



Adopted as Rule: May 2009

**Chemical Name: Alachlor**

**CAS: #15972-60-8**

**Synonyms:** 2-Chloro-N-(2,6-diethylphenyl)-N-(methoxymethyl)acetamide; Alamex; Alanex; Alanox; Ala-Scept; Alazine; Alochlor; Bullet; Cannon; CDMA; CP 50144; Crop Star; Freedom; Lariat; Lasso; Lasso EC; Lasso II; Lasso Micro Tech; Lazo; Methachlor; Methoxymethyl-2',6'-diethylanilide chloroacetate; Micro-tech; Micro-Tech Lasso; Nudor; Partner

**Acute Non-Cancer Health Risk Limit (nHRL<sub>acute</sub>) = Not Derived (Insufficient Data)**

**Short-term Non-Cancer Health Risk Limit (nHRL<sub>short-term</sub>) = 200 ug/L**

$$= \frac{\text{(Reference Dose, mg/kg/d)} \times \text{(Relative Source Contribution)} \times \text{(Conversion Factor)}}{\text{(Short-term intake rate, L/kg-d)}}$$

$$= \frac{(0.10 \text{ mg/kg/d}) \times (0.5) \times (1000 \text{ ug/mg})}{(0.289 \text{ L/kg-d})}$$

$$= 173 \text{ rounded to } \mathbf{200 \text{ ug/L}}$$

Toxicity value: 0.10 mg/kg-d (laboratory animal)  
Source of toxicity value: MDH 2007  
Point of Departure: 10 mg/kg-d (NOAEL, Schroeder et al, 1981 as cited by EPA 1998)  
Human Equivalent Dose Adjustment: Not available  
Total uncertainty factor: 100  
UF allocation: 10 interspecies extrapolation and 10 intraspecies variability  
Critical effect(s): decreased kidney and nephritis (a statistically significant decreased relative ovarian weight was noted in the parental generation. No microscopic evidence was reported and no effect on reproductive parameters was noted. These changes are not included as a short-term additivity endpoint)  
Co-critical effect(s): none  
Additivity endpoint(s): Renal (kidney) system  
Secondary effect(s): none

**Subchronic Non-Cancer Health Risk Limit (nHRL<sub>subchronic</sub>) = 30ug/L**

$$= \frac{(\text{Reference Dose, mg/kg/d}) \times (\text{Relative Source Contribution}) \times (\text{Conversion Factor})}{(\text{Subchronic intake rate, L/kg/d})}$$
$$= \frac{(0.010 \text{ mg/kg/d}) \times (0.2) \times (1000 \text{ ug/mg})}{(0.077 \text{ L/kg-d})}$$
$$= 25.97 \text{ rounded to } \mathbf{30\text{ug/L}}$$

Toxicity value: 0.010 mg/kg-d (laboratory animal)  
Source of toxicity value: MDH 2007  
Point of Departure: 1mg/kg-d (NOAEL, Naylor et al, 1984 as cited by EPA 1998)  
Human Equivalent Dose Adjustment: Not available  
Total uncertainty factor: 100  
UF allocation: 10 interspecies extrapolation and 10 intraspecies variability  
Critical effect(s): hemosiderosis in the kidney and spleen and liver hemolytic anemia (reduced red blood cell counts, hematocrit and hemoglobin levels)  
Co-critical effect(s): increased liver weights  
Additivity endpoint(s): Hepatic (liver) system, hematological (blood) system  
Secondary effect(s): None

**Chronic Non-Cancer Health Risk Limit (nHRL<sub>chronic</sub>) = 5 ug/L**

$$= \frac{(\text{Reference Dose, mg/kg/d}) \times (\text{Relative Source Contribution}) \times (\text{Conversion Factor})}{(\text{Chronic intake rate, L/kg/d})}$$
$$= \frac{(0.0010 \text{ mg/kg/d}) \times (0.2) \times (1000 \text{ ug/mg})}{(0.043 \text{ L/kg-d})}$$
$$= 4.65 \text{ rounded to } \mathbf{5\text{ug/L}}$$

Toxicity value: 0.0010 mg/kg-d (laboratory animal)  
Source of toxicity value: MDH 2007  
Point of Departure: 1 mg/kg-d (NOAEL, Naylor et al, 1984 as cited by EPA 1998)  
Human Equivalent Dose Adjustment: Not available  
Total uncertainty factor: 1000  
UF allocation: 10 interspecies extrapolation, 10 intraspecies variability, 10 for subchronic to chronic extrapolation (comparison of shorter term to longer term duration studies indicates a decrease in the point of departure and additional effects as duration increased)  
Critical effect(s): hemosiderosis in the kidney and spleen and liver hemolytic anemia (reduced red blood cell counts, hematocrit and hemoglobin levels).  
Co-critical effect(s): increased liver weights  
Additivity endpoint(s): Hepatic(liver) system, hematology (blood) system

Secondary effect(s): Ocular effects and increased mortality

**Cancer Health Risk Limit (cHRL) = Not Applicable<sup>1</sup>**

Cancer classification: likely to be carcinogenic at high doses, but not likely at low doses, by all exposure routes

Slope factor: 0.08 per (mg/kg-d), however a nonlinear approach is recommended (EPA 1998, 2004)

Source of slope factor: EPA 1998

Tumor site(s): nasal, stomach and thyroid tumors

<sup>1</sup> Alachlor is a nonlinear carcinogen and the RfD is considered to be protective against cancer.

**Volatile: No**

**Summary of changes since 1993/1994 HRL promulgation:**

The 1993/94 HRL value for alachlor (4ug/L) was based on cancer. Legislation passed in the 2007 regular session ([Chapter 147, Article 17, section 2](#)) established new Health Risk Limit (HRL) values, effective July 1, 2007, for chemicals when the federal standard determined by the United States Environmental Protection Agency (US EPA) is more stringent than the 1993/1994 HRL value. MCLs are federal standards adopted for regulation of public drinking water in Minnesota. However, MCLs incorporate a consideration of the costs required to reduce contaminant concentrations of a given level and the technological feasibility of reaching that level and therefore are not solely based on consideration of human health.

A comparison of 1993/94 HRL values to the current Maximum Contaminant Levels (MCLs) from the US EPA identified eleven chemicals, including alachlor, that had a lower MCL value than a HRL value. The 1993/94 HRL value of 4 ug/L was revised to the MCL value of 2 ug/L as of July 1, 2007.

The draft short-term (200 ug/L), subchronic (30 ug/L) and chronic nHRL (5 ug/L) represents new HRL values. A cancer HRL has not been derived as the result of applying a nonlinear cancer assessment methodology rather than a linear approach as recommended by EPA. The chronic nHRL (5 ug/L) is 2.5-fold higher than the current MCL-based HRL of 2 ug/L as the result of: 1) a more recent toxicological review, including reclassification as a nonlinear carcinogen; 2) use of intake rates that incorporate higher intake during early life; and 3) rounding to one significant digit.

**Summary of toxicity testing for health effects identified in the Health Standards Statute:**

	Endocrine	Immunotoxicity	Development	Reproductive	Neurotoxicity
Tested?	No <sup>1</sup>	No	Yes	Yes	No <sup>4</sup>
Effects?	--	--	No <sup>2</sup>	No <sup>3</sup>	No

Note: Even if testing for a specific health effect was not conducted for this chemical, information about that effect might be available from studies conducted for other purposes. Most chemicals have been subject to multiple studies in which researchers identify a dose where no effects were observed, and the lowest dose that caused one or more effects. A toxicity value based on the effect observed at the lowest dose across all available studies is considered protective of all other effects that occur at higher doses.

**Comments on extent of testing or effects:**

- <sup>1</sup>Although endocrine effects were not tested, in the future EPA will be evaluating whether or not alachlor is an endocrine disruptor. They are developing a screening program for effects in humans caused by toxicants that mimic the effects caused by naturally occurring hormones such as estrogen. Alachlor has been shown to cause increase thyroid weights at doses ~5-fold higher than the subchronic and chronic critical study LOAEL. Thyroid tumors were also observed in rats exposed to doses ~40-fold higher than the subchronic and chronic critical study LOAEL.
- <sup>2</sup>Developmental studies have reported increase resorptions and decreased litter size at dose levels >10-fold higher than the short-term critical study LOAEL and >100-fold higher than the subchronic and chronic critical study LOAEL. The 3-generation study reported renal effects in rat pups at levels 10-fold higher than the subchronic and chronic critical study LOAEL.
- <sup>3</sup>A single multigenerational study has been conducted. No effect on reproductive parameters were reported, however, significant decreases in ovarian weight were observed in the F0, parental generation. No microscopic changes were reported.
- <sup>4</sup>Based on toxicity profile for alachlor, OPP concluded that developmental neurotoxicity study was not needed.

**References:**

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