Carcinogenicity of atrazine, alachlor, and vinclozolin

From October to November, 2025, a Working Group of 22 scientists from 12 countries met at the International Agency for Research on Cancer (IARC) in Lyon, France, to finalise their evaluation of the carcinogenicity of atrazine, alachlor, and vinclozolin.

Atrazine and alachlor were each classified as "probably carcinogenic to humans" (Group 2A), based on combinations of "limited" evidence for cancer in humans with "sufficient" evidence for cancer in experimental animals and "strong" mechanistic evidence in experimental systems. Vinclozolin was classified as "possibly carcinogenic to humans" (Group 2B), based on "sufficient" evidence for cancer in experimental animals and on "strong" mechanistic evidence in experimental systems. These assessments will be published in Volume 140 of the *IARC Monographs*.[1]

For each of these pesticides, factory and agricultural workers have the highest exposures, which can occur during pesticide production and agricultural or horticultural activities, primarily via inhalation and dermal absorption. General population exposure occurs primarily via ingestion of drinking-water and foodstuffs, and is typically estimated to be low. Atrazine and alachlor are environmentally persistent and have been detected in environmental matrices, especially water. Vinclozolin is environmentally non-persistent.

Atrazine is a broad-spectrum chlorinated triazine herbicide that is used primarily in corn, sorghum, and sugarcane cultivation. It has been banned as a pesticide in the European Union and in several other countries but remains in heavy use elsewhere. In the general population, residential turf contact can result in higher short-term incidental dermal and oral doses than from food or drinking-water.

There is "limited" evidence in humans that atrazine causes non-Hodgkin lymphoma (NHL) that is positive for the t(14;18) chromosomal translocation. Two case-control studies reported strong positive associations between exposure to atrazine or triazines (at a time when atrazine was the predominant triazine used in the study area) and translocation-positive NHL (meta-odds ratio, 2·03; 95% CI 1·12–3·68), and no increased risk for translocation-negative NHL cases.[2,3] The t(14;18) translocation was considered relevant because it may hinder cell apoptosis. The Working Group considered that bias and confounding could be ruled out with reasonable confidence but that the role of chance could not. For all other cancer types considered, the available studies in humans did not show consistent positive findings, and the evidence was considered "inadequate".

The "sufficient" evidence for cancer in experimental animals for atrazine was based on an increase in the incidence of malignant neoplasms in female rats in multiple well-conducted studies, including two that complied with Good Laboratory Practice (GLP). Atrazine caused adenocarcinoma of the mammary gland in female Sprague-Dawley rats[4] and adenocarcinoma of the uterus in Fischer 344/LATI rats.

There is "strong" mechanistic evidence that atrazine exhibits key characteristics of carcinogens (KCs). Atrazine induces oxidative stress in experimental systems. Several studies in rodents showed increases in reactive oxygen species production and endpoints of oxidative damage to lipids and DNA in multiple organs, and reduced oxidative stress in the presence of antioxidants. Atrazine induces inflammation in vivo, including increased expression of NF-κB-dependent genes and altered Th1 and Th2 cytokine balance. Atrazine is immunosuppressive; in rodents it decreased leukocyte count and effector T cells, increased regulatory Treg cells, and decreased the delayed-type hypersensitivity response. Atrazine modulates oestrogen-mediated effects in experimental systems. Atrazine increased aromatase activity (CYP19). It modulated the membrane oestrogen receptor mER/GPR30/GPER[5] in various human hormone-sensitive and triple-negative breast cancer cell lines. In male Wistar rats, atrazine reduced serum levels of testosterone, luteinising hormone, and follicle-stimulating hormone.[6] Atrazine also altered cell proliferation and cell death in vitro, and induced hyperplasia in several tissues in rodents.

Alachlor is a chloroacetanilide herbicide that has been widely used on crops such as corn and soybeans. Once one of the most heavily used herbicides globally, alachlor use declined sharply after regulatory restrictions began in the 1990s, and today it is used to a much lower extent.

There is "limited" evidence that alachlor causes laryngeal cancer in humans on the basis of evidence from a single, large, high-quality cohort study of pesticide applicators.[7] A strong exposure–response association between alachlor and laryngeal cancer was found, and it remained unchanged under different latency periods and after adjustment for a range of co-exposures and potential confounders, including other pesticides, smoking, alcohol, and additional laryngeal cancer risk factors. Chance could not be ruled out with reasonable confidence because this was a single finding for a cancer type not commonly associated with pesticide exposure. For all other cancer types, the evidence was considered "inadequate" because the available studies did not show consistent positive findings.

The "sufficient" evidence in experimental animals for alachlor is based on an increase in the incidence of either malignant neoplasms or an appropriate combination of benign

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and malignant neoplasms in both sexes of two species in multiple well-conducted studies, including one that complied with GLP. Alachlor caused bronchioloalveolar adenoma or carcinoma (combined) in male CD-1 albino mice.[8] In male and female Long-Evans rats, alachlor caused malignant tumours of the stomach. In males, alachlor also caused malignant and benign or malignant tumours (combined) of the nasal cavity[9] and of the thyroid gland. In females, alachlor also caused benign or malignant tumours (combined) of the nasal cavity, and malignant tumours of the thyroid gland and of the thymus and uterus.

There is "strong" mechanistic evidence that alachlor exhibits KCs in experimental systems. Alachlor modulates receptor-mediated effects, showing in vitro modulation of the oestrogen-receptor pathway. Alachlor altered cell proliferation in rat and mouse nasal tissue in multiple studies. Hyperplasia was also observed in the nasal tissue[9] and stomach in rodents.

Vinclozolin is a fungicide that has been used mainly on fruit and vegetables, but also on turf. In the 2000s, its use was phased out in the European Union, the USA, and several other countries, but it remains in use elsewhere.

The evidence regarding cancer in humans is "inadequate" for vinclozolin because results reported in the few available studies were largely null.

The "sufficient" evidence for cancer in experimental animals for vinclozolin is based on an increase in the incidence of either malignant neoplasms or an appropriate combination of benign and malignant neoplasms in both sexes of two species in multiple studies that complied with GLP. Vinclozolin caused hepatocellular carcinoma in female C57BL/6/JICO mice.[10] In male Wistar rats, vinclozolin caused hepatocellular carcinoma and benign or malignant Leydig cell tumours (combined). In females, vinclozolin caused adenoma or carcinoma (combined) of the adrenal gland and of the adrenal cortex, benign or malignant C-cell tumours (combined) of the thyroid gland, and adenocarcinoma of the uterus.[11]

There is "strong" mechanistic evidence that vinclozolin exhibits KCs in experimental systems. It induced alterations of epigenetic endpoints associated with carcinogenic pathways in vitro and in vivo. Vinclozolin increases endpoints of inflammation in vitro, and induces chronic inflammation in rodents in many tissues, including prostate.[12] Vinclozolin modulates the androgen-signalling pathway in vitro. In the absence of androgens, vinclozolin or its metabolites induced prostate-specific antigen secretion. Vinclozolin and its metabolites promoted cell proliferation in vitro[13] and induced hyperplasia in rodents in several tissues, including prostate.

We declare no competing interests.

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