International Agency for Research on Cancer

World Health Organization

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IARC Monographs evaluate the carcinogenicity of atrazine, alachlor, and vinclozolin

IARC Monographs Volume 140

Questions and Answers (Q&A)

The meeting for *IARC Monographs* Volume 140: Atrazine, Alachlor, and Vinclozolin, convened by the International Agency for Research on Cancer (IARC), took place between 28 October and 4 November 2025.

The Working Group of 22 international experts from 12 countries met in Lyon, France, to evaluate the carcinogenicity of three pesticides: the herbicides atrazine and alachlor and the fungicide vinclozolin.

More information about the meeting is available on the *IARC Monographs* website: https://monographs.iarc.who.int/iarc-monographs-volume-140/.

The outcome of the assessment has been published in a summary article in *The Lancet Oncology*[1] and will be described in detail in Volume 140 of the *IARC Monographs*, to be published in late 2026 or early 2027.

1. What was evaluated in IARC Monographs Meeting 140?

The Working Group for *IARC Monographs* Volume 140 evaluated the carcinogenicity of three pesticides: the herbicides atrazine and alachlor and the fungicide vinclozolin.

Atrazine, a chlorinated triazine herbicide first registered in the 1950s, is one of the most widely used weed control agents globally. In agricultural settings, atrazine is used primarily in the cultivation of corn, sorghum, and sugarcane. It is also used on turf and lawns in residential or recreational settings.

Alachlor is a chloroacetanilide herbicide, introduced in 1969, that was widely used to control grasses and broadleaf weeds in crops such as corn and soybean.

Vinclozolin is a fungicide that was first registered for use in 1981. It has been used mostly in agriculture, mainly on fruit and vegetables but also on turf.

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[1] Cattley RC, De Roos AJ, Mandrioli D, Pearce N, Pronk A, Soshilov A, et al. (2025). Carcinogenicity of atrazine, alachlor, and vinclozolin. *Lancet Oncol*. Published online 21 November 2025; https://doi.org/10.1016/S1470-2045(25)00702-8

2. Who is exposed to these agents, and how?

For each of these pesticides, workers have the highest exposures, which can occur during pesticide production and agricultural or horticultural activities. Occupational exposure occurs primarily via inhalation and dermal absorption.

Exposure of the general population occurs primarily via drinking-water and the diet, and it is generally estimated to be low. Increased exposure to atrazine via drinking-water has been shown in areas that are contaminated with atrazine from agricultural activities. Contact with treated turf has been estimated to result in increased incidental dermal or oral exposure, especially in children.

3. Are these pesticides still being used?

The use of atrazine has been banned in some countries, including in the European Union, but it is still one of the main herbicides used in many regions of the world, including the USA and many low- and middle-income countries in Africa, Asia, and Latin America.

Alachlor was once one of the most heavily used herbicides globally, but its production and use declined sharply after regulatory restrictions and bans beginning in the 1990s, particularly in regions with stronger regulations.

Globally, the use of vinclozolin was higher before the 2000s than it is today, especially in the USA and Europe. Since 2007, vinclozolin products have been withdrawn from the European Union, as well as Australia and South Africa. The use of vinclozolin has been restricted in the USA and has been continuously decreasing. However, vinclozolin is still used in some other countries.

4. What are the results of the evaluation?

The results of the evaluation are summarized in Table 1.

Table 1. Summary of classifications in *IARC Monographs* Volume 140

Agent	Evidence stream			Overall evaluation
	Cancer in humans	Cancer in experimental animals	Mechanistic evidence	
Atrazine	*Limited (*non-Hodgkin lymphoma that is positive for the t(14;18) chromosomal translocation)	*Sufficient*	*Strong* (in experimental systems)	**Group 2A**
Alachlor	*Limited* (laryngeal cancer)	*Sufficient*	*Strong* (in experimental systems)	**Group 2A**
Vinclozolin	*Inadequate*	*Sufficient*	*Strong* (in experimental systems)	**Group 2B**

5. How did the Working Group reach these results?

Atrazine and alachlor were each classified as *probably carcinogenic to humans* (Group 2A) in two ways: (i) via the combination of *limited* evidence for cancer in humans and *sufficient* evidence for cancer in experimental animals; and (ii) via the combination of *limited* evidence for cancer in humans and *strong* mechanistic evidence in experimental systems.

Vinclozolin was classified as *possibly carcinogenic to humans* (Group 2B) in two ways: (i) via *sufficient* evidence for cancer in experimental animals; and (ii) via *strong* mechanistic evidence in experimental systems. For vinclozolin, the evidence regarding cancer in humans was *inadequate*.

6. How was the Group 2A classification reached for atrazine? What were the key studies?

For atrazine, in two case–control studies, strong positive associations were reported between exposure to atrazine[2] or triazines (at a time when atrazine was the predominant triazine used in the study area)[3] and non-Hodgkin lymphoma (NHL) cases that were positive for the t(14;18) chromosomal translocation, whereas no increased risk was reported for NHL cases that were negative for this translocation. The Working Group thus concluded that the evidence for cancer in humans was *limited* for NHL that is positive for this chromosomal translocation.

The *sufficient* evidence for cancer in experimental animals treated with atrazine was based on an increase in the incidence of malignant neoplasms in female Sprague-Dawley and Fischer 344/LATI rats in multiple well-conducted studies, including two studies that complied with Good Laboratory Practice (GLP). There was *strong* mechanistic evidence in experimental systems that atrazine exhibits the following key characteristics of carcinogens: “induces oxidative stress”, “induces inflammation”, “is immunosuppressive”, “modulates receptor-mediated effects”, and “alters cell proliferation, cell death, or nutrient supply”.

Therefore, atrazine reached a Group 2A classification in two distinct ways: (i) via the combination of *limited* evidence for cancer in humans and *sufficient* evidence for cancer in experimental animals, and (ii) via the combination of *limited* evidence for cancer in humans and *strong* mechanistic evidence in experimental systems.

7. What is t(14;18)-positive non-Hodgkin lymphoma (NHL), and why did the Working Group restrict its evaluation of the evidence for cancer in humans for atrazine as *limited* to this type of NHL only?

Overall, for all types of NHL combined, the evidence regarding an association with atrazine was mixed, and therefore the evidence regarding cancer in humans was considered *inadequate*. However, the Working Group considered that there was *limited* evidence for NHL that is positive for the chromosomal translocation t(14;18). This description, NHL that is positive for the translocation t(14;18), denotes a category of NHL in which the cancer cells carry a characteristic mutation, whereby a portion of chromosome 14 is translocated to chromosome 18. This mutation constitutively activates a gene that normally inhibits programmed cell death, which can eventually lead to tumour formation.

Two case–control studies found strong elevations in the odds of translocation-positive NHL, but not translocation-negative NHL, among users of atrazine[2] or triazine (at a time when atrazine was judged to be the dominant triazine in the study region)[3]. Although confounding by other pesticides was considered, the Working Group judged, with reasonable confidence, that confounding could not account for elevations in risk

[2] Schroeder JC, Olshan AF, Baric R, Dent GA, Weinberg CR, Yount B, et al. (2001). Agricultural risk factors for t(14;18) subtypes of non-Hodgkin’s lymphoma. *Epidemiol*. 12(6):701–9. https://doi.org/10.1097/00001648-200111000-00020
[3] Chiu BC, Dave BJ, Blair A, Gapstur SM, Hoar Zahm S, Weisenburger DD (2006). Agricultural pesticide use and risk of t(14;18)-defined subtypes of non-Hodgkin lymphoma. *Blood* 108(4):1363–9. https://doi.org/10.1182/blood-2005-12-008755

of such magnitude. Similarly, although recall bias was a consideration in the appraisal of case–control studies on all types of NHL overall, this was deemed unlikely to explain the specificity of the elevated risk for translocation-positive NHL, because participants would have been unaware of their chromosomal translocation status (and its significance) when reporting their pesticide use. Nevertheless, the lower number of studies that evaluated translocation status and imprecision of the results meant that chance could not be excluded with reasonable confidence.

8. How was the Group 2A classification reached for alachlor?

There was *limited* evidence that alachlor causes laryngeal cancer in humans (see Question 9 below). There was *sufficient* evidence that alachlor causes cancer in experimental animals. Oral treatment with alachlor led to 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 well-conducted studies, including one that complied with GLP. There was *strong* mechanistic evidence in experimental systems that alachlor exhibits the key characteristics of carcinogens "modulates receptor-mediated effects" and "alters cell proliferation, cell death, or nutrient supply".

Therefore, the Working Group reached a Group 2A classification for alachlor in two distinct ways: (i) via the combination of *limited* evidence for cancer in humans and *sufficient* evidence for cancer in experimental animals, and (ii) via the combination of *limited* evidence for cancer in humans and *strong* mechanistic evidence in experimental systems.

9. How did the Working Group reach its conclusion that there was *limited* evidence for a causal association between alachlor and laryngeal cancer in humans?

The conclusion of *limited* evidence that alachlor causes laryngeal cancer in humans was based on the results of a single large, high-quality cohort study of pesticide applicators – the Agricultural Health Study in the USA.[4] The study found a strong exposure–response association between intensity-weighted days of alachlor exposure and laryngeal cancer. The association 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 risk factors for laryngeal cancer such as exposure to asbestos, dusts (silica, wood, sand, cotton, grain), solvents, engine exhaust, and metal grinding. For the evaluation, the Working Group consulted guidance on appraising single studies within the Preamble to the *IARC Monographs*[5] and the report

[4] Lerro CC, Andreotti G, Koutros S, Lee WJ, Hofmann JN, Sandler DP, et al. (2018). Alachlor use and cancer incidence in the Agricultural Health Study: an updated analysis. *J Natl Cancer Inst.* 110(9):950–958. https://doi.org/10.1093/jnci/djy005

[5] IARC (2019). Preamble to the *IARC Monographs* (amended January 2019). Lyon, France: International Agency for Research on Cancer. Available from: https://monographs.iarc.who.int/iarc-monographs-preamble-preamble-to-the-iarc-monographs/.

of the Advisory Group to Recommend Priorities for the *IARC Monographs* during 2025–2029[6]. One piece of guidance states that "The key issue a Working Group must consider in such a circumstance is whether chance, bias, and confounding can be ruled out when there is only a single study". For alachlor and laryngeal cancer, the consistency of the association across analyses, the study quality, and the comprehensiveness of adjustment allowed the Working Group to consider that there was a credible positive association, and to rule out bias and confounding with reasonable confidence. Nevertheless, in line with guidance from the Preamble, that "[to draw causal inferences,] more than a single study in a single population will almost always be needed", the Working Group could not conclude that a causal relationship had been established. The Working Group highlighted that, for the present evaluation, even the statistically significant findings from the single study did not allow them to rule out chance with reasonable confidence, especially because there was no clear a priori hypothesis. This is also consistent with the suggestion of the Advisory Group to Recommend Priorities for the *IARC Monographs* that "the number of studies required might differ for an interpretation of *limited* versus *sufficient* evidence".

10. How was the Group 2B classification reached for vinclozolin?

For vinclozolin, there was *sufficient* evidence for cancer in experimental animals and *strong* mechanistic evidence in experimental systems. In experimental animals, oral treatment with vinclozolin caused 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. There was consistent and coherent evidence in experimental systems for the following key characteristics of carcinogens: "induces epigenetic alterations", "induces chronic inflammation", "modulates receptor-mediated effects", and "alters cell proliferation, cell death, or nutrient supply".

Therefore, vinclozolin reached a Group 2B evaluation in two distinct ways: (i) via *sufficient* evidence for cancer in experimental animals; and (ii) via *strong* mechanistic evidence in experimental systems.

11. What sources of information were considered by the Working Group in its evaluation of the evidence of cancer in experimental animals?

As described in the current Preamble to the *IARC Monographs* (last revised in 2019)[5], the Working Group reviews publicly available scientific data, including peer-reviewed papers in the scientific literature, and may also review unpublished reports, if they are made available in their final form by governmental agencies and if they contain sufficient detail for critical review by the Working Group. For atrazine, alachlor, and vinclozolin, in addition to scientific research publications, the Working Group was able to retrieve and review several Data Evaluation Reports that were submitted for regulatory authorization to the United States Environmental

[6] IARC (2024). Report of the Advisory Group to Recommend Priorities for the *IARC Monographs* during 2025–2029. Lyon, France: International Agency for Research on Cancer. Available from: https://monographs.iarc.who.int/wp-content/uploads/2024/11/AGP_Report_2025-2029.pdf.

Protection Agency (US EPA) and that were publicly accessible on the US EPA website. Specifically, Data Evaluation Reports reviewed by the Working Group included those for atrazine, alachlor, and vinclozolin. These references are listed below.

Atrazine

US EPA (1987a). Atrazine – submission of toxicity studies to fulfill the requirements outlined in the registration standard. Review of an unpublished report prepared by Hazelette JR, et al. (1987). Washington (DC), USA: United States Environmental Protection Agency. Available from: https://archive.epa.gov/pesticides/chemicalsearch/chemical/foia/web/pdf/080803/080803-109.pdf.

US EPA (1987b). Twenty-four-month combined chronic oral toxicity and oncogenicity study in rats utilizing atrazine 98.9% technical [Accession Nos. 262714–262727]. Review of an unpublished report prepared by Mayhew DA, et al. (1986). Washington (DC), USA: United States Environmental Protection Agency. Available from: https://archive.epa.gov/pesticides/chemicalsearch/chemical/foia/web/pdf/080803/080803-083.pdf.

US EPA (1988). Atrazine, toxicology chapter of the registration standard. Review of an unpublished report prepared by Copley MP (1988). Washington (DC), USA: United States Environmental Protection Agency. Available from: https://www3.epa.gov/pesticides/chem_search/cleared_reviews/csr_PC-080803_9-Dec-88_130.pdf.

US EPA (1994). Transmittal of reviews of carcinogenicity studies in Sprague Dawley and Fischer 344 rats with atrazine. Review of an unpublished report prepared by Thakur AK (1992). Washington (DC), USA: United States Environmental Protection Agency. Available from: https://www3.epa.gov/pesticides/chem_search/cleared_reviews/csr_PC-080803_9-Feb-94_233.pdf.

US EPA (1995). Atrazine reregistration; reregistration case no. 0062; preliminary review of combined chronic feeding/carcinogenicity study in rats using G-34048 technical (hydroxyatrazine) as the test material, guideline 83-5. Review of an unpublished report prepared by Chow E, et al. (1995). Washington (DC), USA: United States Environmental Protection Agency. Available from: https://www3.epa.gov/pesticides/chem_search/cleared_reviews/csr_PC-080803_13-Jul-95_238.pdf.

US EPA (1996). Carcinogenicity peer review on atrazine (5th). Washington (DC), USA: United States Environmental Protection Agency. Available from: https://archive.epa.gov/pesticides/chemicalsearch/chemical/foia/web/pdf/080803/080803-244.pdf.

US EPA (1998). Atrazine – review of a 2-year oncogenicity study, MRID 44544701. Review of an unpublished report prepared by Morseth S (1998). Washington (DC), USA: United States Environmental Protection Agency. Available from: https://www3.epa.gov/pesticides/chem_search/cleared_reviews/csr_PC-080803_4-Jan-99_254.pdf.

Alachlor

US EPA (1981). EPA Reg. #524–316. Review of Monsanto 18-month oncogenic study in mice. Review of an unpublished report prepared by Street RW (1981). Washington (DC), USA: United States Environmental Protection Agency. Available from: https://archive.epa.gov/pesticides/chemicalsearch/chemical/foia/web/pdf/090501/090501-020.pdf.

US EPA (1984). Chronic feeding study in rat. Review of an unpublished report prepared by Scott LD (1983). Project No. ML-80-186; Study No. 800218; MRID No. 00139021. Washington (DC), USA: United States Environmental Protection Agency. Available from: https://www3.epa.gov/pesticides/chem_search/cleared_reviews/csr_PC-090501_2-Nov-84_056.pdf.

US EPA (1996). Carcinogenicity peer review of alachlor – third. Washington (DC), USA: United States Environmental Protection Agency. Available from: https://archive.epa.gov/pesticides/chemicalsearch/chemical/foia/web/pdf/090501/090501-142.pdf.

Vinclozolin

US EPA (1994a). Vinclozolin. Oncogenicity feeding – mouse (83-2). Review of an unpublished report prepared by Mellert W (1994), Carcinogenicity study with Reg. No. 83 258-vinclozolin in C57BL mice administration in the diet for 18 months, Project No. 83-2: Study No. 80S0375/88112; MRID No. 43254704. Washington (DC), USA: United States Environmental

Protection Agency. Available from: https://www3.epa.gov/pesticides/chem_search/cleared_reviews/csr_PC-113201_19-Apr-95_218.pdf.

US EPA (1994b). Reg. 83 258 – vinclozolin. Chronic feeding – rat (83-1a). Review of an unpublished report prepared by Mellert W (1994), Chronic toxicity study with Reg. No. 83 258 – vinclozolin in rats administration in the diet for 24 months. Project No. 83-1a: Study No. 71S0375/88026; MRID No. 43254701. Washington (DC), USA: United States Environmental Protection Agency. Available from: https://archive.epa.gov/pesticides/chemicalsearch/chemical/foia/web/pdf/113201/113201-221.pdf.

US EPA (1994c). Reg. 83 258 – vinclozolin. Chronic feeding – rat (83-1b). Review of an unpublished report prepared by Mellert W (1994), Second chronic toxicity study with Reg. No. 83258 – vinclozolin in rats administration in the diet for 24 months. Project No. 83-1b: Study No. 71S0375/88109; MRID No. 43254702. Washington (DC), USA: United States Environmental Protection Agency. Available from: https://archive.epa.gov/pesticides/chemicalsearch/chemical/foia/web/pdf/113201/113201-220.pdf.

US EPA (1994d). Reg. 83 258 – vinclozolin. Chronic feeding – rat (83-2a). Review of an unpublished report prepared by Mellert W (1994), Toxicology study report: carcinogenicity study with No. 83 258 – vinclozolin in Wistar rats administration in the diet for 24 months. Project No. 83-2a: Study No. 71S0375/88105; MRID No. 43254703. Washington (DC), USA: United States Environmental Protection Agency. Available from: https://www3.epa.gov/pesticides/chem_search/cleared_reviews/csr_PC-113201_14-Jun-95_222.pdf.

12. Were these agents assessed in the past, and if so, why were they re-assessed?

Atrazine was previously classified by the *IARC Monographs* programme as *not classifiable as to its carcinogenicity to humans* (Group 3) in 1998 (Volume 73[7]). In 2024, the Advisory Group to Recommend Priorities for the *IARC Monographs* during 2025–2029[6] had recommended the re-evaluation of atrazine with high priority because new studies were available that have investigated the association between atrazine exposure and cancer in humans and experimental animals, as well as new mechanistic evidence.

Neither alachlor nor vinclozolin have been previously evaluated by the *IARC Monographs* programme.

13. Are these agents persistent in the environment?

Atrazine and alachlor are persistent in the environment. Because of its slow degradation in water, atrazine is consistently detected in surface and groundwater, even in areas where its use as a pesticide has ceased. Vinclozolin is not considered to be environmentally persistent.

14. Are there any specific risks associated with water pollution or other environmental contamination from these pesticides? What can be done to protect people?

IARC is a research organization that generates and evaluates evidence related to the causes of cancer but does not make health or policy recommendations. IARC does not perform risk assessment for any specific exposure scenarios. However, the evaluations made by the *IARC Monographs* programme are often used as a basis for national and international policies, guidelines, and recommendations to minimize cancer risks.

[7] IARC (1999). Some chemicals that cause tumours of the kidney or urinary bladder in rodents and some other substances. *IARC Monogr Eval Carcinog Risks Hum*. 73:1–674. Available from: https://publications.iarc.who.int/91.

15. On the basis of these evaluations, what recommendations does IARC make?

IARC is a research organization that generates and evaluates evidence related to the causes of cancer but does not make health or policy recommendations. However, the evaluations made by the *IARC Monographs* programme are often used as a basis for national and international policies, guidelines, and recommendations to minimize cancer risks.

You can find more information on the *IARC Monographs* evaluation process here: https://monographs.iarc.who.int/wp-content/uploads/2018/07/QA_ENG.pdf.

16. What does the *IARC Monographs* classification mean in terms of risk?

The *IARC Monographs* classification indicates the strength of the evidence that a substance or agent can cause cancer. The *IARC Monographs* programme seeks to identify cancer hazards, meaning agents with the potential for the exposure to cause cancer. However, the classification does not indicate the level of cancer risk associated with exposure at different levels or in different scenarios. The cancer risk associated with substances or agents that are assigned the same classification may be very different, depending on factors such as the type and extent of exposure and the size of the effect of the agent at a given exposure level.

17. What are the different strength-of-evidence evaluation groups used by the *IARC Monographs*?

The strength-of-evidence groups that contribute to each evaluation are summarized in Table 2.

Table 2. Strength-of-evidence groups used by the *IARC Monographs*

Evidence of Cancer in Humans	Evidence of Cancer in Experimental Animals	Mechanistic Evidence	Evaluation
Sufficient			Carcinogenic (Group 1)
	Sufficient	Strong (exposed humans)	
Limited	Sufficient		Probably carcinogenic (Group 2A)
Limited		Strong	
	Sufficient	Strong (human cells or tissues)	
		Strong (mechanistic class)	
Limited			Possibly carcinogenic (Group 2B)
	Sufficient		
		Strong	
	Sufficient	Strong (does not operate in humans)	Not classifiable (Group 3)
All other situations not listed above			

18. What are the four different categories into which agents are classified by the *IARC Monographs*?

Group 1: The agent is *carcinogenic to humans*.

This category is used when there is *sufficient* evidence for cancer in humans. In other words, there is convincing evidence that the agent causes cancer in humans. The evaluation is usually based on the results of epidemiological studies showing the development of cancer in exposed humans. Agents can also be classified in Group 1 on the basis of *sufficient* evidence for cancer in experimental animals supported by *strong* evidence in exposed humans that the agent has mechanistic effects that are important for cancer development.

Group 2:

This category includes agents with a range of evidence regarding cancer in humans and experimental animals. At one extreme of the range are agents with positive but not conclusive evidence regarding cancer in humans. At the other extreme are agents for which evidence in humans is not available but for which there is *sufficient* evidence for cancer in experimental animals. There are two subcategories, which indicate different levels of evidence.

Group 2A: The agent is *probably carcinogenic to humans*.

This category is used in four different scenarios (which can occur simultaneously):

1. When there is *limited* evidence for cancer in humans and *sufficient* evidence for cancer in experimental animals ("*limited* evidence for cancer in humans" means that a positive association has been observed between exposure to the agent and cancer but that other explanations for the observations, technically termed "chance", "bias", or "confounding", could not be ruled out with reasonable confidence);
2. When there is *limited* evidence for cancer in humans and *strong* mechanistic evidence;
3. When there is *sufficient* evidence for cancer in experimental animals and *strong* mechanistic evidence in human primary cells or tissues;
4. When, based on mechanistic considerations, the agent belongs to a class of agents of which one or more is *probably carcinogenic to humans* (Group 2A) or *carcinogenic to humans* (Group 1).

These scenarios may also occur simultaneously within a Group 2A classification, as was seen for atrazine and alachlor.

Group 2B: The agent is *possibly carcinogenic to humans*.

This category is used when there is *limited* evidence for cancer in humans and less-than-*sufficient* evidence for cancer in experimental animals. It may also be used when the evidence regarding cancer in humans does not permit a conclusion to be drawn (referred to as *inadequate* evidence) but there is *sufficient* evidence for cancer in experimental animals. It can also be used when there is *strong* mechanistic evidence.

Group 3: The agent is *not classifiable as to its carcinogenicity to humans*.

This category is used most commonly when the evidence is *inadequate* regarding cancer in humans and *inadequate* or *limited* for cancer in experimental animals, and mechanistic evidence is less than *strong*. "*Limited* evidence for cancer in experimental animals" means that the available information suggests a carcinogenic effect but is not conclusive.

19. How was the evidence reviewed in the *IARC Monographs* evaluation?

During an *IARC Monographs* evaluation, experts critically review the scientific evidence according to strict criteria, which focus on determining the strength of the available evidence that the agent causes cancer. These criteria are described in the Preamble to the *IARC Monographs*, which is available on the *IARC Monographs* website: https://monographs.iarc.who.int/wp-content/uploads/2019/07/Preamble-2019.pdf.

The experts critically review four types of data:

- the situations in which people are exposed to the agent;
- epidemiological studies on cancer in humans exposed to the agent (scientific evidence regarding cancer in humans);
- experimental studies of cancer in laboratory animals treated with the agent (scientific evidence regarding cancer in experimental animals); and
- studies on how cancer develops in response to the agent (scientific evidence on carcinogen mechanisms).

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The International Agency for Research on Cancer (IARC) is part of the World Health Organization. Its mission is to coordinate and conduct research on the causes of human cancer, the mechanisms of carcinogenesis, and to develop scientific strategies for cancer control. The Agency is involved in both epidemiological and laboratory research and disseminates scientific information through publications, meetings, courses, and fellowships. If you wish your name to be removed from our press release emailing list, please write to terrassev@iarc.who.int

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