1. *Bioavailability and metabolism*. DEHA is well absorbed from the gastrointestinal tract of rats, mice, monkeys, and humans (Ref. 2). No data were available concerning the possible absorption of DEHA from the lung or through the skin.

DEHA is rapidly hydrolyzed to adipic acid and 2-ethylhexanol both *in vivo* and *in vitro*. 2-Ethylhexanol is subsequently metabolized to ethylhexanoic acid and other acid and hydroxy acid derivatives and their gluconuride conjugates. Adipic acid is further oxidized to carbon dioxide. Excretion is primarily in the urine, with smaller amounts excreted in the expired air (carbon dioxide) and feces (Ref. 2).

2. *Acute toxicity*. DEHA exhibits slight acute toxicity. The oral median Lethal Dose (LD_{50}) value for rats is greater than 8 grams per kilogram (g/kg), and the dermal LD_{50} value for rabbits is greater than 9 g/kg (Ref. 2). There was no mortality among rats exposed by inhalation to a saturated vapor. DEHA was not irritating to rabbit eyes and skin, and it was not a dermal sensitizer in guinea pigs.

3. *Chronic toxicity*. Several chronic and subchronic feeding studies in rats and mice show that DEHA is not highly toxic. The primary effect in both species appears to be body weight depression. In rats, the Lowest Observed Adverse Effect Level (LOAEL) was 1,125 milligrams per kilogram per day (mg/kg/day) for both the chronic and 13-week studies. In mice, the LOAELs ranged from 2,800 mg/kg/day (chronic study) to 900 mg/kg/day (13-week study) (Ref. 2).

The weight of the evidence from several mutagenicity assays indicates that DEHA is probably not mutagenic (Ref. 2). Although most mutagenicity assays on DEHA are negative, DEHA does produce chromosome mutations in mammalian cells in culture (weakly), increase DNA synthesis in rats *in vivo*, and induce dominant lethals in mice *in vivo*. A positive response in the dominant lethal without collaborating genotoxicity data in assay systems designed to assess basic mutagenicity hazard is not an indication of potential mutagenicity (Ref. 2).

Data on both developmental and reproductive system toxicity are limited (Ref. 2). For developmental toxicity, a standard protocol test is available for only one species. For reproductive toxicity, there is a one-generation test, but not a multi-generation test. The one-generation reproduction study on male and female rats showed a reduction in litter size with administration of approximately 1,080 mg/kg/day of DEHA in feed, but the reduction was small and not statistically significant. The dominant-lethal assay discussed above found a dose-related increase in early fetal death, but the increase was not statistically significant and doses (0.46 to 9.2 g/kg, by single interperitoneal injection) were high.

4. *Carcinogenicity*. The National Toxicology Program tested DEHA for carcinogenicity in male and female rats and mice treated via diet (Ref. 2). Doses were approximately 700 or 1,500 mg/kg/day in the rat and 2,800 or 7,000 mg/kg/day in the mouse. The chemical was carcinogenic for female mice, inducing a significantly increased incidence of hepatocellular carcinomas. A marginally significant increase in hepatocellular carcinomas and adenomas combined was reported for male mice as compared with that of the concurrent controls. DEHA was not carcinogenic for the rats of either sex.

5. *Ecotoxicity*. DEHA is not expected to pose a significant hazard to the environment. Based on structure activity relationships (SARs), no toxic effects are anticipated for both freshwater and saltwater species at saturation (Ref. 2). For sediment species, acute and chronic toxicity are expected to occur only at high concentrations: 1,000 and 100 mg/kg (dry weight), respectively.

C. Environmental Fate

DEHA released to air has an estimated half-life for hydroxy radical oxidation of 5.2 hours. No information was found on photolysis of DEHA in air.

DEHA released to water is expected to undergo biodegradation in the water column with a half-life on the order of days to weeks. It will also partition readily to sediment based on its estimated soil organic carbon partition coefficient of 15,500. Once bound to sediments, DEHA will probably continue to biodegrade, but possibly at a significantly slower rate (halflife on the order of months). Hydrolysis is not expected to be a significant removal process below pH 9 (estimated half-life = 3.2 years at pH 7).

DEHA released to soil is expected to adsorb strongly based on its estimated soil organic carbon partition coefficient (15,500). Biodegradation is possible, and could further mitigate migration through soil. Biodegradation half-life in soils is estimated on the order of weeks.

DEHA is expected to be removed from wastewater in biological wastewater treatment systems by adsorption and biodegradation. Based on available biodegradation data and physical chemistry properties, 90 percent removal in Publicly Owned Treatment Works was estimated.

D. Exposure and Releases

Reported releases of DEHA were retrieved from the Toxic Release Inventory System (TRIS) and used to estimate air and water concentrations using TRIAIR and TRIWATER modeling techniques. The estimated maximum Lifetime Average Daily Potential Dose via inhalation (0.00178 mg/kg/day) is over 300-fold less than the Reference Dose (RfD) (0.6 mg/kg/day). The difference for oral exposure is much greater for water (Ref. 3). Based on this information, releases of DEHA are not expected to result in exposures of concern for human health or the environment.

The Agency believes that exposure considerations are appropriate in making determinations: (1) Under section 313(d)(2)(A); (2) under section 313(d)(2)(B) for chemicals that exhibit low to moderately low toxicity based on a hazard assessment; and (3) under section 313(d)(2)(C) for chemicals that are low or moderately ecotoxic but do not induce well-documented serious adverse effects. The Agency believes that exposure considerations are not appropriate in making determinations: (1) Under section 313(d)(2)(B) for chemicals that exhibit moderately high to high human toxicity based on a hazard assessment; and (2) under section 313(d)(2)(C) for chemicals that are highly ecotoxic or induce well-established adverse environmental effects. Given DEHA's low chronic toxicity and low ecotoxicity, exposure considerations are appropriate for detrminations under sections 313(d)(2)(B) and (C) as part of this proposed rule to delist. A more detailed discussion of EPA's listing determination guidelines is provided in the **Federal Register** of November 30, 1994 (59 FR 61442).

E. Technical Summary

Based on the total weight of available toxicity data, EPA believes that DEHA cannot reasonably be anticipated to cause significant adverse effects on human health or the environment. DEHA exhibits slight acute toxicity and causes adverse chronic effects only at high doses. Furthermore, DEHA is not expected to pose a significant hazard to the environment. In addition, based on EPA's exposure assessment, releases of DEHA are not expected to result in exposures of concern.

IV. Rationale for Proposal to Grant

EPA is granting the petition by proposing to delete DEHA from the EPCRA section 313 list of toxic chemicals. This decision is based on the