

exposures to EGBE of actual persons living in the immediate vicinity of EGBE emission sources would be significantly less than the concentrations estimated by the model. Considering such things as human activity patterns and that predicted ambient concentrations fall significantly from those predicted by the models, we expect that the HQ for most of the surrounding population would be several orders of magnitude less than one.

We also use a Hazard Index (HI) approach to characterize the potential for EGBE exposures to cause adverse effects when combined with typical exposures to pollutants that also affect the circulatory system. In this case, we rely on the 1996 National Air Toxics Assessment (NATA) which estimates risks to certain HAP by census blocks. The NATA results indicate that more than 99 percent of the census blocks have circulatory system HI below 0.1. As such, even when combined with other exposures to circulatory system toxicants, EGBE exposures would result in HI that are well below 1.0 and, therefore, would not be associated with risk of adverse effects.

The reference level we used to determine EGBE's ingestion HQ is the IRIS RfD of 0.5 (mg/kg/day). Based on our analysis, we judge that maximum exposures to EGBE via ingestion of water contaminated with EGBE from air releases is not likely to exceed 0.28 mg/kg/day. The resulting HQ is 0.6. In other words the concentration in the environment is 60 percent of the RfD. Given the conservative nature of the parameters used to derive the average daily intake, we conclude that the actual HQ will be significantly less than 0.6.

Therefore, based on information presented in the petition, EPA's evaluation of data made available after the submission of the petition, and our own supplemental analyses, we have made an initial determination that emissions, ambient concentrations, bioaccumulation or deposition of EGBE may not reasonably be anticipated to cause any adverse effects to human health.

E. Ecological Risk Characterization and Conclusions

We developed an independent ecological risk assessment (ERA) to evaluate the potential environmental impacts of EGBE emissions. We organized our analysis according to EPA's framework for ecological risk assessment and followed a two tiered approach. Under this approach, the tier 1 analysis used conservative point estimates of exposure (maximum possible concentration in the

environment) and effect (e.g., national ambient water quality criterion). If the tier 1 analysis indicated that a conservative estimate of exposure would not exceed a very sensitive effects threshold (i.e., quotient <1), the analysis was terminated. If the tier 1 analysis indicated the potential for effect (i.e., quotient >1), the analysis proceeded to tier 2. In tier 2, more realistic assumptions were made about exposure and effects. If the tier 2 quotients were less than one, the analysis was terminated. However, if one or more of the tier 2 quotients were greater than one, the risk assessment would proceed to a probabilistic risk assessment.

Because EGBE concentrations will be the highest close to the emission source and because it is unlikely to be transported widely due to its short half-life in air and its propensity to partition from air to soil and water, we decided that the appropriate spatial modeling scale for the analysis was local. Using the petitioner's dispersion modeling analysis, we selected the single facility from the inventory that was the source of the largest maximum predicted annual concentration of EGBE as predicted by the ISCST3 model. This maximum annual average concentration was then used in conjunction with a Mackay Level I fugacity model to determine a steady state equilibrium concentration of EGBE in soil, water, and sediment in a simulated environment situated at the fence line. (Due to the relatively short distance from the source to the fence line, we assumed EGBE to disperse in the atmosphere as a passive tracer, not subject to removal through deposition or chemical reaction during transport.)

We developed exposure scenarios for small mammals and aquatic species and derived a quotient to characterize the potential ecological risk. The tier 1 ERA suggested that EGBE may have the potential to cause adverse effects to small mammals and to sensitive aquatic biota residing close to and downwind of the largest emitting source. This determination was, at least in part, due to the conservatism of tier 1 analysis, and the fact the decision criterion for these quotients were derived from very minor effects which were unlikely to be ecologically significant at the population level of ecological organization.

The tier 2 analysis combined a Level III Mackay Model and the ISCST3 outputs for the largest source. The Level III fugacity model takes into account reaction, advection and intermedia exchange after emission to the atmosphere. Based on the fugacity/

ISCST3 approach, the estimated EGBE concentrations in air, soil, and water were determined to be 0.3 mg/m³, 0.07 mg/kg, and 3.64 mg/L, respectively.

The lowest aquatic acute toxicity value available was for the protozoan *Endosiphon sulcatum* which experienced a 5 percent inhibition of cell multiplication at 91 mg/L following a 72-hour exposure. Due to the relatively minor effect reported and because the protozoa were exposed over several generations during the 72-hour period, we applied an acute/chronic adjustment factor of 10 to derive a safe level (i.e., toxicity reference value (TRV)) of 9 mg/L for aquatic biota in water.

The TRV for small mammals was based on the critical mammalian studies identified by IRIS for inhalation and oral exposure. Hemolysis was the critical endpoint of concern. A TRV of 20 mg/kg/day was derived by dividing the most sensitive LOAEL for female rats (59 mg/kg/day) by an uncertainty factor of three to adjust for the absence of a NOAEL.

Exposure scenarios were developed for each species and a quotient was calculated. In both cases, the quotient for aquatic invertebrates and small mammals was determined to be less than one. This suggested that both aquatic organisms and small mammals are not likely to be adversely affected by EGBE emissions to the atmosphere.

Based on our review of these data supplemented by additional environmental modeling, we have made an initial determination that there are adequate data on environmental effects of EGBE to determine that ambient concentrations, bioaccumulation, or deposition of EGBE are not reasonably anticipated to cause adverse environmental effects.

F. Transformation Assessment

Ethylene glycol monobutyl ether is one of many VOC that transform into other HAP after emission into the ambient air. The petition identifies the principal oxidation products of EGBE as n-butyl formate, 2-hydroxyethyl formate, propionaldehyde, 3-hydroxybutyl formate, and several isomeric forms of an organic nitrate compound. Only one of these compounds (i.e., propionaldehyde) is a listed HAP. However, the formate esters are known to transform in the atmosphere into formaldehyde, which is another listed HAP. In addition, propionaldehyde undergoes further transformation to formaldehyde and acetaldehyde (which is also a HAP). Both formaldehyde and acetaldehyde are probable human carcinogens and