DIOXIN-LIKE COMPOUNDS IN BUTTER FROM AUSTRALIA

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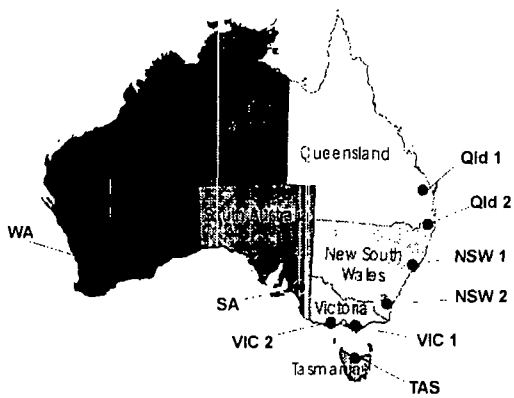
Introduction

An important aspect of human exposure to dioxin-like compounds (ie. polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans (PCDD/Fs) and polychlorinated biphenyls (PCBs)) includes the consumption of contaminated food, such as dairy products [3]. The contamination of dairy products is due primarily to accumulation of these compounds from the atmosphere onto/into plant leaves and subsequent consumption of the leaf material by cows[4]. Studies from Europe suggest that the daily PCDD/F consumption of a lactating cow via food is equivalent to approximately 100000 m^3 of air[5]. During lactation these lipophilic chemicals partition into the milk and it has been shown that the quantity of PCBs that accumulates in 1 g of dairy lipid is equivalent to between about 2.4 and 650 m^3 of air; depending on the congener investigated[10]. Hence, analysis of dairy products may be used to estimate atmospheric concentrations of dioxin-like compounds in a given region, providing that the cows consume food from this region and food additives or dairy processing/packaging do not contribute significantly to the levels of the compounds of interest[2]. In this paper, we present the concentration of dioxin-like chemicals (2,3,7,8-PCDD/Fs and PCBs that are included in the WHO-TEF scheme) in butter obtained from nine different regions in Australia.

Methods

Butter samples were obtained from nine dairy factories in six states of Australia (Fig. 1). Sub-samples were taken from the butter received to ensure minimal contact with packaging material. For analysis butter sub-samples equivalent to about 10 g of lipid were homogenised in a water bath (40 °C) and the lipophilic phase was separated then filtered through anhydrous Na_2SO_4 after which the lipid content was determined gravimetrically.

Samples were spiked with ^{13}C-labelled PCDD/F and PCB standards. For purification of the samples an automated system using activated carbon was employed, in which the PCDD/Fs and co-planar PCBs were separated from the lipid fraction (containing the mono-ortho PCBs). A pre-cleanup was performed for the mono-ortho-PCBs, using approximately 2 g H_2SO_4/SiO_2 (1.5:1) per gram lipid weight to oxidise lipids. After several hours reaction time the samples were filtered and concentrated. Samples were purified using an



acid/base activated silica gel column (from base to top: Na_2SO_4, SiO_2, H_2SO_4/SiO_2, SiO_2, KOH/SiO_2, SiO_2, H_2SO_4/SiO_2, SiO_2, KOH/SiO_2, SiO_2, Na_2SO_4). After clean-up procedures the samples were concentrated to near dryness, transferred to vials and ^{13}C-labelled 1,2,3,4-TCDD recovery standard was added to the PCDD/F and co-planar PCBs fraction. Analysis of tetra- to octa-CDD/Fs, co-planar and mono-ortho PCBs was performed on a GC (DB-5 fused silica column, 60 m, 0.25 mm i.d., 0.1 μm film thickness) interfaced to a VG Autospec mass spectrometer operating on a resolution of approximately 10,000. Identification of 2,3,7,8-substituted PCDD/Fs and PCBs was performed using retention times of the labeled standards and isotope ratios at M^+ and $M+2^+$. For quality control the retention times of the analyte in a sample had to be within 2 s of the retention times of the internal standards. The limit of detection for PCDD/Fs in a given sample was defined by a signal to noise ratio greater than 3 times the average baseline variation and a substance quantity in the sample greater than 3 times the quantity in the respective blank. The limit of detection for the individual PCDD/F congeners was less than 0.02 pg g^{-1} and ranged from 0.01 to 0.81 pg g^{-1}.

Results and Discussion

PCDD/Fs and PCBs were detectable in all butter samples analysed from Australia. The sum of detectable concentrations of PCDD/Fs and PCBs ranged from 0.06 pg WHO TEq g^{-1} fat in a sample from New South Wales (NSW1) to 0.65 pg TEq g^{-1} fat in a sample from Victoria (VIC2) with a mean concentration of 0.25 pg TEq g^{-1} fat. (Note that if the non-quantified compounds are included at half the detection limit the levels ranged from 0.12 pg WHO TEq g^{-1} fat (NSW1) to 0.67 pg TEq g^{-1} fat (VIC2) with a mean concentration of 0.29 pg TEq g^{-1} fat). To the knowledge of the authors the only other published data on PCDD/Fs and dioxin-like PCBs in dairy products from Australia originates from a Greenpeace study[9] that reported the concentration of PCDD/Fs and PCBs in a butter sample that originated from Victoria (personal communication, Matt Ruchel, Greenpeace Australia). The results from the Greenpeace sample obtained in Australia are very similar to the sample VIC 2 (with a mean normalized difference of 16 % for those PCDD/F congeners that were detected in both studies) which showed the highest PCB and PCDD/F concentrations in this study. This sample originated from the Australian state that has the highest population and industrial density. The concentration of PCDD/Fs and PCBs in all other dairy samples from Australia were relatively low compared to concentration in butter from industrialized European countries or the USA but are higher than those reported from New Zealand (for details see Ref.[6]).

The results of the PCDD/F congener and isomer profiles were relatively similar in all butter samples from Australia[6]. In brief, tetra- to octachlorinated 2,3,7,8-substituted PCDFs and tetra- to pentachlorinated PCDDs were usually below 0.1 pg g^{-1} fat. Highest concentrations were found for the 1,2,3,6,7,8-HxCDD, 1,2,3,4,6,7,8-HpCDD and in particular OCDD. Despite the relatively low concentration, the highest contribution to the toxicity equivalency factors was due to 1,2,3,7,8-PeCDD which contributed with 20 – 58 % to the TEqs (based only on chemicals above the LOD). Although the PCDD/F congener profile in the butter samples from Australia are similar to those observed in dairy samples from other industrialised countries it is noteworthy that slightly elevated HpCDD and OCDD concentrations were detected in the sample QLD2. Relatively high concentrations of HpCDD and OCDD have been found in soil and sediment samples collected all along Queensland coastal zone and have been attributed to an unknown source in the area[8,1].

In comparison to the PCDD/Fs of the dioxin-like PCBs only PCB 126 (from <0.7 – 2.1 pg g^{-1} lipid) and PCB 169 (from 0.13 – 0.34 pg g^{-1} lipid) were detectable in the majority of the samples at levels above the detection limit (i.e. 3 times the blank values). In the samples where PCB 126 was detected, PCBs contributed to about 35-45 % of the total WHO-TEqs. (Note that if ½ LOD is used for calculating the Teqs the levels increase by about a factor of 2 in samples WA and NSW1, by 27 % and 17 % for samples SA and QLD2 respectively and by less than 10 % for all other samples).

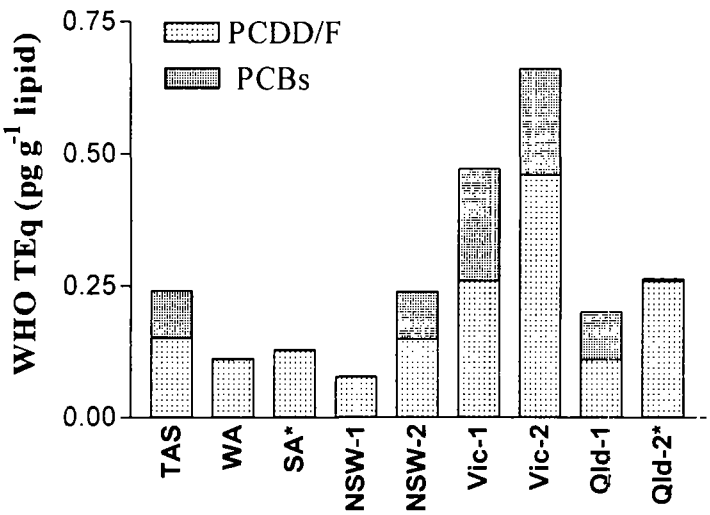


Figure 2 Concentration of detectable PCDD/Fs and PCBs expressed as WHO-TEqs in butter samples collected from Australia. *(In samples from SA and QLD2 PCB 126 was not quantifiable due to interferences. In samples NSW1 and WA PCB 126 was below the limit of detection).

The data from this study can provide some information to assess atmospheric levels of PCDD/Fs and PCBs in various regions of Australia. For example, based on the analysis of butter and assuming that the Australian cows had similar feed ratios and average daily lactation as Bavarian cows the model calculations[5] suggest atmospheric concentrations of 1,2,3,7,8-PeCDD (congener with highest contribution to TEqs) in Queensland equate to approximately 1 (QLD1) to 3 (QLD2) fg m^{-3} air. These data are in good agreement with the limited measured data obtained from suburban Brisbane (Queensland) in 1995/96 (1.0-2.8 fg m^{-3} air)[7]. Overall the measured concentration in the air of suburban Brisbane are very similar or slightly higher than those

predicted from the model of McLachlan[5] for the areas where the dairy samples QLD1 and QLD2 originated from No data on either the input parameters for the model, or for atmosphere-milk transfer factors are available for the PCBs that were quantified in this study thus no atmospheric concentrations were predicted.

In summary the data from this study indicate that the levels of PCDD/Fs and PCBs are usually relatively low in Australia.

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