deaths (13 percent) occurred at 2.5 mg/ kg/day. The developmental toxicity study results are supported by the 1 year dog study in which significant plasma and RBC ChE inhibition occurred as early as 2 weeks following administration of 1.0 and 3.0 mg/kg/ day. In addition, plasma ChE inhibition ranged from 21 to 26 percent in the 0.1 mg/kg/day group at 2 weeks. These studies indicate that effects associated with ChE inhibition occur at levels slightly higher than 0.1 mg/kg/day. Therefore, EPA has determined that the study results support a NOEL of 0.1 mg/ kg/day for calculating margins of exposure for intermediate exposure.

(c) Chronic/long-term exposure. The oral and inhalation toxicity studies that EPA has evaluated resulted in comparable NOELs for assessing chronic dietary and long-term occupational and/ or residential exposure (substantial portion of a lifetime). The inhalation study in rats demonstrated a NOEL of 0.055 mg/kg/day (converted from 0.05 mg/m³) based on statistically significant ChE inhibition in plasma, RBC, and brain at 0.48 mg/m³. The oral study in dogs resulted in a NOEL of 0.05 mg/kg/ day, based on statistically significant plasma, RBC, and brain ChE inhibition at 1.0 mg/kg/day. EPA rounded the inhalation NOEL to 0.05 mg/kg/day for ease in calculating MOEs. In addition, there is uncertainty associated with converting from mg/m3 to mg/kg/day in the chronic inhalation study.

3. Adverse liver effects. The PD 1 also cited a concern for adverse liver effects resulting from exposure to dichlorvos. A 2-year dog feeding study indicated increased liver weight and enlargement of liver cells with a NOEL of 0.08 mg/kg/day. EPA recently reevaluated this study and downgraded its acceptability from minimum to invalid. The study was reclassified because the actual dose ingested by the animals cannot be confirmed, due to impurities and decomposition products in the test material.

In addition, the 1 year oral dog study cited above was reviewed for the purpose of evaluating the validity of the liver effect concern. No liver effects were reported after 1 year of treatment at higher doses than the doses in the invalidated 2–year study. Therefore, this endpoint is no longer of regulatory concern.

C. Exposure Analysis

1. Dietary exposure—i. Background. Dietary exposure to a pesticide depends on two components: the amount of pesticide residue on a commodity and how much of that commodity is consumed. In estimating dichlorvos

residues on food, EPA relied on a variety of data for dichlorvos, including tolerance levels (the legal maximum residue) and field trial data (measured residues resulting from actual application of dichlorvos). In addition, these estimated residues can be further refined by taking into account the effects of processing and cooking on treated foods, and by estimating the percent of the crop that is treated.

The Agency currently uses food consumption values derived from a USDA survey to estimate dietary exposure to pesticides. The USDA conducted a nationwide survey (1977-1978) of the food consumption patterns of 30,770 individuals for 3 days. Based on this survey, EPA can estimate the dietary exposure and risk for the U.S. population and 22 subgroups of the total population using a computer-based tool called the Dietary Risk Evaluation System (DRES). DRES multiplies the average daily consumption values by residue information for each commodity to obtain the total dietary exposure. In the absence of data for residues of dichlorvos on crops and an estimate of the percent of the crop treated with a pesticide, EPA estimates exposure based on the Theoretical Maximum Residue Contribution (TMRC). The TMRC assumes residues on crops are present at tolerance levels (the maximum residue limit allowed by law) and 100 percent of the crop is treated. When EPA has additional data to refine the TMRC, based on residue data and estimates of percent of crop treated, the Agency uses this new information to calculate the Anticipated Residue Contribution (ARC). When available, the ARC is used instead of the TMRC in estimating

Dietary exposure to dichlorvos residues may occur as a result of use on a variety of sites. These sites include greenhouse food crops, food or feed containers, bulk-stored, bagged or packaged nonperishable raw agricultural commodities (RACs) food, and bulk stored, bagged or packaged nonperishable processed commodities, commercial food processing plants, groceries, eating establishments, livestock (direct animal treatment), swine feed (as a dewormer), and food in homes where resin pest strips are

Tolerances and FARs exist for residues of dichlorvos in or on raw agricultural and processed products and on meat, milk, poultry and eggs. As noted in the Registration Standard, even though dichlorvos is registered for use in food handling establishments (including food processing, food manufacturing and eating

establishments), there are no FARs for the related uses.

In estimating dietary exposure for the initiation of Special Review in 1988, the Agency did not have sufficient data on actual residue levels. Therefore, EPA's dietary exposure estimate at that time was based on the assumption that residues were present at tolerance levels (40 CFR 180.235). Residues were adjusted based on cooking data on small grains and on an estimate of percent of crop treated. At the time of the initiation of Special Review, EPA estimated that the average consumer in the U.S. population was exposed to 4.2 x 10⁻² mg/kg/day of dichlorvos. This may have been an overestimate of chronic exposure because tolerance level residues were assumed. However. limited data available at that time suggested that some residues were at or above tolerance levels (nonperishable stored foods). In addition, exposure could have been underestimated because, in the absence of a FAR for food handling uses, the exposure estimate did not consider residues from food handling uses, or any degradation resulting from two related pesticides, naled and trichlorfon.

Amvac recently notified the Agency (Ref. 40) that it is not supporting the reregistration of greenhouse food and nonfood uses and that it requests voluntary deletion of those uses. Therefore, some exposure may be eliminated as a result of these voluntary deletions, or due to cancellation of uses related to the revocation of the FAR for packaged or bagged nonperishable processed food. However, since these actions have not occurred, EPA will continue to consider these residues for this proposed determination.

ii. Naled and trichlorfon. Naled and trichlorfon degrade to dichlorvos through plant metabolism. Three factors will significantly affect dietary exposure to dichlorvos from registered uses of naled and trichlorfon; these include, the preharvest interval (PHI), the condition and length of storage, and cooking and processing. Naled is metabolized to dichlorvos by plants. Plant metabolism studies show that dichlorvos residues are formed 1 to 3 days after treatment with naled and trichlorfon: however. dichlorvos residues are less than the limit of detection (0.01 to 0.05 ppm) 7 days after treatment. In general, registered uses of naled have PHIs of less than 7 days, while trichlorfon registrations have PHIs greater than 7 days. Because of the short PHIs for naled products, measurable residues of dichlorvos may be present in the U.S. diet from naled treated food. EPA does not expect measurable residues from