

Dietary acrylamide intake and risk of cancer: a systematic review

Carolina Capitão^a¹, Raquel Martins^a¹, Thorhallur I. Halldorsson^{b,c}, Tommaso Filippini^d, Marco Vinceti^{d,e}, Osvaldo Santos^af, Ana Virgolino^a, Federica Laguzzi^g

- a EnviHeB Lab, Instituto de Saúde Ambiental, Faculdade de Medicina, Universidade de Lisboa, Lisbon, Portugal b Centre for Fetal Programming, Department of Epidemiology Research, Copenhagen, Denmark c Unit for Nutrition Research, Faculty of Food Science and Nutrition, University of Iceland, Reykjavík, Iceland d Environmental, Genetic and Nutritional Epidemiology Research Center (CREAGEN), Department of Biomedical, Metabolic and Neural Sciences, University of Modena and Reggio Emilia, Modena, Italy
- e Department of Epidemiology, Boston University School of Public Health, Boston, Massachusetts f Unbreakable Idea Research, Cadaval, Portugal
 - g Unit of Cardiovascular and Nutritional Epidemiology, Institute of Environmental Medicine, Karolinska Institute, Stockholm, Sweden

- Corresponding author's contact: carolinacapitao@medicina.ulisboa.pt
- † These authors contributed equally

Background and objective

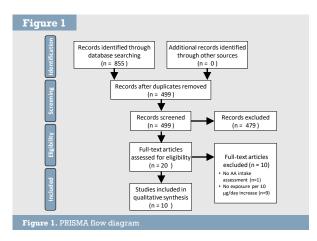
Acrylamide (AA) is a probable human carcinogen. Along with occupational exposure and smoking, diet is the main source of AA. A systematic review was conducted to explore the associations between dietary AA exposure and the risk of developing different cancer types, in prospective studies.

Methods

PubMed, Web of Science and Scopus databases were used to search for studies published in English, until 18 June 2020. Eligible studies included adults, assessment of dietary AA and cancer incidence, reported as hazard ratio (HR) for AA intake as a continuous variable (per 10 µg/day increase). Breast, endometrial, ovarian, and renal cell cancer studies were excluded, since it has been already the focus of recently published systematic reviews^{1,2}. Quality of papers was assessed using the NIH's Quality Assessment Tool for Observational Cohort and Cross-sectional Studies.

Results

In total, 855 records were identified, of which 10 studies met the inclusion criteria (1 cohort³ and 9 case-cohort studies⁴⁻¹²), providing a dataset of 526,151 participants, of which 13,629 developed cancer. The mean follow-up period was 14.5 (7.3 - 17.3) years. Paper selection process is presented in Figure 1.



All studies were considered to have fair quality because AA exposure was only measured once. Dietary AA exposure was assessed by food frequency questionnaire.

Multivariate-adjusted pooled HR per cancer site are presented in Figure 2. No clear association was found for almost any of the included cancer diagnosis, overall and for both sexes, per 10 µg/day increase of AA intake. As exceptions, in men, pooled HR showed an increased association between a consumption increment of 10 µg/day and the following cancers: cutaneous malignant melanoma (HR=1.13, 95%CI: 1.10-1.26), follicular lymphoma (HR=1.28, 95%CI: 1.03-1.61), and multiple myeloma (HR=1.14, 95%CI: 1.01-1.27). In women a decreased risk of lung cancer was noted (HR=0.82, 95%CI: 0.69-0.96).

Irain	Hogervorst et al. (2009a)	MF
Lung	Hogervorst et al. (2009b)	M
Head and neck	Schouten et al. (2009)	MF
Thyroid	Schouten et al. (2009)	MF
Esophageal	Hogervorst et al. (2008a)	MF
Gastric	Hogervorst et al. (2008a)	MF
Pancreatic	Hogervorst et al. (2008a)	MF
	Obón-Santacana et al. (2013)	MF
Colorectal	Hogervorst et al. (2008a)	MF
	Hogervorst et al. (2014)	М
	,	F
Bladder	Hogervorst et al. (2008b)	MF
Prostate	Hogervorst et al. (2008b)	Μ
Adv. prostate	Perloy et al. (2018)	М
Melanoma	Lipunova et al. (2016)	M
CLL	Bongers et al. (2012)	M
DLCL	Bongers et al. (2012)	М
MM	Bongers et al. (2012)	F
	D (2012)	F
FL	Bongers et al. (2012)	M
WMI	Bongers et al. (2012)	M
MCL	Bongers et al. (2012)	M
T-cell	Bongers et al. (2012)	М
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Figure 2. Association between dietary AA intake (continuous) and cancer incidence, by cancer site. MF, male and female patients; M, only male patients; F, only female patients; CLL, chronic lymphocytic leukaemia; DLCL, diffuse large cell lymphoma; MM, multiple myeloma; FL, follicular lymphoma; WMI, Waldenstrom macroglobulinemia and immunocytoma; MCL, mantle cell lymphoma; T-cell, T-cell lymphoma.

Conclusions

This systematic review suggests that dietary AA exposure may increase the risk of cutaneous malignant melanoma, follicular lymphoma and multiple myeloma in men only. More studies are needed to confirm the effect of dietary AA in the development of each cancer type, to ultimately improve current knowledge regarding the overall risks associated to the current exposure of the population to this substance.

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