

Appendix K

Meat, Milk, and Egg Transfer Coefficients

K.1 Chemical Transfer Coefficient (Tco) Derivation Methodology

Meat, cow's milk and eggs can become contaminated when food-producing animals inhale or ingest contaminated materials that then transfer into these food products. The transfer coefficients (Tco) presented in Tables K.1 and K.2 were derived from published studies investigating chemical concentrations in food products resulting from animal intake of the chemical. In most studies, the chemicals were mixed into the animal's feed, although some studies investigated the bioaccumulation of chemicals from contaminated soil in poultry feed. The Tcos, expressed in day/kilogram (d/kg), represent the ratio of contaminant concentration in fresh weight animal product (in mg/kg, for example) to the daily intake of contaminant by the animal (in mg/day). Tcos were determined only for the main food-producing animal sources, including cow's milk, eggs, and meat from cattle, pigs and chickens.

The studies selected to estimate Tcos were usually of long enough duration to allow steady-state concentrations to be reached in milk-, meat- and egg-producing animals. Steady-state concentrations in the tissues are a function of the tissue elimination half-lives (MacLachlan and Bhula, 2008). Assuming a first-order process, an exposure duration that is five times greater than the tissue elimination half-life has been used to represent time to steady-state conditions (i.e., the ratio of the measured concentration at five half-lives to steady-state concentration is 0.968).

Realistically, fast-growing animals used for food may never attain a true tissue steady-state for persistent organic chemicals due to the competing factors of growth, fattening and lactation (Fries, 1996; Hoogenboom, 2005). A steady-state concentration in food-producing animals will likely be reached more quickly than in humans due to these factors and may even show declining levels in fat during the fattening phase of the animals' prior to slaughter (Fries, 1996). The most practical approach is to base the Tco on exposure studies that expose the animal for a majority of the animals' life span up to or near marketable weight. The studies that followed tissue and milk contaminant levels during exposures over most of the animals' productive lifespan have shown that a sufficient semblance of steady-state is reached during the productive life of lactating dairy cattle and laying hens, and in meat animals prior to slaughter.

Default consumption rates of contaminated feed were used for estimating Tcos if no consumption data were provided in the primary studies. Usually, the food-producing animals in biotransfer studies were caged or treated similar to commercial farming practices. However, this exposure assessment document is primarily concerned with small farm or family farm situations in which the food-producing animals may be allowed to roam more freely than in commercial operations. This is particularly relevant for pigs and chickens. Free-range and organic farming will result in greater feed intake, slower

growth, and potentially greater contaminant exposure from range forage and soil ingestion (MacLachlan, 2010).

Specifically regarding poultry food products, the term “poultry” refers to a number of avian species that are food sources for humans. Due to the substantial human consumption of eggs and meat from chickens, the Tcos described here were exclusively based on data from chickens, laying hens (usually Leghorns) for the egg Tcos and usually meat chickens (broilers) for the meat Tcos. However, these values could also be reasonably applied to other home-raised avian species, such as turkeys and quail.

Compared to chickens and dairy cattle, fewer swine and beef cattle exposure studies could be found to estimate the biotransfer of ingested contaminants to muscle tissue. Rather than simply adopting the same cattle Tco values for swine when biotransfer data are lacking, contaminant transfer models are employed by OEHHA to estimate differences in chemical accumulation among livestock. For transfer of organic lipophilic chemicals, MacLachlan (2009) developed Physiologically Based Pharmacokinetic (PBPK) models to derive scaling factors that are used to assist the extrapolation of transfer studies, carried out most often on lactating dairy cows, to beef cattle and pigs. Given the estimated half-life (or extraction ratio for liver) of the chemical in the animal and the ratio of the chemical concentration in milk fat to body fat of dairy cows, the appropriate scaling factor can be selected and combined with the Tco derived from lactating dairy cattle to improve estimates of residues in beef cattle and pigs.

For metal Tcos, a metabolic weight adjustment can be made that accounts for differences in tissue transfer of chemicals in animals of different weight (i.e., a lower metabolic rate is expected in larger animals such as cattle compared to smaller animals such as swine, resulting in slower rates of transfer into tissues). A similar metabolic weight approach has been used to estimate the transfer of metals to dairy cattle from data in sheep (Crout et al., 2004). This adjustment is reasonable considering most of the metal compounds of interest have passive uptake and elimination processes and are subject to little or no metabolism.

The effect of metabolic weight is apparent when comparing the meat Tco values between chicken and cattle in Tables K-1 and K-2. Where published data were used to directly estimate individual chemical Tco values, the chicken Tcos were greater than cattle Tcos. For chemicals in which biotransfer could not be estimated from published reports in pigs, a default meat Tco was estimated with the following formula:

$$\text{Pig Tco}_i = (W_{\text{cow}}^{0.75} / W_{\text{pig}}^{0.75}) \times \text{cow Tco}_i \quad \text{Eq. K-1}$$

Where: $W_{\text{cow}}^{0.75}$ = live-weight in kg of a cow to the 0.75 power

$W_{\text{pig}}^{0.75}$ = live weight in kg of a pig to the 0.75 power

Pig Tco_i = pig meat Tco for chemical i

Cattle Tco_i = cow meat Tco for chemical i

Using average live weights of 500 kg for cattle and 60 kg for swine, the metabolic weight ratio adjustment is 4.8.

Table K.1 Meat, Milk and Egg Transfer Coefficients for Persistent Organic Chemicals

Organic Chemical	Tcos (d/kg) ^a				
	Cow's Milk	Chicken Egg	Chicken Meat	Cattle Meat	Pig Meat
Diethylhexylphthalate	9 x 10 ⁻⁵	0.04	0.002	6 x 10 ⁻⁴	5 x 10 ⁻⁴
Hexachlorobenzene	0.02	20	10	0.2	0.08
Hexachlorocyclohexanes	0.01	7	5	0.2	0.09
PAH's	0.01	0.003	0.003	0.07	0.06
Pentachloropenol	^b	^b	^b	^b	^b
PCB Congeners					
77	0.001	6	4	0.07	0.4
81	0.004	10	7	0.2	0.4
105	0.01	10	7	0.6	0.7
114	0.02	10	7	0.9	0.7
118	0.03	10	7	1	0.7
123	0.004	10	7	0.2	0.7
126	0.04	10	7	2	0.7
156	0.02	10	8	0.9	2
157	0.01	10	8	0.5	2
167	0.02	10	8	1	2
169	0.04	10	8	2	2
189	0.005	10	8	0.2	1
Unspeciated	0.01	10	7	0.2	0.5
PCDD/F's Congeners					
2378-TCDD	0.02	10	9	0.7	0.1
12378-PeCDD	0.01	10	9	0.3	0.09
123478-HxCDD	0.009	10	6	0.3	0.2
123678-HxCDD	0.01	10	6	0.4	0.1
123789-HxCDD	0.007	7	3	0.06	0.02
1234678-HpCDD	0.001	5	2	0.05	0.2
OCDD	0.0006	3	1	0.02	0.1
2378-TCDF	0.004	10	6	0.1	0.02
12378-PeCDF	0.004	30	10	0.1	0.01
23478-PeCDF	0.02	10	8	0.7	0.09
123478-HxCDF	0.009	10	5	0.3	0.1
123678-HxCDF	0.009	10	6	0.3	0.09
234678-HxCDF	0.008	5	3	0.3	0.06
123789-HxCDF	0.009	3	3	0.3	0.03
1234678-HpCDF	0.002	3	1	0.07	0.06
1234789-HpCDF	0.003	3	1	0.1	0.02
OCDF	0.002	1	0.6	0.02	0.03
Unspeciated	0.001	6	5	0.03	0.09

^a All Tco values were rounded to the nearest whole number

^b To be assessed for transfer to meat, milk and eggs

Table K.2 Meat, Milk and Egg Transfer Coefficients for Inorganic Metals and Chemicals

Inorganic Metals	Tcos (d/kg) ^a				
	Cow's Milk	Chicken Egg	Chicken Meat	Cattle Meat	Pig Meat
Arsenic	5 x 10 ⁻⁵	0.07	0.03	2 x 10 ⁻³	0.01 ^b
Beryllium	9 x 10 ⁻⁷	0.09	0.2	3 x 10 ⁻⁴	0.001
Cadmium	5 x 10 ⁻⁶	0.01	0.5	2 x 10 ⁻⁴	0.005
Chromium (VI)	9 x 10 ⁻⁶	NA ^c	NA	NA	NA
Fluoride	3 x 10 ⁻⁴	0.008	0.03	8 x 10 ⁻⁴	0.004 ^b
Lead	6 x 10 ⁻⁵	0.04	0.4	3 x 10 ⁻⁴	0.001 ^b
Mercury	7 x 10 ⁻⁵	0.8	0.1	4 x 10 ⁻⁴	0.002 ^b
Nickel	3 x 10 ⁻⁵	0.02	0.02	3 x 10 ⁻⁴	0.001
Selenium	0.009	3	0.9	0.04	0.5

^a All Tco values were rounded to the nearest whole number.

^b The meat Tco was estimated using the metabolic weight adjustment ratio of 4.8 from cattle to pig

^c NA – no data available or were not applicable

Speciated data existed that allowed the derivation of individual Tcos for polychlorinated biphenyls (PCBs) and polychlorinated dibenzo-p-dioxins and furans (PCDD/F), shown in Table K.1, that are of toxicological concern under the “Hot Spots” program. Tcos for unspiciated mixtures of PCBs and PCDD/Fs have also been calculated by OEHHA from literature sources and are shown in Table K.1. In risk assessments in which only the unspiciated mixture is determined, OEHHA recommends using the Tcos for PCB126 to represent the PCBs, and the Tcos for 2,3,7,8-TCDD to represent the PCDD/F's. These compounds are one of the most persistent and toxic congeners within their respective classes. The unspiciated Tco values in K.1 are for only comparison to the other Tco values. Different emissions sources of these chemicals may result in different mixtures of PCBs and PCDD/Fs, and thus influence the unspiciated Tco value.

K.2 Tco Derivations for Milk, Meat and Eggs

K.2.1 Semi- and Non-Volatile Organic Chemicals

The exposure studies used to derive organic compound Tcos often normalized the muscle tissue, egg and cow's milk contaminant concentrations to their respective fat content. The Tcos presented here are based on fresh, whole meat, egg and milk concentrations of the contaminants. If necessary, the fat concentration of a chemical was adjusted to the average fresh weight concentration using fat content default factors derived from reference sources: 0.11 for egg, 0.07 for chicken meat, 0.19 for beef cattle meat, 0.23 for pig meat, and 0.04 for cow's milk (Malisch et al., 1996; Pirard and De Pauw, 2005; U.S. EPA, 2005). If only the fat concentration of the organic chemical in egg yolk was provided in the key study, the fresh weight whole egg concentration was derived based on a fat content default value of 0.30 for yolk, and a yolk volume of 0.32

for the whole egg. If the study determined the fat content in food products, these were used for adjustment to fresh weight concentration in lieu of the default values.

For chicken meat, organic chemical content in skin was usually not included by the studies, although skin has a higher fat content and is often consumed with the meat. This would suggest that the skin could have a higher contaminant content than the muscle tissue. Due to lack of skin chemical concentration data and potential loss or destruction of organic chemicals in skin when the meat is cooked, the concentration of chemical in skin was considered similar to the concentration of a chemical in muscle for T_{co} derivation.

In general, extensive bioaccumulation of persistent, organic chemicals is not as great in either beef or dairy cattle as might be expected, even though beef cattle have no major fat excretion pathway as dairy cattle do with milk production (McLachlan, 1996). This finding is a result of the short life spans and rapid growth dilution that is characteristic of modern animal husbandry. A beef cow develops 100-150 kg of fat in which to deposit the chemical that it absorbs over its 1.5-year life. While a milk cow might excrete its absorbed contaminant in 300 kg of milk fat over the same period, it consumes more feed (and contaminant) in this time. Hence, the chemical concentrations in milk fat were not always much lower compared to beef fat (McLachlan, 1996; RTI, 2005).

Interestingly, the lower-than-expected bioaccumulation of persistent, hydrophobic chemicals in cow's milk does not translate to human milk (McLachlan, 1996). Persistent, organic chemicals tend to bioaccumulate in human milk by an order of magnitude greater than in cow's milk, presuming similar chemical concentrations in the diet on a mg/kg basis. This pronounced difference in bioaccumulation is due to a more limited capability of humans to excrete these chemicals. In addition, the extent of contaminant absorption from food in the human digestive tract may be greater. For example, nursing human infants absorb over 95% of PCBs and most PCDD/Fs while absorption in cows for these same compounds averages closer to 80%.

K.2.1.1 Diethylhexylphthalate (DEHP)

At high concentrations (1% DEHP in feed), T_{cos} for chicken eggs and breast muscle were estimated by OEHHA to be 0.04 and 0.002 d/kg (Ishida et al., 1981; Ishida, 1993). The low transfer values for DEHP relative to other organic chemicals are likely due to rapid metabolism and excretion of DEHP in the chicken.

In dairy cattle, DEHP was observed to be extensively metabolized prior to secretion into the milk (Bluthgen and Ruoff, 1998). OEHHA surmised that much of the metabolism begins in the rumen, where DEHP ester-bond cleavage would occur. Consequently, steady-state is reached in about 7 days and a low milk T_{co} of 9×10^{-5} d/kg was calculated by OEHHA. Cessation of DEHP administration resulted in nearly undetectable milk levels within 3 days post-exposure. No data could be found regarding residue levels of DEHP in cattle muscle, so a T_{co} of 4×10^{-4} d/kg was estimated after adjusting for the average fat content difference between cow's milk and cattle muscle. PBPK modeling by MacLachlan (2009) observed a ratio of about 1.5 for residues of

highly metabolized lipophilic compounds, such as DEHP, in body fat of non-lactating cows and steers to the same compound in body fat of lactating dairy cows. Thus, the Tco of 4×10^{-4} d/kg was increased by a factor of 1.5 to arrive at a Tco of 6×10^{-4} d/kg for DEHP in meat of beef cattle.

Bioaccumulation data are lacking for DEHP in pigs. Thus, a scaling factor by MacLachlan (2009) was applied for the transfer of lipophilic xenobiotics from lactating cattle to other livestock species. For chemicals such as DEHP that are extensively metabolized in the animal and have a short half-life ($t_{1/2} < 5.8$ d in lactating cows), the ratio of simulated residues in the body fat of pigs to the body fat of lactating dairy cows was essentially equal to 1. Therefore, the dairy cattle muscle Tco determined above (4×10^{-4} d/kg) was only adjusted for the difference in muscle fat content in pig to beef cattle (ratio = 1.2) to arrive at a default Tco of 5×10^{-4} d/kg for pig meat.

K.2.1.2 Hexachlorobenzene (HCB)

HCB in the atmosphere is predicted to be predominantly in the vapor phase (Lane et al., 1992). However, due to the extreme persistence of HCB and other chlorinated organic compounds in the environment, deposition and accumulation of non-volatile forms of these organics onto crops, soil and sediment are significant pathways of exposure (Eisenreich et al., 1981; Kelly et al., 1991; Douben et al., 1997; Horstmann and McLachlan, 1998).

In dairy cattle, two studies recorded nearly identical cow's milk HCB Tcos of 0.015-0.016 d/kg with 60-70 days of exposure (Fries and Marrow, 1976; Firestone et al., 1979). The data suggested near steady-state levels in milk were attained with this duration of exposure. A higher Tco of 0.030 d/kg was recorded in pregnant dairy cattle after about 8 months of exposure (Vreman et al., 1980). Steady-state was reached in milk of the pregnant dairy cattle after about 5 months. The average HCB Tco from these three studies is 0.02 d/kg.

In his review, Kan (1978) provided bioaccumulation data from which to calculate Tcos for HCB. The Tco for egg and chicken muscle were estimated at 16 and 13 d/kg, respectively.

In beef cattle, steady-state levels of HCB were at or near attainment in subcutaneous fat following ten weeks of exposure in the feed (Dingle and Palmer, 1977; RTI, 2005). A muscle Tco estimated from this study was 0.090 d/kg. Exposure to HCB in dairy cattle provided similar Tco values. A muscle Tco of 0.070 d/kg was calculated from HCB concentrations in body fat of lactating dairy cattle following 60 day exposure in the feed (Fries and Marrow, 1976). An eight-month HCB exposure in dairy cattle resulted in a muscle Tco of 0.16 d/kg (Vreman et al., 1980). Because the Vreman study provided a considerably longer exposure overall for cattle, the Tco was based on this study. The PBPK-based scaling factor data by MacLachlan (2009) was applied to estimate the transfer of HCB from lactating cattle to body fat of steers. Using data supplied by Fries and Marrow (1976), a slow elimination half-life of HCB in lactating dairy cattle (average: 50 days) and a small ratio for milk fat concentration over body fat concentration at

steady state (0.04) suggests that the PBPK-generated ratio of simulated HCB level in body fat of steers to body fat of lactating dairy cows would be about 1.5. The final default beef Tco is 0.24 d/kg (0.16 d/kg x 1.5)

No data for HCB accumulation in pig muscle tissue could be found. Therefore, a PBPK-based scaling factor was also applied to estimate the transfer of HCB from lactating cattle to pigs (MacLachlan, 2009). The PBPK model results generated a ratio of 0.5 for the simulated HCB level in body fat of pigs to body fat of lactating dairy cows. The final default pig Tco is 0.08 d/kg (0.16 d/kg x 0.5)

K.2.1.3 Hexachlorocyclohexanes (HCH)

HCH Tcos of 7.3 d/kg for egg and 5.1 d/kg for chicken meat were calculated from contaminated feed data provided by Kan (1978) and Szokolay et al. (1977). The beta-isomer tended to have roughly 10-fold greater bioaccumulation in poultry egg and muscle than the other major isomers (i.e., alpha and gamma isomers), but is generally found to a lesser extent in the environment. Hence, the Tcos represent a mean of the three major HCH isomers. MacLachlan (2008) developed a model that adequately reproduced the pattern of lindane (gamma-HCH) residue levels in fat and eggs of hens consuming contaminated feed. Utilizing the authors' data, the egg and muscle Tcos at steady-state were estimated to be 1.3 and 1.5 d/kg, respectively. These lindane Tcos were similar to those calculated from data by Kan (1978) and Szokolay et al. (1977) for eggs, 1.7 and 4.2 d/kg, respectively, and in muscle, 1.8 and 1.2 d/kg, respectively.

As in eggs and meat, the major isomers of HCH (alpha-, beta-, and gamma-HCH) had different patterns of accumulation in cow's milk. The beta isomer has the largest transfer factor, 0.025 d/kg, but generally is in the smallest proportion relative to the other 2 major isomers found in the environment (van den Hoek et al., 1975; Vreman et al., 1976; Vreman et al., 1980). Average Tco values for the alpha- and gamma- (Lindane) isomers were 0.0054 and 0.0014 d/kg, respectively (Williams and Mills, 1964; van den Hoek et al., 1975; Vreman et al., 1980; Surendra Nath et al., 2000). An average Tco for these three HCH isomers is 0.011 d/kg. Surendra Nath et al. (2000) provided data for the industrial grade HCH isomer mixture resulting in a Tco of 0.003 d/kg. The HCH mixture contained 21% gamma-HCH, but further speciation data were not included.

Vreman et al. (1980) fed dairy cows diets containing alpha- and beta-HCH for up to eight months. The calculated muscle Tcos were 0.045 and 0.19 d/kg for alpha- and beta-HCH, respectively. For lindane (gamma-HCH), a Tco of 0.027 d/kg was calculated from a different study following 12-week exposure in non-lactating dairy cattle (Claborn et al., 1960).

We applied a scaling factor by MacLachlan (2009) to estimate the transfer of HCHs from lactating cattle to beef cattle. Using data supplied by Vreman *et al.* (1980) that showed a cow's milk elimination half-life of 9-19 days for alpha- and beta-HCH, and the data by van den Hoek et al. (1975) that showed similar levels of HCH isomers in milk fat and body fat, the PBPK-generated ratio of simulated HCH levels in body fat of steers to body fat of lactating dairy cows is approximately 2. We multiplied the alpha- and beta-

HCH Tcos of 0.045 and 0.19 d/kg, respectively, which were determined in dairy cattle by the scaling factor of 2. The gamma-HCH Tco remained unchanged since non-lactating cows and steers have similar steady state HCH levels in body fat. The average Tco for these three isomers is 0.17 d/kg and is the recommended Tco for beef cattle.

No data for HCH accumulation in pig muscle tissue could be found, so we used a scaling factor by MacLachlan (2009) to estimate the transfer of HCHs from lactating cattle to pigs. Based on the HCH half-lives and milk fat to body fat ratios in dairy cattle discussed above, the PBPK-generated ratio of simulated HCH levels in body fat of pigs to body fat of lactating dairy cows is very close, or slightly greater, than 1. Thus, Tcos of the three isomers in lactating and non-lactating dairy cows were averaged by us and used as the default for pig meat ($0.045 + 0.19 + 0.027 \text{ d/kg} / 3 = 0.087 \text{ d/kg}$).

K.2.1.4 Polycyclic Aromatic Hydrocarbons (PAH)

Although there are a considerable number of studies investigating PAH exposure in the environment, there are surprisingly few studies that provide reliable data for estimating Tcos in food-producing animals. Exposure of fish, poultry and dairy cattle to a mixture of PAHs results in the presence of mainly low molecular weight PAHs (i.e., three or four cyclic rings) in the fat of meat and milk (Meador et al., 1995; Grova et al., 2000; Grova et al., 2002; Schaum et al., 2003; Lutz et al., 2006). Many of the high molecular weight PAHs with five or more cyclic rings, such as benzo[a]pyrene (BaP), are known carcinogens or possible carcinogens. Bioaccumulation of PAHs declines with increasing number of aromatic rings and the associated increase in K_{ow} , likely due to both lower gut assimilation efficiency and increased metabolism rate. Another factor appears to be that lower levels of the larger carcinogenic PAHs contaminate pastures and feed compared to the smaller PAHs, often resulting in animal milk and tissue concentrations below the detection limits of analysis equipment (EC, 2002). For example, Muhlemann et al. (2006) found that the larger carcinogenic PAHs in contaminated feed comprised only 8.3% of total PAHs, while the smaller PAHs of four rings or less contributed most of the remaining fraction.

Broiler chickens fed a diet containing low levels of PAHs found in de-inking paper sludge did not exhibit increased PAH levels in abdominal fat for nearly all carcinogenic PAHs examined (Beauchamp et al., 2002). However, the low molecular weight PAHs fluoranthene and pyrene showed increasing levels in abdominal fat with increasing levels of PAHs from paper sludge in the diet of broilers. The carcinogenic potential of these PAHs are undetermined, due to inadequate evidence of carcinogenicity in animals. The calculated broiler muscle Tco for total PAHs was 0.003 d/kg (due mainly to accumulation of pyrene and fluoranthene), and the individual PAH Tcos for pyrene and fluoranthene were 0.1 and 0.04 d/kg, respectively. The total PAH Tco of 0.003 d/kg was chosen as a poultry muscle default value for PAHs, as Tcos for the larger carcinogenic PAHs would likely not surpass this value. No data could be found for PAH accumulation in eggs. Thus, the poultry muscle Tco was also applied to the egg Tco.

The presence of PAHs in milk and milk products suggests that these foods can represent a significant part of human intake of PAHs (Schaum et al., 2003). Among PAHs, the lightest and least lipophilic ones, such as naphthalene, phenanthrene, fluoranthene and pyrene, are detected in the greatest amounts in milk from farms exposed to airborne PAHs (Grova et al., 2000; Grova et al., 2002; Cavret et al., 2005; Lutz et al., 2006). Higher molecular weight PAHs with more than four rings, including possible carcinogens or known carcinogens such as BaP, chrysene and benz[a]anthracene, have been largely undetectable in cow's milk. Of the larger carcinogenic and possibly carcinogenic PAHs, only benz[a]anthracene was detected in tank milk (pooled milk from many cows) sampled near several potential contamination sources (Grova et al., 2002). Levels of this PAH in milk fat ranged from 1.9-2.2 ng/g in milk fat (approximately 0.08-0.09 ng/g in whole milk).

Based on the pasture grass concentrations and corresponding cow's milk concentrations of the three most abundant PAHs (phenanthrene, anthracene, and pyrene) from 10 rural and urban farms investigated by Grova et al. (2000), the range of PAH Tco values in milk were 0.02 to 0.002 d/kg. However, some assumptions were made to arrive at this estimate, including pasture grass as the only source of ingested PAHs, and intake of pasture grass ranged between 10 to 100% of the cow's diet.

A cow's milk Tco range of 0.002 to 2×10^{-5} d/kg for total PAHs was calculated by OEHHA from the risk assessment by Muhlemann et al. (2006), based on measurement of total PAHs (roughly 19 PAHs measured) in contaminated feed. Although BaP consisted of only 1.5% of total PAHs, the calculated Tco was within an expected range of 0.013-0.00013 d/kg for BaP. We chose a cow's milk Tco of 0.01 d/kg for total PAHs based primarily on the high-end accumulation of BaP in cow's milk from Muhlemann et al. The recommended Tco is also within the range of 0.02 to 0.002 d/kg estimated for PAHs from data published by Grova et al. (2000).

No data could be found regarding residue levels of PAHs in cattle muscle. The ratio of simulated PAH residues in body fat of steers to body fat of lactating dairy cows for extensively metabolized lipophilic compounds is about 1.4, based on PBPK modeling (MacLachlan, 2009). Assuming equal PAH concentrations in milk fat and body fat of dairy cattle, and application of a scaling factor of 1.4 for dairy cattle to steers, we calculated a default beef Tco for PAHs of 0.067 d/kg ($0.01 \text{ d/kg} \times 0.19/0.04 \times 1.4$).

Accumulation data are also lacking for PAHs in pigs. Using the assumptions from MacLachlan (2009) for transfer of extensively metabolized lipophilic compounds to body fat in livestock, the ratio of PAHs in body fat of pigs to dairy cattle is close to 1. Based on a milk Tco of 0.01 d/kg, adjusting for fat content in pig meat and a scaling factor of 1, we calculate a default pig meat Tco of 0.058 d/kg ($0.01 \text{ d/kg} \times 0.23/0.04 \times 1$).

K.2.1.5 Polychlorinated Biphenyls (PCB)

Specific congener Tcos are recommended due to variation in absorption and metabolism of PCBs in dairy cattle, and also due to the degree of chlorination and the position of the chlorine atoms. Some PCBs are transferred effectively unchanged from grass to milk and dairy products (e.g. PCBs 118, 138, 153, 180), with the cow acting as an efficient conduit to humans, while others (e.g. PCBs 52, 101, 149) are largely removed from the environment and the human food chain if ingested by the dairy cow because they are readily metabolized by the cow (Thomas et al., 1999b). Tcos for individual PCB congeners were estimated from published data and are presented in Table K-1 (Slob et al., 1995; Thomas et al., 1998; Thomas et al., 1999a; Kerst et al., 2004; Huwe and Smith, 2005). Kerst et al. (2004) provided TEQ-adjusted data from which a Tco (WHO-TEQ) of 0.014 d/kg was estimated for unspecified PCBs.

In dairy cattle, Willett et al. (1990) reviewed early studies that examined the transfer of Aroclor 1254 applied to feed to cow's milk. Tcos of 0.008 to 0.009 d/kg were obtained with doses ranging from 3.5-200 mg/d and exposures ranging from 60-107 days. A cow's milk Tco of 0.01 d/kg for unspecified PCBs from data by Thomas et al. (1999a) was calculated for the sum of 28 PCB congeners found both in feed and the milk.

Only one study could be found that allowed development of poultry meat Tcos for a limited number of individual PCB congeners. Pirard and De Pauw (2005) determined bioconcentration factors for coplanar-PCBs (PCBs 77, 81, 126, 169) in chicken breast muscle. Traag et al. (2006) provided bioconcentration data in abdominal chicken fat for all PCBs but exposure lasted only seven days. Because steady-state was not attained, Tcos could not be reliably determined. However, the data do indicate that based on the number of chlorines, the coplanar-PCBs are similarly, or more, bioaccumulative in fat compared to the other PCB congeners with the same number of chlorines. Thus, Tcos for the non-coplanar PCB congeners in Table K-1 were based on the co-planar PCBs with the same number of chlorines.

No reliable data could be found for developing individual congener Tcos for chicken eggs. Thus, the muscle Tcos for individual PCB congeners were also used for eggs, following adjustment for the higher fat content of eggs (11%) compared to muscle (7%).

A general PCB egg Tco of 6.7 d/kg was calculated from a laboratory study in which seven reference congeners (only one of which (#118) is listed in Table K-1) were spiked in the diet of hens (De Vos et al., 2005). Because none of the more bioaccumulative co-planar PCBs were investigated in this study, the co-planar PCB Tco of 10 d/kg was used for unspecified PCBs. Numerous unspecified PCB feed-to-muscle tissue studies have been published in chickens, resulting in a range of Tco values of 2.5 to 7.7 d/kg (Hansen et al., 1983; De Vos et al., 2003; Maervoet et al., 2004; De Vos et al., 2005; Pirard and De Pauw, 2005). A Tco of 7 d/kg for unspecified PCBs was selected as the default value to reflect the median Tco of the individual congeners listed in Table K-1, and because this value is within the range of Tcos for unspecified PCBs.

No reliable data could be found that estimated transfer of PCBs consumed in food to body fat of beef cattle. In dairy cattle, Willett et al. (1990) reviewed early experiments that examined the transfer of Arochlor 1254 from feed to adipose tissue. Fresh weight dairy beef Tcos of 0.013 to 0.027 d/kg were obtained for doses ranging from 10-200 mg/d with 60 day exposures. In another study, a beef Tco of 0.024 d/kg was calculated for dairy cattle following 14-week consumption of PCBs that naturally contaminated pastures (Thomas et al., 1999a).

On a fat weight basis, Thomas et al. (1999b) observed that not only are the PCB concentrations in body fat and milk fat similar, but that the congener patterns were similar as well. Thus, even though comprehensive congener-specific data are lacking for PCBs in muscle, congener-specific beef Tcos can be estimated from the cow's milk Tco data by adjusting for the greater fat content in muscle tissue (19%) compared to the milk fat content (4%).

We applied a PBPK-generated scaling factor developed by MacLachlan (2009) to estimate the transfer of PCBs from body fat of lactating cattle to body fat of beef cattle. Using data by Huwe and Smith (2005) that found a cow's milk half-life of 39-196 days for some co-planar PCBs, and the data by Thomas et al. (1999b) that showed similar levels of PCBs in milk fat and body fat, the ratio of simulated co-planar PCB levels in body fat of steers to body fat of lactating dairy cows is approximately 10. We multiplied the scaling factor of 10 by the PCB milk Tcos in Table K-1 following adjustment for differences in fat content between milk and beef to generate Tcos for beef.

In swine, Arochlor 1254 was added to feed for 6 months resulting in an unspciated PCB Tco of 0.52 d/kg (Hansen et al., 1983). Spciated Tcos for 16 PCBs could be determined from the data, although only one PCB (#118) is currently listed in Table K-1. Thus, Tcos for individual PCBs in Table K-1 were based on the highest calculated PCB Tco with the same number of chlorines from the Hansen et al. study.

K.2.1.6 Polychlorinated Dibenzo-p-Dioxins and Furans (PCDD/F)

Numerous studies have investigated the feed-to-cow's milk transfer of PCDD/Fs. Several of these studies were conducted in the field near municipal solid waste incinerators, or estimated the mass balance of PCDD/F intake resulting from exposure to background or elevated levels of PCDD/Fs in pasture and soil (McLachlan et al., 1990; Slob et al., 1995; Schuler et al., 1997b; McLachlan and Richter, 1998; Lorber et al., 2000). These types of studies likely represent the best data for developing individual congener and overall unspciated transfer factors of PCDD/Fs from "Hot Spots" facilities. Averaged congener Tco values were estimated from these data and are presented in Table K-1.

The milk Tco decreases by an order of magnitude or more for some of the higher chlorinated PCDD/Fs. This trend agrees with models showing that the percent transfer of chemical from feed to milk decreases for compounds with log Kow larger than about 6.5 (McLachlan, 1996). This reduced absorption is attributed to the presence of an aqueous resistance that limits diffusion of very hydrophobic compounds through the

intestinal wall. Thus, a Tco for total PCDD/Fs (unspeciated PCDD/Fs) has not been pursued by researchers in their exposure studies. Nevertheless, a Tco for unspeciated dioxin-like PCDD/Fs of 0.001 d/kg can be calculated from the data by McLachlan et al. (1990).

Several studies provided data from which Tcos could be estimated for individual PCDD/F congeners found in eggs and chicken meat. For eggs, transfer factor data were derived from three studies in which feed was mixed with soil environmentally contaminated with PCDD/Fs (Petreas et al., 1991; Stephens et al., 1995; Schuler et al., 1997a), and one study of feed contaminated with fly ash (Pirard and De Pauw, 2006). Individual congener Tcos among the studies were similar, often within a factor of five between values. An average Tco was calculated for each congener from the four studies and is shown in Table K-1.

Many of the same studies in chickens also estimated accumulation values for the sum of all PCDD/F congeners, or unspeciated PCDD/Fs, in eggs and meat. In egg, four studies in free-range and laboratory chickens exposed to contaminated soil provided an average Tco of 5.5 d/kg (range: 1.9 to 13.1 d/kg) for unspeciated PCDD/Fs (Petreas et al., 1991; Stephens et al., 1995; Malisch et al., 1996; Schuler et al., 1997a). In chicken muscle, three contaminated feed or soil studies provided accumulation data from which an average Tco of 4.6 d/kg (range: 1.0 to 7.6 d/kg) was calculated (Stephens et al., 1995; Iben et al., 2003; Pirard and De Pauw, 2005).

For the controlled laboratory feed-to-egg studies in which PCDD/Fs in fly ash or oil were added to feed (i.e., no contaminated soil was added to the diet), egg Tcos ranged from 8.5 to 17 d/kg with a mean of 12 d/kg (Pirard and De Pauw, 2005; 2006; Van Eijkeren et al., 2006).

For field studies, calculated egg Tcos of free-foraging chickens in various regions with PCDD/F-contaminated soil showed greater variation and was higher (Schuler et al., 1997a; Harnly et al., 2000; Hoogenboom et al., 2006). The Tcos ranged from 12 to 37 d/kg with an average of 23 d/kg. An assumption was made that the PCDD/F source for the free-foraging hens was contaminated soil, and that the soil ingestion rate was 10 g soil/day. There is general support among researchers for this soil ingestion rate by free-foraging chickens (De Vries et al., 2006). The larger egg Tco in field studies compared to controlled laboratory studies may be a result of free-foraging chickens consuming soil organisms and herbs and grass which may also be contaminated. However, greater bioavailability of soil PCDD/Fs in the field, or a higher soil ingestion rate than predicted may also play a role in a larger egg Tco under field conditions.

Overall, the range of mean values for these three types of studies is not large (within a factor of 10), considering the different sources of PCDD/Fs that the poultry were exposed to. A grand mean from the three types of exposure studies (contaminated soil field study, controlled contaminated soil study and contaminated feed study) is 13 d/kg $(3.6 + 23 + 12 \text{ d/kg} / 3)$, which we recommend as the default egg Tco for PCDD/Fs.

For edible muscle tissue (usually thigh or breast tissue), TEQ-adjusted Tcos could be calculated from several studies that investigated PCDD/F concentrations in chickens given contaminated feed. In a controlled laboratory study in which 10% of the diet was PCDD/F-contaminated soil, a Tco of 7.4 d/kg was calculated (Stephens et al., 1995). In three contaminated feed studies where PCDD/Fs in oil or fly ash were added to diet, similar Tcos of 8.6, 9.0 and 4.1 d/kg were calculated (Iben et al., 2003; Pirard and De Pauw, 2005; 2006).

Congener-specific data for development of beef Tcos were not as comprehensive as that for development of cow's milk Tcos. Two long-term pentachlorophenol (PCP) feeding studies in dairy cattle determined body fat concentrations for several PCDD/F congeners (1, 2, 3, 6, 7, 8- and 1, 2, 3, 7, 8, 9-HxCDD, 1, 2, 3, 4, 6, 7, 8-HpCDD, OCDD, 1, 2, 3, 4, 6, 7, 8-HpCDF, and OCDF) that were contaminants in the PCP formulation (Firestone et al., 1979; Parker et al., 1980). Beef Tcos based on dairy cattle for the other congeners and unspciated PCDD/Fs were estimated with the assumption that the fat concentration is similar in milk and beef, and were adjusted upward to account for the greater fat content in muscle tissue (19%) compared to the fat content in milk (4%). As noted above, the concentration of PCBs in milk fat and body fat have been shown to be similar in exposure studies (Thomas et al., 1999b). We then applied scaling factors by MacLachlan (2009) to estimate the transfer of PCDD/Fs from body fat of lactating cattle to body fat of beef cattle. Data by Huwe and Smith (2005) found that half-lives were mostly 30-50 days for the PCDD/Fs; the major exceptions were OCDF ($t_{1/2} = 14$ days) and OCDD ($t_{1/2} = 72.6$ days). A ratio of 7 is estimated for the simulated PCDD/F levels in body fat of steers to body fat of lactating dairy cows for most PCDD/Fs. A ratio of 4 was estimated for OCDF and a ratio of 10 was estimated for OCDD.

Pig Tcos for individual and unspciated PCDD/Fs in Table K-1 were estimated from a comprehensive study in which PCDD/Fs were added to the diet in feed of pigs during the 12-week fattening period (Spitaler et al., 2005). This exposure period represents the last 12-weeks prior to slaughter in the typical 6-month life of a pig. Notably, the researchers did not observe a reduction of residues due to roasting of the meat.

K.2.2 Tcos for Inorganic Metals and Chemicals

The studies used to derive inorganic metal Tcos listed in Table K-2 usually presented data as fresh weight concentrations in muscle, milk and eggs. Occasionally, dry weight concentrations were reported. Unless the study noted the water content of the food source, default factors of 0.87 for cow's milk, 0.35 for chicken egg, 0.25 for chicken meat, and 0.30 for beef and pork were used for adjusting to fresh weight concentration (USDA, 1975).

Biotransfer studies for pig muscle could not be found for most of the metals. As noted in the beginning of this appendix, biotransfer data in cattle were more abundant. Where specific metal biotransfer data were missing in pigs but present in cattle, the pig meat Tco was estimated using a simple metabolic weight adjustment from cattle to pig as shown in Eq. K-1.

In general, low concentrations of inorganic metals are transferred from contaminated feed to muscle tissue, cow's milk and eggs and are not as great a concern relative to other potential sources of heavy metals in multipathway exposures. However, many of the inorganic metals such as cadmium, lead and mercury tend to accumulate over time in organs, particularly kidney and liver. Thus, frequent consumption of organs from exposed food animals may present a much greater toxic hazard to humans than consumption of the meat. Cadmium is of particular concern due to its relatively high toxicity and high potential for accumulation in the kidney and liver. Kidney and liver-specific Tcos for cadmium and a few other metals are presented in the text below for some of these food-producing animals only for comparison purposes. Tcos for accumulation in bone for some of the metals (i.e., lead) are also noted or calculated for some of the food products.

Another toxicological concern is that chickens can convert some of the ingested inorganic mercury in controlled feeding studies to methyl mercury, which is then found primarily in the poultry meat and egg white (Kiwimae et al., 1969). The inorganic mercury Tcos for poultry meat and eggs in Table K-2 represents total mercury, although some will be present as organic methyl mercury. Because methyl mercury is not emitted from facilities (i.e., only inorganic or elemental mercury is emitted), it is not accounted for in health risk assessments. However, Tcos for methyl mercury were calculated by OEHA and presented in Section K.2.2.7 only for comparison to the inorganic mercury Tcos.

K.2.2.1 Arsenic

Only one study could be located that recorded a measurable increase of arsenic in cow's milk following dairy cattle consumption of contaminated feed. We calculated a Tco of 5×10^{-5} d/kg from data in dairy cattle exposed to As(III) as arsenic trioxide for 15-28 months (Vreman et al., 1986).

In poultry, organic arsenic compounds are an approved dietary supplement that can result in increased levels of total arsenic in meat and eggs (Lasky et al., 2004). Both organic and inorganic forms of arsenic are found in poultry, with inorganic forms more toxic than organic forms. Analysis of poultry and meat samples indicates that about 65% of total arsenic is in the inorganic form.

We calculated a Tco of 0.07 d/kg for total arsenic in eggs from hens fed a diet containing arsenic trioxide (Holcman and Stibilj, 1997). In muscle, total arsenic Tcos of 0.06 and 0.02 d/kg were determined in chickens from two studies following addition of arsenic trioxide to feed (Overby and Frost, 1962; Vadnjal et al., 1997). The proportion of arsenic in the inorganic form was not determined. In drinking water, soluble As(V) was added to the water resulting in a total arsenic Tco of 0.2 d/kg in muscle of broiler chickens (Pizarro et al., 2004). However, only 10% of arsenic in muscle was in the inorganic form. Over 50% was present as dimethylarsinic acid, which is considered a methylation detoxification pathway for arsenic. Thus, the inorganic arsenic Tco was 0.02 d/kg. We calculated an average muscle Tco of 0.03 d/kg from the three studies for transfer of arsenic from diet to chicken meat.

In beef cattle, Vreman et al. (1988) administered arsenic trioxide in the feed for 143 days to 16 bulls at about 12.5 mg/d resulting in a muscle Tco of 2.4×10^{-3} d/kg. The same Tco was calculated from data by Ham et al. (1949) that dosed adult steers daily with 270 mg arsenic trioxide for 201 days. In another study in steers, Bruce et al. (2003) estimated the daily intake of arsenic from grazing pasture grass, ingesting dust adhering to pasture, and direct ingestion of soil in an area contaminated with arsenic-laced mine tailings. Based on the daily intake and muscle concentration of arsenic at sacrifice after 237 days of exposure, a Tco of 2.8×10^{-4} d/kg was derived. We calculated an average muscle Tco of 1.7×10^{-3} d/kg from these three studies, which we recommend as the default value for beef cattle. Long-term arsenic feeding studies have also been conducted in lactating dairy cows. A slightly lower muscle Tco of 7.1×10^{-4} d/kg was calculated from these studies (Peoples, 1964; Vreman et al., 1986).

Arsenic exposure in beef and dairy cattle has not shown tissue-specific sequestering in liver or kidney, unlike some of the inorganic metals (e.g., cadmium, lead, and mercury). Similar Tcos were estimated for muscle, liver and kidney (Ham et al., 1949; Peoples, 1964; Vreman et al., 1988).

K.2.2.2 Beryllium

No inorganic beryllium accumulation studies could be found in the literature for poultry. Thus, we calculated poultry egg and meat Tcos for beryllium based on the average Tco value of the other "Hot Spots" divalent, cationic metals in Table K-2 (i.e., cadmium, lead, inorganic mercury, and nickel) providing beryllium Tcos for egg and muscle of 0.09 and 0.2 d/kg, respectively.

No multiple day inorganic beryllium exposure studies have been conducted in cattle or swine. In a single bolus study, Ng (1982) estimated a cow's milk Tco of 9.1×10^{-7} d/kg based on recovery of radiolabeled beryllium chloride given to dairy cattle. For beef, we determined a beryllium Tco of 3×10^{-4} d/kg based on the average Tco value of the divalent, cationic metals cadmium, lead, and inorganic mercury. Beef Tcos for these three metals were determined directly from published studies. A default pork Tco was determined by us by the same method as that used for beef, resulting in a pig meat Tco of 1×10^{-3} d/kg.

K.2.2.3 Cadmium

Very low accumulation of cadmium occurs in cow's milk, and concentrations of cadmium in cow's milk are often below the detection limit. In his review, Stevens (1991) estimated an average Tco of 1.3×10^{-6} d/kg in cow's milk from two long-term cadmium exposure studies by Vreman et al. (1986). More recently, we estimated a milk Tco of 1.3×10^{-5} d/kg from exposure data in a single cow exposed to cadmium for 77 days (Mehennaoui et al., 1999). The average Tco from the three exposure studies is 5×10^{-6} d/kg, which we recommend as a default Tco.

Numerous cadmium accumulation studies have been conducted in poultry. Similar to cow's milk, very low accumulation of cadmium occurred in hen's eggs with exposure in

feed; the levels of cadmium in eggs are sometimes below the detection limit. We calculated an average egg Tco of 0.01 d/kg from the best available data (Leach et al., 1979; Sharma et al., 1979; Hinesly et al., 1985). In muscle, we determined cadmium Tcos in exposed chickens ranging from 0.2 to 1 d/kg (Leach et al., 1979; Sharma et al., 1979; Hinesly et al., 1985; Pribilincova et al., 1995; Bokori et al., 1996). The average value from these studies was 0.5 d/kg, which we recommend as the Tco.

Similar cadmium Tcos in muscle of dairy and beef cattle have been observed in long-term feeding studies lasting 3.5 to 28 months. We calculated an average Tco of 2.0×10^{-4} d/kg with a range of $1.2 - 3.2 \times 10^{-4}$ d/kg (Johnson et al., 1981; Vreman et al., 1986; 1988). A muscle Tco of 6.5×10^{-5} d/kg was obtained from a feeding study by Lamphere et al. (1984) describing cadmium body burden in calves exposed for 60 days. However, the short exposure duration only during growth of the animal may result in an underestimation of the Tco compared to exposure to adulthood.

Cadmium accumulates to a much greater extent in some organs compared to muscle tissue. In poultry, exposure studies suggest that cadmium accumulation in the kidney and liver increases with increasing exposure duration and may not attain a steady-state concentration. Eighty-week exposure to cadmium in chickens resulted in a Tco of 800 d/kg in the kidney and 70 d/kg in the liver (Hinesly et al., 1985). In dairy and beef cattle, cadmium Tcos for liver and kidney did not vary greatly even though exposure durations varied. Average calculated Tcos were about 0.03 d/kg (range: 0.01 to 0.048 d/kg) for liver, and 0.1 d/kg (range: 0.09 to 0.19 d/kg) for kidney (Sharma et al., 1979; Sharma et al., 1982; Vreman et al., 1986; 1988).

Only one study could be found that measured cadmium muscle levels in pigs following exposure to cadmium in feed. Cousins et al. (1973) only found measurable cadmium levels in skeletal muscle at the highest of four doses tested (1350 ppm) following a six-week exposure, but this level caused severe toxicity. More accurate estimates of muscle uptake were found in heart tissue, which exhibited increased tissue concentration with increasing dose and may represent the upper end of the cadmium concentration found in skeletal muscle. The average Tco we calculated in heart muscle was 0.0051 d/kg. In the liver and kidneys of pigs, cadmium Tcos as high as 0.48 and 2.53 d/kg, respectively, were calculated from a study by Sharma et al. (1979).

K.2.2.4 Chromium (Hexavalent)

Only a portion of ingested hexavalent chromium (Cr(VI)), perhaps 1-2%, is expected to be systemically absorbed in the hexavalent form due to rapid reduction to the less soluble and less toxic trivalent chromium in the acidic environment of the stomach (Costa, 1997; NTP, 2008). Trivalent chromium (Cr(III)) is an essential micronutrient, but no cancer potency or noncancer reference exposure level is currently available for this form of chromium. Cr(VI) that is absorbed can then be actively transported into all cells and tissues of the body in place of anions, such as phosphates. Once inside the cell, the Cr(VI) is reduced to various unstable reactive intermediates and, finally, stable Cr(III) is ultimately formed inside the cell.

Current analytical procedures cannot differentiate between the oxidation states of chromium in biological tissues (NTP, 2008). However, it has been advocated that any Cr(VI) transported into meat and eggs would be converted to the more stable Cr(III) form and would presumably not pose a risk for human consumption (Chundawat and Sood, 2005). Based on these findings no Cr(VI) Tco is currently recommended by OEHHA for meat and eggs.

However, a similar situation may not be the case for cow's milk. Lameiras et al. (1998) found Cr(VI) in cow's milk, which was 25-50% of total chromium. In whole milk, the average total chromium concentration was 2.70 ug/L (range: 1.42-5.70 ug/L) and the average Cr(VI) concentration was 0.68 ug/L (range: 0.20-1.20 ug/L). No multiple day Cr(VI) exposure studies in dairy cattle could be found in the literature. Following a single oral dose of radiolabeled sodium chromate (Na_2CrO_4), Van Bruwaene et al. (1984) calculated a steady-state cow's milk Tco of 1.0×10^{-5} d/kg for total chromium. Stevens (1991) estimated a similar Tco of 1.4×10^{-5} d/kg from the same data based on a half-life of 26 days for total chromium in cow's milk. These studies did not attempt to estimate the proportion of total chromium that was secreted as Cr(VI) into milk.

Multiplying the Stevens total chromium Tco by the fraction of total chromium that is Cr(VI) in normal milk (1.4×10^{-5} d/kg \times 0.68/2.70 ug/L) provided a modified Tco of 3.5×10^{-6} d/kg. Until valence-speciated cow's milk data are available from Cr(VI) exposure studies, we chose a midpoint Tco value between the Stevens Tco and this modified Tco adjusted for Cr(VI) content in normal milk (8.75×10^{-6} d/kg) as a health-protective cow's milk default value for Cr(VI).

K.2.2.5 Fluoride

In a series of long-term exposure studies on fluorides' effect on milk production, the fluoride concentration in the milk of dairy cows given fluoride in feed resulted in an estimated cow's milk Tco of 0.0003 d/kg (Stoddard et al., 1963; Harris et al., 1964).

Fluoride in the diet of hens resulted in very low accumulation of fluoride in muscle, and yolk and albumin of eggs (Hahn and Guenter, 1986). We calculated a Tco in whole eggs of 0.008 d/kg from the exposure data. Considerably greater accumulation occurs in egg shell. Muscle accumulation in the fluoride-exposed hens resulted in a Tco of 0.03 d/kg.

Specific data concerning accumulation of fluoride in the skeletal muscle tissue of exposed cattle could not be found. However, in cases of high fluoride intake, fluoride levels in the soft tissue (i.e., brain, liver, kidney, pancreas, intestines, etc.) are reported to increase only two or three times the normal value in meat producing animals. Fluoride does not accumulate in the edible portions of the animal (Suttie et al., 1958; Shupe et al., 1964). However, considerably greater accumulation of fluoride occurred in bone. In heart tissue, we calculated a fluoride Tco of 8.4×10^{-4} d/kg for Holstein cows fed fluoride-contaminated rations for 5.5 years, which we recommend as the default muscle Tco for range cattle (Suttie et al., 1958). It is assumed that similar

pharmacokinetic properties, and similar Tcos, occur for fluoride in both skeletal and heart muscle tissue.

K.2.2.6 Lead

Only three contaminated feed studies observed measurable levels of lead in milk from both control and exposed dairy cows. Based on data from a 15-28 month lead exposure study of dairy cows kept indoors, a cow's milk Tco of 2.6×10^{-5} d/kg was calculated (Vreman et al., 1986). A three-month outdoor lead exposure study by the same researchers produced a Tco of 5.4×10^{-5} d/kg. Stating that the half-life of lead in dairy cows is about 45 days, Stevens (1991) adjusted the Tco of the three-month outdoor study to 7.1×10^{-5} d/kg. However, Willett et al. (1994) observed that steady-state was attained in cow's milk after only 14 days of a 49-day lead exposure study, generating a Tco of 7.9×10^{-5} d/kg. Using the steady-state-corrected Tco by Stevens (1991) for the outdoor Vreman study, we recommend an average Tco of 5.9×10^{-5} d/kg from these three studies.

An average Tco of 0.4 d/kg in muscle was calculated by OEHHA for lead in broiler chicks fed contaminated feed for 20 days (Stoddard et al., 1963; Harris et al., 1964; Latta and Donaldson, 1986a; 1986b). For comparison, a roughly 10-fold higher Tco was calculated for lead in kidney. However, lead tends to accumulate most in bone, generating a Tco of 70 d/kg. Lead in bone is not expected to be a problem, unless contaminated bone is ground into bone meal and fed to animals. Accumulation of lead in eggs was very low, generating a Tco of 0.04 d/kg (Meluzzi et al., 1996).

Vreman et al. (1988) administered lead acetate in feed to young bulls for 143 days during the fattening period. The resulting muscle Tco was 2.7×10^{-4} d/kg. A slightly lower muscle Tco of 6.7×10^{-5} d/kg in lactating dairy cows fed lead mixed with their feed (Vreman et al., 1986).

Roughly 10- to 100-fold greater accumulation of lead occurs in the kidney and liver of cattle compared to their muscle tissue. We calculated Tcos of 4.8×10^{-3} and 1.4×10^{-2} d/kg for liver and kidney, respectively, in the bulls from the Vreman et al. (1988) study. In addition to liver and kidney, lead was also found to accumulate in bone. In a three-month feeding study in dairy cattle, a bone Tco of 0.02 d/kg was calculated from the data by Sharma et al. (1982). In one of the few biotransfer studies conducted in pigs, a liver Tco of 1.4×10^{-2} d/kg was recorded in pigs fed diets containing either 5 or 25 ppm lead acetate for 90 days (Sharma and Street, 1980).

K.2.2.7 Inorganic Mercury

Addition of inorganic mercury (Hg(II)) to the feed of hens for 140 days resulted in a muscle tissue Tco of 0.1 d/kg (Kiwimae et al., 1969). However, some Hg(II) was converted to methyl mercury (MeHg) in the chickens, resulting in a muscle Tco of 0.09 d/kg for MeHg. When only MeHg is added to the diet in prolonged feeding studies, an average Tco of 10 d/kg was calculated with virtually all the mercury in the muscle as MeHg (Kiwimae et al., 1969; Soares et al., 1973; Hilmy et al., 1978). Some Hg(II) added

to feed is also endogenously methylated in the hens and transported to the eggs. Addition of Hg(II) to the feed of hens for 140 days resulted in a calculated egg Tco of 0.8 d/kg for total mercury, and 0.5 d/kg for MeHg (Kiwimae et al., 1969). An average egg Tco of 11 d/kg was calculated when only MeHg was added to feed (Scott et al., 1975; Hilmy et al., 1978).

Vreman et al. (1986) observed a small, but statistically insignificant increase in mercury in cow's milk with exposure of dairy cattle to inorganic mercury in feed for 15-28 months. The Tco range was 7 to 40 x 10⁻⁵ d/kg with an average of 2 x 10⁻⁴ d/kg. Stevens (1991) calculated Tcos of 9.2 x 10⁻⁶ and 1.3 x 10⁻⁵ d/kg from oral single bolus studies of radiolabeled inorganic mercury by Mullen et al. (1975) and Potter et al. (1972). The steady-state Tcos were calculated by use of study-specific half-lives of 1.2 (Potter et al., 1972) or 5.5 days (Mullen et al., 1975) for mercury. We calculated an average Tco of 7 x 10⁻⁵ d/kg from the three studies, which we recommend for transfer of inorganic mercury to cow's milk.

Similar to cow's milk, only a small, but statistically insignificant increase in inorganic mercury could be measured in muscle tissue following long-term exposure of dairy and beef cattle to soluble mercury (Vreman et al., 1986; 1988). Calculated maximum muscle Tco values from these two studies were 6.7-18 x 10⁻⁴ d/kg, but we lack confidence in this value due to the detection limit of these studies. To calculate the biotransfer of ingested mercury to muscle, Stevens (1992) relied on three oral bolus dose studies that determined the half-life of inorganic mercury in blood of dairy cattle (Potter et al., 1972; Ansari et al., 1973; Mullen et al., 1975). Operating on a reasonable assumption that muscle is a well-perfused tissue and shares the same kinetic compartment as blood, Stevens calculated an average muscle Tco of 3.5 x 10⁻⁴ d/kg (range: 1.8-4.4 x 10⁻⁴ d/kg). This value is comparable with the Tcos estimated from the Vreman studies, which we recommend as the point estimate Tco for inorganic mercury in beef.

Although it is not anticipated that human exposure to methyl mercury via cow's milk and beef would be a significant pathway (e.g., as compared to fish), biotransfer information is included here for completeness. There are few published data that investigated ruminant methylmercury uptake and accumulation. However, background exposure and accumulation of inorganic and methylmercury in meat products are reported to be very low (U.S. EPA, 1997). In their risk assessment guidelines, U.S. EPA (2005) suggests that only 13% of total mercury in ruminants is present as methylmercury, an indication that ruminants have little exposure to methylmercury.

In vitro, cow rumen microflora does not methylate added inorganic mercury (as HgCl₂) to methylmercury (Kozak and Forsberg, 1979). On the other hand, rumen microflora was found to demethylate up to 40% of added methylmercury to elemental (Hg⁰), or metallic, mercury, which would then be presumably excreted with little or no absorption. This finding suggests that ruminants can detoxify some of the ingested methylmercury.

Stevens (1991) estimated that the Tco for methylmercury in cow's milk is roughly one order of magnitude greater than that for inorganic mercury (i.e., 7 x 10⁻⁴ d/kg). His

finding was based on a study by Neathery et al. (1974), in which two dairy cows were given a bolus dose of radiolabeled methylmercuric chloride and followed for the appearance of label in milk for 14 days. A milk excretion half-life of 6 days was calculated from the data. It was suspected that the lipophilic nature of methyl mercury resulted in its accumulation in milk fat. Of the labeled methylmercury that was absorbed, 72% of the total body burden was found in muscle tissue 15 days after the single bolus dose. However, there are insufficient data to estimate the biotransfer of ingested methylmercury in cattle and pigs with chronic exposure.

K.2.2.8 Nickel

Only two studies were found in the literature that attempted to estimate the nickel concentration in cow's milk following 1.5 to 2 month exposure of the dairy cattle to inorganic nickel-contaminated feed (Archibald, 1949; O'Dell et al., 1970). Neither study used analysis methods that were sensitive enough to record measurable increases of nickel in the cow's milk. Stevens (1991) used the maximum value approach by dividing the detection limit (0.1 ppm) of the studies by two, arriving at an average cow's milk Tco of 2.7×10^{-5} d/kg. Until more sensitive studies are conducted, we recommend this Tco as the default value for inorganic nickel.

Limited data for nickel indicate low accumulation of this metal occurs in eggs and tissues of chickens (Ling and Leach, 1979; Meluzzi et al., 1996). We calculated Tcos of 0.02 d/kg for both eggs and muscle tissue of hens fed inorganic nickel mixed in their diet. As with other inorganic metals, greatest nickel accumulation occurred in the kidney (Tco = 0.68 d/kg), resulting in a Tco over 30-fold higher than that found in muscle or eggs.

No adequate studies investigating biotransfer of ingested inorganic nickel to beef or pork could be located. As with the approach used for beryllium, we determined a beef Tco based on an average of the three divalent cationic metal Tcos (i.e., cadmium, lead and inorganic mercury) that had sufficient biotransfer data available in the literature. The resulting beef Tco was 3×10^{-4} d/kg. We then developed a pig meat Tco of 0.001 d/kg based on the cow-to-pig metabolic weight ratio adjustment (Eq. K-1). OEHHA recognizes that these Tcos developed for beef and pork are more uncertain than would be desirable. However, the data available in other food-producing animals and similar Tcos developed for other cationic metal contaminants indicates the nickel muscle Tco is likely not underestimated in cattle and pigs.

K.2.2.9 Selenium

The selenium concentration in milk tends to increase as intake of selenium increases from about 2 to 6 mg/day (Fisher et al., 1980; Maus et al., 1980; Beale et al., 1990). Secretion of selenium into milk then appears to reach a temporary limit when selenium intake is about 6 to 12 mg/day. The mammary gland is either limited in the limited amount of selenium it can secrete into milk, or, more likely, the net absorption of selenium from the gut is controlled in the face of increased selenium intake. Only when selenium intake increases above 50-100 mg/day does the ability of the protection

mechanism become exceeded, resulting in selenium toxicity and increased selenium concentration in milk. We calculated a Tco of 0.009 d/kg based on the average value for studies that supplemented feed with 6 mg/d selenium or less.

Optimum levels of selenium in the diet of poultry are about 0.1 to 0.2 ppm (Arnold et al., 1973; Moksnes and Norheim, 1982). Concentrations of selenium above 3 ppm may result in toxicity. At concentrations of 1 to 9 ppm selenite in the feed, we calculated an average egg Tco of 3 d/kg (Arnold et al., 1973; Ort and Latshaw, 1978; Moksnes and Norheim, 1982; Davis and Fear, 1996). In broiler chicks, an average Tco of 0.9 d/kg for muscle was calculated (Moksnes and Norheim, 1982; Echevarria et al., 1988a; 1988b). Laying hens had a lower Tco of 0.4 d/kg for muscle tissue, possibly due to eggs acting as an elimination pathway for selenium (Arnold et al., 1973; Ort and Latshaw, 1978; Moksnes and Norheim, 1982). Thus, the muscle Tco for selenium is based on the findings in meat (broiler) chickens.

In beef cattle, groups of calves were fed sodium selenite in a milk replacer at concentrations of 0.2 to 5 ppm for six weeks (Jenkins and Hidioglou, 1986). We calculated an average muscle Tco of 6.6×10^{-2} d/kg from the exposure data. In another study, inorganic selenium was intraruminally administered in beef cows through two soluble-glass boluses to slowly release Se over approximately 11 months (Hidioglou et al., 1987). We calculated a Tco of 7.1×10^{-3} d/kg in the muscle tissue. The average muscle Tco from the two studies is 0.037 d/kg, which we recommend as the default selenium transfer factor. Jenkins and Hidioglou (1986) also observed greater accumulation of selenium in the liver and kidney cattle compared to muscle, resulting in calculated Tcos of 2.7 and 0.25 d/kg, respectively.

In pigs, selenium muscle concentrations have been measured following unsupplemented intake or supplementation of selenium in diets. No studies could be located that estimated tissue levels of selenium following prolonged intake of toxic or near-toxic levels of selenium. Using a study by Ku et al. (1972), we calculated an average muscle Tco of 0.61 d/kg in groups of adult pigs that had been fed diets containing selenium at levels ranging from 0.027 to 0.493 ppm. A positive correlation between selenium level in the diet and muscle concentration was observed. Using another study, which exposed pigs to diets containing 0.78-0.88 ppm selenium during the growth phase, we calculated a muscle Tco of 0.35 d/kg in pigs at market weight (Jenkins and Winter, 1973).

Similar to the phenomena observed in dairy cattle, supplementation of pig diets with selenium (0.1 to 1.0 ppm) did not always result in an increase in tissue selenium levels. Tcos based on these studies are as much as 10-fold lower compared to Tcos calculated from baseline levels of selenium found in feed (Groce et al., 1971). However, it is not known if this protective mechanism also operates at higher selenium levels in feed that may produce toxic effects in pigs. Thus, we recommend a default pig Tco based on the average Tco (0.48 d/kg) determined using Ku et al. (1972) and Jenkins and Winter (1973), which covered a range of baseline selenium intakes in feed from 0.027 to 0.88 ppm.

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Technical Support Document for Exposure Assessment and Stochastic Analysis,
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