HEALTH RISK ASSESSMENT OF DIOXIN EXPOSURE: THE NEERLAND-WIJK (WILRIJK, BELGIUM)

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Introduction

At the moment, about 60 % of the municipal refuse in Flanders are disposed of by incineration in 13 operational waste incinerators (situation 1997). However waste incineration has generated considerable public concern, mainly due to the emission of toxic substances, which present potential human health hazards.

In recent years residents of the Neerlandwijk regularly complained of different types of health problems. This urban district is surrounded by several sources of environmental pollutants, among them two very busy traffic arteries, a metal smelting and refining industry, a crematorium and two waste incinerators. Several children with congenital anomalies were born from mothers who were inhabitants of the area. Profound medical investigations revealed that most of these congenital malformations were of a multi-factorial origin [1]. Local people linked those cases to the presence of the nearby waste incinerators, which were in full operation since the seventies (waste incinerator IHK) and the eighties (waste incinerator ISVAG). Both waste incinerators were shut down in November 1997 due to exceeding of the dioxin emission standards (0,1 ng TEQ dioxin/m³) and considerations of health.

This paper focuses on the exposure evaluation and the risk characterization of the PCDD and PCDF contamination in the Neerlandwijk. International toxic equivalent factors (I-TEFs) are used in order to estimate the risks associated with exposure to mixtures of PCDDs/PCDFs. The major routes of human exposure and the relative contribution of each route for the period 1971 - 1997 were identified.

Materials and Methods

The dioxin emissions of both waste incinerators amounted to 18,9 TEQ/year in 1980 and to 3,1 TEQ/year in 1997. These emissions are based on available data from measurements as much as possible while missing data are estimated on the basis of validated emission factors and figures on production and use quantities.

The geographical dispersion of the total dioxin deposition as a consequence of the emissions of both municipal waste incinerators was calculated using the Operational atmospheric transport and deposition model for Priority Substances (OPS-model) [2].

This resulted in a maximum deposition field value of 1,21 ng TEQ/m² year (dry deposition 0,89 ng TEQ/m² year, wet deposition 0,22 ng TEQ/m² year, background deposition 0,1 ng TEQ/m² year) for 1997. A deposition of 1,31 ng TEQ/m² year (dry deposition 1,05 ng TEQ/m² year, wet deposition 0,26 ng TEQ/m² year) in the Neerlandwijk was measured during April-May 1997 using the Bergerhoff-method. The deposition for background areas in Flanders, is estimated to be 0,1 ng TEQ/m² year based on calculations [1,3]. The background deposition was not considered for the situation in 1980 due to lack of information but must have been significantly higher. Starting from the emissions of both municipal waste incinerators in 1980, a maximum value of

6,62 ng TEQ/m² year (dry deposition 5,18 ng TEQ/m² year, wet deposition 1,44 ng TEQ/m² year) in the Neerlandwijk was estimated.

The accumulation of PCDDs and PCDFs in environmental media was calculated, based on the results of the deposition modeling. Accumulation in the water compartment was not relevant for the Neerlandwijk. Atmospheric substances are partitioned onto soil and vegetation and may bioaccumulate in livestock (cow milk and meat). This results in a number of indirect sources for human exposure [2].
Topsoil samples were collected by Kerremans et al. at 15 different locations in the Neerlandwijk and analyzed isomer-specifically for PCDDs and PCDFs [4]. Exposure is calculated using the highest reported soil concentration (27,7 ng TEQ/kg dm). Although the municipal waste incinerators are held responsible for the PCDD/PCDF contamination, the deposition modeling does not correspond with the soil contamination pattern. This indicates that the contamination originates at least partially from other sources.
The concentrations of dioxin in vegetables, cow milk and meat were calculated using transfer factors according to Prinz et al. [5]. Samples of cow milk taken in January 1992 and May 1998 on behalf of the Ministry for Agriculture contained respectively 7,9 and 1,5 pg TEQ/g fat, which corresponded well with the model calculations using transfer factors. Recently in November 1997 two isolated cases showed concentrations of 11 pg TEQ/g fat in milk of a Jersey Cow and 10 pg TEQ/g fat in milk of a single normal cow. Exposure in 1997 is calculated using the highest (11 pg TEQ/g fat) and lowest (1,5 pg TEQ/g fat) reported concentration for cow milk. For the state in 1980 the result of the model calculation was used.

Three exposure scenarios were examined based on different exposure patterns. Although a worst case pattern (scenario I) is highly unlikely as revealed from inspection of the site, this scenario has been included to estimate exposure of individuals consuming foods (meat, milk and vegetables) produced entirely in the impact area and living in this area. The common case (scenario II) consists of individuals whose exposure pattern would be representative of the general population. These individuals would consume products sold commercially which show background concentrations of dioxins. In the intermediate scenario III it was assumed that people lived in and consumed 25 % crops and 50 % meat produced in the impact area.
If one assumes that the Dutch and Belgian diet are roughly the same, the daily background exposure to dioxins in Belgium amounts to 1 pg TEQ/kg bw for adults and to 2,4 pg TEQ/kg bw for children [6]. This dietary background exposure is taken into account proportionally in each exposure scenario.
It is assumed that the receptor population is exposed to emissions for a 70 year lifetime, divided into two age groups: children (up to 6 years old, 15 kg average body weight) and adults (age 15-70 years old, 70 kg body weight). Exposure to the sources was estimated in each group and a time-weighted average daily dose was calculated from the results.
The human exposure to PCDD/PCDF emissions from the municipal waste incinerator was calculated based on the concentration of TCDD equivalents (TEQ) in various environmental media and biota. The exposure pathways considered in this assessment include inhalation, dermal absorption from soil, ingestion of soil, vegetation, beef, water and cow's milk. Exposure was calculated as the average daily intake of TCDD equivalents per unit body weight and was estimated using the exposure formulas present in VLIER-HUMAAN [7].

Results and Discussion

The risk-index was calculated by dividing the summation of the background and lifelong exposure by the Tolerable Daily Intake as defined by the World Health Organization (WHO). Consequently, a TDI of 10 pg TEQ/kg bw day was used [8]. A risk-index < 1 means that there is no indication for a risk. On the contrary, a risk-index $\geq$ 1 indicates a risk.

Intake via food represents the most important dioxin exposure pathway for scenarios I and III. Consumption of milk, meat and vegetables contributes for more than 95 %. The relative part of inhalation is less than 2 %. Although in scenario II inhalation represents one of the major intake routes, this exposure pathway is more than 100 times smaller than the background intake. For this most common scenario in the Neerlandwijk, the main route of exposure is not associated with consumption of homegrown vegetables, meat and milk. Consequently, the exposure in this case is only slightly different from that of the general population in Flanders.

Comparison of the state in 1980 with this in 1997 shows a significant improvement of the situation (see table 1 and 2). The lifelong average exposure has even decreased with a factor 3 in scenario I. Influences are most apparent in this scenario because all exposure pathways are completely taken into account. The exposure and concomitant the risk-index increases from scenario II, III to I due to the increasing consumption of food produced in the impact area. Just residing in the impact area does not result in a meaningful risk (scenario II, risk-index = 0,12 for 1980 and 0,11 for 1997). Since the background deposition must have been significantly higher in 1980 than in 1997, the exposure for 1980 is probably underestimated and consequently could come up to or exceed the TDI in some cases (scenario I).

The calculated exposure is always higher for children than for adults. This has to be at least partially attributed to the different consumption behavior (milk) and lower bodyweight of the former. Therefore, risks for children are raised and effects cannot be excluded, in particular for the highly exposed scenario I in 1980 (16,62 pg TEQ/kg bw day). This again confirms that if one is willing to drive back the exposure for children and to a lesser extent for adults, the dioxin content in milk should be properly controlled. The calculations for scenario III in 1997 with a dioxin concentration of 11 pg TEQ/g fat resulted in an exposure 8,17 pg TEQ/kg bw day for children and 1,87 pg TEQ/kg bw day for adults. It should be noted that these results are not related to a normal milk-cow and had only implications for a few families.

The risk-index for the local population is < 1 in all cases, indicating that there are no risks if the present reference value (TDI = 10 pg TEQ/kg bw day) of the WHO is accepted as the standard. However the WHO intends to lower this value at least by half [9]. In the latter case there might be a serious indication for concern in the highly exposed scenario I.

Table 1: Calculated exposure in the Neerlandwijk for 1980 (pg TEQ/kg bw day)

scenario	child + background	adult + background	lifelong + background	risk-index (exposure/TDI)
I	16,62	5,09	6,08	0,61
II	2,48	1,05	1,17	0,12
III	6,65	1,70	2,22	0,22

Table 2: Calculated exposure in the Neerlandwijk for 1997 (pg TEQ/kg bw day)

scenario	child + background	adult + background	lifelong + background	risk-index (exposure/TDI)
I	4,93	1,71	1,99	0,20
II	2,42	1,02	1,14	0,11
III	3,42	1,26	1,45	0,15

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