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JOINT UNEP/FAO/WHO FOOD CONTAMINATION MONITORING AND ASSESSMENT PROGRAMME (GEMS/FOOD)

# ASSESSMENT OF DIETARY INTAKE OF CHEMICAL CONTAMINANTS

**UNITED NATIONS** ENVIRONMENT PROGRAMME



FOOD AND AGRICULTURE **ORGANIZATION** 



**WORLD HEALTH** ORGANIZATION

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# **ASSESSMENT OF DIETARY INTAKE OF CHEMICAL CONTAMINANTS**

Joint UNEP/FAO/WHO Food Contamination Monitoring and Assessment Programme (GEMS/Food)

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## **ASSESSMENT OF DIETARY INTAKE OF CHEMICAL CONTAMINANTS**

### Charles F. Jelinek, Ph.D





Annex 9 Dietary Intake of Polychlorinated Biphenyls by Adults 75

#### **ASSESSMENT OF DIETARY INTAKE**

#### **OF CHEMICAL CONTAMINANTS**

#### **Charles F. Jeilnek, Ph.D**

#### **1. INTRODUCTION**

Over ten million unique chemical entities are now known to scientists and around several hundred thousand are in common use throughout the world. While the technologies associated with the use of these chemicals have resulted in a dramatic improvement in living conditions for most of the world's population, these technologies and related human activities have also resulted in the introduction of numerous chemicals, directly or indirectly, into the food supply. Only a few of these chemicals have been fully characterized in terms of their potential toxicities to animals and humans, particularly in relation to their long-term effects. Similarly, many naturally occurring chemicals known to be toxic to animals and humans have yet to be adequately assessed.

An assured and adequate supply of safe, wholesome food is essential for health. Food contamination monitoring is an essential component of ensuring the safety of food supplies and managing scarce environmental and agricultural resources, since it provides information on the levels and sources of contaminants in foods, on the amounts of contaminants ingested by humans, and on trends in contamination levels.

Chemical contaminants present in food may result from their natural occurrence in soil (e.g. cadmium, lead, and mercury) or from mycotoxin contamination (e.g. aflatoxins), from pollution arising from industrial and other human activities (e.g. lead, mercury, cadmium, and polychiorinated biphenyls (PCBs)), from agricultural practices (e.g. pesticides such as lead arsenate, fertilizers, and drugs used in food animals), and from food processing and packaging (e.g. nitrosamines, certain polycydic aromatic hydrocarbons, lead and PCBs). These contaminants are likely to enter the food supply and present a potential hazard for human health.

These concerns led to the establishment of the Joint UNEP/FAO/WHO Food Contamination Monitoring and Assessment Programme (GEMS/Food) in 1976, with the initial participation of 13 countries. At present, 40 countries participate in GEMS/Food. These countries are listed in Annex 1. GEMS/Food is a part of the United Nations system-wide Global Environment Monitoring System (GEMS), which also includes components in air and water contamination monitoring as well as total human exposure assessments.

The main objective of the Programme is to inform governments, the Codex Alimentarius Commission, other relevant institutions, and the public on levels and trends of contaminants in food, their contribution to the total human exposure and significance with regard to public health and trade. The Programme is conducted globally, regionally, and nationally, as well as in local areas of special concern that serve as models - where appropriate - for remedial actions, food control and resource management. Supporting

**components of the Programme involve technical cooperation, training, analytical quality assurance studies and information exchange.** 

**A major activity of this Programme is to compile food contamination monitoring data from the different countries for world-wide presentation, synthesis and evaluation.**  Three data summaries have been published for the period 1980-1988 (UNEP/FAO/WHO, **1986a, 1988a, 1991a).** 

**Most of the data submitted by the participating institutions to the Programme have been concerned with levels of priority chemicals in foods of major dietary importance. The chemicals and foods to be reported upon have been selected by the GEMS/Food Programme Management Committee which meets periodically to provide overall guidance to the Programme. WHO serves as the lead agency for** *GEMS/Food.* 

**A significant portion of the data also submitted to the Programme concerns the estimated total dietary intakes of these selected contaminants for further comparison with toxicologically acceptable or tolerable intake levels. Dietary intake data have been included in** *GEMS/Food* **since 1980, when intake data were first requested.** 

**This report is an assessment of global data reported on estimated total dietary intakes of pesticides, PCBs, lead, cadmium and mercury for the 1980s. The data submitted to GEMS/Food are the major source of information for this report. Other sources of data are reports prepared for the Codex Alimentarius Commission and its various subsidiary bodies under the Joint FAO/WHO Food Standards Programme and papers and reports in the open literature.** 

#### **2. BACKGROUND**

#### **2.1 Priority foods and chemicals selected for assessment**

**The choice of foods and contaminants to be monitored and assessed varies from country to country and, occasionally, from place to place within the same country. It may also change with time as dietary habits shift, or as other contamination problems become apparent.** 

**The following factors are of importance when selecting priority foods and contaminants for assessment:** 

- **the potential risk for human health posed by the contaminant, including the**   $(a)$ **severity of the possible adverse effects (e.g. neurotoxic, carcinogenic, mutagenic, and hepatotoxic) together with any information on current human exposure and the population at risk;**
- **the frequency with which a food/contaminant combination is implicated in**   $(b)$ **intoxications in humans and animals and the levels of the contaminant found in different foods;**
- **the importance of the food in the total diet; staple foods including mother's**   $(c)$ **milk deserve special attention;**
- the feasibility of measuring the level of the contaminant in a reliable  $(d)$ manner in an adequate number **of food samples;**
- the persistence, ubiquity and abundance of the contaminant in the  $(e)$ environment, including its resistance to degradation, its possible conversion to more toxic substances, and its accumulation in the food chain;
- the amount of the contaminant being discharged into the environment by  $(f)$ industry, agriculture and urban centers; and
- the economic importance of the food in international trade.  $(g)$

#### **2.2 Dietary intakes of chemical contaminants**

To assess the health risk to the consumer, it is necessary to estimate the actual dietary intake of a contaminant for comparison with toxicologically acceptable levels (Section 2.4). This estimate is also important in determining whether there is a relationship between any observed effects in human beings and the intake of a particular contaminant. Thus, the estimation of the dietary intake of a contaminant is indispensable for risk assessment and management. Such estimates carried out periodically provide information on trends of exposure and effectiveness of control measures.

Because of the importance of developing estimates of dietary intake of contaminants and the complexity involved in obtaining such information, guidelines for organizing and conducting dietary intake studies were prepared under the auspices of GEMS/Food (UNEP/FAO/WHO, *1985a).* 

Three basic approaches can be used to determine intake:

- total diet studies (analysis of a "standard" market basket), usually prepared as for customary consumption;
- consideration of food consumption information together with data on contaminant levels in foods of major dietary importance generated from monitoring programmes; and
- analysis of duplicate portions of food consumed by selected individuals.

The main characteristics of dietary intake studies carried out in various countries are given in Annex 2. The composition of the diet, the consumer to whom the study is geared, the preparation for analysis, the total weight of the diet and the study approach vary widely from country to country and study to study. Because of such differences, comparisons between countries are often difficult to make, and only trends within a country may at times be established.

The review of dietary intake of chemical contaminants on a global basis is hampered by a number of factors, including:

- the disparity in the nature of dietary surveys and the number of years in  $(i)$ which they have been carried out;
- lack of data currently available from less developed countries; and  $(ii)$
- $(iii)$ inadequate assurance of the quality of some of the data.

Nevertheless, the available information can provide an indication of the nature, levels and trends in dietary intake of the different chemical contaminants.

#### **2.3 Estimates of Infant Intakes**

Estimates of the dietary intake of contaminants by the breast-fed infant may be obtained from human milk data. For the first three months of life, an infant consumes on average 120 g per day of human milk per kg body weight, while after three months of age, the volume consumed per unit weight decreases with increasing age (WHO, *1985).*  By multiplying the concentration ( $\mu$ g/kg or  $\mu$ g/l) of a given contaminant in the whole milk by 0.12, the approximate daily intake of the contaminant in  $\mu$ g/kg body weight may be estimated, and this in turn may be compared to international guidelines (see Section 2.4). If contaminant concentration in the milk fat is reported, it is assumed that the average fat content of the milk is *3.5%* unless, of course, the authors report the fat content of the samples analyzed.

The reported levels of certain pesticides and other contaminants in human milk at times yield estimated intakes by the breast-fed infant that exceed toxicologically acceptable intakes. However, acceptable intakes are usually developed on the basis of lifetime exposure, while consumption of contaminants from human milk is limited to a few months in a lifetime. In considering the balance of risks and benefits posed by human milk containing contaminants, WHO noted that it is not known whether such limited intakes are actually detrimental to the child's progress in terms of physical and mental development. But any concern about contaminated breast milk must be seen in perspective because the problem is not simply one of breast-feeding. For example, a child taken off breast-feeding might continue to ingest contaminants through the daily diet just as the mother who had passed them on in her breast milk. In the long term, there are two ways of effectively combating the problem:

- by developing compounds that are easily excreted; and  $(a)$
- $(b)$ by identifying the most harmful compounds and strictly controlling their use (WHO, *1985).*

#### **2.4 Comparison with standards and guidelines**

Whenever possible, monitoring data from dietary intake studies are compared with acceptable or tolerable levels recommended by the Joint FAO/WHO Meeting on Pesticide Residues (JMPR), or the Joint FAO/WHO Expert Committee on Food Additives (JECFA). The Acceptable Daily Intake (ADI) level of a chemical is the daily intake which, during a lifetime, would pose no appreciable risk to the consumer, on the basis of all facts known at the time. It is expressed in milligrammes per kilogramme of body weight. The Provisional Tolerable Weekly Intake (PTWI) represents permissible human weekly exposure to those contaminants unavoidably associated with the consumption of otherwise wholesome and nutritious foods. The term "provisional" expresses the tentative nature of JECFA's evaluation and "tolerable" signifies permissibility rather than acceptability for the intake of trace contaminants which have no necessary functions in food in contrast to those of permitted pesticides or food additives. For cumulative toxicants, such as lead, cadmium and mercury, the tolerable intakes are expressed on a

weekly basis to allow for daily variations in intake levels, the real concern being prolonged exposure to the contaminant (WHO, 1987a).

#### *2.5* **Scope of present assessment**

#### 2.5.1 GEMS/Food

Countries participating in GEMS/Food are asked to submit data on the median, mean and 90th percentile daily intakes. The data obtained permit a reasonable assessment of median/mean intakes, but less often of the 90th percentile values.

GEMS/Food collects residue and intake data on the following 19 priority contaminants:

- Organochlorine pesticides (8)--aldrin/dieldrin, DDT complex, heptachior and heptachior epoxide, hexachlorobenzene, total hexachiorocyclohexane (HCH) isomers, gamma-HCH (lindane), endosulfan, and endrin
- Organophosphorus pesticides (5)--diazinon, fenitrothion, malathion, parathion, and parathion-methyl
- Industrial chemicals (5)--Polychlorinated biphenyls (PCBs), lead, cadmium, mercury and tin
- Mycotoxins (1)--Aflatoxins

These are all discussed in this report, except for aflatoxins and tin. Aflatoxins will be the subject of a separate assessment report. Tin is not included because it has only recently been included in GEMS/Food.

The global storage and processing centre for data is located at WHO Headquarters in Geneva. Dietary intake data are included in summary reports covering data submitted for the years 1980-88 (UNEP/FAO/WHO 1986a, 1988a, 1991a). Because of the volume of data received, these dates are not discussed in detail in this report. The data summary reports should be consulted for further details, if desired. The 1980-1985 GEMS/Food dietary intake data have been assessed with regard to levels, trends and possible cause of these trends (UNEP/FAO/WHO 1986b, 1988c). The present report includes an overall assessment of intake data submitted for the period 1980-1988.

#### 2.5.2 Codex Alimentarius Commission

Established under the auspices of the Joint FAO/WHO Food Standards Programme, the Codex Alimentarius Commission is an intergovernmental body which was established in 1963 to promote consumer protection and to facilitate international trade in food by providing generally accepted food standards, codes of practice and other guidance, including recommendations on the safe levels of pesticide residues, food additives and contaminants in food.

In addition to the systematic data collection from institutions participating in GEMS/Food, the Codex Committee on Pesticide Residues (CCPR) and the Codex

Committee on Food Additives and Contaminants (CCFAC) have, on an ad hoc basis, requested *GEMS/Food* **to** gather information from members **of Codex on** subjects of specific interest to these Codex Committees. However, while there are over 140 countries which belong to Codex, in most cases only a few have responded to such requests. GEMS/Food has, nonetheless, collected useful information on the dietary intakes of lead, cadmium, mercury, PCBs and pesticides for these Codex Committees.

#### *2.5.3* Open literature

The published results referenced in this report are not an exhaustive compendia of all dietary intakes which have been reported publicly. Generally, citations are limited to major publications in the scientific literature.

#### **2.6 Validity and comparabifity of the data**

Institutions submitting data to GEMS/Food are asked to state, in general terms, the reliability of their analytical results. In some, but not all of the countries, the institution indicated that studies to validate the performance of an analytical method were carried out and/or that check samples, such as the United States National Bureau of Standards reference materials, were used to evaluate the validity of the data. In many cases, quality of the analytical results was checked by participation in international collaborative studies.

Analytical Quality Assurance (AQA) studies have been conducted regularly by GEMS/Food to promote the quality and comparability of the data submitted (UNEP/FAO/WHO, 1981, 1983, 1985b, 1988b, 1991b). An analysis of recent AQA studies (UNEP/FAO/WHO, 1988b) indicates that the results from a number of laboratories are not reliable, especially those for heavy metals. A number of laboratories, in both industrialized and developing countries, have problems with the analytical quality of their data, and in some cases the quality of the data has remained poor. Accordingly, a concerted effort is being made to improve the quality of GEMS/Food data by making participation in international or national inter-laboratory AQA programmes a prerequisite for submission of monitoring data.

Through 1983, participating countries were requested to report levels above the "limit of detection". This is dependent on the sensitivity of the analytical method used, and all values reported would represent residues which were detectable and identifiable. Since 1983, this has been changed to "limit of determination", which denotes the level at or above the limit of detection, where the residue can be quantitatively measured with accuracy and precision. This is also dependent on the method of analysis, and can be somewhat of an arbitrary judgement by the individual laboratory. Consequently, the limits reported in GEMS/Food for the same contaminant in the same food will often vary from country to country. Also, a given contaminant level reported by a given country may not have been detected by a second country, even when present at the same level, if the second country's method is not sufficiently sensitive. Therefore, even though intracountry comparisons of data can be made, inter-country comparisons should be made cautiously.

Regarding sampling, GEMS/Food does not specify the sampling methods to be used, but assumes proper procedures were followed. The total number of market baskets, samples, or individual diets studied also is not specified and may be quite small in some cases. In general, no attempt has been made in this report to relate numbers of samples analyzed to individual sets of data.

As far as the data reported outside GEMS/Food are concerned, even less is known about their reliability and the limits of detection or determination. In addition, the authors of the various reports and papers have used different formats in the presentation of their data.

Taking these factors into account, it is clear that considerable judgement must be used in drawing conclusions concerning levels of dietary intakes of the different contaminants and the trends involved, especially when making comparisons between countries. In addition, it should be kept in mind that most of the data reported are from industrialized countries. Where possible, trends and other evaluations given in this report will include qualifying information to provide greater insight concerning the validity and comparability of the data.

#### 3. LEAD

Lead is one of the earliest metals used by man. It is recovered from naturally occurring suiphide ores, and is used in storage batteries, as an ingredient in anti-knock motor fuel additives, for ammunition, solder, in pigment colours, galvanizing and plating. It is also a constituent or a contaminant in houseware materials, such as crystal and pewter (IARC, 1980).

Lead is a cumulative poison. It produces a continuum of effects, primarily on the hematopoietic system, the nervous system and the kidneys. Adverse effects on intelligence and behavior have been noted in infants and young children, even with very low levels of lead in the blood.

Exposure of the general population occurs by inhalation and by ingestion of food **and water. Where lead pipes or lead** solder **are used, exposure from drinking** water can be considerable. Brass plumbing fixtures and fittings are also a source of lead in the water. In addition, particular behavioral characteristics of children, such as heightened hand-to-mouth activity and the ingestion of non-food items. (pica) may result in appreciable ingestion of lead from non-food sources (WHO, 1972).

In 1972, the Joint Expert Committee on Food Additives (JECFA) established a Provisional Tolerable Weekly Intake (PTWI) for lead from all sources of 50  $\mu$ g/kg body weight for adults (WHO, 1972). Because of the special concern for infants and children, JECFA evaluated the health risks of lead to this population group in 1986 and established a PTWI of 25  $\mu$ g/kg body weight. Infants and young children are more vulnerable to exposure to lead than adults because of metabolic and behavioral differences (WHO, 1987b and c). It was recognized that the evaluation of health effects of lead relates to exposure from all sources and that any increase in lead from non-food sources (e.g.

drinking water, air, soil and *pica)* will reduce the amount that can be tolerated in food (WHO, 1987b).

**Kitchenware, especially improperly glazed ceramic ware, can be a major source of lead in the diet. The International Organization for Standardization (ISO) adopted limits of release for lead from ceramic foodware of 1.7 mg/dm2 for flatware and** *2.5-5.0* **mg/i**  of extraction solution for hollow-ware (ISO, 1981).

#### **3.1 Dietary intake of lead**

Many more countries have produced information on the dietary intake of lead than on pesticides, perhaps because of its pervasive occurrence and out of concern for its effects on the young. In addition to the factors which render it difficult to compare the dietary intakes reported in different countries, and, indeed, in different studies carried out in the same country (Section 2.2), the control of laboratory contamination by lead is much more difficult than for any other contaminant considered here. Much greater care must be taken to prevent contamination of the analytical samples from the atmosphere and laboratory environment because of the ubiquitous presence of lead in old paint, in petrol emissions, and in dust and soils. Thus, even greater care must be observed in comparing the dietary intakes of lead from different studies. Even in sophisticated laboratories, some of earliest reported levels of lead have become suspected of being too high because of such difficulties in the analytical procedures. -

#### 3.1.1 Dietary intake, adults

Information from 26 countries, mostly developed, on the dietary intakes of lead by their adult populations was available for review. These data, together with their references, are listed in Annex 3. The average weekly lead intakes for adults which were obtained in the more recent surveys in each of the various countries are depicted in descending order in Figure 1. A strict comparison of the levels between the various countries obviously cannot be made. The differences in years in which the studies were carried out and in the study **approaches, plus inadequacies in analytical** quality control may account for much of the differences in intakes.

As a general rule, the *95th* percentile consumers of food in general have an intake of food that is twice the average consumption of the population as a whole, while the ratio between the average and the *95th* percentile consumption of a particular food appears to be roughly three times the mean consumption (UNEP/FAO/WHO, 1985a). Therefore, the intake of a contaminant by the average adult population should be viewed with some concern if it approaches the PTWI, since a significant segment of the population may exceed the PTWI because of varying dietary habits.

The average adult dietary intakes of lead reported by Cuba, India and Thailand were essentially equal to or exceeded the PTWI, and the intakes reported from Italy, Guatemala, Germany and New Zealand were one-half or more of the PTWI. The lowest intakes were reported by Sweden, Yugoslavia, Finland and the USA.



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In a 1980 study **reported to GEMS/Food** by Denmark, the average weekly adult lead intake was about 8 **ag/kg body** weight. An appreciable increase in lead intake was noted with consumption of a quarter of a liter of wine per day  $(24 \mu g/kg$  body weight), in areas where vegetables are grown near heavy traffic  $(30 \mu g/kg$  body weight) and in areas where adults live near a secondary lead smelter outside of Copenhagen (32.6  $\mu$ g/kg body weight).

Mean lead levels in wine of about 20 to 100  $\mu$ g/kg have been reported from Belgium (Fouassin and Fondu, 1981), the UK (Sherlock et al, 1986) and the Netherlands (Edel et al. 1983). The UK investigators reported levels up to 1890  $\mu$ g/kg lead in the wine first poured from bottles using lead caps. Conso et al (1984) reported lead poisoning in a patient who had consumed wine with a content of around  $2,000 \mu g/kg$ .

Data from Poland indicate an increase in dietary intakes near industrial areas. In connection with the contribution of dietary lead from traffic, lower intakes were noted in rural areas in Japan (Ikeda et al, 1989) and in the Republic of Korea (Watanabe, Cha et al, 1987).

The presence of lead plumbing can cause a dramatic increase in dietary lead intake. Sherlock et al  $(1982)$  noted a mean weekly intake of around  $47 \mu g/kg$  body weight, in a study in Ayr, as compared to the UK national average of around  $7 \mu g/kg$ body weight. In this study, the 90th percentile intake was 114  $\mu$ g/kg body weight/wk, more than twice the PTWI.

Of countries which reported lead intakes for several years, a downtrend probably occurred in Finland, the UK, and the USA (Figure 2). In the USA the trend is more apparent in recent years than in the other countries.

3.1.2 Dietary intake, infants and young children

The data on weekly dietary intake of lead by infants and young children from 16 countries are presented in Annex 4. The PTWI for lead by infants and children refers to the maximum intake from all sources (Section 3). Thus, not only the median and mean, but even the 90th percentile intakes should be well below 25  $\mu$ g/kg body weight, because some of the young will ingest substantial amounts of lead from paint, dust and soil by hand-to-mouth activities.

One of the highest mean weekly intakes  $(118 \mu g/kg$  body weight) was reported from Austria (Haschke and Steffan, 1981) in a study based on lead levels in selected foods. Higher intakes were noted after introduction of mixed feeding of adult foods such as vegetables at the age of around 4 months. Environmental contamination appeared to be the main reason for the high lead content noted in some of the food, especially vegetables.

The average lead intakes from the more recent surveys are shown in Figure 3. Average intakes slightly below the PTWI were reported from Switzerland and Finland while intakes well over 50% of PTWI were noted in the Germany and Poland. The





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lowest intakes were reported by investigators from the USA, Niger, Zaire and Guatemala.

In Poland, the weekly intake for children living in an industrial area was  $33 \mu g/kg$ body weight, about twice that for children living in a non-industrial area. Mean intakes for infants of around 105  $\mu$ g/kg body weight were obtained in a study in Germany (Mueller and Schmidt, 1983) with high lead content in the drinking water. Likewise, mean infant intakes of around 360  $\mu$ g/kg body weight were obtained in a study conducted in the UK in Ayr, where some dwellings have lead plumbing (Sherlock et al, 1982). This increase in dietary lead results from the water used to dilute dehydrated infant formula and infant cereals as well as that which is consumed directly.

In cases where canned foods such as formula are used, lead-soldered seams can be a major source of lead. Dabeka (1989) reported a mean intake of about 38  $\mu$ g/kg body weight from a diet which included infant formula in lead-soldered cans as compared to about 8  $\mu$ g/kg body weight where non-soldered cans were used. Aside from foods in lead-soldered cans, other major sources of lead in the diet of the very young are cereal products, vegetables and milk.

Very low intakes of around 2-3  $\mu$ g/kg body weight were reported for 0-1 month old infants fed breast milk in studies in Sweden (Larsson et al, 1981) and Canada (Dabeka and McKenzie, 1988). The Canadian investigators noted a correlation of lead level in the breast milk with heavy traffic and with occupancy in older houses (Dabeka g al, 1986).

Very few countries reported intakes for enough years to determine whether a trend has occurred. The data in Figure 4 indicate a probable downtrend from 1980 to 1987 in Australia, as the 1987 data are for 95th percentile consumers. A definite decrease in lead intake has occurred in the USA from 1980 to 1988, even when one takes into account the change in composition of the total diet that occurred during 1982 (Gunderson, 1988). This downtrend coincides with the total conversion in that country to non-soldered containers for infant formula, infant juices, pureed baby foods and evaporated milk (Jelinek, 1982; Capar and Rigsby, 1989) and with a drastic reduction in lead content in petrol.

#### **3.2 Summary Assessment**

Lead intakes near or above the PTWI for adults and infants and young children were reported from both developed and developing countries. A downtrend probably occurred during the conversion from lead-soldered to non-soldered cans and during the phasing out of lead additives in petrol. However, higher dietary intakes occur in industrial areas, in areas of high traffic density, in areas with high lead concentration in tap water and (in the case of adults) in the consumption of wine, probably from bottles with lead caps.

Fewer dietary surveys have been carried out for infants' and children's dietary intakes of lead, especially over continuing periods by a consistent approach. However, a definite decreasing trend was noted in the USA. The lowest intakes among the countries was noted with breast-fed infants. A correlation of lead level in the breast milk occurred



with proximity to heavy traffic and with occupancy in older houses. Lead intake by infants and young children is far in excess of the PTWI of  $25 \mu g/kg$  body weight in localities where tap water contains elevated lead levels.

In view of the relative frequency that median and mean intakes of lead approached or exceeded the PTWI, more countries, both developed and developing, should carry out dietary intake studies for lead. These surveys should be conducted on a continuing basis to determine whether a trend is occurring in intakes on a national basis. More duplicate diet studies should be carried out in areas with heavy industrial activities, especially mining and metal processing, in areas with high lead levels in the tap water and in areas of high traffic density.

Since dietary intakes of lead relative to body weights are highest for infants and young children, who are also especially sensitive to the toxic effects of lead, and since lead passes the placental barrier readily, every effort should be made to reduce the levels of lead in foods for infants and children, in foods consumed by women of child-bearing age and in tap water. In addition, more duplicate diet studies for lead should be conducted with pregnant and nursing women and with infants and young children.

#### **4. CADMIUM**

The major uses of cadmium are in electroplating, as stabilizers for plastics and in pigments. Exposure to cadmium occurs from the working environment, soil and dust, ambient air, drinking water, tobacco and food. Food is the main source of cadmium intake in non-occupationally exposed persons (IARC, 1976). The main sources of cadmium entering food include cadmium-containing soil (Sherlock, 1984), industrial heavy-metal emissions (Auermann et al, 1980), phosphate rock used in fertilizers, the mining and metal industries, the use of municipal sludge and compost on agricultural land, waste incineration and the combustion of fuel. The use of cadmium-plated and galvanized equipment in food processing, cadmium-containing enamel and ceramic glazes, and cadmium-based pigments or stabilizers in plastics may be significant sources of food contamination (WHO, 1992a).

Cadmium has an extremely long half-life in man. The kidney, in particular the kidney cortex, is the target organ in relation to chronic exposure to relatively low levels of cadmium. The first adverse functional change is usually a low molecular weight proteinuria. In 1988, JECFA established a Provisional Tolerable Weeldy Intake for cadmium of  $7 \mu g/kg$  body weight, applicable to adults as well as to infants and children. The PTWI was derived from estimated accumulation over a period of 50 years at an exposure rate equivalent to 1  $\mu$ g/kg body weight/day for adults. Intakes above this figure may therefore be tolerated provided that they are not sustained for long periods of time (WHO, 1989a).

The International Organization for Standardization (ISO) adopted limits of release for cadmium from ceramic foodware, varying from  $0.17 \text{ mg/dm}^2$  for flatware to  $0.25$  -0.50 mg/l of extraction solution for hollow-ware (ISO, 1981).

#### **4.1 Dietary intake of cadmium**

#### 4.1.1 Dietary intake, adults

Data obtained on weekly adult dietary intakes of cadmium by investigators from 24 countries are given in Annex *5.* The average intakes from the more recent surveys in each country are presented in descending order in Figure *5.* As with lead, strict comparisons cannot be made because of such factors as variations in the protocols of surveys and differences in the degree of quality control.

The average intake reported from Thailand  $(17.7 \mu g/kg$  body weight/week) was well above the PTWI. Those from New Zealand and Germany approached the PTWI, while those from Poland, Guatemala, France, Italy, Japan, and the Republic of Korea were around *50%* of the PTWI. At the lower end, the intakes from Finland, the USA, Sweden, Yugoslavia, China, Australia, Turkey and Hungary were near or below  $1 \mu g/kg$ body weight/week.

Canada, Denmark, Finland, the Netherlands and the USA identified cereals and their products, followed by potatoes and other vegetables, as the largest contributors to this intake. Higher cadmium levels are found in animal kidneys, mollusks and crustacea, but these foods constitute much less of the average diet.

Comparatively few countries reported data from nationally representative dietary intake surveys for enough years to determine whether a trend exists. Of those that have, it appears that there has probably been a downtrend in adult dietary intake of cadmium in Japan, the UK and the USA (Figure 6).

In Belgium, it was noted that once-a-week consumption of mussels or kidneys would result in a mean intake that approximates the PTWI (Fouassin and Fondu, 1980). Similarly, it was noted in Denmark that above average consumption of beef kidney, mussels from contaminated water, or wild mushrooms would result in intakes that would exceed the PTWI (Andersen, 1981) In another study, meals which included wild mushrooms or liver led to higher intakes of cadmium (Louekari, et al. 1987).

The intakes of cadmium of those living in industrial areas in Poland and the UK were much higher than those living in a non-industrial area. Similarly, consumption of home-grown vegetables or fruits in an area in Denmark near a lead smelter led to an increased cadmium intake (Andersen, 1981). On the other hand, studies in Belgium, China, Cuba, Poland, Sweden, the UK and Yugoslavia would indicate that urban surroundings per se do not lead to elevated cadmium intakes (Annex 5).

In the UK, a survey was conducted in Shipham after a finding of substantial soil contamination by cadmium had caused concern about its possible effect on the health of the residents. The cadmium is part of the remains of an old zinc mine which had operated from Elizabethan times to the mid-19th century. The results of the survey showed that the dietary intake of the Shipham inhabitants studied was higher than the national average during the season when locally grown produce was consumed. At that time, about 6% of the survey population had estimated intakes higher than the PTWI. By





comparison, none of the households in the study who did not consume Shipham-grown produce had estimated intakes exceeding the PTWI (Morgan et al., 1988).

#### 4.1.2 Dietary intake, infants and young children

Information on intakes of cadmium by infants and young children were obtained from reports and publications from 10 countries (Annex 6), compared to 16 references in the case of lead. The greater interest in ingestion of lead by the very young results from the fact that lead causes intelligence and behavioral problems in this population group. On the other hand, elevated intake of cadmium above the PTWI may be tolerated provided it is not sustained for a long time, since the PTWI is derived from exposure over a period of 50 years (WHO, 1989).

The average weekly intakes of cadmium by infants and young children reported in the more recent surveys by the 10 countries are shown in descending order in Figure 7. The average intake reported from Cuba and Poland exceed the PTWI. The intakes reported from Finland, Germany, the UK, Canada and Niger were around *50%* of the PTWI. The lowest average intake (around 2  $\mu$ g/kg body weight/week), was reported from the USA, the Philippines and Australia. There may have been a slight downtrend in Japan and the USA (Annex 6), even when account is made for the change in study design in the USA in 1982.

The average dietary intakes of adults and of infants from comparable studies in the various countries indicate that those of infants and children are higher on a body weight basis than those of adults. As pointed out before, this must be considered from the standpoint that the PTWI was derived from the accumulation of cadmium over a period of 50 years.

In studies in Canada, the cadmium intake by infants consuming soy-based infant formula was greater than those drinking milk-based formula (Dabeka, 1989). The cadmium intake levels by infants rises appreciably with increasing levels of drinking water and may then exceed the PTWI (Mueller and Schmidt, 1983). Intake of cadmium by the breast-fed infants is low (Larsson et al, 1981; Dabeka and McKenzie, 1988). This would indicate that cadmium does not accumulate in human milk.

In studies carried out in Berlin (Radisch et al, 1987) on the effect of smoking on cadmium levels in human milk, the median level of cadmium was  $0.07 \mu g/kg$  in the milk of non-smoking mothers and  $0.12 \mu g/kg$  in those who smoked 10 cigarettes per day. These results indicate that smoking increased the cadmium content in breast milk, but the resultant level is still less than in infant formula. These levels in human milk agree well with those reported by Canadian investigators (Dabeka et al. 1986; Dabeka and McKenzie, 1988).

#### 4.2 **Summary Assessment**

In many of the reporting countries, the median or mean cadmium intakes constitute an appreciable percentage of the PTWI. Where the 90th percentiles were reported, they approached or exceeded the PTWI in some cases.



**Exposure to** cadmium in the diet is of public health concern. Nationally representative dietary intake studies should be carried out by more countries and appropriate measures should be taken to minimize its occurrence in the diet.

Well-designed duplicate diet studies should be conducted in locations of probable cadmium contamination, such as those near mining and metal extraction operations, phosphate fertilizer plants, high-cadmium bearing strata in the soil, municipal sludge-deposition areas, and shellfish areas affected by improperly treated industrial or municipal discharges. Caution should be observed in consuming meat organs, shellfish, vegetables, fruit and grains from areas of known cadmium contamination, and appropriate measures should be taken to minimize its occurrence in these areas.

#### 5. **MERCURY**

Mercury is used primarily in the chlor-alkali, electrical and paint industries. Inorganic mercury is converted to the much more toxic methylmercury by anaerobic organisms in the aquatic environment, and leads to the contamination of fish with this toxic organometallic compound (Wood et al. 1968). The toxic effects of methylmercury occur primarily in damage to the central nervous system; the fetus and infant are more sensitive to such toxic effects than are adults (GESAMP, 1986; WHO, 1990, 1991a).

JECFA established a Provisional Tolerable Weekly Intake of 300  $\mu$ g, of which no more than 200 **ig should be** present as methylmercury. These amounts are equivalent to  $5 \mu g/kg$  body weight and  $3.3 \mu g/kg$  body weight respectively (WHO, 1972). In 1988, the Committee reassessed methylmercury as new data were only then available on this compound, and confirmed the previously recommended PTWI of 200  $\mu$ g (3.3  $\mu$ g/kg body weight) methylmercury for the general population. Further, it also noted that pregnant women and nursing mothers are likely to be at greater risk to adverse effects from methylmercury. The available data were considered insufficient to recommend a tolerable methylmercury intake specific for this segment of the population (WHO, 1989).

#### **5.1 Dietary intake of mercury**

#### *5.1.1* Dietary intake, adults

Data reported from 16 countries on weekly adult dietary intake were available for assessment. The relevant information is presented in Annex 7. The average mercury intakes from each of the countries are presented in Figure 8. The highest average intakes, from Poland and Denmark, are about 60% of the PTWI of 3.3  $\mu$ g/kg body weight for methylmercury, or about 40% of the PTWI for total mercury. The average intakes from Germany, Guatemala, Belgium, and Cuba, was around *50%* of the PTWI for methylmercury. The lowest average intakes were reported from Finland and Sweden  $(0.27$  and  $0.23 \mu g/kg$  body weight, respectively). In Belgium, the dietary intake exceeded the PTWI for total mercury in about 2% of the participants in the study (Buchet et al, 1983). Not enough data were reported by any country to determine whether there have been any trends in intakes.



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Data from Belgium, Germany, the Netherlands, France, the United Kingdom, Finland and the USA indicate that fish contributes from a high of about *85%* of the total dietary intake of mercury in Finland and the USA to a low of around 20% in Belgium, Germany and the Netherlands, with France and the UK in-between. Thus, the general assumption that fish is the main contributor to the total intake of mercury may not at times be justified.

The mean weekly intake of mercury by the average population in the UK is about  $0.3 \mu$ g/kg body weight. In coastal communities receiving discharges high in mercury, the mean intake was about 1.45  $\mu$ g/kg body weight (Sherlock et al, 1982). The average fish consumption was about 50 g/day, as compared to about 20 g/day for the whole country. About 12% of the survey population had mercury intakes in excess of the PTWI for methylmercury.

#### *5.1.2* Dietary intake, infants and young children

The data available concerning weekly total mercury intakes by infants and children are well within the PTWI of  $5.0 \mu g/kg$  body weight and are given in Table 1. Intakes of total mercury were highest in Poland and Sweden followed by Finland and Zaire. The intakes were much lower in Australia, and the USA. Comparison of the mercury intakes per kg body weight in this table with those for adults in Figure 8 shows that for the young they are equal to or higher than the adults in the countries concerned.

In a survey in Sweden of breast milk, mainly with fishermen's wives who consumed fish from coastal areas of the Baltic Sea, the Gulf of Bothnia or Lake Malar, average levels of 3.1  $\mu$ g/kg total mercury and 0.6  $\mu$ g/kg methylmercury were found in the milk (Skerfving, 1988). Using the standard conversion factor of 0.12 and multiplying by 7 days (See Section 2.3), these levels would be equivalent to an average weekly intake by the infant of 2.6  $\mu$ g/kg body weight of total mercury and 0.5  $\mu$ g/kg body weight of methylmercury for the infants. These intakes would be well within the PTWI's for the total mercury and methylmercury.

#### *5.2* **Summary Assessment**

Even when account is made for the fact that GEMS/Food requested dietary intake data for mercury at a later date than for lead and cadmium, it appears that fewer dietary intake studies have been conducted on mercury than on the other two metals. In the data reviewed from 16 countries, the average adult intake did not exceed the PTWI in any instance. However, in several countries, it amounted to an appreciable portion of the PTWI. In addition, the 90th percentile intake exceeded the PTWI in most studies where they were reported. In view of these results, dietary intake studies should be continued in order to follow up on results obtained to date, and should be carried out in additional countries.

Most of the studies reviewed have been carried out in developed countries. In the results to date, there did not appear to be any correlation with the state of development of the country, but not enough data were available from developing countries to make a valid comparison.



#### **Table 1. Dietary Intake of Total Mercury by Infants and Children**  (FAO/WHO Provisional Tolerable Weekly Intake: 5.0  $\mu$ g/kg body weight)

Not many results were available concerning dietary intakes of mercury by infants and young children, the age group most at risk. However, in all cases the intakes were equal to or higher than for adults.

Representative studies on the dietary intakes of infants and children should be carried out in more countries. In addition, well-designed duplicate portion studies should be conducted in more countries with targeted groups, such as infants, children, pregnant and nursing women, those who consume greater amounts of fish and those living near areas of metal pollution.

#### **6. PESTICIDES**

The GEMS/Food programme has been collecting data on pesticides in foods since 1976. Data on levels of residues of organochlorine and organophosphorus

pesticides have been submitted for a wide variety of foods by a total of twenty nations since 1980. Few countries have submitted data on all the pesticides requested and fewer yet on all the requested food groups. Certainly a major reason for this is the non-usage or, in some cases, trivial usage of pesticides monitored by GEMS/Food in many countries. On the other hand, participation of developing countries is very low, even though in recent years they have been the main users of organochiorine pesticides, which have been banned in many of the developed countries. Data have been submitted to GEMS/Food by even fewer countries on dietary intakes of pesticides.

In comparison to the twenty nations which have reported on levels in foods, only nine countries have submitted data for 1980-1988 dietary intakes of the pesticides selected by GEMS/Food as shown below:



It should be noted that no country submitted intake data for every year. Also, DDT is the only pesticide for which all eight countries submitted data, and the USA is the only country which reported intake data on all thirteen pesticides.

#### **6.1 Organochiorine Pesticides**

In many countries, restrictions have been placed on the use of organochlorine pesticides for non-food as well as food applications, because of their persistence in the fatty tissue in organisms. In foods, they thus occur primarily in milk and dairy products, eggs, meat and animal fat, and fish. Even when they are used only in nonfood applications, they can enter the food supply not only by direct drift onto crops, but they can ultimately contaminate animal-derived foods because of their chemical stability in the soil and waterways.

#### 6.1.1 Aidrin and dieldrin

The use of aidrin as a pesticide has been restricted or banned in many countries. One continuing use is in treatment of soil around buildings for control of termites. Aidrin is readily converted to dieldrin by plants and animals (IARC, 1974; WHO, 1989b). Thus, mainly dieldrin residues are detected in food. The Joint

FAO/WHO Meeting on Pesticide Residues (JMPR) have recommended an Acceptable Daily Intake for aldrin/dieldrin of 0.1  $\mu$ g/kg body weight.

The data on mean daily adult dietary intakes were reported to GEMS/Food by several countries. Published literature provided information on two countries, Egypt (Abel-Gawaad, 1989) and Switzerland (Wuthrich et al, 1985). The 1988 mean intake reported by Egypt was well above the FAO/WHO ADI of 0.1  $\mu$ g/kg body weight and amounted to  $1.36 \mu g/kg$  bw. The mean intake reported from other countries did not exceed *15%* of the ADI in the most recent data from each country.

Of those countries reporting data for more than one year, definite downtrends occurred in the intakes of aldrin/dieldrin from Australia, Guatemala, Japan, Thailand, the UK and the USA, while residues were not detected in the 1984 and 1985 studies in the Netherlands.

Dietary intakes for infants and children were reported to GEMS/Food and also were published in the literature by investigators from Australia (National Health and Medical Research Council, 1990) and the USA (Gartrell et al, 1985a, 1986, 1988; US Food and Drug Administration, 1988, 1989, 1990). These intakes were in the same general range as those for adults. The dietary intakes of aldrin/dieldrin from human milk are given in Table 2.

The estimated median intake from Denmark was about 80% of the **AD!** of 0.1  $\mu$ g/kg body weight, while those for Australia, Germany, Hong Kong, Iraq, Japan, Tunisia and the UK were above the ADI with estimated intakes from Iraq  $(2.28 \mu g/kg)$ body weight) being the highest. In the Australian study, infants in houses which had recently been treated with the pesticide had median intakes of 1.56  $\mu$ g body weight. The pesticide was not detected in the survey in Guatemala.

During the 1980s, the most recent average daily intakes of aldrin/dieldrin for the general population were well below the AD!, with the exception of Egypt in 1988. Downward trends in exposure existed in those countries which reported results for more than one year. In the case of the USA, this continues the downtrend noted in the 1970s as the use of this pesticide was being phased out.

However, the average intakes calculated for infants from human milk were well above or approaching the ADI, with the exception of Guatemala. The highest levels were reported from some developing countries where pesticide-treated houses were studied. In view of these findings, it is important that monitoring for this pesticide in diets and commercial foods be continued, especially in developing countries and that monitoring in human milk be intensified in all countries.

#### 6.1.2 DDT complex

DDT was introduced as an insecticide in the 1940s and was used very extensively during the following decades in agriculture and in public health programmes to control diseases such as malaria, sleeping sickness and yellow fever.



#### **Table 2. Dietary Intake of Aldrin/Dieldrin by Infants from Human Milk**  (FAO/WHO Acceptable Daily Intake:  $0.1 \mu g/kg$  body weight)

Many countries have now banned or restricted the use of DDT, except in some cases where it is needed for the protection of health. Most developed countries and some developing countries, such as China and Thailand, have banned its use in agriculture, whereas it remains one of the main pesticides used in India (Asian Development Bank, 1987). For vector control, DDT is used particularly to protect against endemic and epidemic malaria. The greater cost of alternatives such as malathion or propoxur would result in decreased coverage of control programmes in some countries (WHO, 1979).

The toxicity of DDT and its derivatives has been reviewed by various expert bodies (IARC, 1974; WHO, 1979). DDT and some of its derivatives are stored in adipose tissues which can be followed by excretion in urine and milk. No confirmed ill-effects of DDT have been reported in infants, even in communities where the highest residue levels in human milk have been reported (WHO, 1979). JMPR established an Acceptable Daily Intake (ADI) of 20  $\mu$ g/kg body weight for the DDT complex. However, a recent epidemiology study has suggested that DDT exposure may be related to an increased incidence of pancreate cancer (Garabrant et al, 1992).

The data on adult dietary daily intakes are presented in Table 3. The mean intake reported from Egypt was about 70% of the ADI, much higher than those reported from the other countries. The mean intakes reported from India were about 20% of the ADI. The 1983 study described by Kaphalia et al was a market basket survey of foods representing the average Indian diet. About 88% of the intake was accounted for by wheat flour. High DDT levels were also noted in milk, meat, fish and eggs. The major contribution of wheat flour to the dietary intake of DDT is probably accounted for by the widespread agricultural use of DDT -- DDT and HCH formed more than 70% of the total insecticide usage during the mid-80's (ICS, 1986) -- and by the importance of flour in the diet.

In contrast, the 1981 duplicate diet study carried out in Punjab (Singh et al. 1988) indicated that fatty foods accounted for about *85%* of the dietary intake of DDT. The intake of DDT by vegetarians was essentially the same as for nonvegetarians, probably because cheese is an important item in the diet of the vegetarians studied.

The DDT intakes reported from other countries were far below the AD!, with evidence of a downtrend in these low levels in some of the reporting countries. The intakes of DDT by infants and toddlers, as reported by Australia and the USA (cited in 6.1.1) were at the same general level as for adults.

The intakes of DDT in human milk, as calculated from levels reported to *GEMS/Food* by Denmark, Germany, Guatemala, Japan and the UK, are given in Table 4. In addition, investigators from Australia, Brazil, Finland, Greece, Hong Kong, India, Iraq, Sweden, and Tunisia published data on the levels of DDT in human milk (references noted in Table 4). The median or mean daily intakes of DDT by infants are generally much higher than the adult intakes. However, they are well below the ADI, with the exception of Guatemala, Hong Kong, India, Iraq and Tunisia which reported intakes of 41.4, 47.2, 36.7, 19.2 and 17.4  $\mu$ g/kg body weight, respectively.

In the case of Hong Kong, local production of vegetables accounts for about 40% of the total consumption and persistent pesticides have been known to be employed in agriculture in the Hong Kong area. Seafood is probably another source of persistent pesticides as high concentrations have been found in mussels in that locality.

The samples in India collected in Ahmedabad showed DDT levels higher than those found in most of the earlier studies in India, and probably reflect continued use of persistent pesticides in that country.

The high levels found in Baghdad reflect the use of DDT for malaria control programmes in Iraq for more than 25 years, as well as for some agricultural purposes, before it was banned in 1976.



**Table 3. Dietary Intake of DDT by Adults**  (FAO/WHO Acceptable Daily Intake:  $20 \mu g/kg$  body weight)

In summary, the most recent daily dietary intakes of DDT reported by the different countries from studies of regular diets were less than 1 % of the ADI, with the exception of the Egyptian and Indian studies which reported average intakes of about 70% and 20% of the ADI, respectively. Much higher intakes were estimated





for infants from human milk. The average estimated intakes reported from studies in Tunisia, Iraq, India, Hong Kong and Guatemala ranged from just below the ADI to substantially above, while the intakes reported from other countries were about 10- 30% of the ADI.

These results indicate that DDT and its derivatives were contaminating the food supply directly in countries where DDT is still in use or where it has been
banned only recently, and *it* is entering the food chain from the environment even in countries where its use has been prohibited for some years. In view of these findings, its monitoring should be increased in human milk in all countries and in foods, including total diets, in most of the developing countries.

# 6.1.3 Total HCH isomers

Technical grade hexachiorocyclohexane (HCH) consists of a mixture of alpha-, beta-, gamma-, and delta-isomers (WHO, 1991b). Technical HCH has been banned or severely restricted for use as an insecticide in most developed countries (IARC, 1979) and in some developing countries, e.g., China. In India, however, HCH is one of the major pesticides used (Asian Development Bank, 1987). No FAO/WHO acceptable daily intake has been established for the technical product. However, an ADI of 8  $\mu$ g/kg body weight has been established for the gamma-isomer (lindane) and will be discussed in paragraph 6.1.4.

Table *5* presents the median and mean dietary daily intakes for total HCH isomers. With the exception of findings in India, where intakes of around 2 and 20  $\mu$ g/kg body weight were reported, the mean daily intakes were generally less than  $0.04 \mu g/kg$  body weight. There appears to be a general decline in the intakes found in Guatemala, Japan, the UK and the USA. Intakes for infants and children reported by the USA were about the same as for adults.

In the 1981 study in India (Singh et al, 1988), conducted with vegetarians and non-vegetarians in Punjab, similar intakes were obtained with both population groups. The residues were distributed fairly evenly through the food groups, with the fatty foods contributing higher amounts. The intakes from the foods exclusive to vegetarian diets (peas and cheese) and those exclusive to the non-vegetarian (meat and eggs) were about the same.

In the other Indian study, carried out in Lucknow (Kaphalia et al., 1985), wheat flour accounted for about 97% of the total dietary intake of total HCH. The intakes from both vegetarian and non-vegetarian diets were essentially the same.

The estimated average daily dietary intakes of total HCH isomers by infants from human milk are generally around  $1-5 \mu g/kg$  body weight (Table 6), much higher than the general intake of around  $0.04 \mu g/kg$  reported for all age categories from ordinary diets. With the exception of Yugoslavia (Jan, 1983) and the 1980 Indian survey in Punjab (Kaira and Chawla, 1983), these studies were also cited in Table 4. In the case of the studies near Sao Paulo in Brazil, higher levels were found in rural than in urban areas. This difference was ascribed to the fact that most of the mothers in the rural zone worked in cotton, sugar-cane and coffee plantations that frequently used HCH. In addition, house spraying in the rural zone was usually conducted by public health authorities as part of a campaign, where HCH was more widely used. Much higher estimated dietary intakes of around 22  $\mu$ g/kg body weight in Punjab, 27  $\mu$ g/kg body weight in Ahmedabad, India and 61  $\mu$ g/kg body weight in Hong Kong were reported.



# Table 5. Dietary Intake of Total HCH Isomers by Adults **(No FAOIWHO** Acceptable Daily Intake established)

In view of the higher intakes of total HCH isomers reported from India and Hong Kong, increased monitoring for this insecticide in diets and commercial foods should be carried out in those countries where HCH is still in use or where its use has been discontinued recently. Also, more frequent monitoring for HCH in human milk



# **Table 6. Dietary Intake of Total HCH Isomers by Infants from Human Milk**  (No FAO/WHO Acceptable Daily Intake established)

should be conducted in all countries, but especially in those countries where *it* has been recently employed for agricultural or public health purposes.

# 6.1.4 Gamma-HCH (lindane)

Lindane, consisting of about 99% gamma-HCH, is now restricted in use and is chiefly applied to residential buildings against household pests (WHO, 1991c). FAO/WHO has established an ADI of 8  $\mu$ g/kg body weight for this pesticide.

Investigators from Guatemala, Japan, the Netherlands, Switzerland, the UK and the USA reported mean daily dietary intakes of gamma-HCH which were around 0.1 % or less of the **AD!.** In two studies conducted in India, intakes of around *5 %* 

(Singh et al, 1988) and 38% (Kaphalia et al, 1985) of the ADI were found. Little difference was noted between the dietary intakes from vegetarian and non-vegetarian diets. In Egypt, the reported intake was about *125%* of the ADI (Abdel-Gawaad, al, 1989).

The median or mean estimate daily dietary intakes of gamma-HCH from human milk reported by Brazil, Denmark, Germany, Greece, Hong Kong, India, Japan, and the UK were around 2% or less of the AD!. The estimated intake in Baghdad was around *45%* of the AD!.

With the exception of the data from Egypt, the dietary intakes have been low in the reporting countries during the 1980s, both from ordinary diets and from human milk. Downtrends were noted in the intakes found in some countries. These low levels probably result from the fact that gamma-HCH is only a minor portion of commercial HCH, and the uses for the purified gamma-isomer (lindane) have been restricted in many countries.

# 6.1.5 Hexachlorobenzene (HCB)

Use of HCB as a fungicide, particularly on cereal grains, has almost ceased, but it had been widely used until the 1970s. Contamination of food can arise from the residues of the fungicide and also from HCB emitted by the production and waste disposal operations in the manufacture of such non-pesticidal organochiorine products as perchloroethylene. A temporary ADI of 0.6  $\mu$ g/kg body weight, previously established for HCB, was withdrawn in 1978.

Daily dietary intakes of HCB were submitted to GEMS/Food by Guatemala, Japan, the Netherlands, the UK and the USA. In addition, intakes were reported by investigators from Finland and Switzerland (see references cited in table 3). All mean dietary intakes for adults were less than  $0.025 \mu g/kg$  body weight. Data reported by the USA for intakes by infants and young children were in the same range.

Data on levels of HCB in human milk were submitted to GEMS/Food by Denmark, Germany, Japan and the UK and were also reported by investigators from Australia, Finland, Greece, Hong Kong, Israel, Tunisia and Yugoslavia. The daily dietary intakes of HCB by infants, calculated from these levels, are presented in Table 7.

Intake data from both regular diets and from human milk were reported only by investigators from Finland, Japan and the UK so that only limited comparisons can be made. Nevertheless, it is of interest that the lowest estimated intakes from human milk were around  $0.2$ -0.5  $\mu$ g/kg body weight, and thus were substantially higher than any of those obtained in intake studies of regular diets. It should also be noted that much higher estimated daily average intakes, in the range of  $1-4 \mu g/kg$  body weight, were reported from Australia, Greece, Germany and Tunisia.

Most of the data on dietary intakes of HCB has come from industrial countries. In view of the much higher estimated dietary intakes from human milk as compared to



# **Table** 7. **Dietary Intake of HCB by Infants from Human Milk**  (FAO/WHO Acceptable Daily Intake:  $0.6 \mu g/kg$  body weight (withdrawn)

intakes from commercial foods, increased monitoring for HCB in human milk should be carried out in both developed and developing countries, and in regular diets in countries where HCB is found in significant levels in foods of animal origin. In countries where HCB has been banned or greatly curtailed in the past years, the current sources of this chemical are probably mainly from chemical industries and waste disposal operations.

# 6.1.6 Heptachlor and Heptachlor Epoxide

Heptachior was used widely for seed treatment and on foliage, fruits and cereals up to the early 1970s. Since then, restrictions have been imposed on its usage, primarily to reduce the resultant contamination of milk and animal products. Heptachlor is converted in the environment to heptachlor epoxide (IARC, 1979; WHO, 1984a). The ADI for heptachlor plus its epoxide, previously 0.5  $\mu$ g/kg body weight, was lowered to 0.1  $\mu$ g/kg body weight in 1991 by JMPR.

Australia, Guatemala, Japan, the Netherlands, Thailand and the USA submitted data on dietary intakes of heptachlor and heptachior epoxide to GEMS/Food. In

addition, investigators from Finland and Switzerland reported data in published papers (see references in Table 3) on dietary intakes of this pesticide. The highest mean daily intake for adults was  $0.031 \mu g/kg$  body weight or about 30% of the ADI. Most of the intakes were well below this level. Intakes for infants and young children reported by Australia and the USA were in the same range. Daily intakes of heptachlor plus heptachlor epoxide for infants, calculated from levels in human milk reported from six countries, are presented in Table 8.

# **Table 8. Dietary Intake of Heptachior and its Epoxide by Infants from Human Milk**  (FAO/WHO Acceptable Daily Intake:  $0.1 \mu g/kg$  body weight)



The estimated average intakes from human milk were generally below the ADI with the exception of Australia and Iraq. In the Australian study, the houses of some of the participants had been treated previously with heptachlor.

Almost all the dietary intake data on heptachlor and its epoxide originated from developed countries. The very low dietary intakes of heptachlor and its epoxide from regular diets probably reflect the restrictions or bans on its use enforced in many countries. Nations which do not have restrictions on heptachlor usage or have instituted them in recent years should monitor for it at least in animal-derived foods and conduct dietary intake studies. The higher dietary intakes, as calculated from the pesticide levels in human milk, would indicate that more countries should still plan for appropriate monitoring in this food.

# 6.1.7 Endosulfan

Endosulfan was introduced in the mid-1950s. It is used as a broad-spectrum insecticide to control pests on fruit, vegetables, tea, and on non-food crops such as tobacco and cotton. It is also used in the control of the tse-tse fly. After DDT was banned, its use increased rapidly until the mid-1970s (WHO, 1984b). Data were first requested by GEMS/Food in 1980. FAO/WHO established an ADI of 6  $\mu$ g/kg body weight for endosulfan in 1989.

The available adult dietary intakes for endosulfan are summarized below:



#### *\*95th* percentile consumers.

These results were reported to GEMS/Food except for the data from Switzerland (Wuthrich et al, 1985). These adult intakes from the above countries are appreciably lower than the ADI. The daily intakes reported by Australia and the USA for infants and young children are at the same general level as for adults. No data were submitted to GEMS/Food or were available in the literature on levels of endosulfan in human milk.

The intakes of endosulfan reported were all well below the ADI. However, the data are very limited. Data from more countries and for more years on dietary intake and on levels in human milk would be necessary to determine whether there may be significant contamination, especially in countries where endosulfan has been used recently.

#### 6.1.8 Endrin

Endrin is used as an insecticide chiefly on non-food crops, mainly cotton (WHO, 1992b). GEMS/Food first requested data in 1980. The FAO/WHO ADI for endrin is  $0.2 \mu g/kg$  body weight. The data on mean adult daily intakes are presented in Table 9.

The highest mean intake was reported from Egypt and amounted to 1200% of the ADI. Thailand reported a much lower intake (about 17% of the ADI), while the intakes reported by Guatemala, Switzerland and the USA are much below this. The intakes for infants and children in the USA were in the same general levels as for adults. In 1983, endrin was not detected in human milk in Guatemala.

From the very limited data available, the dietary intakes of endrin are low. However, data are insufficient to make any conclusion. Because a potential exists for very high levels to appear in food, more countries should monitor for residues of endrin.



# **Table 9. Dietary Intake of Endrin by Adults**  (FAO/WHO Acceptable Daily Intake: 0.2 µg/kg body weight)

### 6.1.9 Other organochiorine insecticides

In the studies conducted in various countries, estimated daily dietary intakes were reported for pesticides other than those requested by GEMS/Food. In many cases the levels of particular pesticides are determined by multiresidue methods, which analyze a family of pesticides by the same procedure. Thus, intakes of other pesticides will be determined along with those which have been reported to GEMS/Food. This is the case with the organochiorine insecticides, where intakes have been reported on other pesticides of this category than the nine included in GEMS/Food. In addition, dietary intake studies may be carried out for a selected pesticide whose levels are not determined accurately by a multiresidue method.

In the case of human milk, published data were found only for chlordane, in addition to the pesticides selected by GEMS/Food. These data are summarized below:



The mean calculated chlordane intake in the study in Finland was about 16% of the FAO/WHO ADI, of  $0.5 \mu g/kg$  body weight while the intake from the survey in Australia was 140% of the **AD!.** In this study, however, one of the donors lived in a house which had recently been treated with chlordane.

In regard to dietary intakes of pesticides from regular foods, Part A of Annex 8 lists the mean adult dietary intakes of four other organochiorine insecticides, reported by investigators from Australia, Finland, Japan, Switzerland, Thailand and the USA. All the intakes were less than 2.5% of their respective FAO/WHO ADIs, most of them well under 1%.

This tabulation is not a complete listing of all the other organochlorine insecticides or of all the countries in which such determinations have been carried out. Nevertheless, it does indicate that, in general, the dietary intakes in these countries of organochiorine pesticides other than those selected by GEMS/Food are well below the ADIs set by FAO/WHO.

On the other hand, the sparse amount of data available on levels of other organochlorine pesticides in human milk indicate that elevated levels may occur, especially with insecticides which have been used in or near houses.

## **6.2 Organophosphorus pesticides**

The increasing use of organophosphorus insecticides led to their inclusion in GEMS/Food from 1980 onwards. Organophosphorus pesticides generally are not stable in the environment and are metabolized extensively by animals. Thus, residues would be expected to occur mainly in raw crops, but not in foods of animal origin (WHO, 1986). In addition to dietary intakes, data are requested by GEMS/Food for residues in cereal grains, vegetables and fruits of dietary importance. Few data have been submitted as compared to organochiorine insecticides. As with the organochiorine pesticides, dietary intake data are compared with FAO/WHO AD! values (see Section 2.4).

### 6.2.1 Diazinon

Major uses of diazinon, which was first produced in the early 1950s, have included the control of pests of public health importance and on fruit and vegetables. Major areas of use have included control of pests on maize and alfalfa. The JMPR has established an ADI of 2  $\mu$ g/kg body weight for diazinon.

Only Japan, New Zealand and USA have submitted data to GEMS/Food on daily dietary intakes of diazinon. In addition, investigators from Australia (National Health and Medical Research Council, 1990) and Switzerland (Wuthrich et a1, 1985) reported intake data in the literature. The mean adult intakes were generally below  $0.01 \mu g/kg$  body weight/day. An exception occurred in Japan in 1983, when a mean intake of 0.16  $\mu$ g/kg weight/day was reported because of the inclusion of a heavily contaminated vegetable sample. Daily intakes of this pesticide by infants and children in the USA were also generally below  $0.01 \mu g/kg$  body weight.

#### 6.2.2 Fenitrothion

Fenitrothion is a broad-spectrum insecticide with much lower acute mammalian toxicity than similar insecticides. It has been used widely on fruit, vegetables, grains, tea, coffee and cotton as well as to control public health pests (WHO, 1992c). The FAO/WHO ADI for fenitrothion is  $5 \mu g/kg$  body weight.

Data have been submitted to GEMS/Food by Australia for 1986 and 1987 and the USA for 1988 on daily dietary intakes of fenitrothion. In addition, information has been published in the literature by investigators in Japan on fenitrothion intakes (Matsumoto et al, 1987). The mean dietary intakes reported from Japan and the USA for adults were less than  $0.03 \mu g/kg$  body weight. The mean intake reported by Australia for the 95th percentile consumers was 2.2  $\mu$ g/kg body weight (about 44% of the ADI). Grains and cereal products are the main sources of fenitrothion in the diet in Australia (National Health and Medical Research Council, 1990). The dietary intakes from Australia for infants and young children were in the same range as for adults from that country, while those submitted by the USA were about  $0.003 \mu g/kg$ body weight or less. Fenitrothion continued to be the most frequently detected organophosphorus residue in Australia, and the 1987 intakes were similar to those found in 1986.

#### 6.2.3 Malathion

Malathion was introduced commercially in 1950. It is used as a broadspectrum insecticide and acaricide in a wide variety of agricultural and nonagricultural uses, including the control of lice, flies and mosquitoes. However, it is applied mainly to agricultural crops (IARC, 1983). The FAO/WHO ADI for malathion is 20  $\mu$ g/kg body weight.

Data were reported on the daily dietary intake of malathion to GEMS/Food by Guatemala, Japan, New Zealand, Thailand and the USA, and published in the literature by investigators from Australia and Switzerland (cited in 6.2.1).

The daily intakes in Japan rose slightly from  $0.02 \mu g/kg$  body weight in 1980 to 0.04 in 1984 and then during 1985-1988 decreased to lower levels than previously found. The adult mean intakes submitted to GEMS/Food by the five countries were generally less than 1% of the ADI. The intakes reported by the USA were somewhat higher than those from the other countries. They declined from  $0.24 \mu g/kg$  body weight in 1982 to about 0.1  $\mu$ g/kg in 1988. The mean intakes from the USA for infants and young children were in the same general range.

In summary, the dietary intakes reported from these seven countries were well below the FAO/WHO ADI. The chief source of malathion in the diet studies in Japan and the USA are grains and cereal products (Matsumoto et al, 1987; Gunderson, 1988).

# 6.2.4 Parathion

Parathion (parathion-ethyl) was introduced as a commercial pesticide in 1947. It is used as a broad-spectrum insecticide in agricultural applications (IARC, 1983). The use of parathion has been restricted generally in favor of the somewhat less toxic methyl parathion. An ADI of  $5 \mu g/kg$  body weight has been established for parathion.

Data on daily dietary intakes of parathion were submitted to GEMS/Food by five countries (Guatemala, Japan, New Zealand, Thailand and the USA). Investigators from Australia and Switzerland (cited in 6.2.1) reported data elsewhere.

Mean daily intakes of less than  $0.002 \mu g/kg$  body weight were generally found. Parathion was not detected in surveys reported from Australia, Japan and Switzerland, and in most of the surveys from Guatemala. In the USA the mean dietary intakes for infants and children were in the same general low range as for adults.

#### *6.2.5* Parathion-methyl

Parathion-methyl is used as a broad-spectrum insecticide in a wide variety of agricultural applications. It is particularly valuable in the control of cotton insects (IARC, 1983). The FAO/WHO ADI for parathion-methyl is 20  $\mu$ g/kg body weight.

Daily dietary intakes of parathion-methyl were reported by Guatemala, Japan, Thailand and the USA to GEMS/Food. Dietary intakes of less than  $0.002 \mu g/kg$  body weight/day were found by the USA. Parathion-methyl was not detected in the diets in Japan and Thailand, and it was found in only one of the four studies reported by Guatemala. The dietary intakes for infants and young children in the U\$A were also very low, at the same general levels as for adults.

# 6.2.6 Other organophosphorus pesticides

Part B of Annex 8 lists the mean adult daily dietary intakes reported by investigators from Australia, Japan, Switzerland and the USA for eighteen organophosphorus pesticides not included in GEMS/Food. One reason for the greater number of other organophosphorus as compared to organochlorine pesticides is that, beginning in the early 1970s, developed countries placed restrictions, including outright bans, on the use of the organochlorine pesticides because of concerns about their persistence in the environment, their bioaccumulation in fatty tissues and their adverse effects noted in animal studies. These governmental actions resulted in a halt in the development of new organochlorine insecticides with a subsequent increase in development and use of new organophosphorus pesticides, which are less persistent and which do not accumulate in fatty tissues.

The daily intakes of all the organophosphorus insecticides listed in Annex 8 were less than about 0.1  $\mu$ g/kg body weight and far below their respective FAO/WHO ADI.

#### 6.2.7 Summary Assessment

In the data available for organophosphorus insecticides, the daily dietary intakes were almost always far below the ADIs for the five pesticides included in GEMS/Food and the eighteen reported elsewhere, with the sole exception of the intake for fenitrothion in Australia. In this case, the mean intake was about *45%* of the ADI for the 95th percentile consumers. The USA reported higher intakes of malathion than the other countries, but even so the mean intakes were no more than about 1% of the ADI.

However, very few countries have reported data on dietary intakes of these pesticides, and almost all of these have been developed nations. More countries should be encouraged to conduct dietary intake surveys on these pesticides, especially the developing countries where their use may be on the upswing as organochlorine insecticides are phased out.

## **6.3 Other categories of pesticides**

Annex 8 also presents data on the following categories of pesticides for which daily dietary intakes have been reported in the literature:



The higher intakes were all less than about 1% of the relevant ADIs, and almost all were well below this.

## 7. **POLYCHLORINATED BIPHENYLS (PCBs)**

The commercial production of PCBs started in the 1930s. Because of their high stability and good dielectric properties, the largest uses of PCBs are as dielectric fluids in transformers and capacitors. They have also found application as heat transfer and hydraulic fluids, in the formulation of lubricating and cutting oils, and as plasticizers in paints, copying paper and other products (Van der Kolk, 1984; WHO, 1976; IARC, 1978).

Contamination of edible oil with PCBs led to large-scale intoxications incidents in Japan in 1968 and Taiwan in 1979 (WHO, 1987d). Limited evidence of carcinogenicity to humans was obtained from the incident in Japan as well as from studies of occupationally-exposed populations. However, there is sufficient evidence of PCB carcinogenicity in experimental animals, and as such it is prudent to regard PCBs as presenting a carcinogenic risk to humans (IARC, 1987). Recent evidence suggests that transplacental exposure to PCBs may result in developmental delays and effect congnitive functioning (Yu et al, 1991; Jacobson et al, 1990).

No tolerable PCB intakes for man have been established by FAO/WHO. The United States Food and Drug Administration (U.S. FDA) has suggested a consumption maximum of 1  $\mu$ g/kg body weight per day for adults (Swain, W.R., 1988). It should be noted that PCBs exist as a mixture of congeners which differ in their toxicological properties. Nevertheless, GEMS/Food only monitors total PCB5.

### 7.1 **Dietary intake of PCBs**

## 7.1.1 Dietary intake, adults

The available data from 9 countries on mean daily dietary intakes of PCBs are summarized in Annex 9. Since fish generally contain higher levels of PCBs than any other food category, diets containing higher amounts of fish may be expected to lead to higher PCB intakes.

Only Japan and the USA reported intakes over several years. Both countries reported mean intakes of less than  $0.05 \mu g/kg$  body weight/day over the 1980-1988 period. The USA intakes were an order of magnitude lower than those of Japan, probably because of the lower amount of fish in the USA diet. Very low intakes, similar to those of the USA, were reported from Australia and the United Kingdom while those from Guatemala were somewhat higher. Some of the intakes from Finland, the Netherlands and Switzerland were in the range of 0.1 to 0.2  $\mu$ g/kg body weight/day.

Substantially higher daily intakes in New Zealand were due primarily to the high PCB intake from dairy products (Pickston et al, 1985). In this case, the mean intake of 0.9  $\mu$ g/kg body weight/day approaches the maximum of 1  $\mu$ g/kg body weight suggested by the U.S. FDA, while the daily mean intake of  $1.5 \mu g/kg$  body weight for male teens exceeds it.

#### 7.1.2 Dietary intake, infants and young children

Intakes reported from Australia and the USA for infants and young children were in the same range as those they reported for adults. The average daily intake of PCBs for infants, as estimated from levels in human milk reported from 8 countries, are presented in Table 10. The data from Denmark, Germany, Japan and the UK were submitted to GEMS/Food. The estimated intakes reported from all countries are above the U.S. FDA-suggested consumption maximum of  $1 \mu g/kg$  body weight, except for India where the use of PCBs is greatly restricted (Jani et al. 1988).



**Table 10. Dietary Intake of PCBs by Infants from Human Milk**  (U.S. FDA-suggested daily consumption maximum:  $1\mu g/kg$  body weight)

A mean intake of about 13  $\mu$ g/kg body weight, calculated from data reported from PCB content in the breast milk of Inuit women from the Hudson Bay region of Northern Quebec, was obtained in a study of these Eskimo women and also of women from southern Quebec near the St. Lawrence River (Dewailly et al, 1989). The higher PCB intake in the Inuit population, about 13  $\mu$ g/kg body weight as compared to  $3.1 \mu g/kg$  in the southern Quebec group, is ascribed to the markedly higher consumption of fish and marine mammals by Inuit mothers as compared to mothers living in Southern Canada. No difference was noted between Inuit settlements. A major cause of the presence of PCBs in the Arctic is the long-range atmospheric transport from the more industrialized and populous regions (Norstrom and Muir, 1988). Calculated mean intakes above 10  $\mu$ g/kg body weight were also noted from levels found in Germany. In this connection, it should be noted that high PCB levels have been reported in fish and meat in certain areas of Germany, indicating localized high levels of environmental contamination (UNEP/FAO/WHO, 1988c; Schueler et al, 1985; Anhalt, 1985; Brunn et al, 1985).

Intakes of around 2-3.5  $\mu$ g/kg were calculated from levels reported from Denmark, Finland (Wickstrom et al, 1983), Hong Kong (Ip et al, 1989), Japan and the United Kingdom. Multi-year data from Japan indicated that a slight decrease may have occurred in the more recent years.

# 7.2 **Summary Assessment**

The available information on dietary intake of PCBs by adults is almost exclusively from developed countries. The intakes are usually well below the U.S. FDA guidance figure of 1  $\mu$ g/kg body weight per day. However, only Japan and the USA submitted data over a period of years.

Monitoring of PCBs in animal-derived foods, especially fish, should be carried out in all countries to determine possible sources of this contaminant in the diet, and dietary intake studies should be carried out, including areas of probable high intake of PCB-contaminated food.

The estimated intake of PCBs by the breast-fed infant was usually far in excess of this guidance value in virtually all reporting nations. In view of the high levels of PCBs found in human milk, surveys of this food should be conducted on a more regular basis in both industrialized and developing countries.

## **8. CONCLUSIONS AND RECOMMENDATIONS**

The data collected by GEMS/Food has allowed an overall assessment of the dietary intake of chemical contaminants which indicates that dietary exposures are generally within the maximum levels established by FAO/WHO. However, in certain local areas and for certain groups of high consumers, these maximum levels may be approached or exceeded. In addition, the global picture is far from complete. Many developing countries, especially those introducing new technologies which might lead to unsafe chemical residues in food, need to establish basic monitoring and assessment programmes for at least those contaminants of priority concern. These programmes can supply the essential information necessary for developing effective food control strategies and efficient management of environmental resources.

All countries, including those with active monitoring programmes, are encouraged to participate in GEMS/Food and to share their knowledge and expertise to address common problems of food contamination, many of which go beyond national and regional boundaries. Central to all GEMS/Food activities is the improvement of the analytical quality of the data submitted to the programme. Participating institutions should make every effort to strengthen analytical quality assurance programmes in their laboratories, and make use of the technical assistance offered by GEMS/Food.

## **8.1 Heavy metals**

Participation in the dietary intake studies in GEMS/Food has been much greater for the heavy metals than for the pesticides, probably because there are many more instances where the intakes of the metals (especially lead and cadmium) approached or exceeded the FAO/WHO intake limits. These cases occurred in both the developed and developing countries. In view of these results, more nations should be encouraged to carry out

dietary intake studies for lead, cadmium and mercury. Since the very young are especially vulnerable to the toxic effects of lead and mercury, more countries should also carry out studies on the dietary intakes of these two metals by infants and young children as well as by pregnant and nursing women.

#### **8.2 Organochiorine pesticides**

Far fewer countries have participated in GEMS/Food with respect to dietary intake studies for organochlorine and organophosphorus insecticides than for the metals, and even somewhat less than for PCBs. The participation has been less for the developing countries even though the persistent organochiorine insecticides are still in use or steps to phase them out have been taken only recently. in addition, it is noteworthy that pesticide intake data were available in the open literature from about as many countries as reported to GEMS/Food. This low participation in GEMS/Food may result from the fact that the intakes of pesticides from ordinary diets have almost never approached the ADI. Whether the lack of *GEMS/Food* data from the developed countries is the result of their not carrying out intake studies or of their not reporting results because of lack of a perceived contamination problem is not known.

More countries, especially the developing countries, should be encouraged to carry out dietary intake studies of the organochiorine insecticides. Also, all participating countries should plan for appropriate monitoring of these pesticides in human milk. In the case of the persistent organochlorine insecticides, studies indicate that their intake by breast-fed infants is very often above the ADIs.

# **8.3 Organophosphorus pesticides**

Fewer countries have reported on dietary intakes of the organophosphorus than for the organochiorine pesticides. Even though the intakes have been far below the ADIs, in at least one instance, the intake was about *50%* of the ADI for *95th* percentile consumers. In order to obtain a more representative picture, more countries, developed and developing, should conduct intake studies of these insecticides.

### **8.4 Other pesticides**

Of the data available in the literature on organochiorine and organophosphorus insecticides not included in GEMS/Food and also on other categories of pesticides, the intakes were far below the ADIs. None of these data would indicate that additional pesticides should be included in GEMS/Food at this time. However, information on dietary intakes of pesticides not now included in GEMS/Food should be solicited from GEMS/Food participating institutions and Codex Contact Points to obtain a broader base for establishing priorities.

## 8.5 **Polychlorinated Biphenyls**

Intake studies have been carried out for PCBs in only a few countries, almost exclusively the industrialized nations. Even though the intakes from regular diets were usually well below the U.S. FDA's suggested consumption maximum, the estimated intake of PCBs by the breast-fed infant was usually far in excess of this value.

In view of these results, market basket and duplicate diet studies in areas of probable high intake of PCB-contaminated food should be carried out in both developed and developing countries.

#### **REFERENCES**

**ABEL-GAWAAD, A.A.Z. SHAMS EL DINE, A.** (1989). Insecticide residues in total diet samples. J. Egypt. Soc. Toxicol. 4: 79-89.

ABDULLAH, **M., JAGERSTAD,** M., KOLAR, K., **NORDEN,** A., SCHUTZ, A. and SVENSSON, S. (1983). Essential and toxic inorganic elements in prepared meals -- 24 hour sampling techniques. In: Trace Element Analytical Chemistry in Medicine and Technology, Vol. 2. Bratter, **P.** and Shramel, **P.** (eds). Walter de Gruyter and **Co. Berlin.** 

**AL-OMAR, M. A., ABDUL-JALIL, F. H., AL-OGAILY,** N. A., TAWFIK, S. J. **AND AL-BASSOMY, M. A.** (1986). A followup of maternal milk contamination with organochlorine insecticide residues. Environ. Pollution. Series A 42: 79-91.

**ANDERSEN, A.** (1981). Lead, cadmium, copper and zinc in the Danish diet. Publication No. *52,* Miljoministeriet, Statens Levnesmiddelinstitut, Soborg, DK.

ANHALT G. (1985). Monitoring concerning foods of animal origin under aspects of the consumer risk and the causality. Dtsch. Tieraertzl. Wschr. 92: 283-286.

ASIAN DEVELOPMENT BANK (1987). Handbook on the use of pesticides in the Asian-Pacific Region. Asian Development Bank, Manila, Philippines.

AUERMANN, H. G., DAESSLER, H. **G.,** JACOBI, *I.,* CUMBROWSKI, **J.** and MECKEL, U. (1980). Studies on heavy metal contents of cereals and potatoes. Die Nahrung. 24: *925-927.* 

**BRUNN, H., BERLICH, H.D. & MUELLER, F.J.** (1985). Residues of pesticides and polychiorinated biphenyls in game animals. Bull. Environ, Contam. Toxicol, 34: *527-532.* 

BUCHET, **J. P.,** LAUWERYS, **R., VANDEVOORDE, A.** and PYCKE, **J.** M. (1983). Oral daily intake of cadmium, lead, manganese, copper, chromium, mercury, calcium, zinc and arsenic in Belgium: a duplicate meal study. Fd. Chem. and Toxic. 21: 19-24.

CAPAR, S. G. and **RIGSBY, E. J. (1989).** Survey of lead in canned evaporated milk. J. Assoc. Off, Anal. Chem. 72: 416-417.

CONSO, F., TULLIEZ, M., FAUVARQUES, M. 0. and NENNA, A. D. (1984). Anémie sidéroblastique saturnisme et yin du Bordelais. Ann, Med. Interne. 135: 43- *45.* 

**CONSULTATION OF THE EUROPEAN COOPERATIVE RESEARCH NETWORK ON TRACE ELEMENTS** (1989). Lausanne, Switzerland, *5-8* September, 1989. FAO, Rome.

**DABEKA, R. W.** (1989). Survey of lead, cadmium, cobalt and nickel in infant formulas and evaporated milks and estimation of dietary intakes of the elements by infants 0-12 months old. Sci. Total Environment. 89: 279-289.

**DABEKA, R. W., KAPINSKI, K. F., McKENZIE, A. D. and BAJDIK, C. D. (1986). Survey of lead,** cadmium and fluoride in human milk and correlation with environmental and food factors. Fd. Chem, **Tox.** 24: 913-921.

**DABEKA, R. W., and McKENZIE,** A. D. (1988). Lead and cadmium levels in commercial infant foods and dietary intake by infants 0-1 year old. Food Additives and Contaminants. *5:* 333-342.

**DABEKA, R.** W., McKENZIE, A. **D. and LACROIX,** M. A. (1987). Dietary intakes of lead, cadmium, arsenic and fluoride by Canadian adults: a 24-hour duplicate diet study. Food Additives and **Contaminants. 4: 89-102.** 

**DAVIES, D. I. A., THORNTON, J. M., WATF, E. B., CULBARD, E. B.,**  HARVEY, **P. G., DELVES, H. T., SHERLOCK, J. C., SMART,** G. A., THOMAS, J. F. A. and QUINN, M. J. (1990). Lead intake and blood lead in two-year old U.K. urban children. Sci. Total Environment. 90: 13-29.

DEWAILLY, E., NANTEL, A., **WEBER, J. P. and MEYER, F. (1989). High**  levels of PCBs in breast milk of Inuit women from Arctic Quebec. Bull. Environ, Contam. Toxicol. 43: 641-646.

**EDEL, W., KREMERS, G. J., P1ETERS, J. J. L., SCHUDEBOOM, L. J. and**  STAARINK, T. (1983). Surveillance programme "Man and Nutrition", results up to and including 1980. Ministry of Welfare and Cultural Affairs, Government Printing Office, The Hague.

ELLEN, **G., EGMOND, E.,** VAN LOON, **J. W., SAHERTIAN, E.** T. and TOLSMA, K. (1990). Dietary intakes of some essential and non-essential trace elements, nitrate, nitrite and N-nitrosoamines by Dutch adults; estimated via a 24-hour duplicate portion study. Food Additives and Contaminants. 7: 207-221.

**ERARD, M., MJSEREZ,** A. and **ZIMMERLI, B.** (1982). Infants' exposure to lead, cadmium and selenium by food. Tran. Chim, Aliment, Hyg. 73: 394-411.

FOOD AND DRUG ADMINISTRATION (1988). Food and Drug Administration pesticide program, residues in foods - 1987. J. Assoc. Off, Anal, Chem, 71: *156a-*174a.

FOOD AND DRUG ADMINISTRATION (1989). Food and Drug Administration pesticide program, residues in foods - 1988. J. Assoc. Off, Anal, Chem. 72: 133a-152a.

**FOOD AND DRUG ADMINISTRATION** (1990). Food and Drug Administration **pesticide program, residues in foods - 1989. J. Assoc.** *Off,* Anal, Chem, 73: 127a-146a.

FOUASSIN, A. and FONDU, M. (1990). Evaluation de la teneur moyenne en plomb et en cadmium de la ration alimentaire en Belgique. Arch. Belges de Med. Soc.. Hyg.. Med. du Travail et Med. Legale. 38: *453-467.* 

FYTIANOS, **K., VASIKIOTIS, G.,** WElL, L., KAVLENDIS, E. and LASKADIDIS, N. *(1985).* Preliminary study of organochlorine compounds in milk products, human milk and vegetables. Bull. Environ. Contam. Toxicol. 34: 504-508.

GARABRANT, D.H., HELD, J., LANGHOLZ, B., PETERS, J.M. and MACK, T.M. (1992). DDT and related compounds and risk of pancreatic cancer. J. Natl. Cancer Inst. 84(10): 746-71.

GARTRELL, M. C., CRAUN, J. C., PODREBARAC, **D. S.** and GUNDERSON, E. L. *(1985).* Pesticides, selected elements and other chemicals in infant and toddler total diet samples, October, 1979 - September, 1980. J. Assoc. *Off,* Anal. Chem, 68: 1163-1183.

GARTRELL, M. C., CRAUN, J. C., PODREBARAC, D. S. and GUNDERSON, E. L. (1986). Pesticides, selected elements and other chemicals in infant and toddler total diet samples, October, 1980 - March, 1982. J. Assoc. Off, Anal, Chem, 69: 123-144.

GERMANY (1987). Cadmium in German foods. Intakes of cadmium. Submitted to **WHO.** 

**GESAMP** (1986). Reports and studies No. 28. Review of potentially harmful substances -- arsenic, mercury and selenium.

GUNDERSON, E. L. (1988). FDA Total Diet Study, April, 1982-April 1984, dietary intakes of pesticides, selected elements and other chemicals. J. Assoc. Off, Anal. Chem, 71: 1200-1209.

HASCHKE, F. and STEFFAN, I. (1981). Lead intake with food of young infants in the years 1980/1981. Wiener Klinische Wochenschrift. 93: 613-616.

IARC (1974). IARC monographs on the evaluation of the carcinogenic risk of chemicals to man, some organochiorine pesticides, Lyon.

IARC (1976). IARC monographs of the evaluation of carcinogenic risk of chemicals to man, Vol. 11. Cadmium, nickel, some epoxides, miscellaneous industrial chemicals and general considerations of volatile anaesthetics, Lyon.

IARC (1978). IARC monographs of the evaluation of carcinogenic risk of chemicals to humans, Vol. 18. Polychlorinated biphenyls and terphenyls, Lyon.

IARC (1979). IARC monographs of the evaluation of carcinogenic risk of chemicals to humans, Vol. 20. Some halogenated hydrocarbons, Lyon.

IARC (1980). IARC monographs of the evaluation of carcinogenic risk of chemicals to humans, Vol. 23, some metals and metallic compounds, Lyon.

IARC (1983). IARC monographs of the evaluation of carcinogenic risk of chemicals to humans, Vol. 30, miscellaneous pesticides, Lyon.

IARC (1987). IARC monographs of the evaluation of carcinogenic risks to humans. Supplement 7, overall evaluations of carcinogenicity: An updating of IARC monographs, Vols. 1 to 42, Lyon.

ICS (1986). Demand pattern of pesticides during the seventh five-year plan. In: Indian Chemical Statistics, 1986-87, Ministry of Industry, Government of India, New Delhi: 136-139.

IKEDA, M., WATANABE, T., KOIZUMI, A., FUJITA, H., NAKATSUKA, H. and KASAHARA, M. (1989). Dietary intake of lead among Japanese farmers. Arch. Environ, Health. 44: 23-29.

ISO (1981). International Standard 6486/2. Ceramic ware in contact with food. Release of lead and cadmium - Part 2: Permissible limits, Geneva, ISO.

IP, H. M. H. and PHILLIPS, D. J. H. (1989). Organochiorine residues in human breast milk in Hong Kong. Arch, Environ. Toxicol. 18: 490-494.

JACOBSON, J.L., JACOBSON, S.W., and HUMPHREY, H.E. (1990). Effects of in utero exposure to polychiorinated biphenyls and related contaminants on congnitive functioning in young children. J. Pediatr, 116(1) 38-45.

JAN, J. (1983). Chlorobenzene residues in human fat and milk. Bull. Environ. Contam. Toxicol, 30: 595-599.

JAM, J. P., PATEL, J. S., SHAH, M. P., GUPTA, S. K. and KASHYAP, S. K. (1988). Levels of organochiorine pesticides in human milk in Ahmedabad, India. mt. Arch. Environ, Health. 60: 111-113.

JATHAR, V. S., PENDARKAR, P. B., RAUT, S. J. and PANDAY, V. K. (1981). Intake of lead through food in India. J. Food Sci. and Technology, India. 18: 240- 242.

JELINEK, C. F. (1982). Levels of lead in the United States food supply. I. Assoc. Off, Anal, Chem, 65: 942-946.

JEMAA, Z., SABBAH, S., DRISS, **M. R.** and BOGUERRA, M. L. (1986). Hexachlorbenzene in Tunisian mother's milk, cord blood and foodstuffs. In: Hexachlorobenzene: Proceedings of an International Symposium, Morris, C. P. and Cabral, J. R. P. (eds). IARC Scientific Publication No. 77, International Agency for Research on Cancer, Lyon, 139-142.

KALRA, R. L. and CHAWLA, R. P. (1983). Studies on pesticide residues and monitoring of pesticidal pollution. Final technical report PL-480 Project, Dept. Entomology, Punjab Agricultural University, Ludhiana.

KAPHALIA, B. 5, SIDDIQUI, F. S. and SETH, T. D. *(1985).* Contamination levels in different food items and dietary intake of organochlorine pesticide residues in India. Indian J. Med. Res, 81: 71-78.

LARSSON, B., SLORACH, **S. A.,** HAGMAN, U. and HOFVANDER, Y. (1981). WHO collaborative breast feeding study H. Levels of lead and cadmium in Swedish human milk. Acta Pediatr. Scand. 70: 281-284.

LLERANDI, G. B., GRILLO, M., TORRES, 0. and GARCIA ROCHE, M. 0. (1989). Estimation of the daily intake of cadmium which may be consumed by students 12-17 years old in secondary schools in the city of Havana. Die Nahrung. 33: 315-318.

LOUEKARI, K., JOLKKONEN, L. and VARO, P. (1987). Exposure to cadmium from foods, estimated by analysis and calculation, comparison of methods. Food Additives and Contaminants. 5: 111-117.

LUCAM (LABORATORIO UNTFICADO DE CONTROL DE ALIMENTOS Y MEDICAMENTOS (1989). Dietary intakes of contaminants in Guatemala. Ministry of Public Health and Social Assistance, Guatemala City.

MARZEC, Z. and BULINSKI, R. (1988). Evaluation of cadmium, mercury and lead intake with reproduced diets. Roczn. PZH. 39: 344-348.

MATSUMOTO, H., MURUKAMI, Y., KUWABARA, K., TANAKA, R. and KASHIMOTO, T. (1987). Average daily intake of pesticides and polychlorinated biphenyls in total diet samples in Osaka, Japan. Bull, Environ, Contam. Toxicol. 38: 954-958.

MINISTRY OF AGRICULTURE, FISHERIES AND FOOD (1987). Survey of mercury in food: second supplementary report, London.

MOILANEN, R., PYYSALO, H. and KIMPULAINEN, J. (1986). Average total dietary intakes of organochlorine compounds from the Finnish diet. Z. Lebensm. Unters, Forsch. 182: 484-488.

MUELLER, J. and SCHMIDT, E. H. F. (1983). Heavy metals in the infant diet. In: Health Evaluation of Heavy Metals in Infant Formula and Junior Food. Schmidt, E. H. F. and Hildebrandt, A.G. **(eds).** Springer-Verlag, Berlin, Heidelberg.

MYKKANEN, H., RASANEN, L., AHOLA, M. and KIMPPA, S. (1986). Dietary intakes of mercury, lead, cadmium and arsenic by Finish children. Human Nutrition: Applied Nutrition. 40A: 32-39.

NATIONAL HEALTH AND MEDICAL RESEARCH COUNCIL (1990). The 1987 market basket survey. Australian Government Publishing Service. Canberra: 1-84.

NORSTROM, R. J. and MUIR, D. C. G. (1988). Long range transport of organochiorines in the Arctic and sub-Arctic: Evidence from analysis of marine mammals and fish. In: Toxic Contamination in the Great Lakes. Vol. I:Chronic effects of toxic contaminants in large lakes. Schmidtke, N.W. (ed). Lewis Pub!. Inc., Chelsea, Mich.

PICKSTON, N. L., BREWERTON, H. V., DRYSDALE, J. M., HUGHES, J. T., SMITH, J. M., LOVE, J. L., SUTCLIFFE, **E. R.** and DAVIDSON, F. *(1985).* The New Zealand diet: a survey of elements, pesticides, colours and preservatives. New Zealand J. of Technology. 1: 81-89.

RADISCH, B., LUCK, W. and NAU, H. (1987). Cadmium concentration in milk and blood of smoking mothers. Toxicol. Letters. 36: 147-152.

SANT'ANA, L. S., VASSILIEFF, I. and JOKL, L. (1989). Levels of organochlorine insecticides in milk of mothers from urban and rural areas of Botucatu, SP, Brazil. Bull. Environ, Contam. Toxicol, 42: 911-918.

SCHUELLER, W., BRUNN, H. and MANZ, D. (1985). Pesticides and polychlorinated biphenyls in fish from the Lahn River. Bull, Environ, Contam, Toxicol. 34: 608-616.

SEKITA, H., TAKEDA, M. and UCHIYANA, M. (1985). Studies on the analysis of pesticide residues in foods (XLV). Surveillance of daily intake of chlordane congeners from total diet in 1983. Bull, Nat, Inst. Hyg, Sci, 103: 143-145.

SHERLOCK, J. S. (1984). Cadmium in the foods and the diet. Experientia. 40: 152-156.

SHERLOCK, J. S., LINDSAY, **D. G.,** HISLOP, J. E., EVANS, W. H. and COLLIER, T. R. (1982). Duplication diet study on mercury intake by fish consumers in the United Kingdom. Arch, Environ, Health. 37: 271-278.

SHERLOCK, J. C., PICKFORD, C. J. and WHITE, G. F. (1986). Lead in alcoholic beverages. Food Additives and Contaminants. 3: 347-354.

SHERLOCK, J., SMART, **G.,** FORBES, G. I., MOORE, **M. R.,** PATFERSON, W. **J., RICHARDS, W. N.** and WILSON, T. S. (1982). Assessment of lead intakes and dose-response for a population in Ayr **exposed** to a plumbosolvent water supply. Human Toxicol, 1: 115-122.

SINGH, P. P. and CHAWLA, **R. P.** (1988). Insecticide residues in total diet samples in Punjab, India. Sci. of Total Environ. 76: 139-146.

SKERFVING, S. (1988). Mercury in women exposed to methylmercury through fish consumption, and in their newborn babies and breast milk. Bull. Environ, Contam, Toxicol, 41: 475-482.

SLORACH, S., GUSTAFSON, T.- B., JORHEM, L. and MATTSON, M. (1983). Intakes of lead, cadmium and certain other metals via a typical Swedish weekly diet. Var. Foeda, Suppl. 1: 3-16.

SMART, G. A., SHERLOCK, J. C. and NORMAN, I. A. (1987). Dietary intakes of lead and other metals: a study of young children from an urban population in the UK. Food Additives and Contaminants. 5: 85-93.

STACEY, C. I. and TATUM, T. (1985). House treatment with organochiorine pesticides and their levels in human milk - Perth, Western Australia. Bull, Environ. Contam. Toxicol, 35: 202-208.

SWAIN, **W. R.** (1988). Human health consequences of consumption of fish contaminated with organochiorine compounds. In: Acquatic Toxicology. Malins, D.C. and Jensen, A. (eds). Vol. 11, Elsevier Science Publishers, Amsterdam.

SZYMCZAK, J., REGULSKA, B., ILOW, R. and BIERNAT, J. (1984). Cadmium, lead and mercury levels in meals from canteens for young people. Roczn. PZH. *35:*  328-332.

SZYMCZAK, J., REGULSKA, B., ILOW, R. and ZECHALKO, A. (1987). Cadmium, lead and mercury in daily food rations of children aged 1-7 years. Roczn. PZH. 38: 230-236.

UNEP/FAO/WHO (1981). Analytical Quality Assurance of monitoring data, Geneva, WHO.

UNEP/FAO/WHO (1983). Analytical Quality Assurance II, Geneva, WHO.

UNEP/FAO/WHO (1985a). Guidelines for the study of dietary intakes of chemical contaminants, (WHO offset publication No. 87) (A, F, **E, 5).** Geneva, WHO.

UNEP/FAO/WHO (1985b). Analytical Quality Assurance III, Geneva, WHO.

UNEP/FAO/WHO (1986a). Summary of 1980-83 Monitoring Data, Geneva, WHO.

UNEP/FAO/WHO (1986b). Chemical contaminants in foods: 1980-1983, Geneva, WHO.

UNEP/FAO/WHO (1988a). Summary of 1984-85 Monitoring Data, Geneva, WHO.

UNEPIFAO/WHO (1988b). Analytical Quality Assurance Studies, 1985-1987, Geneva, WHO.

UNEP/FAO/WHO (1988c). Assessment of Chemical Contaminants in Food, Nairobi, UNEP.

UNEP/FAO/WHO (1991a). Summary of 1986-1988 Monitoring Data, Geneva, WHO.

UNEP/FAO/WHO (1991b). Analytical Quality Assurance Studies, 1989-1990, Geneva, WHO.

VAHTER, M., BERGLUND, M., FRIBERG, L., JORHEM, L., LIND, B., SLORACH, S. and AKESSON, A. (1990). Dietary intake of lead and cadmium in Sweden. Var, Foeda. 44: Suppl. 2: 1-16.

VAHTER, M. and SLORACH, S. (1990). Exposure monitoring of lead and cadmium: An international pilot study within the WHO/UNEP Human Exposure Assessment Location (HEAL) Programme. WHO and UNEP, Nairobi.

VAN DER KOLK, J. (1984). Consideration of a Codex approach to contamination of foodstuffs with polychiorinated biphenyls **(PCBs).** *CX/PR* 84/10. Codex Committee on Pesticide Residues.

VAN DOKKUM, **W., DE VOS, R. H., MUYS,** T. and WESSTRA, J. A. (1989). Minerals and trace elements in total diets in the Netherlands. Brit, J. Nutr. 61: *7-15.* 

VONGBUDDHAPITAK, A., SUNGWARANOND, B., THOOPHOM, G. and ATISOOK, K. (1983). Pesticide residues intake in Bangkok. The Bulletin of the Department of Medical Sciences. B.E. 2526. *25:* 131-141.

WATANABE, T., ABE, H., KIDO, K. and IKEDA, M. (1987). Relationship of cadmium levels among blood, urine and diet in a general population. Bull. Environ, Contam, Toxicol, 38: 196-202.

WATANABE, T., CHA, C.W., SONG, D.B. and IKEDA, M. (1987). Pb and Cd levels among Korean populations. Bull. Environ, Contam, Toxicol, 38: 189-195.

WATANABE, T., KOISUMI, A., FUJITA, H., KUMAI, M. and IKEDA, M. *(1985).* Dietary cadmium intakes of farmers in nonpolluted areas in Japan, and the relation with blood lead levels. Environ. Res, 37: 33-43.

WEIGERT, P., MUELLER, J., KLEIN, H., ZUFELDE, **K. R.** and HILLEBRAND, J. (1984). Arsenic, lead, cadmium and mercury in and on foods. ZEBS - Hefte 1/1984: 33-36.

WEISENBERG, E. (1986). Hexachlorobenzene in human milk: a polyhalogenated risk. In: Hexachlorobenzene: Proceedings of an International Symposium. Morris, C.R. and Cabral, J.R.P. (eds). IARC Scientific Publication No. 77, International Agency for Research on Cancer, Lyon, 193-200.

WHO (1972). Toxicological evaluation of certain food additives and the contaminants: mercury, lead and cadmium (Technical Report Series *505),* Geneva, WHO.

WHO (1976). Environmental Health Criteria No. 2: Polychiorinated biphenyls and terphenyls, Geneva, WHO.

WHO (1977). Environmental Health Criteria No. 3: Lead, Geneva, WHO.

WHO (1979). Environmental Health Criteria No. 9: DDT and its derivatives, Geneva, WHO.

WHO (1984a). Environmental Health Criteria No. 32: Heptachlor, Geneva, WHO.

WHO (1984b). Environmental Health Criteria No. 40: Endosulfan, Geneva, WHO.

WHO *(1985).* The quantity and quality of breast milk, Geneva, WHO.

WHO (1986). Evrironmental Health Criteria No. 63: Organophosphorus insecticides: a general introduction, Geneva, WHO.

WHO (1987a). Environmental Health Criteria No. 70: Principles for the safety assessment of food additives and contaminants in food, Geneva, WHO.

WHO (1987b). Toxicological evaluation of certain food additives and contaminants (Technical Report Series *751),* Geneva, WHO.

WHO (1987c). Toxicological evaluation of certain food additives and contaminants. WHO Food Additives Series: 21, Cambridge, Cambridge University Press.

WHO (1987d). PCBs, PCDDs and PCDFs: Prevention and control of accidental and environmental exposures, Copenhagen, WHO Regional Office for Europe.

WHO (1989a). Toxicological evaluation of certain food additives and contaminants. WHO Food Additives Series: 24, Cambridge, Cambridge University Press.

WHO (1989b). Environmental Health Criteria No. 91: Aldrin and dieldrin, Geneva, WHO.

WHO (1990). Environmental Health Criteria No. 101: Methylmercury, Geneva, WHO.

WHO (1991a). Environmental Health Criteria No. 101: Inorganic mercury, Geneva, WHO.

WHO (1991b). Environmental Health Criteria No. 123: Alpha- and betahexachiorocyclohexane, Geneva, WHO.

WHO (1991c). Environmental Health Criteria No. 124: Lindane, Geneva, WHO.

WHO (1992a). Environmental Health Criteria No. 134: Cadmium, Geneva, WHO.

WHO (1992b). Environmental Health Criteria No. 130: Endrin, Geneva, WHO.

WICKSTROM, K., PYYSALO, H. and SIMES, M. A. (1983). Levels of chiordane, hexachlorobenzene and DDT compounds in Finnish human milk in 1982. Bull. Environ. Contam, Toxicol. 31: *251-256.* 

WOOD, J. M., KENNEDY, F. S. and ROSEN, C. G. (1968). The synthesis of methylmercury compounds by methanogenic bacteria. Nature. 220: 173-174.

WUTHRICH, C., MUELLER, F., BLASER, 0. and MARER, B. *(1985).* Pesticides and other chemical residues in Swiss diet samples. Mitt. Gebiete Lebensm. Hyg. 76: 260-276.

YENGAR, G.V. and PARR, R.M. (1985). Trace element considerations in human milk from several global regions. Int. Workshop on Composition and Physiological Properties of Human Milk, Kiel, Germany.

YU, M.L., HSU, B.C., BLADEN, B.C. and ROGAN, W.J. (1991). In utero PCD/PCDF exposure: relation of developmental delay to dysmorphology and dose. Neurotoxicol. Teratol, 13(2): 195-202.

# **Countries with Institutions Participating in GEMS/Food**



Characteristics of Various National Studies on the Intake of Contaminants Characteristics of Various National Studies on the Intake of Contaminants



Annex 2

# **Dietary Intake of Lead by Adults**  (FAO/WHO Provisional Tolerable Weekly Intake: 50 μg/kg body weight)







# **Annex 3 (Continued)**



'GEMS/Food unless otherwise referenced. b Non-smoking adult females, duplicate diet.

Geometric mean. Three-fold decrease from 1978.

Maximum.

**Dietary Intake of Lead by Infants and Children**  (FAO/WHO Provisional Tolerable Weekly Intake: 25 µg/kg body weight)



Intake  $(\mu$ g/kg bw/wk)

Country	Year	Age	Med.	<b>Mean</b>	90th perc.	Reference <sup>®</sup> /Remarks
Niger	1983	3 mo.	3.6			
Philippines	1983	3 mo.	12.8			
Poland	1984	1-3 yr.	5.4	33.4	96.6	<b>Industrial</b> area x
	1984	1-3 yr.	2.7	17.8	61.3	Non-industrial area
	1985	$1-3$ yr.	14.6	32.3	85.4	Industrial area
	1984-85	$1-3$ yr.		20.0		Szymczak et al (1987). Wroclaw
Sweden	1979	3 mo.		1.7		Larsson et al (1981). Breast-fed
	1983	3 mo.	14.1			Yengar & Parr (1985)
Switzerland	1982	10 mo.		23.5		Erard et al (1982)
UK	1981	$<$ 4 mo.	226.1	361.2	807.8	Sherlock et al (1982). High-Pb H <sub>2</sub> 0, Ayr
	1982	3.7 yr.	6.0	7.3	15.9	
	1982	4 yr.	5.6	6.8	16.1	Asian non-vegetarian
	1982	3.6 yr.	8.3	8.9	15.4	Caucasian non vegetarian
	1982	3.4 yr.	6.36	6.83	12.6	
	1985	2 уг.	12.4	15.3	24.9	Smart et al (1987); Davies et al (1990). Birmingham
<b>USA</b>	1980	6 mo.		29.0		
	1982	6 mo.		16.8		
	1985	6-11 mo.		12.2		New study design
	1986	6-11 mo.		5.0		
	1987	6-11 mo.		3.6		
	1986-88	6-11 mo.		3.2		
Zaire	1983	3 mo.		2.7		

Intake (µg/kg bw/wk)

**'GEMSIFOOd unless otherwise referenced.** 

**<sup>b</sup>Non-lead-soldered cans.** 

**Lead-soldered cans.** 

# **Dietary Intake of Cadmium by Adults**  (FAO/WHO Provisional Tolerable Weekly Intake:  $7 \mu g/kg$  body weight)







 $\hat{\mathcal{D}}$
### Annex *5* **(Continued)**



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Intake  $(\mu$ g/kg bw/wk)

'GEMS/Food unless otherwise referenced.

<sup>b</sup> Non-smoking females.

<sup>e</sup> Geometric means

<sup>d</sup> Maximum intake.

**Dietary Intake of Cadmium by Infants and Children (FAOIWHO Provisional Tolerable** Weekly **Intake: 7 pg/kg body weight)** 



Intake  $(\mu$ g/kg bw/wk)

Country	Year	Age	Med.	<b>Mean</b>	90th perc.	<b>Reference</b> <sup>a</sup> /Remarks
Sweden	1983	3 mo.		0.1		Larsson et al (1981). Breast-fed
UK	1982	3.7 yr.	2.0	2.0	2.7	
	1982	4 уг.	1.9	1.9	3.1	Asian non-vegetarian
	1982	3.6 yr.	1.8	2.4	3.6	Caucasian non-vegetarian
	1982	3.4 yr.	2.5	2.5	3.8	
	1985	2 yr.	2.7	2.9	3.7	Smart et al (1987). Birmingham
<b>USA</b>	1980	6 mo.		9.8		
	1982	6 mo.		9.4		
	1985	6-11 mo.		3.5		New study design
	1986	6-11 mo.		2.9		
	1987	6-11 mo.		2.2		
	1986-88	6-11 mo.		2.3		

Intake (µg/kg bw/wk)

'GEMSIFOOd unless otherwise referenced.

#### **Dietary Intake of Mercury by Adults (FAO/WHO Provisional Tolerable Weekly Intake: Total Mercury -5 pg/kg body weight Methylmercury - 3.3 pg/kg body weight)**



### **Annex** 7 **(Continued)**



\* GEMS/Foodunless otherwise referenced.

b Maximum.

#### **Dietary Intakes of Pesticides not Included in GEMS/Food by Adults**  (All **values given in pg/kg** body weight/day)

# A. Organochlorine Insecticides



# B. Organophosphorus Insecticides



# C. **Fungicides**



# D. N-Methyl Carbamates



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#### E. Pyrethroid Insecticides



### F. Substituted Urea Insectides



#### G. Other



'Australia - 1987 **data. (National Health and Medical Research Council,** 1990).

<sup>b</sup> Finland - 1984 data. (Moilanen et al, 1986).

<sup>c</sup> Japan - 1983 data for chlordane (Sekita et al, 1987). 1985 data for other pesticides: Osaka study (Matsumoto et al, 1987).

<sup>d</sup> Switzerland - 1983 data. Berne region (Wuthrich et al. 1987).

'Thailand - 1980 data. Bangkok study crhailand to FAO/WHO, 1984).

USA - Highest intake from 1987, 1988, 1989 (Food and Drug Administration, 1987, 1988, and 1989).

AD! not established.

<sup>h</sup> Includes other related chemicals.

Temporary ADI.

Parent chemical only.

k Temporary AD! withdrawn

**Annex 9** 





\* GEMS/Foodunless otherwise referenced.

b ND: Not detected