol 5 mg/g creatinine, end of shift, end of work week (1991-92)  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

### *Exhaled Air:*

No data given.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**Substance:** PENTACHLOROPHENOL

**Disease/Effect:** Increased metabolic rate up to collapse and death.

**Air Standards:**

Excessive exposure can occur through skin absorption.

HSC UK : OES  $0.5 \text{ mg/m}^3$  TWA (8h) STEL  $1.5 \text{ mg/m}^3$ . Sk (1992)  
ACGIH : TLV  $0.5 \text{ mg/m}^3$  TWA (8h) Sk (1991-92)  
DFG : MAK  $0.05 \text{ mg/m}^3$  TWA (8h). Peak Limit Value Category III (1989)  
FINLND :  $0.5 \text{ mg/m}^3$  TWA (8h).  $1.5 \text{ mg/m}^3$  STEL Sk (ILO 1991)  
FRANCE :  $0.5 \text{ mg/m}^3$  TWA (8h) Sk (ILO 1991)  
SWEDEN:  $0.5 \text{ mg/m}^3$  TWA (8h).  $1.5 \text{ mg/m}^3$  STEL Sk (ILO 1991)  
USSR :  $0.1 \text{ mg/m}^3$  Sk (ILO 1991)

**Blood Standards:**

Conflicting data on half life in plasma (reports give 30 h to 16 days). Cases of poisoning suggest the longer half life, hence end of week or shift cycle appropriate

HSC UK :  
ACGIH : BEI. Free PCP in plasma, 5 mg/l, end of shift (1991-92)  
DFG : BAT. PCP in plasma/serum 1000  $\mu\text{g/l}$ , unspecified sample time (1989)  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**PENTACHLOROPHENOL**

## PENTACHLOROPHENOL

### *Urine Standards:*

74% excreted as PCP, 12% as the glucuronide. Similar comments to blood as regards half life

HSC UK :

ACGIH : BEI. PCP 2 mg/g creatinine, before last shift of work week (1991-92)

DFG : BAT. PCP 300  $\mu$ g/l unspecified sampling time (1989)

FINLND : PCP 3 mg/l (Ho and Dillon 1987)

FRANCE :

SWEDEN:

USSR :

### *Exhaled Air:*

No data given.

HSC UK :

ACGIH :

DFG :

FINLND :

FRANCE :

SWEDEN:

USSR :

**Substance: PHENOL**

**Disease/Effect:** Absorbed through skin. Accidental exposures may cause death some hours after exposure.  
Repeated lower exposures cause dermatitis.

**Air Standards:**

Inhalation much less important than skin absorption. Control by procedures including PPE.

HSC UK : OES 5 ppm TWA (8h). STEL 10 ppm Sk (1992)  
ACGIH : TLV 5 ppm TWA (8h). Sk (1991-92)  
DFG : MAK 5 ppm TWA (8h) Peak Limit Value Category I (1989)  
FINLND : 5 ppm TWA (8h). 10 ppm STEL Sk (ILO 1991)  
FRANCE : 5 ppm TWA (8h) Sk (ILO 1991)  
SWEDEN: 1 ppm TWA (8h). 2 ppm STEL Sk (ILO 1991)  
USSR : 5 ppm TWA (8h). 0.3 mg/m<sup>3</sup> STEL Sk (ILO 1991)

**Blood Standards:**

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**PHENOL**

## PHENOL

### *Urine Standards:*

Based on excretion of phenol.

HSC UK :  
ACGIH : BEI. Total phenol 250 mg/g creatinine, end of shift (1991-92)  
DFG : BAT. Phenol 300 mg/l, end of shift (1989)  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

### *Exhaled Air:*

No data given.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**Substance:** SELENIUM (excluding hydrogen selenide)

**Disease/Effect:** Contact burns from dioxide, skin and eyes. Acute respiratory irritant from anhydride. Garlic breath reported, probably due to tellurium as impurity.

**Air Standards:**

Site of deposition and intake depends on particle size and compound. Risk usually from acute accidental exposures, placing greater emphasis on working procedures and protection than on air sampling.

HSC UK : OES Compounds and hexafluoride 0.1 mg Se/m<sup>3</sup> TWA (8h) (1992)

ACGIH : TLV Selenium and compounds 0.2 mg Se/m<sup>3</sup> TWA (8h) (1991-92)

Selenium hexafluoride as Se 0.05 ppm TWA (8h) (1991-92)

DFG : MAK selenium compounds 0.1 mg Se/m<sup>3</sup>. Peak Limit Value Category III. TWA (8h) (1989)

FINLND : 0.1 mg/m<sup>3</sup> TWA (8h). 0.3 mg/m<sup>3</sup> STEL: hexafluoride 0.4 mg/m<sup>3</sup> TWA (8h). 1.2 mg/m<sup>3</sup> STEL (ILO 1991)

FRANCE : Hexafluoride 0.2 mg/m<sup>3</sup> TWA (8h) (ILO 1991)

SWEDEN: Compounds 0.1 mg/m<sup>3</sup> TWA (8h) (ILO 1991)

USSR : Selenium (IV) dioxide 0.1 mg/m<sup>3</sup> STEL (ILO 1991)

**Blood Standards:**

No standards to date - problems as with urine.

HSC UK :

ACGIH :

DFG :

FINLND :

FRANCE :

**SELENIUM (excluding hydrogen selenide)**



## SELENIUM (excluding hydrogen selenide)

### *Urine Standards:*

Selenium is a normal soil trace element, to give high dietary intake in some areas, and so high excretion. All data must be referred to a local baseline. Early recommendations were 0.1 mg Se/l (Elkins 1959), 0.07 mg Se/l (Stokinger 1955) quoted by Ho and Dillon (1987).

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

### *Exhaled Air:*

No data given

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN :  
USSR :

**Substance: STYRENE**

**Disease/Effect:** Respiratory irritant with carcinogenic potential from its metabolic routes. 80% excreted in urine as mandelic and phenylglyoxylic acids. Short half life of hours only.

**Air Standards:**

Readily absorbed through intact skin.

HSC UK : MEL 100 ppm TWA (8h). STEL 250 ppm (1992)  
ACGIH : TLV 50 ppm TWA (8h). STEL 100 ppm. Sk (1991-92)  
DFG : MAK 20 ppm TWA (8h) Peak Limit Value Category II (1989)  
FINLND : 20 ppm TWA (8h). 100 ppm STEL (ILO 1991)  
FRANCE : 50 ppm TWA (8h) (ILO 1991)  
SWEDEN: 25 ppm TWA (8h). 75 ppm STEL (ILO 1991)  
USSR : 50 ppm TWA (8h). 30 mg/m<sup>3</sup> STEL (ILO 1991)

**Blood Standards:**

Not in general use because of short half life.

HSC UK :  
ACGIH : BEI styrene 0.55 mg/l end of shift. Styrene 0.02 mg/l prior to next shift (1989/90) (Sq)  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**STYRENE**

## STYRENE

### *Urine Standards:*

Based on metabolism to mandelic acid and phenylglyoxylic acid. Affected by alcohol.

HSC UK : Quotes DFG and ACGIH lists (1987)

ACGIH : BEI mandelic acid in urine: End of shift 800 mg/g creatinine (Ns)

Prior to next shift 300 mg/g creatinine (Ns)

Phenylglyoxylic acid in urine: End of shift 240 mg/g creatinine (B, Ns)

Prior to next shift 100 mg/g creatinine

DFG : BAT 2 g/l mandelic acid, 2.5 g/l mandelic plus phenylglyoxylic acids, end of shift. (1989)

FINLND :

FRANCE :

SWEDEN:

USSR :

### *Exhaled Air:*

HSC UK :

ACGIH : BEI styrene 40 ppb in mixed exhaled air prior to next shift (1990-1)

DFG :

FINLND :

FRANCE :

SWEDEN:

USSR :

**Substance: TETRACHLOROETHYLENE**

**Disease/Effect:** Similar to trichloroethylene but lower vapour pressure. Majority (90%) excreted in exhaled air over days, a small amount metabolised to trichloroacetic acid.

**Air Standards:**

Difficult to evaluate exposure with some exposure patterns.

HSC UK : OES 50 ppm (TWA 8h). STEL 150 ppm (1992)  
ACGIH : TLV 50 ppm TWA (8h). STEL 200 ppm (1991-2)  
DFG : MAK 50 ppm TWA (8h) (IIIB). Peak Limit Value Category II (1989)  
FINLND : 50 ppm TWA (8h). 75 ppm STEL Sk (ILO 1991)  
FRANCE : 50 ppm TWA (8h). (ILO 1991)  
SWEDEN: 10 ppm TWA (8h). 25 ppm STEL (ILO 1991)  
USSR : 50 ppm TWA (8h). 10 mg/m<sup>3</sup> STEL (ILO 1991)

**Blood Standards:**

HSC UK : Quotes BAT (1985)  
ACGIH : BEI 1 mg/l prior to last shift of work week (1991-92)  
DFG : BAT 100 µg/100 mls beginning of next shift (1989)  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**TETRACHLOROETHYLENE**

## TETRACHLOROETHYLENE

### *Urine Standards:*

Based on metabolism to trichloroacetic acid. Affected by alcohol.

HSC UK :

ACGIH : BEI 7 mg trichloroacetic acid/1 end of work week (1991-92) (Ns, Sq)

DFG :

FINLND :

FRANCE :

SWEDEN:

USSR :

### *Exhaled Air:*

HSC UK : Quotes BAT (1987)

ACGIH : BEI 10 ppm in end exhaled air prior to last shift of work week (1991-92)

DFG : BAT 9.5 ppm beginning of next shift (1989)

FINLND :

FRANCE :

SWEDEN:

USSR :

**Substance: TOLUENE**

**Disease/Effect:** Narcotic. 20% excreted in air, rapid drop after exposure. Fatty tissue accumulation (half life of days). Majority excreted as hippuric acid in urine (half life in hours).

**Air Standards:**

HSC UK : OES 50 ppm TWA (8h). STEL 150 ppm Sk (1992)  
ACGIH : TLV (100) ppm TWA (8h). STEL 150 ppm Sk (1991-92).  
Intended change 50 ppm TWA (8h) Sk  
DFG : MAK 100 ppm TWA (8h) Peak Limit Value Category II (1989)  
FINLND : 100 ppm TWA (8h). 150 ppm STEL Sk (ILO 1991)  
FRANCE : 100 ppm TWA (8h). 150 ppm STEL (ILO 1991)  
SWEDEN: 50 ppm TWA (8h). 100 ppm STEL Sk (ILO 1991)  
USSR : 100 ppm TWA (8h). 50 mg/m<sup>3</sup> STEL (ILO 1991)

**Blood Standards:**

Metabolism affected by alcohol.

HSC UK : 20  $\mu$ mol/l end of shift corresponds to 100 ppm air (1987)  
ACGIH : BEI toluene 1 mg/l end of shift (1991-92) (Sq)  
DFG : BAT toluene 170  $\mu$ g/100 mls end of shift (1989)  
FINLND : 300  $\mu$ g/100 mls (Ho and Dillon 1987)  
FRANCE :  
SWEDEN:  
USSR :

**TOLUENE**

## TOLUENE

### *Urine Standards:*

Based on metabolism to hippuric acid, normal levels very variable, limiting its use at low exposures.

HSC UK : Does not recommend hippuric acid (1987)

ACGIH : BEI hippuric acid 2.5 g creatinine end of shift, last 4 h of shift (1991-92).  
Intent to establish o-cresol 1 mg/g creatinine (Ns)

DFG :

FINLND :

FRANCE :

SWEDEN:

USSR :

### *Exhaled Air:*

About 15% of absorbed toluene is exhaled, with a biphasic exponential curve.

HSC UK : Recommends, quotes ACGIH value (1987)

ACGIH : BEI 20 ppm during shift (1989/90) (Sq)

DFG :

FINLND :

FRANCE :

SWEDEN:

USSR :

**Substance: 1 1 1 TRICHLOROETHANE (METHYL CHLOROFORM)**

**Disease/Effect:** Narcotic. 90% excreted in exhaled air, remainder slowly metabolised and excreted as trichloroacetic acid.

**Air Standards:**

Exposures can arise from misuse for hand cleaning.

HSC UK : MEL 350 ppm TWA (8h). STEL 450 ppm (1992)  
ACGIH : TLV 350 ppm TWA (8h). STEL 450 ppm (1991-92)  
DFG : MAK 200 ppm TWA (8h) Peak Limit Value Category II (1989)  
FINLND : 100 ppm TWA (8h). 250 ppm STEL (ILO 1991)  
FRANCE : 300 ppm TWA (8h). 450 ppm STEL (ILO 1991)  
SWEDEN: 50 ppm TWA (8h). 90 ppm STEL (ILO 1991)  
USSR : 200 ppm TWA (8h). 20 mg/m<sup>3</sup> STEL (ILO 1991)

**Blood Standards:**

HSC UK : Quotes BAT, but pre-shift end of week samples (1987)  
ACGIH : BEI trichloroethanol 1 mg/l end of shift, end of work week (1991-92) (Ns)  
DFG : BAT 1 1 1 trichloroethane 55 µg/100 mls beginning of shift, end of work week (1989)  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**1 1 1 TRICHLOROETHANE (METHYL CHLOROFORM)**



## 1 1 1 TRICHLOROETHANE (METHYL CHLOROFORM)

### *Urine Standards:*

Based on metabolism to trichloroacetic acid and trichloroethanol.

HSC UK :

ACGIH : BEI trichloroacetic acid 10 mg/l end of work week. Total trichloroethanol 30 mg/l end of shift and end of work week (1991-92) (Ns, Sq)

DFG :

FINLND :

FRANCE :

SWEDEN:

USSR :

### *Exhaled Air:*

HSC UK :

ACGIH : 40 ppm prior to last shift of work week (1991-92)

DFG : BAT 20 ppm beginning of next shift, end of work week (1989)

FINLND :

FRANCE :

SWEDEN:

USSR :

**Substance: TRICHLOROETHYLENE**

**Disease/Effect:** De-fats the skin plus narcotic and cardiovascular effects. 10% lung excretion, rest metabolised to trichloroethanol (half life 12 hours) and trichloroacetic acid (half life 3 days).

**Air Standards:**

Short term narcosis and addiction. Often intermittent exposures.

HSC UK : MEL 100 ppm TWA (8h). STEL 150 ppm Sk (1992)  
ACGIH : TLV 50 ppm TWA (8h). STEL 200 ppm. (1991-92)  
DFG : MAK 50 ppm TWA (8h) III B. Peak Limit Value Category II (1989)  
FINLND :  
FRANCE : 75 ppm TWA (8h). 200 ppm STEL (ILO 1991)  
SWEDEN: 10 ppm TWA (8h). 25 ppm STEL (ILO 1991)  
USSR : 50 ppm TWA (8h). 10 mg/m<sup>3</sup> STEL (ILO 1991)

**Blood Standards:**

Depends upon variability of exposure - early am may not be quantified by late pm sample.

HSC UK :  
ACGIH : BEI 4 mg free trichloroethanol/1 end shift, end of week (1991-92) (Ns)  
DFG : BAT 500 µg/100 mls trichloroethanol, end shift for long-term exposure (1989)  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**TRICHLOROETHYLENE**

## TRICHLOROETHYLENE

### *Urine Standards:*

Based on metabolism to trichloroacetic acid. Affected by alcohol.

HSC UK : Quotes BAT value (1985)

ACGIH : BEI 100 mg trichloroacetic acid/g creatinine end of work week. 300 mg trichloroacetic acid plus trichloroethanol/g creatinine, end of shift and end of work week (1991/92) (Ns)

DFG : BAT 100 mg/1 trichloroacetic acid, end shift and end of work week (1989)

FINLND :

FRANCE :

SWEDEN:

USSR :

### *Exhaled Air:*

No data given.

HSC UK :

ACGIH :

DFG :

FINLND :

FRANCE:

SWEDEN:

USSR :

**Substance: XYLENE**

**Disease/Effect:** De-fatting of skin and narcotic. Absorbed through skin and metabolised and excreted as methyl hippuric acid, affected by alcohol and aspirin.

**Air Standards:**

Excessive intake can occur through skin absorption.

HSC UK : OES 100 ppm TWA (8h). STEL 150 ppm Sk (1992)  
ACGIH : TLV 100 ppm TWA (8h) ppm. STEL 150 ppm  
DFG : MAK 100 ppm TWA (8h) Peak Limit Value Category II (1989)  
FINLND : 100 ppm TWA (8h). 150 ppm STEL (ILO 1991)  
FRANCE : 100 ppm TWA (8h). 150 ppm STEL (ILO 1991)  
SWEDEN: 50 ppm TWA (8h). 100 ppm STEL (ILO 1991)  
USSR : 100 ppm TWA (8h) (ILO 1991)

**Blood Standards:**

HSC UK :  
ACGIH :  
DFG : BAT Xylene 150 µg/100 mls, end of shift (1989)  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

**XYLENE**

## XYLENE

### *Urine Standards:*

Based on metabolism to methyl hippuric acid. Affected by alcohol and aspirin. Two phase methyl hippuric acid excretion.

HSC UK : Quotes FINLAND (1987)  
ACGIH : BEI hippuric acids 1.5 g/g creatinine end of shift, 2 mg/min last 4 h of shift (1991-92)  
DFG : BAT toluric acid 2 g/l end of the shift (1989)  
FINLND : hippuric acid 700  $\mu$ mol/mmol creatinine corresponds to 50 ppm xylene TWA (8h) (1981)  
FRANCE :  
SWEDEN:  
USSR :

### *Exhaled Air:*

No data.

HSC UK :  
ACGIH :  
DFG :  
FINLND :  
FRANCE :  
SWEDEN:  
USSR :

# Clayton

## ENVIRONMENTAL CONSULTANTS

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Tel: 021 359 5951  
Fax: 021 359 7606.

*Contact: Mr B. Owen.*

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## BIOLOGICAL MONITORING OF HUMAN EXPOSURE TO TRACE ELEMENTS

The *Supra-regional Assay Service (SAS) Trace Element Laboratories of the NHS and their Associated Laboratories* offer collectively a comprehensive range of quantitative clinical trace element assays in blood, urine and other body tissues and fluids. Some of these assays are useful for *Occupational Monitoring*.

The elemental assays offered are:

Al, Ag, Au, B, Ba, Be, Bi, Cd, Co, Cr, Cu, F, Fe, Hg,  
Mn, Mo, Ni, Pb, Pt, Sb, Se, Sn, Sr, Te, Ti, Tl, V, Zn.

For further details including cost, turn-around time, sample requirements, plus availability of common internal quality control protocols and of participation in external quality assessment schemes and of other relevant assays available, contact the laboratory Director.

### SAS AND ASSOCIATED\* LABORATORIES

LOCATION	DIRECTOR	TELEPHONE
Birmingham, Dudley Road Hospital	Dr SS Brown	021-554-3801 #4135 Fax: 021-554-7386
Glasgow Royal Infirmary*	Dr GS Fell	041-304-4635 Fax: 041-553-1703
Guildford, St Luke's Hospital	Dr A Walker	0483-502742 Fax: 0483-503517
London, King's College Sch of Medicine and Dentistry	Prof TJ Peters	071-326-3009 #4127 Fax: 071-737-7434
London, Poisons Unit, New Cross Hospital	Dr B Widdop	071-635-8032 Fax: 071-639-2101
Leeds, General Infirmary*	Mr K Newton	0532-431751 #5659 Fax: 0532-335672
Southampton General Hospital	Dr HT Delves	0703-796419 Fax: 0703-704062

**National Poisons Unit,  
Guy's and Lewisham NHS Trust**

**OCCUPATIONAL and ENVIRONMENTAL  
TOXICOLOGY SERVICES**

*The National Poisons Unit*, established in 1963, offers a wide range of services for the occupational physician and hygienist. *The National Poisons Information Service* operates a computerized database management system which can be searched, for example, to retrieve toxicity data on particular groups of compounds on behalf of manufacturers. *The Poisons Unit Laboratory*, which incorporates a *Supra-regional Assay Service (SAS) Trace Element Laboratory*, offers analytical facilities for biological monitoring of a wide range of compounds including:

Chlorinated pesticides (lindane, pentachlorophenol)  
Organophosphate pesticides  
Solvents (acetone, ethylbenzene, methanol, methyl ethyl ketone, methyl isobutyl ketone, tetrachloroethylene, toluene, 1,1,1-trichloroethane, trichloroethylene, xylenes)

Additional indirect assays available include:

Carboxyhaemoglobin (carbon monoxide, dichloromethane)  
Cholinesterase activity (organophosphate and carbamate pesticides)  
Methaemoglobin (aniline, nitrobenzene)

Pre-employment and employment urine screening for drugs of abuse including amphetamines, cannabis, cocaine, ethanol, and opiates can also be arranged.

A booklet describing the services offered is available from:

Dr RJ Flanagan, National Poisons Unit, Guy's and Lewisham NHS Trust, Avonley Road, London SE14 5ER.  
Tel: 071-635 0858 (direct line); Fax: 071-639 2101.





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IR1, Types and Effects 22 minutes; IR2, Origins and Control 27 minutes.

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VHS format.

M1, Scientific Background 18 m; M2, Units for Radiation Protection 20 m;

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by Peter Dewell

These programs are offered on 3½" DD (720k) PC discs. Many have been in use for more than ten years.

A *Manual* is provided to guide you through the programs. This is *not* a statistics book. You will find many of the statistics behind the programs in *Some Applications of Statistics in Occupational Hygiene*, P. Dewell, BOHS Technical Handbook No.1 from H & H Scientific Consultants, P.O. Box MT27, Leeds, LS17 8QP.

## MANUAL CONTENTS

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## SCREEN MENU OF PROGRAMS

**FILPLOT**, Test for (log)nor dist, parameters, screen/HP plot  
**FILZERC**, Cut down FILPLOT, for zero & below detectable level values  
**FANTTEST**, F & t-tests on (log)normal data loaded from file or keyboard  
**FTDMAN**, Manual input of Arith & Geom Means & SDs for F- & t-tests  
**PAIREDT**, Paired t-tests, differences and ratios + unmatched t-tests  
**HIST**, Sorting data for histogram plotting, Lognor & Nor, no plot  
**CHICUM**, Chi square test of goodness of fit for HIST  
**CHISQU**, Chi square for 2x2 and other tables, OBS. and OBS./EXP.  
**SAMCON**, Cross links Group & Sample size, 1 in top t% & % confidence  
**SAMPsize**, Simple program for sample size given Mean, SD % error  
**STEL**, NIOSH test for compliance of STELs.  
**RNDTIMES**, Random sampling timetables for STEs, at 10 min intervals  
**RNDCONC**, Random (log)normal concentrations: analysis, plot & file  
**CONTCHTS**, An approach to the use of Control Charts in OH.



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