EXECUTIVE SUMMARY

Di-tert-Butyl Peroxide – Oral Risk Assessment CAS # 110-05-4			
PARAMETER	**LEVEL**	**UNITS**	**CALCULATED:**
NOAEL (no-observed-adverse-effect level)	Not identified		
Oral RfD (oral reference dose)	Not identified		
TAC (total allowable concentration)	0.01	mg/L	Based on a qualitative risk assessment under NSF/ANSI 61 (2002).
SPAC (single product allowable concentration)	0.01	mg/L	Based on a qualitative risk assessment under NSF/ANSI 61 (2002).
STEL (short term exposure level)	0.01	mg/L	Based on a qualitative risk assessment under NSF/ANSI 61 (2002).

KEY STUDIES	Zeiger, E., B. Anderson, S. Haworth, T. Lawlor, and K. Mortelmans. 1988; and Microbiological Associates, Inc. 1996.
CRITICAL EFFECT	There were insufficient data to identify a critical toxicological effect for this chemical.
UNCERTAINTY FACTORS	Since a quantitative risk assessment was not performed, no uncertainty factors were used.
TOXICITY SUMMARY	Di-tert-butyl peroxide was negative in two *Salmonella* reverse mutation assays. A mouse bone marrow micronucleus assay produced weakly positive results at one dose level in two assays, but results of one of the assays was within the range of historical control results. Di-tert-butyl peroxide was also a weak inducer of short-term indices of tumor promotion, although it was not a tumor promoter in a 60-week dermal study. However, lack of human data and of oral data in laboratory animals, as well as very weak positive responses in several genetic toxicity studies, indicate that *data are inadequate for an assessment of human carcinogenic potential* of di-tert-butyl peroxide. Floyd and Stokinger (1958) reported results of numerous experiments on the acute and subacute non-cancer effects of di-tert-butyl peroxide. The acute toxicity of the chemical is low, with an oral LD_{50} > 25,000 mg/kg and an intraperitoneal LD_{50} of 3,210 mg/kg. In a seven-week study during which five rats were dosed three times per week at one fifth of the oral LD_{50} (5,000 mg/kg), one rat died during the second week and another during the third week. The three survivors failed to gain weight normally. Histopathology of rats dosed with organic peroxides revealed mild liver effects of questionable relevance to treatment. Non-lethal effects of orally administered di-tert-butyl peroxide are therefore limited to decreased body weight gain and possible liver effects, based on available studies. The number of toxicological endpoints examined was not sufficient to meet current guidelines for a repeated dose study from which a critical effect could be selected and an oral RfD calculated.
CONCLUSIONS	The available data are inadequate to quantitatively describe the risk to human health posed by di-tert-butyl peroxide. Based on genetic toxicity and dermal tumor promotion data suggesting that the carcinogenic potential of the chemical is weak or absent, a qualitative risk level of 0.01 mg/L is adequately protective of human health based on a weight of evidence approach consistent with NSF/ANSI 61 (2002), Table A3.