Dibromoacetic acid meets the criteria for listing as known to the State to cause cancer under the Safe Drinking Water and Toxic Enforcement Act of 1986 (Health and Safety Code Section 25249.5 et seq.), more commonly known as Proposition 65, via the authoritative bodies mechanism. The regulatory requirements for listing by this mechanism are set forth in Title 22, California Code of Regulations, section 12306. The regulations include the criteria for evaluating the documentation and scientific findings by the authoritative body that the Office of Environmental Health Hazard Assessment (OEHHA) uses to determine whether listing under Proposition 65 is required.

The National Toxicology Program (NTP) is one of five institutions which has been identified as an authoritative body for identification of chemicals as causing cancer for the purposes of Proposition 65 (Section 12306(l)). NTP has identified dibromoacetic acid as causing cancer. The Office of Environmental Health Hazard Assessment (OEHHA) has found that this chemical is “formally identified” as causing cancer according to the regulations covering this issue (Section 12306(d)). Dibromoacetic acid is the subject of a report published by NTP that concludes that the chemical causes cancer. Also, the document specifically and accurately identifies the chemical, and the document meets one or more of the criteria outlined in Section 12306(d)(2).

OEHHA also finds that the criteria given in regulation for “as causing cancer” (Section 12306(e)) have been satisfied for dibromoacetic acid. In making this evaluation, OEHHA relied upon the discussion of data by the NTP in making its finding that dibromoacetic acid causes cancer. A brief discussion of the relevant carcinogenesis studies providing evidence for the finding is presented below. The statement in bold reflects data and conclusions that satisfy the criteria for the sufficiency of evidence for carcinogenicity (Section 12306(e)). The full citation for the document is given in this report.

1 All further references are to Title 22 of the California Code of Regulations unless otherwise indicated.
Dibromoacetic acid (CAS No. 631-64-1)

**Increased incidence of malignant and combined malignant and benign tumors in male and female mice with tumors at multiple sites in male mice.**

The NTP (2007) has concluded that there is clear evidence of the carcinogenic activity of dibromoacetic acid in male and female B6C3F1 mice.

The NTP (2007) exposed male and female F344/N rats and B6C3F1 mice (50 animals/group/sex) to dibromoacetic acid in drinking water for two years. In male mice, statistically significant increases in the incidences of hepatocellular adenoma (18/49, 37/50 [p<0.001], 37/50 [p<0.001] and 42/50 [p<0.001] for control, low-, mid- and high-dose groups, respectively), hepatocellular carcinoma (14/49, 9/50, 19/50 and 26/50 [p<0.05]) and combined hepatocellular adenoma or carcinoma (28/49, 41/50 [p<0.01], 42/50 [p<0.001] and 47/50 [p<0.001]) all occurred with positive trends [p<0.001]. The increased incidence of hepatoblastoma (0/49, 4/50, 6/50 [p<0.05] and 18/50 [p<0.001]) and the combined incidence of hepatocellular adenoma, carcinoma or hepatoblastoma (28/49, 41/50 [p<0.01], 43/50 [p<0.001], and 48/50 [p<0.001]) also occurred with positive trends [p<0.001]. In addition, incidences of alveolar/bronchiolar adenoma (7/49, 5/50, 17/50 [p<0.05], and 12/50) and alveolar/bronchiolar adenoma or carcinoma (12/49, 12/50, 22/50 [p<0.05], and 17/50) were significantly increased in mid-dose male mice. NTP considered the increase in lung tumors in male mice to be related to dibromoacetic acid exposure.

In female mice, statistically significant increases in the incidences of hepatocellular adenoma (19/49, 26/50, 32/50 [p<0.01] and 35/49 [p<0.001]), hepatocellular carcinoma (3/49, 3/50, 12/50 [p<0.01], and 8/49) and combined hepatocellular adenoma or carcinoma (22/49, 28/50, 37/50 [p<0.001] and 37/49 [p<0.001]) occurred with positive trends (p<0.001, for hepatocellular adenoma and combined hepatocellular adenoma or carcinoma; p<0.05, for hepatocellular carcinoma). Female mice also had an increase in combined alveolar/bronchiolar adenoma or carcinoma (2/50, 5/50, 5/50, and 7/50), and NTP concluded that the increase in lung tumors in female mice may have been related to dibromoacetic acid exposure.

NTP (2007) concluded that there was some evidence of carcinogenicity in male rats based on an increased incidence of malignant mesothelioma (all organs) (3/50, 1/50, 0/50 and 10/50 [p<0.05]); NTP found that the increase in mononuclear cell leukemia (17/50, 31/50, 24/50 and 13/50) in male rats may have been related to dibromoacetic acid exposure. NTP (2007) concluded that there was some evidence of carcinogenicity in
female rats based on an increased incidence and positive trend of mononuclear cell leukemia (11/50, 13/50, 16/50 and 22/50 [p<0.05]).

NTP (2007) tested dibromoacetic acid with *Salmonella typhimurium* strains TA98 and TA100. Dibromoacetic acid was mutagenic in strain TA100 with or without liver metabolic activation enzymes. No activity was found in strain TA98 with or without liver metabolic activation enzymes. Dibromoacetic acid tested negative in a mouse peripheral blood micronucleus test.

**Reference**