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Trends in diet and exposure to chemicals in Dutch children

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Summary

This report presents an overview of trends in dietary patterns and exposure to selected chemicals in children aged 0.5-12 years living in the Netherlands, with the aim to get insight in possible trends in exposure over the last two decades. The compounds included are acrylamide, dioxins, nitrate, organophosphate insecticides, sulphite, sweeteners and some mycotoxins. For this data from literature and the three Dutch National Food Consumption Surveys (DNFCS-1 (1987/1988), DNFCS-2 (1992) and DNFCS-3 (1997/1998)) were used. The reported consumption of very young children (aged 8-18 months) was described based on two studies conducted in 2000/2001 and 2002. Due to the relatively short time period between these two latter studies, it was not feasible to report of a time trend for very small children.

For most of the chemicals a decrease in (mean/median) exposure levels is predicted relative to the most recent exposure assessments reported in the literature. For sweeteners, an increase is more likely, due to an increasing trend in the consumption of light soda's. For some compounds, (T_2/HT_2 and sulphite) no conclusions could be drawn.

The results are shortly discussed in relation to differences in food consumption survey methods used over time and developments of methodologies used to assess exposure levels. Also the quality of the residue data used in the exposure assessment is addressed.

Trends in themselves provide only information on whether exposure levels are likely to increase or decrease in time. Also actual levels of exposure should be addressed and compared to the relevant toxicological reference levels. The comparison of the highest, most recent estimated exposure reported in the literature with a relevant reference dose per chemical demonstrated that for some compounds (acrylamide, DON and dioxins) the reference value was exceeded and that it remains to be seen whether a decreasing trend will result in acceptable exposure levels in children in time.

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1 Introduction

Risks related to chemicals in food, especially in relation to health effects in children, have been extensively discussed. It is known that children have higher food and water consumption levels compared to adults when expressed per kg body weight (National Research Council, 1993; SCF 1998), resulting in relatively higher exposure levels to toxic compounds. Also specific dietary patterns of children may contribute to a higher exposure to food contaminants. Apart from higher exposure levels, the physiology of children differs from that of adults. This is especially important for children under 1 year, since their enzymatic activity and therefore their ability to break down chemical compounds is reduced. These toxicokinetic differences largely disappear after the age of 1. Older children may even have a higher metabolism than adults. Overall children can either be more or less sensitive to certain compounds than adults, which will depend on the compound itself and the developing phase of the child (Health Council of the Netherlands, 2004). Due to these differences between young children and adults, internationally established chemical risk assessments procedures may need improvement when dealing with young children.

In 2006 a new food consumption survey has been conducted in the Netherlands among children aged 2-6 years (the so-called ‘VCP-kinderen’). With the results of this survey, exposure assessments will be performed for a selected number of compounds resulting in an insight in the present variation of daily exposure to these compounds. The exposure calculations are planned for the second half of 2007 and will include nitrate, organophosphate insecticides, sweeteners, mycotoxins (DON, T_2/HT_2, patulin, aflatoxin B_1, ochratoxin A), sulphite, acrylamide and dioxins.

The aim of this study is to get insight in possible trends in the dietary exposure to these chemicals in children aged 0.5 to 12 years over the last two decades. For this a literature study was performed, studying first changes in dietary consumption patterns of foods contributing largely to the exposure of these compounds, because trends in these patterns may influence chemical exposure. The main data used for this purpose were those of the three first Dutch National Food Consumption Surveys conducted (DNFCS) in the Netherlands which included children aged 1-12 years: DNFCS-1 (1987/1988), DNFCS-2 (1992) and DNFCS-3 (1997/1998). Two other studies, conducted in 2000/2001 and 2002, investigated dietary consumption patterns of children aged 8-12 months, and 9, 12 and 18 months respectively. Of these surveys the present consumption is reported. Due to the relatively short time period between these two studies, it was not feasible to report a time trend for very young children. Trends in exposure to the various chemicals were studied based on literature and reports on exposure calculations for Dutch children.

This report describes the results of a limited study into trends in dietary exposure and eating habits. Because of the limited time period, we did not perform new exposure assessments and the discussion of the results and the effect of differences in methodologies used (e.g. exposure calculations, food consumption surveys, contaminant/residue analyses) on the observed trends was kept limited.

2 Methods

2.1 Selection of foods

Based on general knowledge, we selected foods and food groups that were expected to contribute most to the selected chemicals mentioned in Chapter 1 (Table 2.1).

Table 2.1. Selected compounds and corresponding food groups studied for trends in dietary patterns.

Compound	Food (groups)
Acrylamide	Crisps, chips, biscuits, Dutch honey cake
Aflatoxin B_1	Peanut butter
Dioxins	Animal products
DON, T_2/HT_2, OTA	Wheat, bread, breakfast cereals, wheat
Nitrate	Leafy vegetables
Organophosphate insecticides	Vegetables, fruit
Patulin	Apple juice, apple sauce/compote
Sulphite	Potatoes, dried fruit
Sweeteners	Light soft drinks, lemonade

When other foods or food groups not listed in Table 2.1 were identified in the literature research as important contributors of the chemicals listed, they were also taken into account.

2.2 Food consumption data

To study trends in food consumption of selected foods in children living in the Netherlands results of three food consumption surveys were used. These surveys include the three Dutch National Food Consumption Surveys (DNFCS) conducted in 1987 – 1998, 1992 and 1997 – 1998 (Anonymous, 1988, 1993, 1998), a research conducted in 2000 – 2001 among infants aged 8 – 12 months (EU baby research; Boon et al., 2004) and a survey conducted in 2002 among infants aged 9 and 12 months and toddlers aged 18 months (VIO = Voedingsstoffen Inname Onderzoek; De Boer et al., 2006). Below we give a description of these food consumption surveys.

2.2.1 Dutch National Food Consumption Surveys

The Dutch National Food Consumption Surveys (DNFCS) are comprehensive surveys of food consumption in representative samples of the Dutch population (sample size ca. 6000). The first DNFCS was conducted in 1987-1988 by order of the Ministry of Public Health, Welfare and Sports and the Ministry of Agriculture, Fisheries and Nature Management. That survey was followed by DNFCS-2 in 1992 and DNFCS-3 in 1997-1998. The Netherlands Nutrition Centre (Voedingscentrum) commissioned DNFCS-3. DNFCS respondents came from a representative panel of households with a main housekeeper younger than 75 years. In DNFCS-3, the sample was extended with individuals living in households with a main housekeeper aged 75 or over. Institutionalized people, individuals with insufficient command of the Dutch language and infants under age 1 were excluded from the surveys. The food consumption data were collected through a 2-day dietary record method on two consecutive days. The foods consumed indoors were recorded in a household diary for all members of the household by the person usually engaged in preparation of the meals. Outdoor consumption was recorded by every participant in a personal diary (children younger than 13 years were assisted by a parent). In each survey food consumption data were collected during 40 weeks per year. The days were evenly distributed over the season and the days of the week. No field work was conducted during (public) holidays because low response levels were expected on these days and periods. Specially trained dieticians were responsible for the field work, including contacting participants, instructions regarding completion of the diaries, checks on completeness of data and estimates of household utensils (common household measures and foods regularly used were weighed), and coding the data.

In all three surveys food frequency questionnaires (FFQ) were administered. In these questionnaires the frequency of consumption and the number of portions was asked for various food or food groups. A standard portion size was used to estimate the amount consumed. In DNFCS-1 and DNFCS-2 the questionnaire was administered among the total population, while in DNFCS-3 two questionnaires, one about foods of animal origin and one of vegetable origin, were administered among a subpopulation.

DNFCS-3 provides the most up-to-date information on consumption of foods and the calculated intake of energy and nutrients for the total Dutch population including children. The three surveys conducted provide information over a period of 10 years, ranging from 1986/87 up to 1997/98.

2.2.2 VIO

VIO is a food consumption survey in a stratified random sample of Dutch infants (9 months of age) and toddlers of 12 and 18 months of age (De Boer et al., 2006). Children were included when their birth weight exceeded 2500 g, their gestational age at birth was at least 38 weeks and no birth defects or inborn diseases were present. Due to practical reasons breastfed children and children with parent(s) or carers not fluent in Dutch were excluded from the study. The present study population comprised a total of 941 children (response rate 82.5%). Final sample sizes per age were 333 infants of 9 months, 306 infants of 12 months and 302 toddlers of 18 months.

Parents or carers were asked to record all foods and drinks the infant or toddler consumed in a pre-structured diary. They were given written instruction on how to record and describe in detail the consumption of food and beverages, and especially to note all foods wasted and leftovers. The diary included different eating occasions and the amounts consumed could be reported by using household measures, units, grams and ml. To assist participants in accurately estimating quantities of foods consumed also two different (standard) spoons, a measuring jug and a set of photographs to assess the amount of spreading and cooking fats consumed was provided. If children were in day care, parents asked the carers present to report the child's food consumption in the diary. Parents were asked to record the consumption of their child on two non-consecutive days, one being a weekday and one a weekend day. Body weight was recorded as the weight measured during the last visit at the child health centre.

2.2.3 EU baby research

The EU baby research is a study into the food consumption habits of young infants (Boon et al., 2004). In this study parents or carers of 373 infants aged 8 – 12 months were asked to record all the foods consumed by the children during one day of the week. Amounts consumed were weighed accurately. The survey was conducted between September 2000 and September 2001. During this survey emphasis was placed on the collection of data on brand names, ingredients, processing practices and cooking recipes. Breastfed children were excluded from the survey due to practical reasons. The survey was distributed equally over the 7 days of the week and over 1 year (excluding holidays). Body weight was recorded as the weight measured during the last visit at the child health centre prior to the collection day and was corrected for the days between the visit and the measurement.

Apart from recording the food consumption in a food diary during one day, carers or parents of the children were requested to prepare a duplicate portion of all meals consumed by the infant during the study day. Duplicate portions were collected in two different clean, leak–proof 1 L polyethylene containers. These duplicate portions were among others used to assess the actual exposure to DON, T_2/HT_2 and 18 pesticides.

Results of this study have been reported by Boon et al. (2004). However, in this report the consumption levels of the infants were reported at the level of the raw agricultural commodity. No consumption levels at food or food group level were reported. For the description of the food consumption levels of the infants in this report and to be able to compare these levels with those reported in the VIO study, we analysed the EU baby research data anew at the level of food (groups). These results have not been published elsewhere and were especially generated for this report.

2.3 Food conversion table

To assess the trend in consumption of wheat (relevant for the exposure to DON, T_2, HT_2 and OTA reported here), we used the conversion model developed at RIKILT – Institute of Food Safety (Van Dooren et al., 1995). With this model foods as consumed are translated to the consumption of raw agricultural commodities based on several sources of information, including recipes of cookery books, and information from either the literature or product label. So for example, the consumption of 100 g of the food product ‘bread, whole meal’ is converted to the consumption of 60 g raw ‘wheat’.

2.4 Exposure calculations

Results of the exposure assessments reported here are derived from the literature and reports. Three types of exposures will be cited, namely short-term (= acute) exposure, long-term (= chronic) exposure, and mean (observed) exposure. **Short term exposure** relates to exposure during one arbitrary day, and is calculated by multiplying individual daily food consumption patterns with randomly selected concentration levels out of a range of levels per food. Summing over foods per day results in an estimate of the short-term daily exposure distribution for a certain chemical. For the estimation of the **long-term exposure**, all daily consumption patterns are multiplied with the average level of the contaminant per food, and summed over foods per day. This results in a set of daily mean exposure levels, which are then analysed with statistical models for long-term exposure, such as the method of Nusser et al. (1996) and of Slob (1993; for more details see Annex 1).

Apart from long-term and short-term exposure also **mean (observed) exposure** levels are reported. In these assessments mean daily exposure levels are calculated. No attempt is however made to extrapolate these estimates to long-term exposure levels. For the long-term exposure the interest is in the variation between the individuals, not between the two days for the same individual. Therefore, a distribution of the long-term exposure will be considerably narrower than the distribution of daily mean exposure. Mean daily exposure levels tend therefore to overestimate long-term exposure levels (see e.g. Van Klaveren et al., 2006).

3 Results

3.1 Consumption patterns

3.1.1 Overall trends in consumption patterns in children based on DNFCS data

The three DNFCS were used to provide information regarding trends in food consumption patterns of children living in the Netherlands. The results of these three surveys were reported in separate reports (Anonymous, 1988, 1993, 1998). These reports can however not be used as such to describe trends in consumption over time, due to differences in coding systems used. In 1998, a report was published describing the trend in consumption patterns of food groups reported in the three surveys taking into account these differences in coding (Hulshof et al., 1998). This trend report was used to examine a possible trend in the consumption of specific food groups important for the exposure to at least one of the studied chemicals (Table 2.1). It should be noted that the difference in food group consumptions between the three DNFCS was not statistically analysed in that report.

In Figure 3.1 the trends in mean consumption levels of specific food groups are presented for the different age groups (for the actual levels of consumption, see Annex 2). For some food groups there was a visible decreasing trend in consumption, such as for potatoes, fruits and vegetables. For other groups, such as non-alcoholic drinks, an increasing trend in consumption was visible or only within a certain age group, e.g. for 'pastry and cookies' and 'nuts, seeds and snacks' both in the age group 10-12 and 'fish' for the age groups 4-6 and 7-9 years. However, all these trends are, due to the high levels of the standard deviations (Annex 2), not likely to be statistically significant.

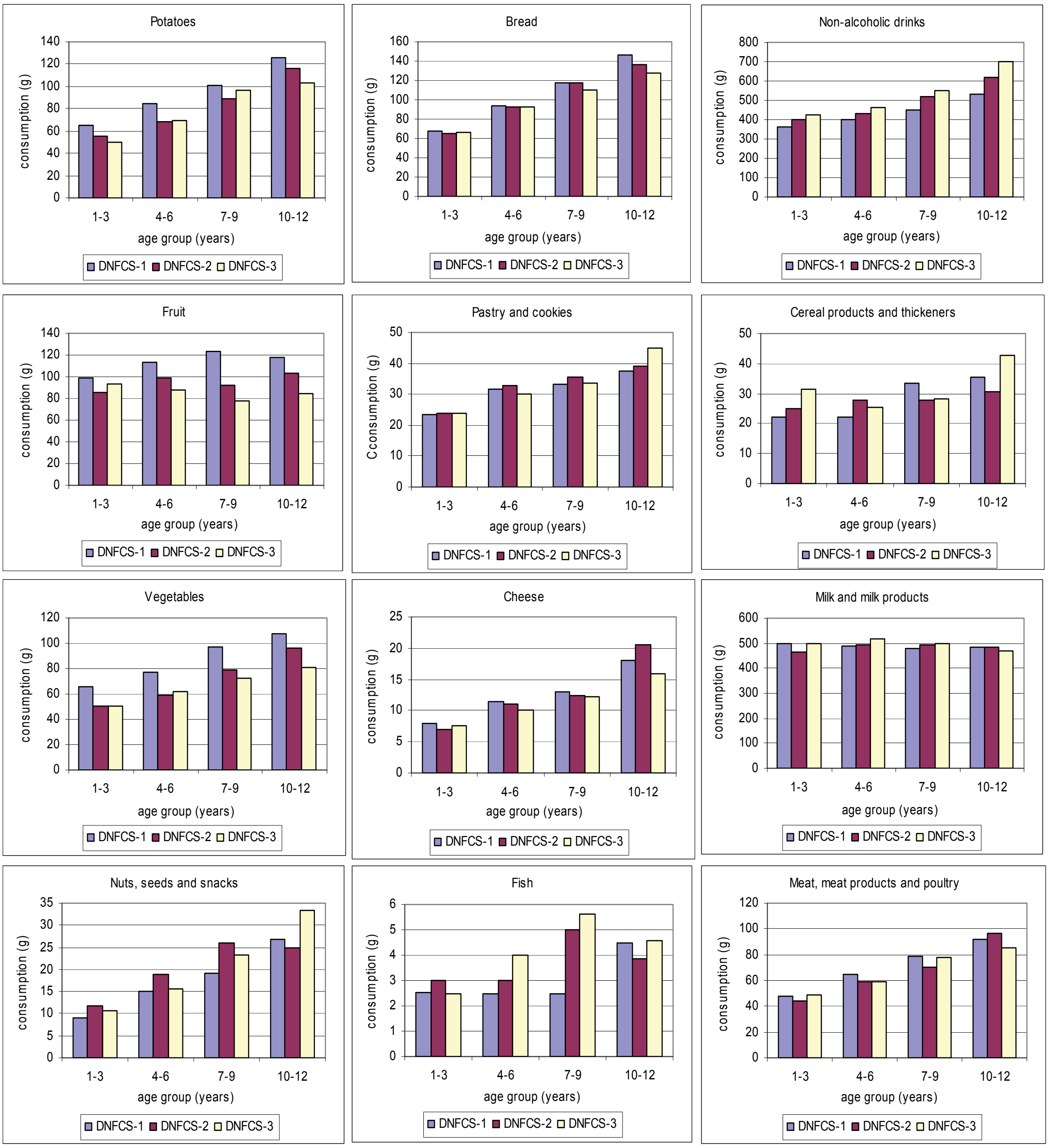

Figure 3.1. Trends in mean consumption (g per day) for various food groups for different age groups, including non-users. Data were obtained from three subsequent Dutch National Food Consumption Surveys (DNFCS; Hulshof et al., 1998).

3.1.2 Consumption patterns in infants and toddlers

As mentioned in Chapter 2, two studies have been conducted among young children aged 8-12 months (Boon et al., 2004) and 9, 12 and 18 months (De Boer et al., 2006). A brief description of actual levels of food consumption reported in both studies for the relevant food groups is given in Table 3.1.

Table 3.1 Mean consumption of different food groups in g per day for young children, including non-users[1]. Data were obtained from the VIO (De Boer et al., 2006) and EU baby research (Boon et al., 2004).

	VIO			EU baby research
Food groups	9 months (N=333)	12 months (N=306)	18 months (N=302)	8 – 12 months (N=373)
Potatoes	21	24	33	29
Bread	27	41	51	24
Non-alcoholic drinks[2]	133	213	387	528
Fruit	168	161	148	118
Pastry and cookies	9	15	25	7
Cereal products and thickeners	19	21	21	17
Vegetables	26	32	37	37
Cheese	6	7	8	4
Milk and milk products	630	608	548	142
Nuts, seeds and snacks	1	2	4	0.1
Fish	1	1	2	1
Meat, meat products and poultry	12	18	29	9

[1] *Estimating the consumption of food groups including the non-users indicates that the food consumption data of all participants is used, even when they did not consume a food from the specific food group.*

[2] *Including water, tea and coffee*

Table 3.1 shows that there is a higher milk consumption and lower consumption of non-alcoholic drinks, mainly water, in the VIO study compared to the EU baby research. An explanation for this difference is that, while in the EU baby research the consumption of infant formula was coded separately as formula and water, in the VIO study this was coded as milk.

In the VIO study a significant increase or decrease in consumption levels with age were detected for all food groups at a significance level (p) below 0.05. For most food groups the consumption was either lower or equal in both infant/toddler studies compared to the 1-3 year group of the three DNFCS, except for milk and milk products (including infant formula) and fruit which both tended to be higher in the infant/toddler surveys.

3.2 Selected chemicals

3.2.1 *Acrylamide*

Introduction

In 2002 acrylamide was found to be present in high concentrations in heat-treated food products rich in carbohydrates (Tareke et al., 2002), which has led to worldwide concern due to its very likely carcinogenic effects (IARC, 1994). This has triggered the interest in analysing levels of acrylamide in al kinds of foods and calculation of the corresponding exposure levels (Bilau et al., 2003; Boon et al., 2005; Dybing et al., 2005; Konings et al., 2003; Svensson et al., 2003).

Trends in consumption

Exposure calculations of acrylamide in the Dutch population and children aged 1-6 years performed in 2002 demonstrated that foods with the highest contribution to the exposure were crisps, chips, snacks and gingerbread (Konings et al., 2003). Besides Dutch honey cake (or gingerbread), also biscuits were shown to be an important source of acrylamide with a contribution of 17% to the total acrylamide exposure in children aged 1-6 years (Boon et al., 2005).

The food groups to which these foods belong are ‘pastry and cookies’ and ‘nuts, seeds and snacks’. Chips belong to the group ‘potatoes’. These food groups are however too broad to examine the trend in specific foods relevant for acrylamide exposure. We therefore retrieved consumption data on crisps, chips, Dutch honey cake and cookies from the three DNFCS and the EU baby research databases. For the results of the three DNFCS see Figure 3.2. Please notice that these trends are not corrected for changes in food coding systems used in the surveys. There is no clear trend, either increasing or decreasing, in consumption levels of these four food products.

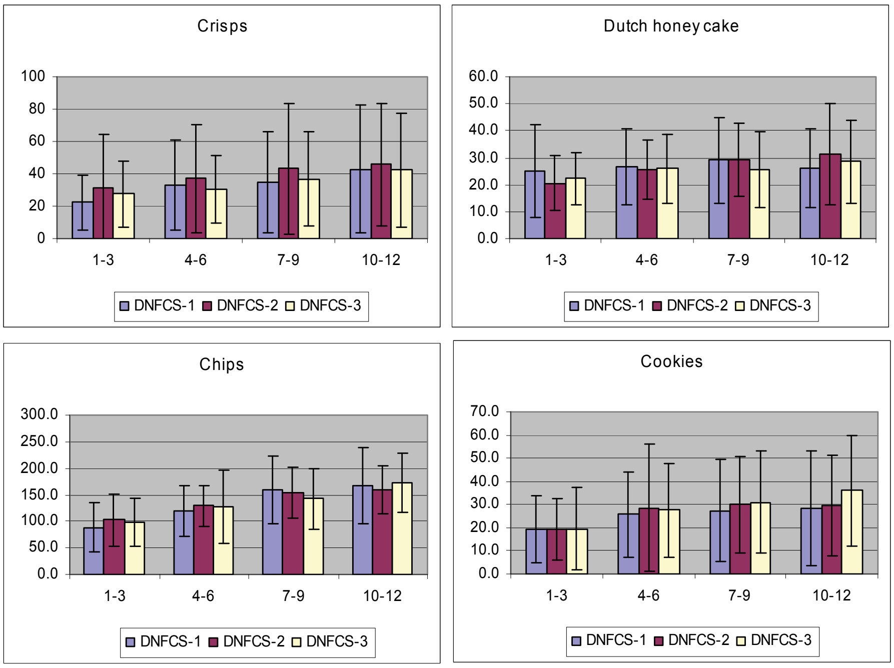

Figure 3.2. Mean daily consumption (g per day) of crisps, chips, Dutch honey cake and cookies for children aged 1-12 years as reported in DNFCS-1, DNFCS-2 and DNFCS-3. Bars indicate standard deviations.

The mean daily consumption of the specific foods for the infants of the EU baby research was 3.5 g per day for crisps, 4 g per day for chips, 18.1 g per day for Dutch honey cake and 10.2 g per day for cookies. No data were available on consumption levels of individual foods from the VIO research (De Boer et al., 2006).

Trends in exposure

Since acrylamide was only detected in 2002, there is only one official publication on acrylamide exposure in the Netherlands by Konings et al. (2003). The year acrylamide was detected a large survey was conducted by the Dutch Food and Consumer Product Safety Authority (VWA[1]) to analyse the acrylamide content of different foods. Using these data, the exposure was calculated for the total Dutch population and children (aged 1-6 years and 7-18 years) by linking these data to DNFCS-3. The estimated acrylamide exposure levels are listed in Table 3.2.

Table 3.2. Long-term exposure to acrylamide (in μg/kg bw per day) in children aged 1-6 years and 7-18 years as reported by Konings et al. (2003).

	Age class (years)	
Exposure	1-6	7-18
Mean	1.04	0.71
P50	0.3	0.2
P90	0.8	0.7
P95	1.1	0.9
P97.5	1.3	1.1
P99	1.7	1.5

There are indications that exposure levels to acrylamide have decreased since 2002, since mitigation measures have been taken to control the formation of acrylamide. Examples of these measures are the use of sodium hydrogen carbonate instead of ammonium hydrogen carbonate during baking procedures of e.g. Dutch honey cake, avoiding excess baking and using frying temperatures below 175 °C (EU, 2003). Preliminary results of new acrylamide analyses performed in the Netherlands in 2006 by the VWA show that levels in especially crisps and Dutch honey cake have decreased resulting in a significant decrease in acrylamide exposure in the total Dutch population (about 20-40%). It is likely that this is also true for children.

Conclusion

Based on trends in consumption of relevant foods no trends in exposure are to be expected for acrylamide exposure. However, based on different mitigation measures taken since the detection of acrylamide, it is likely that the exposure to acrylamide has decreased in children since the first exposure calculations using acrylamide levels of 2002.

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[1] The term ‘Dutch Food and Consumer Product Safety Authority’ also covers the former Dutch Health Inspectorate.

3.2.2 Aflatoxin B_1

Introduction

Aflatoxins are produced by many species of Aspergillus and are known for their carcinogenic, mutagenic and teratogenic properties (Abbas, 2005). Aflatoxins are found as contaminants in human and animal food as a result of fungal contamination both pre- and post-harvest. The rate and degree of contamination is dependent on temperature, humidity, soil and storage conditions. Although a wide range of foods may be contaminated with aflatoxins, they have been most commonly associated with groundnuts, dried fruit, tree nuts, spices, figs, crude vegetable oils, cocoa beans, maize, rice, cottonseed and copra (JECFA, 1998). However, even in the most tropical climates, many lots of these crops do not contain detectable levels of aflatoxins. Aflatoxin B_1 is the most abundant of the aflatoxins (JECFA , 1998).

Trends in consumption

The most important foods determining the exposure to aflatoxin B_1 in the Netherlands are groundnuts and nuts (Van Egmond, personal communication, February 2007). As a very likely representative of this group we examined a possible trend in the consumption of peanut butter, a product consumed frequently by children. The mean daily consumption of this food did not change over the years 1988-1998 and was about 2 g per day for all children aged 1-12 years. The consumption of peanut butter by 8-12 months-old infants was 0.6 g per day. The reported consumption of nuts by children was low (for 0-4 year-olds the number of consumers (N) was less than 15 per age class, for older age classes N=20-60 and the consumption was 1-2 g per day). A time trend could not be observed from these data.

Trends in exposure

There is no information about the dietary exposure of Dutch children to aflatoxin B_1. Neither are concentration data of aflatoxin B_1 available in Dutch food products. In 2006, duplicate diets of children aged 2-6 years were collected. These will be analysed in 2007 for aflatoxin B_1 (and also for OTA and trichotecenes; Van Egmond, personal communication, February 2007).

A duplicate diet study in adults performed in 1994 reported an average exposure of < 0.03 ng/kg bw per day for aflatoxin B_1 (N=123; Sizoo and Van Egmond, 2004), while a similar study carried out in 2004 showed a somewhat higher (median) exposure of 0.04 ng/kg bw per day (N=61; Van Egmond, 2006). As the variation was relatively high (e.g. exposure of 2004 ranged from < 0.005 to 2.3 ng/kg per day), a trend with time cannot be observed.

Conclusion

A time trend of the consumption of nuts and peanut butter could not be observed. Furthermore, due to the absence of Dutch data on aflatoxin B_1 levels in food commodities, exposure calculations are not (yet) available. Two duplicate diet studies with adults do not show a trend with time in aflatoxin B_1 exposure from 1994 to 2004.

3.2.3 Dioxins

Introduction

The term dioxins refers to polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs) and dioxin-like PCBs (polychlorinated mono-ortho and non-orthobiphenyls). These compounds have the same type of toxicity and are therefore mostly studied as a group. Dioxins are persistent chemicals present in the environment. The major source of human exposure to these compounds is through the diet, which accounts for > 90% of their exposure (Huwe, 2002; European POPs Expert Team, 2002).

Trends in consumption

The main products through which exposure to dioxins occur are meat, fish and dairy products (Baars et al., 2004). Together they account for about 75% of the total exposure in the total Dutch population. The trends in consumption of the relevant food groups ('meat, meat products and poultry', 'fish', 'milk and milk products' and 'cheese') for the three DNFCS databases are shown in Section 3.1. Dairy and meat consumption are rather constant throughout the years for all four age groups. Fish consumption seems to increase with time for children between 4 and 10 years.

Because dioxins are lipophilic compounds, they accumulate in the food chain and are mainly present in the (animal) fat part of foods. Due to a lack in animal fat intake data, we studied the trend in total fat intake (Figure 3.3). The total fat intake seems to decrease in all age groups (Hulshof et al., 1998). This was also true for the contribution of fat to the energy intake (Hulshof and Kistemaker, 1995).

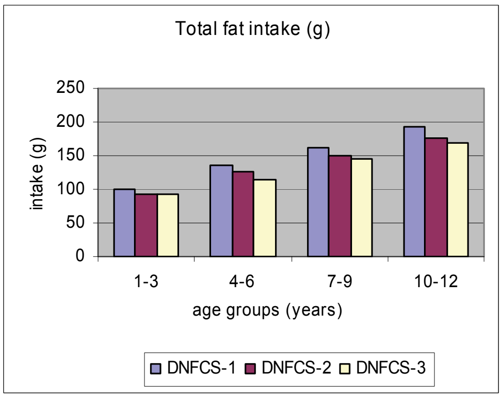

Figure 3.3. Mean intake (g per day) of total fat for children aged 1-12 years as reported in DNFCS-1, DNFCS-2 and DNFCS-3 (Hulshof et al., 1998).

Trends in exposure

It is generally known that the levels of dioxins are decreasing in the environment, resulting in lower levels in food and corresponding lower exposure levels (Huwe, 2002). See also Table 3.3 (mean decrease of total dioxins with 46% in five years). In the Netherlands the intake of PCDDs and PCDFs decreased on average with 50% between 1990 and 1998/1999 and 60% for dioxin-like PCBs. This decrease in intake was mainly due to lower levels of these contaminants in foods rather than changes in food consumption (EU-SCOOP, 2000).

Table 3.3. Mean total dioxin concentrations in 1999 (Baars et al., 2004) and 2004 (De Mul et al., 2008) in pg WHO-TEQ/g fat.

	Year of analysis		
Food category	1999	2004	% decrease
Beef	2.05	1.31	36
Pig	0.47	0.38	19
Poultry	2.78	0.44	84
Milk	1.26	1.00	21
Cheese	1.53	1.03	33
Fatty Fish	3158[1]	19901	37
Lean Fish	593[1]	601	90

[1] *in pg WHO-TEQ/kg product*

The most recent published exposure calculations of dioxins in children reported a median intake for a 2-year-old of 2.8 pg WHO-TEQ/kg bw per day and for a 10-year-old of 1.5 pg WHO-TEQ/kg bw per day (Baars et al., 2004). These exposure levels were estimated using dioxin concentrations analysed in 1999 combined with food consumption data of DNFCS-3.

Figure 3.4 shows the declining trend in median dioxin exposure from around 9 pg WHO-TEQ/kg bw per day in the late seventies to 0.7 pg WHO-TEQ/kg bw per day in 2004 for a 40-year-old adult (De Mul et al., 2008). This declining trend seems however to level off since 1990. Although the declining trend in exposure was performed for a 40 year adult, it may be assumed that the same declining trend applies to children. In 2004 the median dioxin intake for a 40-year-old was 0.7 WHO-TEQ/kg bw per day which indicates a decrease of 36% compared to 1999. If we assume the same declining trend for children this would mean a median intake of 1.8 pg WHO-TEQ/kg bw per day for 2-year-olds and 0.95 pg WHO-TEQ/kg bw per day for 10-year-olds in 2004.

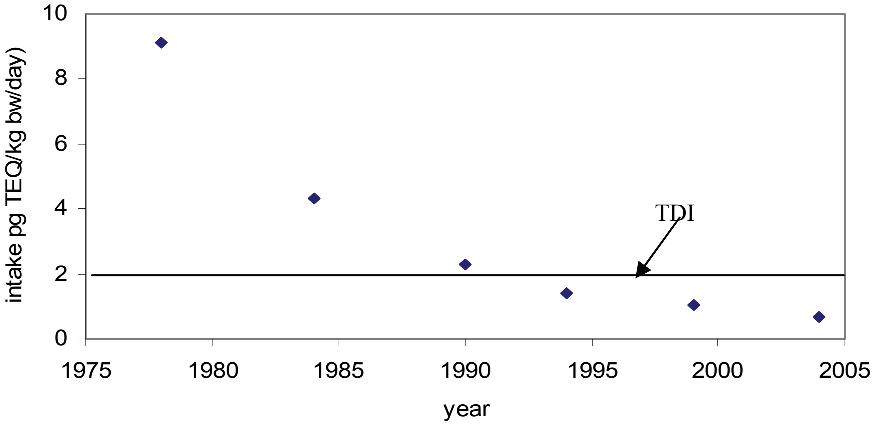

Figure 3.4. Median long-term exposure (in pg WHO-TEQ/kg bw per day) for dioxins and dioxin-like PCBs (non-detects are assigned a concentration of zero) by adults. Data from 1978, 1984 en 1994 are from duplicate diet studies (reviewed by Liem and Theelen, 1997), while data from 1990, 1999 (Baars et al., 2004) and from the present study are from total-diet studies. Derived from De Mul et al. (2008). TDI = Tolerable Daily Intake.

Conclusion

A decreasing trend could be observed in the total fat intake in children aged 1-12 years from 1987 up to 1998. Dioxin exposure levels are declining since the mid-seventies, including the exposure levels. It is however well known that the decrease in dioxin levels (and exposure) is levelling off.

3.2.4 DON

Introduction

Deoxynivalenol (DON, vomitoxin) belongs to the group of trichothecenes, which are mycotoxins mainly produced by fungi of the Fusarium genus. DON may occur in various cereal crops (wheat, maize, barley, oat, and rye). Upon ingestion it can cause severe toxicosis in humans and farm animals. Acute effects of food poisoning in humans are abdominal pains, dizziness, headache, throat irritation, nausea, vomiting, diarrhoea and blood in the stool. DON affects the immune system and alters various blood parameters; in addition it is a gastrointestinal irritant. The main long-term effects at low dietary concentrations are growth retardation and reduced appetite. There are no indications for carcinogenic and/or mutagenic properties of DON (Pieters et al., 2004).

Trends in consumption

Although, as mentioned above, DON may be present in different kinds of cereals, wheat appears to be the only one contributing to the exposure to DON in Dutch children (Pieters et al., 2001, 2004). In the present study, the wheat consumption was calculated for children of different age classes (1 year, 2-4 years, 5-8 years and 9-12 years) using consumption data of the three DNFCS and the food conversion table (see Section 2.3). About 100% of the children in these surveys reported to have consumed foods containing wheat as an ingredient. The mean daily consumption of wheat by children appears to be stable over the time period 1988-1998 (Table 3.4). The mean daily wheat consumption of the infants of the EU baby research is 20 g per day (Boon et al., 2004).

Table 3.4. Average consumption of wheat (g per day) ± standard deviation in the three Dutch National Food Consumption Surveys (DNFCS) for children of different age classes.

Age class (years)	DNFCS-1	DNFCS-2	DNFCS-3
1	47 ± 21	52 ± 27	49 ± 26
2-4	60 ± 29	61 ± 29	68 ± 30
5-8	78 ± 37	81 ± 37	82 ± 35
9-12	103 ± 48	100 ± 45	107 ± 47

Trend in exposure

As mentioned above, two surveys on the exposure to DON in the Netherlands have been carried out, one with concentration data from 1998/1999 (Pieters et al., 2001) and one with concentration data from 2000-2002 (Pieters et al., 2004). In both studies consumption data of DNFCS-3 were used. In 1998 and 1999, wheat contained high levels of DON in the Netherlands, resulting in high exposure levels (see Table 3.5 for median and 95th percentiles (P95) of exposure). After the detection of these high DON-levels, the Dutch government enforced national action limits. Separate from this, the primary sector and the grain processing industry took several contamination-reducing measures such as checking plant material for fungal contamination, improving methods for cultivation, harvest and storage, and removing contaminated lots from the food production process. As a result, the DON contamination of wheat was reduced by 55%. The reduction of the DON concentration in some other foods was even higher (up to 90% for baby food). Exposure calculations using these reduced concentrations of 2001-2002 resulted in a reduction of about one-third of the previous exposure levels (Table 3.5).

Table 3.5. Median long-term exposure (µg/kg bw per day) of DON by children aged 1-12 years. Calculation with occurrence data of 1998-1999 (Survey 1; Pieters et al., 2001) and 2000-2002 (Survey 2; Pieters et al., 2004). Between brackets the 95th percentiles of exposure are reported. The last column reports the decrease in median exposure over the time period (%).

Age class (years)	Survey 1	Survey 2	% decrease
1	1.4 (2.7)	0.5 (1.0)	64
2	1.3 (2.4)	0.4 (0.9)	69
3	1.1 (2.2)	0.4 (0.8)	64
4	1.0 (2.0)	0.3 (0.7)	70
5	1.0 (1.8)	0.3 (0.7)	70
8	0.8 (1.5)	0.2 (0.5)	75
10	0.7 (1.3)	0.2 (0.5)	71
12	0.6 (1.2)	0.2 (0.5)	67

In another study on the dietary exposure to DON, duplicate diet portions collected in the EU baby research were analysed for DON to estimate the real exposure in infants aged 8 – 12 months. For this the diets of 74 infants with the highest cereal consumption levels were analysed for DON (Schothorst et al., 2005). DON was detected in all analysed samples, resulting in daily DON exposures that ranged from 0.03 µg/kg bw per day to 1.98 µg/kg bw per day (median 0.66 µg/kg bw per day). Note that these exposure levels relate to the exposure on one specific day, so-called short-term exposure. Using the food consumption data from the EU baby research as recorded in the food diaries, the exposure to DON was also calculated by linking the consumption levels to concentration data of DON of 2000-2001 omitting the higher concentration data of 1999. This resulted in a median short-terms DON exposure (of the children with relatively high cereal consumption) of 0.41 ug/kg bw per day. This is somewhat lower than the result of the duplicate diets (Schothorst et al., 2005). The reason for this is not clear. The short-term exposure calculation of high cereal consuming infants was comparable with the median long-term exposure calculation of 1-year-olds by Pieters et al. (2004; Table 3.5) in which concentration data of the same time period were used.

Conclusion

Based on consumption data of wheat by children no changes in the exposure of DON due to changes in eating habits are to be expected if the observed trend can be extended to the present. However, the results from the surveys of Pieters et al. (2001, 2004) and from Schothorst et al. (2005) demonstrated that the exposure to DON has declined since about 2000, due to a decline of DON-concentrations in wheat. As new legislation has been in force since 2005[2] (especially for baby food), consequently the exposure to DON in 2007 is expected to show a further decrease.

3.2.5 Nitrate

Introduction
Nitrate is not very toxic of itself. However, in the human body it is converted into nitrite. Nitrite can decrease the availability of oxygen in the blood ('blue baby syndrome'). In addition, it can react with the amines in fish products to become nitrosamines (especially N-nitrosodimethylamine NDMA), which are potentially carcinogenic.

Trends in consumption
Nitrate occurs naturally in vegetables, for example endive, beets, spinach, salad and cabbages, and drinking water. The concentration of nitrate in various foods, specifically vegetables and fruit, depends on the season. Furthermore, the production methods and preparation of foods influences the concentration of nitrate in foods. A recent study of Westenbrink et al. (2005) demonstrated that for children (1-12 years) vegetables contributed most to the exposure, approximately 55%. A second important source was formed by potatoes, about 23%. Of the vegetables, the most important types were spinach (about 18%) and lettuce (about 11%). In the age groups 4-6 years and 7-12 years, also beets were important (about 15%).

For the two most important food groups, vegetables and potatoes, the consumption reported in the three DNFCS is presented in Section 3.1. Overall, the consumption of potatoes and vegetables tended to decrease in all age groups over time. Because of the small number of users of the individual vegetables, it was not possible to study the trends in the consumption of the individual foods. Levels of consumption in infants and toddlers were lower than those reported in all DNFCS for children aged 1-3 years (Figure 3.1 and Table 3.1), which is partly due to the high consumption of jarred baby food in this age group. These jars are coded under the food group 'mixed dishes'.

Trends in exposure
Exposure to nitrate in children was calculated with food consumption data of DNFCS-1 and DNFCS-3. Brussaard et al. (1996) estimated the exposure to nitrate using consumption data of DNFCS-1 and concentration data on nitrate of analyses of 226 foods. These foods were purchased during the whole and covered all seasons. They represented 95% of the total weight eaten and 80% of the total energy of the diet. Concentration levels of nitrate for foods not analyzed were estimated based on published data. Table 3.6 presents the resulting mean exposure levels for various age groups.

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[2] The European Commission set action limits in 2000 of 500 µg DON per kg food product for direct human consumption and of 750 µg DON per kg flour as an ingredient for food products. Thes action limits have been enforced within the European Union since 2000, although legislative status was not achieved until 2005 (EC, Regulation 856/2005). For unprocessed cereals the limits were set at 1250 µg/kg (durum wheat and oats 1750 µg/kg, no limit for maize). The same regulation gave specific low limits for processed cereal based foods for babies and young children and babyfood, namely 200 µg/kg. Additionally, in 2006 a limit was set for unprocessed maize (1750 µg/kg, EC, Regulation 1881/2006).

Table 3.6. Observed exposure to nitrate (mg per day) ± standard deviation (SD) using food consumption data of DNFCS-1 (Brussaard et al., 1996).

	Boys		Girls	
Age class (years)	N	Mean ± SD	N	Mean ± SD
1-3	163	33 ± 46	140	31 ± 38
4-6	128	41 ± 52	128	40 ± 48
7-9	120	58 ± 76	133	41 ± 51
10-12	148	60 ± 65	138	44 ± 55

Food consumption data of DNFCS-3 were used by Westenbrink et al. (2005) to estimate the exposure to nitrate in children. Nitrate concentrations were based on average concentration levels in raw agricultural products from 2000-2002 as recorded in the KAP database (Van Klaveren, 1999; see also Chapter 4). These levels included concentrations from summer and winter and were extended with information on processing, drinking water and other foods. Table 3.7 presents the observed exposure to nitrate in mg per day and the long-term exposure to nitrate in mg/kg bw per day in different age groups, respectively. The observed exposure is reported for a possible trend analysis in exposure compared with the exposure reported by Brussaard et al. (1996). The long-term exposure is reported as the most up-to-date exposure estimation for nitrate available.

Table 3.7. Observed (mg/kg) and long-term exposure (mg/kg bw per day) ± standard deviation (SD) to nitrate based on DNCFS-3 (Westenbrink et al., 2005).

Age class (years)	N[1]	Mean ± SD	P5	P10	P50	P90	P95	P97.5
Observed exposure								
1-3	156	29 ± 24	5	8	23	58	85	103
4-6	289	37 ± 31	7	9	27	82	95	127
7-12	477	50 ± 41	9	12	37	105	134	156
Long-term exposure								
1-3	156	2.44 ± 1.03	1.05	1.29	2.32	3.78	4.33	5.12
4-6	289	2.14 ± 0.96	0.86	1.11	1.97	3.52	4.04	4.42
7-12	477	1.71 ± 0.85	0.64	0.79	1.53	2.83	3.41	3.98

[1] *Data on body weight from all participants was not available. Therefore the number of participants is lower.*

Among the infants and toddlers from the VIO study the mean long-term exposure to nitrate varied from 1.8 mg/kg bw per day (9 months) to 2.7 mg/kg bw per day (12 and 18 months) (De Boer, personal communication, February 2007). Nitrate exposure for the infants of the EU baby research equalled 4 mg/kg bw per day using all nitrate data available in the KAP database (from 1987 onwards; unpublished data). Note that this refers to exposure during one day (= short-term exposure).

Westenbrink et al. (2005) compared also nitrate concentration data from 1994-1996 with concentration data from 2000-2002 for lettuce (iceberg and raw) and spinach. Overall there was a tendency of lower concentrations of nitrate in 2000-2002.

Conclusion
There is a declining trend in the consumption of vegetables and potatoes during the 10-year period from 1987/1988 to 1997/1998. Together with a possible decline in nitrate levels in vegetables this suggests that the exposure to nitrate has a tendency to decline[3].

3.2.6 Organophosphate pesticides

Introduction
Dietary exposure assessments to pesticides are traditionally performed for single compounds. However, there are groups of compounds that should be addressed simultaneously because of a common mechanism of action. When ingested during the same time period (say one day) accumulation of effects may occur. One group of such chemicals are the organophosphorus esters (OPs), widely used insecticides, which inhibit acetylcholinesterase (AChE) by phosphorylation, resulting in a spectrum of acute cholinergic effects (ILSI, 1999; Mileson et al., 1998; Pope, 1999). OPs are used as insecticides in agriculture to control certain pests. Presently about 25 different OPs are detected in monitoring programmes (KAP), which are present in a wide range of fruits and vegetables.

Trends in consumption
To describe trends in exposure to OPs using food consumption data, fruits and vegetables are the most likely candidates to study. Based on the results reported in Section 3.1 it is obvious that consumption of fruits and vegetables tended to decrease over the period 1987 up to 1998 in the age groups 1-12 years.

Trends in exposure
The recognition that groups of compounds should be addressed simultaneously is relatively new. Since 2000 three reports have been published reporting acute percentiles of exposure to OPs in children aged 1-6 years. For other age groups no exposure levels are reported. The first report on cumulative exposure to OPs dates from 2000, in which consumption data of DNFCS-3 were linked to residue data of 40 OPs as analysed by the Dutch Food and Consumer Food Safety Authority (VWA) from 1997 until 1999 (Luijk et al., 2000). In the second report the same consumption data were combined with concentration data from 2000/2001 (Boon and Van Klaveren, 2003). Another study concerns the exposure to OPs using residue levels of 2003, 2004 and 2005 linked again to consumption data of DNFCS-3 (Van Klaveren et al., 2006). In this last report also the exposure to young infants, using consumption data of the EU baby research, were reported. For the exposure levels see Table 3.8.

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[3] Exposure to NDMA: Zeilmaker et al. (2004) estimated the exposure to NDMA resulting from simultaneous ingestion of nitrate-rich vegetables and fish combining information derived from an in vitro model with that of DNFCS-3. More recently, the matrix effects relating to the meal were taken into account as well and the resultant calculation showed that the life-long exposure of a significant part of the Dutch population (especially children) to NDMA arising from fish/vegetable consumption exceeds the chronic health-based limit value for NDMA (Zeilmaker et al., in prep.).

Table 3.8. Acute exposure to organophosphate pesticides (in µg/kg bw per day) using DNFCS-3 and cumulative residue levels from 1997 – 1999 (Luijk et al., 2000), 2000/2001 (Boon and Van Klaveren, 2003), and 2003, 2004 and 2005 (Klaveren et al., 2006).

	Children (1-6 years)					Infants (8-12 months)		
Percentile	1997 - 1999	2000/2001[1]	2003	2004	2005	2003	2004	2005
P95	1.1	0.83	0.7	0.5	0.4	0.6	0.5	0.4
P97.5			2.2	1.7	1.5	2.0	1.7	1.6
P98	2.0	2.0						
P99	3.1	4.0	7.1	5.9	4.5	6.3	5.4	5.2
P99.9	18.9	31.9	83	104	25	45	111	27

[1] *Exposure levels reported were calculated using residue levels of both organophosphorus pesticides and carbamates*

To examine a trend in acute exposure to OPs via food attention should be focussed preferably on the P95 and P97.5 (Van Klaveren et al., 2006). These percentiles are less dependent on accidental outliers than higher percentiles. Examining these percentiles, and ignoring differences in calculation methods applied between the three reports (RPF values used, selected index compound, variability, processing, etc.), there seems to be a declining trend in acute exposure to OPs for children aged 1-6 years of age in the time period 1997-2005. For example, the P95 decreased from 1.1 µg/kg bw per day to. 0.4 µg/kg bw per day. The methodology used to calculate the cumulative acute exposure using residue data of 2003, 2004 and 2005 was identical. Examining only these exposures showed that the acute cumulative exposure tended to be lower in 2005 than 2003 for both the young children and the infants (Table 3.8).

Conclusion

We observed a decreasing trend in the consumption of vegetables and fruits in children aged 1-12 years. The cumulative exposure calculations performed so far also show also a decreasing trend in the exposure to these compounds. However, the trend is weak and further assessments are needed to confirm this.

3.2.7 OTA

Introduction

OTA (ochratoxin A) is a mycotoxin produced by several fungi of the Aspergillus or Penicillium families. It is principally found to occur as a result of poor storage of commodities or poor agricultural practice during drying of produce (Gilbert et al., 2001). OTA is predominantly found in cereals (wheat) and cereal products, but also in raisins, grape juice, wine, coffee, cocoa and in some pulses and nuts (EU-SCOOP, 2002a). As OTA can survive many food processing operations, products derived from these raw materials, such as instant coffee and raisins, can be contaminated as well. OTA has been shown to be nephrotoxic in mammals (JECFA, 2001).

Trends in consumption

For the time trend in the consumption of wheat, see Table 3.4. The raisin consumption between 1988 and 1998 did not show a clear time trend. The consumption ranged from 7 g per day for consumers of 1-2 years (~30% is consumer) to about 5 g per day for 9-12 year-olds (6% is consumer). Consumption of raisins by 8-12 months-old infants equalled 9 g per day (3% is consumer). The consumption of grape juice by children is so low (n < 5 per age group) that it is not possible to observe a time trend. In the EU baby research only one infant consumed grape juice.

Trends in exposure

The exposure of the Dutch population to OTA has been calculated using DNFCS-3 and concentration data of the Dutch Food and Consumer Product Safety Authority from 1995-2001, combined with data from the Netherlands and from other countries from EU-SCOOP (2002a) and JECFA (2001; Bakker and Pieters, 2002). For children, the resulting median long-term exposure ranged from 2.8 ng/kg bw per day for 1-year-olds to 1.2 ng/kg bw per day for 12-year-olds (Table 3.9).

Table 3.9. Long-term dietary exposure to OTA (in ng/kg bw per day) for children, using concentration data from 1995 to 2001 and consumption data of DNFCS-3 (Bakker and Pieters, 2002).

	Exposure (ng/kg bw per day)		
Age (years)	median	P90	P95
1	2.8	4.4	4.9
2	2.4	3.8	4.3
3	2.1	3.4	3.8
4	1.9	3.0	3.4
5	1.8	2.8	3.1
7	1.5	2.4	2.7
10	1.3	2.0	2.3
11	1.3	2.0	2.2
12	1.2	1.9	2.1

For the whole population, the contribution of cereals to the total exposure of OTA was 57%, while coffee, beer and red wine were also large contributors (5-9% each). The consumption of raisins (1 g per day) contributed to a smaller extent to OTA exposure (2%). As children do not consume coffee, wine and beer, consequently, the OTA-exposure in children will be largely dominated by the consumption of cereals (mainly wheat). The contribution of raisins to the exposure of OTA in children is expected to be higher than that for the whole population.

In a duplicate diet study with adults in 1994 a median intake of OTA of 0.7 ng/kg bw per day (N=123) was reported, while for a similar study in 2004 the median intake was reduced to 0.4 ng/kg bw per day (N=61; Van Egmond, 2006).

Conclusion

It is not possible to predict a possible increase or decrease in OTA-exposure when examining trends in consumption of relevant products, including wheat, raisins and grape juice. Two duplicate diet studies with adults indicate a decreasing time trend in the period 1994-2004. Furthermore, current product limits, especially for food products for babies and young children, are stricter than in 2001[4]. For these reasons, it is to be expected that the current exposure of children to OTA is lower compared to the values reported in Table 3.9.

3.2.8 Patulin

Introduction

Patulin is a mycotoxin produced by fungi belonging to several genera, including Penicillum and Aspergillus. It has been mainly isolated from apples and apple products (apple juice, apple sauce) contaminated with the common storage-rot fungus of apples, Penicillium expansum. Patulin has been found to cause a range of adverse effects in laboratory animals. The current health limits are based on a study on reproductive toxicity/long term toxicity/carcinogenicity (JECFA, 1990). No evaluation could be made of the carcinogenicity of patulin to humans (IARC, 1986).

Trend in consumption

In all three DNFCS, for the lower age class (1-4 years) about 30% of the children consumed apple juice, sauce and/or compote. There is a decrease in the daily consumption amount of these products with 30-50% from 1988 to 1998: for 1-year-olds the consumption decreases from 42 to 22 g per day, and for 2-4 year-olds from 55 to 39 g per day). For the other age groups in the DNFCS no time trend in the consumption of these food products was visible. In the baby research the mean consumption level of apple juice, sauce and/or compote was 65 g per day. These levels are much higher than those reported in the DNFCS.

Trend in exposure

No exposure calculations of patulin have been performed in the Netherlands. Neither are there Dutch occurrence data available in the report of EU-SCOOP (Scientific Co-operation on Questions relating to food) on patulin (EU-SCOOP, 2002b). Also no data are available in the KAP database on this compound.

Conclusion

There is no information on the trend in concentrations of patulin. Neither is information available on the exposure of Dutch children to this toxin. Because of the stricter legislation for patulin in fruit juice and products since 2003[5], it may be expected that the concentrations in products and therefore the exposure of children to patulin has decreased since 2003. Furthermore, as for the lower age groups (1-4 years) the consumption of processed apple products has decreased between 1988 and 1998, the decline in exposure may be even more prominent for these age groups.

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[4] Concentration limits for OTA were set by the EU in 2002 for unprocessed cereals (5 µg/kg), processed cereals (3 µg/kg) and dried vine fruit (10 µg/kg), (EC 2002). New, additional, limits were adopted by the EU in 2005 (EC Regulation 123/2005): for coffee, wine and grape juice (2-10 µg/kg) and for baby foods and processed cereal-based foods for infants and young children (0.5 µg/kg).

[5] In 2003 the following product limits for patulin were adopted by the EC (2003): fruit juice 50 µg/kg, apple sauce/compote 25 µg/kg, baby food (including fruit juice and apple sauce especially for children 10 µg/kg).

3.2.9 Sulphite

Introduction

Sulphite is an additive (E220-228) that is used as a preservative in various foods. Some individuals can be allergic for sulphite resulting in asthmatic symptoms, especially young children and people with asthma. The directive 95/2/EC on food additives other than colouring and sweeteners regulates in which foods sulphite may be used as a preservative and the maximum concentration allowed. Sulphites may be added to approximately 60 categories of foods, for example peeled potatoes, various dried vegetables or fruits, jams and juices (directive 95/2/EC). Sulphite is not allowed to be present in meat and meat products since around 1960. The regulation states that sulphite at levels below 10 mg/kg or 10 mg/L is assumed not to be present.

Trends in consumption

Based on two surveys examining the exposure to sulphite in young children, potatoes bought as peeled (Hulshof, 2001) and especially dried fruit (raisins; Hulshof and Doest, 2003) were identified as the most important sources of sulphite exposure. The consumption of dried fruit in the three DNFCS is presented in Table 3.10. It is clear that consumption levels are rather low and stable in time.

Table 3.10. Mean consumption (in g per day) ± standard deviation (SD) of dried fruit for the three DNFCS (Hulshof and Doest, 2006).

	DNFCS-1		DNFCS-2		DNFCS-3	
Age class (years)	N	Mean ± SD	N	Mean ± SD	N	Mean ± SD
1-3	303	9 ± 8	351	8 ± 6	254	8 ± 6
4-12	795	9 ± 11	838	7 ± 6	750	7 ± 6

Based on the VIO study (Breedveld and Hulshof, 2002), the consumption of preserved fruits, including fruits on syrup and dried fruits, was estimated. The mean daily consumption was 3 g per day (9 months), 8 g per day (12 months) and 13 g per day (18 months). The number of users of preserved fruits was estimated at 19% (9 months), 39% (12 months) and 53% (18 months). Infants aged 8 – 12 months from the EU baby research had a mean consumption level of 9 g per day for raisins. The number of users was however low, 3%.

No information regarding the consumption of potatoes bought as peeled could be extracted from the literature.

Trends in exposure

Hulshof (2001) estimated the exposure to sulphites for children under the age of three years using a Tier-2 approach. In this approach the mean consumption of food groups or separate foods, based on the DNFCS-3, was multiplied with the maximum permitted concentration of sulphite using a standard body weight of 15 kg. This approach assumes that all foods in a specific category contain sulphite at the maximum permitted level. Mean daily exposure was assessed at 0.78 mg/kg bw per day. Since no information was available on the use of potatoes bought as peeled, it was assumed that all cooked potatoes were bought as peeled. This implies that the estimated exposure is expected to be higher than the true exposure.

In 2003, another exposure estimation to sulphite in children was performed based on DNFCS-3 but now using a more realistic approach to determine in which foods sulphites were used (Hulshof and Doest, 2003; Table 3.11). In this approach not all potatoes were considered to be bought as peeled. Furthermore, information from producers on the internet was used to determine in which food groups sulphite was used. If sulphite was used, the concentration was assumed to be at the maximum permitted level.

Table 3.11. Estimated observed exposure to sulphite (mg/kg body weight) ± standard deviation (SD) in children in DNFCS-3 (Hulshof and Doest, 2003).

Age class (years)	N	Mean ± SD	Median	P95	P97.5
1-2	156	0.33 ± 0.75	0.04	1.72	3.21
3-6	289	0.13 ± 0.33	0.04	0.69	0.92
6-12	480	0.09 ± 0.21	0.03	0.48	0.71

Conclusion

In the Netherlands two exposure assessments of sulphite were performed that do not permit to study the trend in exposure to this compound, due to the use of the same food consumption data, the different type of input levels used for sulphite and different calculation methods. Data regarding the consumption of one of the sources of sulphite, dried fruit, does not indicate a change in food consumption habits from 1986/1987 until 1997/1998.

3.2.10 Sweeteners

Introduction

There are two different groups of sweeteners, extensive and intensive sweeteners. Extensive sweeteners are as sweet as regular sugar or even less sweet. Examples are isomalt, maltitol and xylitol. Intensive sweeteners are sweeter than regular sugar. Examples are acesulfame-K, aspartame, cyclamate and saccharine. The use of sweeteners is regulated in directive 94/35/EC. A high consumption of extensive sweeteners is related to intestinal complaints. Potential health risks of high consumption of intensive sweeteners depend on the type of sweetener. For example, saccharine might be associated with cancer among smokers. On the other hand, there are no known side effects related to acesulfame-K (www.food-info.net).

Trends in consumption

In a study examining the exposure to various sweeteners in children under the age of three years using mean consumption of food groups or separate foods, based on DNFCS-3, and maximum permitted levels, savoury sauces and energy-reduced/light drinks were identified as important sources of exposure (Table 3.12; Hulshof, 2001). It should be noticed that table-top sweeteners were not taken into account, although use of these products in children up to 3 years is probably small. This last assumption was confirmed by Hulshof et al. (Hulshof, 1989; Hulshof et al., 1995) who determined the use of table-top sweeteners based on the DNFCS-1 and DNFCS-2 in different age groups up to 15 years.

Table 3.12. Important foods/food groups for the exposure to sweeteners among children under the age of three years in DNFCS-3 (Hulshof, 2001).

Sweetener	Foods or food groups
Acesulfame-K	Sauces[1] (56.5%) Water-base flavoured drinks, energy reduced or with no added sugar (21.7%)
Aspartame	Sauces (43.3%) Water-base flavoured drinks, energy reduced or with no added sugar (30.0%)
Cyclamic acid and its salts	Water-base flavoured drinks, energy reduced or with no added sugar (54.5%)
Saccharine	Sauces (66.7%)
Neohesperidine dhydrochlcone	Sauces (66.7%)

[1] *Sauces are savoury sauces which might contain sweeteners*

An important food group is energy-reduced/light drinks, including light soda's. In 2005, the use of light soda's in the three DNFCS was determined (Hulshof, 2005). For the results see Table 3.13 that shows that the consumption of light soda's tended to increase from DNFCS-1 to DNFCS-3.

Table 3.13. Consumption of light soda's (in g per day) ± standard deviation (SD) in the three DNFCS by different age-gender groups (Hulshof, 2005).

	DNFCS-1			DNFCS-2			DNFCS-3		
	All	Users		All	Users		All	Users	
Age class (years) and gender	Mean ± SD	%	Mean ± SD	Mean ± SD	%	Mean ± SD	Mean ± SD	%	Mean ± SD
Children 1-3	0 ± 0	0		5 ± 35	4	126 ± 137	6 ± 31	4	126 ± 137
Boys 4-8	2 ± 19	2	120 ± 65	17 ± 79	8	219 ± 192	11 ± 49	7	167 ± 105
Boys 9-13	2 ± 17	1	144 ± 69	17 ± 81	6	276 ± 181	45 ± 123	17	262 ± 177
Girls 4-8	1 ± 7	1	78 ± 0	11 ± 47	8	141 ± 99	11 ± 41	7	146 ± 64
Girls 9-13	8 ± 51	3	291 ± 112	24 ± 89	12	199 ± 179	50 ± 169	13	368 ± 312

The number of toddlers using foods with sweeteners and/or table-top sweeteners varied from 3% at 9 months to 17% at 18 months. Fruit juices with saccharine and cyclamate were used most often; 9 months: 3.0%, 12 months: 5.5% and 18 months: 15.6%. Milk products with acesulfame-K and aspartame were used by 3 children (1 at age 12 months, 2 at age 18 months). In every age group one child used table-top sweeteners (De Boer et al., 2006).

No information regarding the consumption of savoury sauces with sweeteners was found in the literature.

Trends in exposure

Hulshof et al. (1992) estimated the exposure to aspartame, cyclamate, saccharine and acesulfame-K based on both the 2-day diary and the food frequency questionnaire of the DNFCS-2. Concentration data was based on maximum permitted levels or non-alcoholic drinks and data from producers regarding the table-top sweeteners. Table 3.14 presents the exposure to the sweeteners. No percentiles are presented due to the low number of users.

Table 3.14. Mean exposure to sweeteners (mg/kg bw per day) ± standard deviation (SD) in DNFCS-2 based on the two-day diary (diary) and the food frequency questionnaire (FFQ) among users of foods containing the sweetener (Hulshof et al., 1992).

		Diary		FFQ	
Sweetener	Age class (years) and gender	N[1]	Mean ± SD	N	Mean ± SD
Aspartame	Children 1-3	13	4.6 ± 7.5	17	12.3 ± 13.6
	Children 4-6	17	2.3 ± 2.5	36	5.9 ± 6.8
	Children 7-9	27	4.4 ± 3.9	50	4.9 ± 5.4
	Boys 10-15	19	2.1 ± 1.5	39	3.7 ± 3.1
	Girls 10-15	28	2.2 ± 2.1	45	2.9 ± 2.8
Cyclamate	Children 1-3	5	2.8 ± 1.2	11	4.7 ± 6.9
	Children 4-6	8	2.6 ± 1.8	15	2.9 ± 3.5
	Children 7-9	11	3.0 ± 3.3	13	2.8 ± 2.9
	Boys 10-15	6	1.0 ± 0.8	11	1.5 ± 1.9
	Girls 10-15	10	1.7 ± 1.8	18	2.2 ± 1.8
Saccharine	Children 1-3	7	0.5 ± 0.3	24	1.6 ± 1.5
	Children 4-6	8	0.6 ± 0.5	23	1.1 ± 1.3
	Children 7-9	11	0.6 ± 0.7	19	0.7 ± 0.8
	Boys 10-15	6	0.2 ± 0.2	14	0.3 ± 0.4
	Girls 10-15	10	0.3 ± 0.4	21	0.5 ± 0.5
Acesulfame-K	Children 1-3	1	0.1	0	
	Children 4-6	1	0.1	1	0.1
	Children 7-9	1	0.1	3	0.1
	Boys 10-15	0		5	0.2 ± 0.1
	Girls 10-15	0		5	0.2 ± 0.2

[1] N represents the number of users. The number of participants in each age-gender group is: children 1-3: 351, children 4-6: 329, children 7-9 254, boys 10-15: 255, girls 10-15: 252.

A second study, using consumption data of DNFCS-3, estimated the exposure to various sweeteners for children under the age of three years using a Tier-2 approach (Hulshof, 2001). In this approach the mean consumption of food groups or separate food was multiplied with the permitted concentration of the sweeteners in the specific food group or food. To calculate the mean exposure per kg body weight, a standard body weight of 15 kg was used. This approach assumes that all foods in a specific category contain sweeteners at the maximum permitted level. Table 3.15 lists the exposure to various sweeteners in children under the age of three years based on this approach. It should be noticed that table-top sweeteners were not taken into account.

Table 3.15. Mean exposure to sweeteners (in mg/kg bw per day) among children under the age of three years in DNFCS-3 (Hulshof, 2001).

Sweetener	Mean exposure level
Acesulfame-K	0.23
Aspartame	0.3
Cyclamic acid and its salts	0.11
Saccharine	0.09
Neohesperidine dhydrochlcone	0.03

In an advice for the Dutch Food and Consumer Product Safety Authority (VWA), RIVM-RIKILT reported long term exposure levels to aspartame, using consumption data of DNFCS-3 and analysed aspartame concentrations by the VWA in 2003.The median long-term exposure to aspartame of children aged 1-6 years was 0.6 mg/kg bw per day, while the 95th percentile amounted to 2.1-2.8 mg/kg bw per day (Boon and Donkersgoed, 2006). The VWA also analysed, apart from aspartame, levels of saccharine, acesulfame-K and cyclamic acid in different relevant foods purchased in 2003. Combining these analyses with food consumption data of DNFCS-3 the mean exposure was calculated for various age groups (Van Rooij-van den Bos et al., 2004). The results are listed in Table 3.16.

Table 3.16. Mean observed exposure to sweeteners (mg/kg bw per day) in DNFCS-3 based on analyses of sweeteners performed in 2003 among users of foods containing the sweetener (Van Rooij-van den Bos et al., 2004).

Sweetener	Age class (years)	Mean exposure level
Aspartame	1-4	0.2
	4-7	0.1
	7-10	0.1
	10-13	0.1
Cyclamate	1-4	0.6
	4-7	0.3
	7-10	0.1
	10-13	0.1
Saccharine	1-4	0.03
	4-7	0.02
	7-10	0.00
	10-13	0.02
Acesulfame-K	1-4	0.2
	4-7	0.2
	7-10	0.1
	10-13	0.1

Among the toddlers from VIO the observed mean exposure to cyclamate was 11.9 mg per day at 9 months, 36.6 mg per day at 12 months and 35.2 mg per day at 18 months among users. The mean exposure to saccharine was 1.0 mg per day at 9 months, 2.6 mg per day at 12 months and 3.0 mg per day at 18 months among users (De Boer, personal communication, February 2007).

Conclusion

The most important food groups for the consumption of sweeteners in young children are sauces with sweeteners, and energy-reduced/light drinks. From the DNFCS-1 to DNFCS-3 the consumption of light soda's increased. Exposure to sweeteners was estimated for children based on the DNFCS-2 and DNFCS-3. However, it is very difficult to compare these exposures due to differences in food consumption data (individual data versus mean consumption, taking table-top sweeteners into account or not), differences in concentration data (permitted use/actual use, analysed levels/maximum permitted levels), differences in body weight (standard body weight or individual body weight) and whether the exposure was estimated for users or total group. The increasing consumption of light soda's over the years suggests that the exposure to sweeteners has increased during this time period when assuming that the concentrations of the sweeteners remained at the same level. This does not yet take into account the probable broader use of sweeteners in other food groups, for example milk drinks.

3.2.11 T_2 and HT_2

Introduction

T_2 toxin (T_2) and HT_2 toxin (HT_2) belong, like DON, to the group of trichothecenes, a group of closely related chemical compounds produced by several fungi (mainly of the Fusarium species). T_2 and HT_2 are type A trichothecenes, which are more toxic than the related type B trichothecenes (e.g. DON, nivalenol). Their major effects – related to their concentration in the food commodity – are vomiting and immuno-suppression.

Trends in consumption

Just like DON, T_2 and HT_2 may occur on cereals (wheat, rye and oats). As the consumption of wheat in the Netherlands is far more important than that of other cereals, wheat is expected to be the major cereal that contributes to the intake of T_2 and HT_2 in Dutch children. The time trend of the consumption of wheat is reported in Table 3.4.

Trends in exposure

In the duplicate diet study with 8-12 months-old babies, 74 duplicate diet samples of children with relatively high cereal consumption were analysed for T_2 and HT_2 (Schothorst et al., 2005). T_2 and HT_2 were detected in 69% of the samples. The exposure ranged from 0.01 to 0.16 µg/kg bw per day (mean 0.04 µg/kg bw per day). Note that in general the amounts of DON and T_2/HT_2 did not correlate in the diets. This may be due to different strains of Fusarium which actually produce DON and T_2/HT_2. In a duplicate diet study with 61 adults (2004), HT_2 was detected in only 3 samples (exposure of 3.3, 1.3 and 2.7 µg per day respectively), while T_2 was detected in only 1 sample (0.8 µg per day; Van Egmond, 2005).

Conclusion

There are no data on concentrations of T_2 and HT_2 in Dutch food products. A duplicate diet study with 8-12 months-old babies is the only exposure study for children in the Netherlands. This study indicates that babies with relatively high wheat consumption have a mean intake of 0.04 µg/kg bw per day (range 0.01 to 0.16 µg/kg bw per day). Based on the information available, a conclusion on the time trend of the exposure to T_2 and HT_2 cannot be drawn. However, contamination levels of European cereals with T_2/HT_2 have been increasing in the last few years, especially in Nordic countries (Van Egmond, personal communication, March 2007).

4 Discussion and conclusions

4.1 Trends in consumption and exposure

In this document we reported on trends in the consumption of foods relevant for the exposure to selected chemicals present in food and trends in exposure to these chemicals. For an overview of the results see Table 4.1. It is clear that for most compounds, based on (observed) trends in consumption and/or contamination levels and/or exposure (e.g. due to new legislation), a decrease in exposure is expected relative to the last reported exposure levels in the literature. For sweeteners however, an increase is more likely due to an increasing trend in consumption of light soda's and the probable broader use of these compounds in foods. For T_2/HT_2 an increase in exposure might be likely due to possible rising levels in cereals. For the exposure to aflatoxin B_1 a trend with time is not expected, based on the two duplicate diet studies with adults. For aflatoxin B_1, OTA, DON and T_2/HT_2 exposure levels are expected in 2007 for children aged 2-6 years based on duplicate diet portions. It should be noted that the reported trends in eating habits are based on three food consumption surveys conducted in the past and that an examination into the extension of these trends in the present was not part of this study.

Table 4.1. Overview in trends of exposure in children (0.5-12 years) living in the Netherlands for selected chemicals.

Chemical	Expected	Reason
Acrylamide	Decrease	Mitigation measures
Aflatoxin B_1[1]	Stable	No trend in consumption of relevant foods No trend in exposure based on duplicate diet study adults
Dioxins	Decrease	Decreasing contamination levels in foods, but levelling off Decreasing reported exposure levels, but levelling off
DON	Decrease	Decreasing contamination levels due to stricter legislation
Nitrate	Decrease	Decreasing trend in the consumption of vegetables and fruits (Probable) decreasing trend in concentration levels
OPs[2]	Decrease	Decreasing trend in the consumption of vegetables and fruits (Weak) decreasing trend in exposure
OTA[3]	Decrease	Decreasing contamination levels due to stricter legislation Decreasing trend in duplicate diet study adults
Patulin[1]	Decrease	Decreasing contamination levels due to stricter legislation Decreasing consumption of processed apple products in children
Sulphite	Unknown	No trend in consumption of relevant foods Lack of comparable exposure assessments over time
Sweeteners	Increase	Increasing trend in the consumption of light soda's and the potential broader use of sweeteners in other food groups, for example milk drinks.
T_2/ HT_2[4]	Increase?	Possible rising levels in cereals

[1] *No actual exposure levels in children were available*

[2] *OPs = organophosphorus pesticides*

[3] *OTA = ochratoxin A*

[4] *Only exposure levels in children based on a duplicate diet study in infants (Schothorst et al., 2005).*

Due to a report published by Hulshof et al. (1998) we were fairly well able to report on trends in consumption over the 10-year period in which the three DNFCS were conducted. The methodology of food consumption data collection used in these three surveys is fairly comparable and differences in the coding systems were accounted for. To report on trends on food consumption patterns it would have been better to examine data collected on trends by e.g. market research bureaus (as GfK) and product boards. However these data are not freely available and were left outside this project.

For the exposure in the different years, comparisons are more difficult to make due to differences in methodology of assigning concentrations to foods (e.g. nitrate, acrylamide, sulphite, sweeteners), differences in exposure models applied (e.g. dioxins, sweeteners), differences in Toxic Equivalency Factors (dioxins) and Relative Potency Factors (OPs) used to calculate cumulative levels, selection of compounds included in the assessment (e.g. OPs) and the development of models to perform can, as for example for sulphite, not be made. Further elaboration on these differences is however outside the scope of this project.

4.2 Actual levels of exposure

Apart from a trend in possible exposure levels which may indicate whether a health problem related to a certain chemical either emerges, increases or decreases, also the actual levels of exposure to these chemicals should be addressed. For this we listed in Table 4.2 the most recent reported exposure level for the age group with the highest exposure level and compared these with the most appropriate toxicological reference value (Table 4.2). This comparison shows that for some compounds the exposure exceeds a certain safety limit, namely for acrylamide, dioxins, DON (P95) and sulphite (P95; although the latter exposure estimation is a worst-case estimation). In addition, the combined health limit for T_2/HT_2 may be exceeded, as the maximum exposure level (0.16 µg/kg bw per day) analysed in the duplicate diet was higher than the TDI (Table 4.2). However, since we deal here with a short term estimate and the adverse effect of the mycotoxins is long-term, these two cannot be directly compared. Whether the long-term exposure to T_2/HT_2 exceeds the health limit is therefore uncertain.

Whether for the first three substances mentioned the expected declining trends in exposure as listed in Table 4.1 will result in a decrease in exposure below the health limits needs to be examined. For example, the predicted exposure for 2-years-old of 1.8 pg WHO-TEQ/kg bw per day in 2004, based on trends observed in a 40-year-old, is only a prediction and verification of this declining trend is necessary (Section 3.2.3). This is especially so in view of the fact that the decline in dioxin levels in foods has been levelling off since 1990. Another example is acrylamide, a genotoxic and carcinogenic compound, for which a Margin of Exposure (MoE)[6] of 10.000 may be deemed acceptable. Furthermore even with a decreasing trend in (mean/median) exposure levels, there will still be children at the upper tail of the distribution whose exposure levels exceed a reference dose. These children may be at risk and should be considered.

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[6] The Margin of Exposure (MoE) is the ratio between the estimated exposure to a compound (e.g. mean 90th percentile) and the effect dose at which a just acceptable adverse effect occurs (e.g. 5% reduction in body weight, 20% reduction in enzyme activity). A low MoE means high risk and a high MoE a low risk.

Table 4.2. Exposure levels to different chemicals for the age groups with the highest levels of exposure, including relevant safety limits for exposure.

Chemical	Age group[1]	Best estimate of exposure	Type of exposure	Relevant reference value[2]	Unit
Acrylamide	1-6 years	1.04 (mean) 1.1 (P95)	Long-term	300 (BMDL; JECFA, 2006)	µg/kg bw per day
Aflatoxin	-	-	-	-	-
Dioxins	2 years	2.8 (median)	Long-term	2 (TDI; SCF, 2001)	WHO-TEQ/kg bw per day
DON	1-2 years	0.5 (median) 1.0 (P95)	Long-term	1 (SCF, 2002)	µg/kg bw per day
Nitrate	1-1½ years	2.7 (mean)	Long-term	3.65 (Speijers, 1996)	mg/kg bw per day
OPs[3]	8 -12 months	27 (P99.9)	Short-term	50 (ARfD; FAO, 2002)	µg/kg bw per day
OTA[4]	1 year	2.8 (median) 4.9 (P95)	Long-term	100 (JECFA, 2001)	ng/kg bw per day
Patulin	-	-	-	-	-
Sulphite	1-3 years	0.33 (mean)[5] 1.72 (P95)	Means[6]	0.7 (ADI)[7]	mg/kg bw per day
Sweeteners					
Acesulfame-K	1-2 years	0.23 (mean)	Mean[6]	15 (ADI)[7]	mg/kg bw per day
Aspartame	1-2 years	0.3 (mean)	Long-term	40 (ADI)[7]	mg/kg bw per day
Cyclamic acid and its salts	1-2 years	0.11(mean)	Mean[6]	7[7]	mg/kg bw per day
Saccharine	1-2 years	0.09 (mean)	Mean[6]	5[7]	mg/kg bw per day
Neohesperidine dhydrochlcone	1-2 years	0.03 (mean)	Mean[6]	5[7]	mg/kg bw per day
T_2/HT_2	8-12 months	0.04 (mean)	Short-term	0.06 (SCF, 2002)	ug/kg bw per day

[1] *Age group selected per compound with the highest exposure level reported.*

[2] *BMDL = lower bound one-sided confidence limit (95%) of the benchmark dose (for further explanation see text). TDI = tolerable daily intake; ARfD = acute reference dose.*

[3] *OPs = organophosphorus pesticides*

[4] *OTA = ochratoxin A*

[5] *Exposure estimate is a worst case assessment using maximum permitted levels.*

[6] *In these assessments no statistical method was used to extrapolate daily mean exposure levels to long-term exposure levels (for more details see Section 2.4).*

[7] *www.food-info.net*

4.3 Residue and contaminant levels in foods and/or raw agricultural products

Another relevant issue to address when examining trends in and actual levels of exposure to chemicals in food is the residue or contaminant data used in the assessments. Most exposure assessments reported in this report were performed using data from the KAP database (Quality Programme for Agricultural

Products[7]; Van Klaveren, 1999), for example OPs, nitrate, and acrylamide. This database contains levels of all kinds of chemicals as analysed in Dutch monitoring programmes, predominantly by the Dutch Food and Consumer Product Safety Authority (VWA). Most of these analyses are performed on raw agricultural commodities (e.g. fish, cereals, milk, meat, fruit and vegetables), while a small part of the results is related to consumer products such as bread and biscuits (e.g. for acrylamide and DON). These analyses are mainly of high quality and comply with ISO17025 (Van der Schee, 2004).

Sampling in monitoring programmes is partly targeted (Van Klaveren et al., 2006). Yearly sampling procedures are adjusted based on the experience of previous years and emphasis is more or less on those products for which the probability of exceeding a legal residue limit is highest. This is true for all monitored compounds and understandable from a cost control point of view. However, it is commonly (rightly) assumed that within the sampled products those with high levels are not beforehand known. Due to this, exposure levels calculated with residue or contaminant levels from monitoring programmes may be higher than those encountered in real life. Furthermore, the more or less adjustment of product analysis every year can hamper possible trend analyses in exposure. Another factor that should be pointed out when interpreting exposure levels derived from monitoring analyses is that monitoring programmes are primarily directed to major products consumed with only minor capacity reserved for minor products. For realistic exposure assessments it would therefore be advisable that more representative samples were monitored and evenly distributed over the products available on the market containing these chemicals. Another point is that most analyses are performed in composite samples. When dealing with one compound, this is effective. However, when dealing with the exposure to more than one chemical at a time composite samples may not be ideal (Boon and Van Klaveren, 2003). If a sample contains more than one pesticide with a common mechanism of action the question can be asked whether these pesticides are present on one unit or whether they are present on different units in one sample. It is now assumed that all chemicals analysed in a composite sample are present in one sample, which is a worst-case assumption. Analyses performed on individual units would then result in better estimates of exposure.

For patulin, and T_2/HT_2 we were not able to establish trends in exposure because these compounds were until recently not included in Dutch monitoring programmes. Also for sulphite actual levels in relevant foods were missing. Exposure calculations up-to-now have been performed using maximum permitted levels, because data regarding the concentration in foods is limited. When using the maximum permitted levels, the exposure is likely to be overestimated. The use of label information is a limited option for sulphite because sulphite is not declared when the concentration is below 10 mg/kg or 10 mg/L. Therefore when using information from labels or regulation as concentration data, foods with less than 10 mg/kg or 10 mg/L sulphite are not taken into account. Currently, the FSA (Food Standard Agency, UK) has a research programme involving the development and validation of appropriate methodology to measure levels of additives, including sulphite in foods. For dioxins, the exposure calculations rely on a relative small number (max. N=10 in Baars et al., 2004, N=4-25 in De Mul et al., 2008) of samples, which are then mostly pooled in one composite sample. Whether these samples represent the actual average levels in foods is uncertain.

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[7] www2.rikilt.dlo.nl/kap/index.html

4.4 Conclusions

- For most compounds a decreasing trend in exposure in Dutch children aged 1-12 years is expected, due either to changes in eating habits and/or concentration levels.
- For sweeteners the expectation is that the exposure has increased since the last exposure assessment, primarily due to changes in eating habits, but also due to a probable broader use of sweeteners in food products. For T_2/HT_2 an increase in exposure might also be likely due to possible rising levels in cereals.
- Trends in themselves provide only information on whether exposure levels are increasing or decreasing in time. Therefore also actual levels of exposure were addressed and compared to the relevant toxicological reference value. This showed that for acrylamide, dioxins, DON and sulphite (although the sulphite exposure estimate is a worst-case estimate) these values were exceeded. The combined reference value for T_2/HT_2 may be exceeded as well. However more information is needed to confirm this. Whether a possible expected decreasing trend in the exposure of acrylamide, dioxins and DON will result in an exposure level below these reference values needs to be examined.
- Monitoring samples available for exposure assessments are (or will very soon be made) available via the KAP-database and are of high quality, although more or less targeted. This should be taken into account when interpreting the results of exposure assessments using these types of data.
- For a proper trend analysis and to estimate the ‘real’ exposure as good as possible, it is recommendable that sampling of products is performed at random (so not risk-driven), comparable between years, and covers the whole diet.
- For some mycotoxins no monitoring data are yet available. If analysed, these results should be made available for exposure assessment purposes. This is also true for sulphite for which now maximum permitted levels are used in assessments.

5 References

Abbas HK (Ed) (2005). Aflatoxin and Food Safety. Taylor and Francis, Boca Raton.

Anonymous (1988). Wat eet Nederland. Resultaten van de voedselconsumptiepeiling 1987-1988 (Results of the Dutch National Food Consumption Survey 1987-1988). Rijswijk.

Anonymous (1993). Zo eet Nederland 1992. Resultaten van de Voedselconsumptiepeiling 1992 (Results of the Dutch National Food Consumption Survey 1992). Voorlichtingsbureau voor de Voeding, Den Haag.

Anonymous (1998). Zo eet Nederland. Resultaten van de Voedselconsumptiepeiling 1997-1998 (Results of the Dutch National Food Consumption Survey 1997–1998). Voedingscentrum, Den Haag.

Baars AJ, Bakker MI, Baumann RA, Boon PE, Freijer JI, Hoogenboom LAP, Hoogerbrugge R, Van Klaveren JD, Liem AKD, Traag WA, De Vries J (2004). Dioxins, dioxin-like PCBs and non-dioxin-like PCBs in foodstuffs: occurrence and dietary intake in The Netherlands. Toxicology Letters 151: 51-61.

Bakker M, Pieters MN (2002). Risk assessment of ochratoxin A in the Netherlands (388802025). National Institute for Public Health and the Environment (RIVM), Bilthoven. Available at <www.rivm.nl>.

Bilau M, Matthys C, Vinkx C, De Henauw S (2003). Probabilistic exposure assessment for acrylamide in Flemish adolescents. Toxicology Letters 21: s60.

Boon PE, Van Donkersgoed G (2006). Risicobeoordeling inzake aanwezigheid van aspartaam in voedingsmiddelen: innameberekeningen (Risk assessment concerning the presence of aspartame in food: intake calculations). RIVM-RIKILT Front Office Voedselveiligheid, Bilthoven, Wageningen.

Boon PE, Van Klaveren JD (2003). Cumulative exposure to acetylcholinesterase inhibiting compounds in the Dutch population and young children (2003 003). RIKILT - Institute of Food Safety, Wageningen UR, Wageningen. Available at <www.rikilt.wur.nl>.

Boon PE, De Mul A de, Van der Voet H, Van Donkersgoed G, Brette M, Van Klaveren JD (2005). Calculations of dietary exposure to acrylamide. Mutation Research 580: 143-55.

Boon PE, Tjoe Nij EIM, Koopman N, Van Klaveren JD (2004). Dietary habits and exposure to pesticides in Dutch infants (2004.017). RIKILT - Institute of Food Safety, Wageningen UR, Wageningen. Available at <www.rikilt.wur.nl>.

Breedveld BC and Hulshof KFAM (2002). Zo eten jonge peuters in Nederland (Consumption habits of young children in the Netherlands). Voedingscentrum, Den Haag.

Brussaard JH, Van Dokkum W, Van der Paauw CG, De Vos RH, De Kort WLAM, Löwik MRH (1996). Dietary intake of food contaminants in the Netherlands (Dutch Nutrition Surveillance System). Food Additives and Contaminants 13: 561-573.

De Boer EJ, Hulshof KFAM, Ter Doest D (2006). Voedselconsumptie van jonge peuters (Food consumption of young children) (V6269). TNO-Quality of Life, Zeist.

De Boer WJ, Van der Voet H (2006). MCRA, a web-based programme for Monte Carlo Risk Assessment, Release 5. Biometris and RIKILT, Wageningen UR, Wageningen. Available at <mcra.rikilt.wur.nl/Documentation/Documentation.aspx>.

De Mul A, Bakker MI, Zeilmaker MJ, Traag WA, Van Leeuwen SP, Hoogenboom RL, Boon PE, Van Klaveren JD (2008). Dietary exposure to dioxins and dioxin-like PCBs in The Netherlands anno 2004. Regulatory Toxicology and Pharmacology 51: 278-287.

Dybing E, Farmer PB, Andersen M, Fennell TR, Lalljle SPD, Muller DJG, Olin S, Peterson BJ, Schlatter J, Scholz G, Scimeca JA, Slimani N, Tornqvist M, Tuijtelaars S, Verger P (2005). Human exposure and internal dose assessments of acrylamide in food. Food and Chemical Toxicology 43: 365-410.

EFSA (2005). Opinion of the Scientific Committee on a request from EFSA related to a harmonised approach for risk assessment of substances which are both genotoxic and carcinogenic. The EFSA Journal 282: 1-31.

EU (2003). Information on ways to lower the levels of acrylamide formed in food. Note of the meeting of experts on industrial containments in food: Acrylamide workshop 20-21 October 2003. Available at <europa.eu/food/food/chemical-safety/contaminants/acryl_guidance.pdf>.

European POPs Expert Team (2002). Preparatory actions in the field of dioxins and PCBs. European Commission, Brussels. Available at <ec.europa.eu/environment/dioxin/>.

EU-SCOOP (2000). Assessment of dietary intake of dioxins and related PCBs by the population of EU Member States. Task 3.2.5, Final Report SCOOP/DIOX/REPORT/1, Directorate General Health & Consumer Protection, European Commission, Brussels. Available at <ec.europa.eu/dgs/health_consumer/library/pub>.

EU-SCOOP (2002a). Assessment of dietary intake of Ochratoxin A by the population of EU Member States. Task 3.2.7. Directorate General Health & Consumer Protection, European Commission, Brussels. Available at <ec.europa.eu/food/fs/scoop/index_en.html>.

EU-SCOOP (2002b). Assessment of dietary intake of Patulin by the population of EU Member States. Task 3.2.8. Directorate General Health & Consumer Protection, European Commission, Brussels. Available at <ec.europa.eu/food/fs/scoop/index_en.html>.

FAO (2002). Pesticide residues in food - 2002. Report of the Joint Meeting of the FAO Panel of Experts on Pesticide Residues in Food and the Environment and the WHO Core Assessment Group, FAO Plant Production and Protection Paper 172. Food and Agricultural Organisation, Rome. Available at <www.fao.org/ag/agp/AGPP/Pesticid>.

Gilbert J, Brereton P, MacDonald S (2001). Assessment of dietary exposure to ochratoxin A in the UK using a duplicate diet approach and analysis of urine and plasma samples. Food Additives and Contaminants 18: 1088-1093.

Health Council of the Netherlands (2004). Pesticides in food: assessing the risk to children (2004/11). Health Council of the Netherlands, The Hague. Available at <www.gr.nl>.

Hulshof, KFAM (1989). Het gebruik van zoetstoffen door de Nederlandse bevolking, naar leeftijd en geslacht. Voedselconsumptiepeiling 1987-1988 (The use of sweeteners by the Dutch population, stratified by age and sex. Dutch National Food Consumption Survey 1997-1998) (V89.600). TNO-Voeding, Zeist.

Hulshof KFAM (2001). Assessment of the intake of additives that have not passed Tier 1 in the Netherlands (V3450). TNO-Voeding, Zeist.

Hulshof KFAM (2005). De consumptie van frisdranken, suikervrije frisdranken, siropen en vruchtensappen/-dranken over een periode van 10 jaar (1987/88-1997/98) (The consumption of soft drinks, sugar-free soft drinks, syrups and fruit juices during a period of 10 years (1987/88-1997/98)) (FCRA 05-0644 HUK/diw). TNO-Voeding, Zeist.

Hulshof KFAM, Ter Doest D (2003). Onderzoek naar de inneming van geselecteerde additieven fase 2 en 3 (A study into the intake of selected additives phase 1 and 2) (V5483). TNO-Voeding, Zeist.

Hulshof KFAM, Ter Doest D (2006). De consumptie van groenten en fruit in meer detail op basis van de voedselconsumptiepeilingen in Nederland (The consumption of vegetable and fruit in more detail based on the Dutch National Food Consumption Surveys) (V6831). TNO-Voeding, Zeist.

Hulshof KFAM, Kistemaker C (1995). De inneming van energie en voedingsstoffen door 1-, 2-, 3-, 4- en 5-jarige kinderen in Nederland. Voedselconsumptiepeiling 1992 (The intake of energy and nutrients by children aged 1, 2, 3, 4 and 5 year in the Netherlands. Dutch National Food Consumption Survey 1992) (95.521). TNO-Voeding, Zeist.

Hulshof KFAM, Kistemaker C, Beemster CJM, Bouman M (1998). De inname van energie en voedingsstoffen over een periode van tien jaar - Resultaten van drie voedselconsumptiepeilingen: 1987-1988, 1992, 1997-1998 (The intake of energy and nutrients during a period of 10 years - Results of three Dutch National Food Consumption Surveys: 1987-1988, 1992, 1997-1998) (98.816). TNO-Voeding, Zeist.

Hulshof KFAM, Kistemaker C, Bouman M (1992). Het gebruik van diverse typen zoetstoffen door verschillende bevolkingsgroepen. Voedselconsumptiepeiling 1992 (The use of different types of sweeteners by different groups of the population. Dutch National Food Consumption Survey 1992) (V95.301). TNO-Voeding, Zeist.

Huwe JK (2002). Dioxins in food: A modern agricultural perspective. Journal of Agricultural and Food Chemistry 50: 1739-50.

IARC (1994). Acrylamide. International Agency for Research on Cancer, Lyon, France.

ILSI (1999). A framework for cumulative risk assessment, Institute for Life Sciences, Washington, DC.

JECFA (1998). Safety evaluation of certain food additives and contaminants, WHO Food Additives Series 40. The 49th meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA), World Health Organization, Geneva.

JECFA (2001) Safety evaluation of certain mycotoxins in food. Prepared by the 56th Meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). WHO Food Additives Series L: 46. Ochratoxin A, pp. 281-415. World Health Organization, International Programme on Food Safety, Geneva.

JECFA. (2006). Evaluation of certain food contaminants (64th report of the Joint FAO/WHO Expert Committee on Food Additives), World Health Organisation, Geneva.

Konings EJ, Baars AJ, Van Klaveren JD, Spanjer MC, Rensen PM, Hiemstra M, Van Kooij JA, Peters PW (2003). Acrylamide exposure from foods of the Dutch population and an assessment of the consequent risks. Food and Chemical Toxicology 41: 1569-79

Liem AKD, Theelen RMC (1997). Dioxins. Chemical Analysis, Exposure and Risk Assessment. Thesis, Utrecht University, Utrecht.

Luijk R, Schalk S, Muilerman H (2000). Verliezen we het verstand? Restanten zenuwgif schadelijk voor de hersenontwikkeling van onze kinderen. Consumentenbond en Stichting Natuur en Milieu.

Mileson BE, Chambers JE, Chen WL, Dettbarn W, Ehrich M, Eldefrawi AT, Gaylor DW, Hamernik K, Hodgson E, Karczmar AG, Padilla S, Pope CN, Richardson RJ, Saunders DR, Sheets LP, Sultatos LG, Wallace KB (1998). Common mechanism of toxicity: A case study of organophosphorus pesticides. Fundamental and applied toxicology 41: 8-20.

National Research Council (1993). Pesticides in the diets of infants and children. National Academy Press, Washington.

Nusser SM, Carriquiry AL, Dodd KW, Fuller WA (1996). A semiparametric transformation approach to estimating usual daily intake distributions. Journal of the American Statistical Association 91: 1440-1449.

Pieters MN, Bakker MI, Slob W (2004). Reduced intake of deoxynivalenol in The Netherlands: a risk assessment update. Toxicology Letters 153: 145-153.

Pieters MN, Freijer J, Baars BA, Slob W (2001). Risk Assessment of deoxynivalenol in food. RIVM report 388802022, National Institute for Public Health and the Environment (RIVM), Bilthoven. Available at <www.rivm.nl>.

Pope CN (1999). Organophosphorus pesticides: do they all have the same mechanism of toxicity? Journal of Toxicology and Environmental Health Part B Critical Reviews 2: 161-81.

SCF (1998). Further advice on the opinion of the Scientific Committee for Food expressed on the 19th September 1997 on a Maximum Residue Limit (MRL) of 0.01 mg/kg for pesticides in foods intended for infants and young children (adopted by the SCF on 4 June 1998), European Commission, Brussels.

SCF (2001). Opinion of the SCF in the risk assessment of dioxins and dioxin-like PCBs in food. Update based on new scientific information available since the adoption of the SCF opinion of 22nd November 2000, European Commission, Brussels.

SCF (2002). Opinion of the SCF on Fusarium toxins Part 6: Group evaluation of T_2 toxin, HT_2 toxin, nivalenol and deoxynivalenol, February 2002, European Commision, Brussels.

Schothorst RC, Van Egmond HP, De Mul A, Boon PE, Van Klaveren JD, Speijers GJA (2005). Trichothecenes in baby food. RIVM report 310301002, National Institute for Public Health and the Environment (RIVM), Bilthoven. Available at <www.rivm.nl>.

Sizoo EA, Van Egmond HP (2004). Analysis of 24-hour duplicate diet samples for aflatoxin B_1, aflatoxin M1 and ochratoxin A. Food Additives and Contaminants 22: 163-172.

Slob W (1993) Modeling long-term exposure of the whole population to chemicals in food. Risk Analysis 13: 525-530.

Speijers GJA (1996). Nitrate. In: Toxicological evaluation of certain food additives and contaminants in food. WHO Food Additives Series: 35. WHO/IPCS, Geneva.

Svensson K, Abramsson L, Becker W, Glynn A, Hellenas KE, Lind Y, Rosen J (2003). Dietary intake of acrylamide in Sweden. Food and Chemical Toxicology 41: 1581-1586.

Tareke E, Rydberg P, Karlsson P, Eriksson S, Törnqvist M (2002). Analysis of acrylamide, a carcinogen formed in heated foodstuffs. Journal of Agricultural and Food Chemistry 50: 4998-5006

Van Dooren MMH, Boeijen I, Van Klaveren JD, Van Donkersgoed G (1995). Conversie van consumeerbare voedingsmiddelen naar primaire agrarische produkten (Conversion of consumed foods into raw agricultural commodities) (95.17). RIKILT-DLO, Wageningen. Available at <www.rikilt.wur.nl>.

Van Egmond HP (2005). Letter report to Food Safety Authority (Jeuring), Trichothecenen in duplicaat 24-uurs voedingen (Trichothecenes in duplicate 24-h diets) (2005\0590 ARO Sch/sh), June 2005.

Van Egmond HP (2006). Letter report to Food Safety Authority (Jeuring), Eindrapportage onderzoek duplicaatmaaltijden op aflatoxinen en ochratoxine A (Final report on the analysis of aflatoxins and ochratoxin A in duplicate diets) (0523/2006 ARO vE/mk), July 2006.

Van Klaveren JD (1999). Quality programme for agricultural products. Results residue monitoring in the Netherlands. RIKILT, Wageningen.

Van Klaveren JD, Boon PE, De Mul A (2006). Modelling of dietary exposure to acrylamide. In: Skog K, Alexander J (Eds). Acrylamide and other hazardous compounds in heat-treated foods. Woodhead Publishing Limited, Cambridge, UK, p 195-213.

Van Klaveren JD, Noordam M, Boon PE, Van Donkersgoed G., Ossendorp BC, Van Raaij MTM, Van der Roest J (2006). Trends in normoverschrijdingen, overschrijdingen van de acute referentiewaarde en gesommeerde blootstelling (Trends in exceedances of the limit and the acute reference dose, and cumulative exposure) (2006.011). RIKILT-Institute of Food Safety, Wageningen UR, Wageningen. Available at <www.rikilt.wur.nl>.

Van Rooij-van den Bos L, Konings EJM, Heida P, Hamersveld ICM, Van der Wielen J, Kooijman M (2004). Onderzoek naar de kunstmatige zoetstoffen saccharine, aspartaam, acesulfaam-K en cyclamaat in levensmiddelen (Study into the presence of the artificial sweeteners saccharine, aspartame, acesulfame-K and cyclamic acid in food). The Food and Consumer Product Safety Authority (VWA), region South and region North. Available at <www.vwa.nl>.

Van der Schee HA (2004). Report of pesticide residue monitoring results of the Netherlands for 2003, NW03E11, The Food and Consumer Product Safety Authority (VWA), Amsterdam. Available at <www.vwa.nl>.

Westenbrink S, Jansen MCJF, Ter Doest D, Bausch-Goldbohm RA (2005). De inneming van nitraat en nitriet in Nederland (The intake of nitrate and nitrite in the Netherlands) (V6372). TNO-Voeding, Zeist.

Zeilmaker MJ, Bakker MI, Schothorst R, Krul C, Havenaar R, Slob W (2004). N-nitrosodimethylamine from fish and vegetables: exposure assessment and risk assessment, Proceedings of the Jubilee Annual meeting of the Netherlands Society for Toxicology, Veldhoven.

Zeilmaker MJ, Bakker MI, Schothorst R, Krul C, Havenaar R, Slob W (in prep.). N-nitrosodimethylamine from a fish/vegetable meal: In vitro formation and comparison with Health Based Limit Values.

Annex I Statistical methods to convert short-term exposure to long-term exposure

The method developed by **Nusser et al. (1996)** at Iowa State University (ISU):
Briefly, in this method the exposures are transformed using the Box-Cox approach, and subsequently by a spline function in order to approach normality of the distribution of transformed values. For these assumed normally distributed values the variance components within individuals and between individuals are calculated. The between-individual variance component is used to construct a normal distribution of usual intake at the transformed scale, which is subsequently back-transformed to the original intake scale. The Nusser-method is applied at RIKILT – Institute of Food Safety in the Monte Carlo Risk Assessment software (MCRA release 5.0, Boer & Voet 2006) and at TNO.

The **Statistical Exposure Model (STEM)** developed by Slob (1993):
STEM is intended to model the mean dietary exposure as a function of age. It combines regression analysis on age by fitting a regression curve to the daily exposure data, and nested variance analysis to separate within-subject variance from between-subject variance. The within-subject variance is estimated by analysing the differences between the exposures on the two (consecutive) days for each person. By subtraction of the within-subject variance from the total variance an estimate can be made of the long term between-subject variance. STEM has been applied at the RIVM until 2005. After this, an adapted version of this model was implemented in MCRA (Boer & Voet 2006).

Annex II Mean consumption of important foods groups (g per day) ± standard deviation among boys and girls in various age classes, including non-users[1,2].

	1-3 years			4-6 years			7-9 years			10-12 years		
Gender and food groups	DNFCS-1	DNFCS-2	DNFCS-3	DNFCS-1	DNFCS-2	DNFCS-3	DNFCS-1	DNFCS-2	DNFCS-3	DNFCS-1	DNFCS-2	DNFCS-3
Boys	N=163	N=149	N=135	N=128	N=164	N=138	N=120	N=127	N=104	N=148	N=136	N=112
Potatoes	66 ± 48	55 ± 45	54 ± 42	83 ± 63	72 ± 51	69 ± 54	105 ± 72	94 ± 67	98 ± 69	133 ± 96	118 ± 70	108 ± 76
Bread	68 ± 34	67 ± 29	68 ± 34	103 ± 41	100 ± 43	96 ± 41	124 ± 59	129 ± 52	123 ± 50	157 ± 62	144 ± 60	140 ± 60
Non-alcoholic drinks [3]	351 ± 234	411 ± 248	431 ± 278	414 ± 221	459 ± 222	459 ± 277	465 ± 245	549 ± 311	605 ± 333	536 ± 284	643 ± 318	713 ± 373
Fruit	99 ± 96	89 ± 68	92 ± 74	122 ± 87	99 ± 93	84 ± 85	112 ± 114	91 ± 84	85 ± 116	110 ± 103	94 ± 96	86 ± 88
Pastry and cookies	22 ± 21	25 ± 22	26 ± 22	34 ± 37	36 ± 36	31 ± 29	30 ± 30	36 ± 36	34 ± 27	37 ± 37	38 ± 35	45 ± 39
Cereal products and thickners	25 ± 34	28 ± 29	35 ± 44	24 ± 40	27 ± 39	29 ± 37	38 ± 53	26 ± 45	30 ± 44	40 ± 60	32 ± 58	47 ± 74
Vegetables	70 ± 54	52 ± 43	53 ± 35	77 ± 55	58 ± 51	63 ± 50	102 ± 76	83 ± 67	76 ± 60	114 ± 75	90 ± 62	87 ± 60
Cheese	8 ± 12	7 ± 10	8 ± 10	13 ± 17	10 ± 13	10 ± 14	13 ± 18	12 ± 15	11 ± 17	16 ± 20	20 ± 22	17 ± 20
Milk and milk products	522 ± 240	467 ± 197	520 ± 228	497 ± 196	513 ± 250	529 ± 244	488 ± 215	509 ± 254	497 ± 225	505 ± 236	492 ± 240	500 ± 264
Nuts, seeds and snacks	11 ± 18	10 ± 20	11 ± 18	16 ± 25	22 ± 32	16 ± 23	17 ± 24	29 ± 37	25 ± 38	24 ± 34	23 ± 30	38 ± 54
Fish	3 ± 11	3 ± 12	2 ± 10	2 ± 8	3 ± 13	3 ± 10	3 ± 15	4 ± 14	9 ± 28	4 ± 15	2 ± 10	3 ± 11
Meat, meat products and poultry	49 ± 34	42 ± 28	51 ± 32	65 ± 37	61 ± 46	63 ± 34	83 ± 44	72 ± 45	82 ± 47	97 ± 51	106 ± 61	91 ± 47

	1-3 years			4-6 years			7-9 years			10-12 years		
Gender and food groups	DNFCS-1	DNFCS-2	DNFCS-3	DNFCS-1	DNFCS-2	DNFCS-3	DNFCS-1	DNFCS-2	DNFCS-3	DNFCS-1	DNFCS-2	DNFCS-3
Girls	N=140	N=202	N=119	N=128	N=165	N=138	N=133	N=127	N=134	N=138	N=119	N=124
Potatoes	63 ± 47	56 ± 45	46 ± 39	87 ± 56	65 ± 50	69 ± 53	97 ± 69	83 ± 61	95 ± 66	118 ± 91	114 ± 72	99 ± 74
Bread	66 ± 30	64 ± 33	65 ± 32	85 ± 38	86 ± 39	89 ± 36	111 ± 49	106 ± 42	101 ± 45	134 ± 56	127 ± 47	116 ± 47
Non-alcoholic drinks [3]	375 ± 236	388 ± 223	422 ± 257	381 ± 233	400 ± 219	464 ± 265	432 ± 242	484 ± 234	512 ± 269	525 ± 274	585 ± 280	683 ± 306
Fruit	99 ± 76	83 ± 80	95 ± 80	105 ± 82	99 ± 80	91 ± 71	133 ± 98	94 ± 90	73 ± 73	125 ± 99	115 ± 101	82 ± 87
Pastry and cookies	25±26	23 ± 21	21 ± 19	29 ± 27	30 ± 26	29 ± 26	36 ± 30	35 ± 33	33 ± 29	38 ± 40	40 ± 37	45 ± 34
Cereal products and thickners	19 ± 25	23 ± 29	27 ± 29	20 ± 34	29 ± 40	22 ± 35	29 ± 41	30 ± 46	27 ± 43	31 ± 52	29 ± 64	39 ± 58
Vegetables	61 ± 39	49 ± 38	47 ± 35	77 ± 57	60 ± 44	61 ± 44	93 ± 68	76 ± 49	69 ± 46	100 ± 86	103 ± 76	75 ± 58
Cheese	8 ± 11	7 ± 10	7 ± 10	10 ± 11	12 ± 15	10 ± 13	13 ± 17	13 ± 16	13 ± 19	20 ± 25	21 ± 25	15 ± 18
Milk and milk products	467 ± 198	465 ± 207	473 ± 252	477 ± 198	476 ± 204	505 ± 236	472 ± 216	480 ± 233	496 ± 217	459 ± 247	477 ± 257	445 ± 246
Nuts, seeds and snacks	7 ± 13	13 ± 25	10 ± 15	14 ± 20	16 ± 23	15 ± 24	21 ± 33	23 ± 34	22 ± 26	30 ± 52	27 ± 33	29 ± 38
Fish	2 ± 10	3 ± 12	3 ± 10	3 ± 12	3 ± 11	5 ± 18	2 ± 10	6 ± 15	3 ± 11	5 ± 16	6 ± 25	6 ± 19
Meat, meat products and poultry	47 ± 31	46 ± 32	46 ± 31	65 ± 34	58 ± 42	56 ± 32	75 ± 45	68 ± 39	74 ± 53	86 ± 47	85 ± 52	80 ± 49

[1] *Estimating the consumption of food groups including the non-users indicates that the food consumption data of all participants is used, even when they did not consume a food from the specific food group.*

[2] *Data were obtained from three subsequent Dutch National Food Consumption Surveys (DNFCS; Hulshof et al., 1998).*

[3] *Including water, tea and coffee.*