ACROLEIN

Comments on the **environmental sections** of the CEPA PSL Draft Assessment Report on Acrolein were provided by:

1. Degussa-Huels AG, Hanau-Wolfgang, Germany
2. DaimlerChrysler Canada Inc., Windsor, Ontario

Comments and responses are summarized below by Environment Canada. (All were based on the English version of the report).

Comment[(source)]	**Response**
Choice of the Cassee *et al.* (1996) study for long-term exposure is questioned.[(1)]	Environment Canada concurs with Health Canada's rationale for using this study (see third comment in Health Canada's summary table of public comments and responses on the draft acrolein report).
The range for estimated releases for road motor vehicles is provided in the assessment report. If gasoline-fuelled vehicles are typically below the detection limit, how were these values derived and apportioned for the existing fleet?[(2)]	The assessment report does list the reference literature that was used to make the release calculations. The Supporting Document describes how the calculations were made.
The Ministers' Expert Advisory Panel, 1995 states that, "Photooxidation of diesel and gasoline exhaust are other sources" (of acrolein). The assessment report does not produce the information that apportions the secondary formation of acrolein.[(2)]	The assessment report indicates that non-combustion sources of acrolein are limited and lists releases as unknown.
With the introduction of low sulphur fuel in Canada over the next 6 years, lower total emissions may reduce direct acrolein emissions and precursor emissions.[(2)]	This comment will be forwarded to risk managers for their information.

ACROLEIN

Comments on the **health-related sections** of the CEPA PSL Draft Assessment Report on Acrolein were provided by:

- Degussa-Huels AG, Hanau-Wolfgang, Germany
- Canadian Petroleum Products Institute, Ottawa, Ontario

Comments and responses are summarized below by Health Canada. (All were based on the English version of the report).

Comment	Response
Based upon information provided in the assessment report, ambient air should not be considered an "important" source of human exposure to acrolein.	Monitoring data described and analyzed in the report specifically indicate that ambient air may be an important source of exposure for individuals residing in the vicinity of point sources or in locations heavily impacted by vehicular traffic.
The mentioned option to reduce exposure from vehicle exhaust should be accompanied by an acknowledgement that such a recommendation would likely not result in significant reductions in human exposure.	Reduction in emissions from vehicular sources is anticipated to have a significant impact for individuals residing in the vicinity of point sources or in locations heavily impacted by vehicular traffic, since these sources contribute significantly to exposure. However, detailed analysis of various options to reduce exposure to acrolein will be undertaken in the subsequent risk management phase, with respect to sources controllable under CEPA.
The derived Tolerable Concentration (TC) [inhalation] for acrolein is overly conservative and is based upon a study that is not suitable for the derivation of such values for long-term exposure. The uncertainty factor applied in the derivation of the TC [ingestion] is overly conservative.	A strong rationale is provided in the assessment report for use of a short term study in this case. It includes the sensitivity of the critical study, and nature of the changes which are similar following short and long term exposure. Moreover, the value is compared to those developed from longer term studies. These points were extensively discussed by an independent panel of scientific experts who unanimously agreed with both the choice of the critical study selected for the exposure-response analysis, as well as the uncertainty factors employed in derivation of the TCs for inhalation and ingestion. Data are

Comment	Response
	not available to justify use of less than default values for inter- and intra-species variation for the uncertainty factor for the TC for ingestion.
CNS effects are only observed following exposure to high concentrations of acrolein.	Neurological effects are addressed briefly in the text since they are not considered critical. (Critical effects are those of biological significance expected to occur at lowest dose or concentration). Doses which cause neurological effects were indicated.
The NOAEL in the Parent et al. (1992a) study should be 0.5 mg/kg bw/day.	A statistically significant (dose-related) increase in mortality was noted amongst male and female rats at doses of 0.5 mg/kg bw/day. The NOEL currently identified in the report is appropriate and was considered and agreed during written and panel peer reviews.
The study on subjective effects in humans exposed to acrolein reported by Darley et al. (1960) should not be used as a basis for the risk assessment.	The assessment report indicates that the hazard characterization and dose-response are based primarily on studies conducted in laboratory animals. Reference in the assessment report to the fact that the derived TCs [inhalation] are some orders of magnitude less than putative thresholds for subjective effects in humans identified from the limited studies reported by Darley et al. (1960) is included to illustrate the "protective" nature of the derived TC.
Concerns with respect to the genotoxicity of acrolein at the site of first contact should be put into perspective, based upon other data.	Uncertainties associated with the available database on the genotoxicity were outlined within the assessment report and agreed upon by an independent panel of scientific experts.
A number of minor editorial suggestions were presented.	Editorial changes were made where considered appropriate.