Appendix J

1. CONSIDERATION OF BREAST MILK STUDIES AND DIOXINS IN CARTON MILK.

The mean concentration of 2,3,7,8-TCDD in breast milk in the 1988 survey was 5.12 ng/kg fat and that for the 1998 survey was 1.22 ng/kg fat. The subjects selected for the two surveys are were first time mothers who had been breast feeding for between 5 and 8 weeks. This relatively short period of breast feeding means that the 2,3,7,8-TCDD concentrations are likely to be comparable with those for women of similar age who have not breast fed children, and therefore it might be expected that the 2,3,7,8-TCDD concentrations in the breast milk would give a reasonable check on the accuracy of predictions by the toxicokinetic model over the 1988-1998 period.

The present version of the model using the Lorber expression for 2,3,7,8-TCDD half lives gives a concentration of 3.8 ng/kg fat for 25 year-old women in 1988 and 2 ng/kg fat for 1998. Accordingly, the actual decline in 2,3,7,8-TCDD levels between 1988 and 1998 is significantly larger than predicted by the model in its present form.

As a result of using carton milk collected in 1988 for laboratory quality control purposes during the 1988 breast milk study, it was identified that cartons used at that time added substantial quantities of dioxins to the milk contained in them. 2,3,7,8-TCDF made the largest contribution, but the dietary intake of 2,3,7,8-TCDD from this source for the population average milk intake of two litres/week (about 10 pg/person/day) was very significant, being essentially the same as the optimised 2,3,7,8-TCDD intake predicted for that time by the present version of the toxicokinetic model.

Identification of this source of dioxin exposure led to prompt action by the carton manufacturers and it appears that by about 1991 this source of 2,3,7,8-TCDD exposure had been effectively eliminated.

This means that, whereas the present "background function" used to estimate optimise 2,3,7,8-TCDD intakes shows a gradual fall in intakes from a peak in the early-mid-1960s through to the present, there would have been a sharp decrease in 2,3,7,8-TCDD intakes over the couple of years between about 1989 and 1991. This could readily explain why the model does not indicate as rapid a decline in serum 2,3,7,8-TCDD levels as indicated by the breast milk studies. The model allows for additional intakes to be incorporated over any chosen period. As I recall it, cartons came into widespread use for milk in the early 1980s. We will now incorporate an additional 10 pg/person/day 2,3,7,8-TCDD intake over the period, say, 1982-1990 and re-optimise the model. This will change the shape of the background function, presumably resulting in lower intakes during the 1990s than for the present version. This is expected to give substantially better indications of the likely rate of decrease in 2,3,7,8-TCDD concentrations for the various age groups over the 1990s. The breast milk study data will provide a useful check on the likely accuracy of these predictions.

It is important to take account of rapid and substantial changes in 2,3,7,8-TCDD intakes, such as this. If we are able to identify and quantify any other such changes, the model can incorporate these in a further optimisation.

The gradual, but substantial decline in the 2,3,7,8-TCDD intakes indicated by the model between the early-mid- 1960s and now appears quite likely to be associated with the extent of use of 245-T in agriculture to control weeds such as gorse and thistles, combined with the known decreases over the years in the 2,3,7,8-TCDD content of 245-T. As I recall it, 245-T use decreased greatly during the 1980s and has been negligible in the 1990s.

It is also possible that there may have been a substantial decrease in exposure to dioxins in general resulting from stopping use of pentachlorophenol in the timber industry. However, as I recall it, the dioxins involved are mostly more heavily chlorinated congeners and accordingly the contribution to 2,3,7,8-TCDD levels may have been quite small. Although wood stains, for example for interior use, quite commonly contained pentachlorophenol as a preservative in the 1970s/1980s, and therefore could result in domestic exposure, this does not appear to be as direct a route as from milk cartons or from use of 245-T in grazing agricultural land

While rapid changes in intake such as those from carton milk obviously affect the accuracy of the background function for estimation of general population levels of 2,3,7,8-TCDD over time, they will not affect the ability of the model to back-project "excess" 2,3,7,8-TCDD if found in Paritutu subjects. Changes such as from carton milk are incorporated into the general population levels as indicated by the adjusted 1997 survey results, and we do not really need to know these in order to establish when an individual shows "excess" 2,3,7,8-TCDD in 2003/4. Back-projection of the "excess" to what the person's exposures might have been in the 1960s and 1970s to give rise to the current excess inevitably includes the assumption that everyone is subject to the same "background" dioxin exposures. Accordingly, it does not matter what the pattern of the modelled background 2,3,7,8-TCDD intakes is, as long as it is held constant once optimised on the best available data.

It is possible that some people might have had a much higher 2,3,7,8-TCDD intake over the 1980s because of higher than average consumption of carton milk. If so, it seems likely that to ratio of 2378 TCDF to 2,3,7,8-TCDD would indicate this possibility, since carton milk contained about 10 times more 2,3,7,8-TCDF than 2,3,7,8-TCDD in the late 1980s.