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Review

Risks of dietary acrylamide exposure: A systematic review

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ABSTRACT

Acrylamide (AA) is a probable human carcinogen found in carbohydrate-rich foods that have been heated to high temperatures. AA dietary exposure has been associated to development of health problems. We perform a systematic review to elucidate the association of dietary AA exposure and human health problems. Articles were screened by reading titles and abstracts before the full text of eligible articles was read ($\kappa = 0.824$). Data were harvested by two reviewers and checked by a third. Forty-one articles were analyzed and assessment of dietary exposure proved to be far from uniform and suffered from limitations that possibly impact on the validity of outcomes with relation to human health. Risk assessment of dietary acrylamide exposure is in need of high quality methods for evaluating dietary exposure and validated acrylamide content databases.

Contents

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1. Introduction

Acrylamide (AA) is a colourless, odourless, crystalline solid with low molecular weight that is formed by hydration of acrylonitrile, with a melting point of 84.5 °C. It is a biodegradable compound that is soluble in water, acetone and ethanol and which exhibits high mobility in soil and groundwater (McCollister, Oyen, & Rowe, 1964; Smith, Prues, & Oehme, 1996). It has been used in industry since the 1950s as an intermediate step in the production of polyacrylamides (Hogervorst et al., 2010), but currently its primary application is for electrophoresis and chromatography in laboratory research (Friedman, 2003).

In 2002, AA was found in certain foods that had been subjected to high temperatures (Stadler, Blank, Varga, et al., 2002), having been formed at concentrations in the order of μg/kg during processes involving temperatures >120 °C, such as cooking, frying, toasting, roasting or baking of foods that are rich in carbohydrates (Tareke, Rydberg, Karlsson, Eriksson, & Törnqvist, 2002). This is primarily the result of the Maillard reaction between the amino acid asparagine and sugars such as glucose and fructose (Mottram et al., 2002; Stadler et al., 2002; Zyzak et al., 2003). Foods that are rich in these two precursors and, as a result, become major sources of AA when cooked, are primarily derived from products of vegetable origin, such as potato, cereals, peanuts, lentils and asparagus, but apparently not from products of animal origin (Friedman & Levin, 2008).

Acrylamide and its metabolite glycidamide (GA) are conjugated with glutathione and eliminated as by-products of mercapturic acid in urine, which is the primary route of excretion of AA metabolites in humans (Boettcher, Schettgen, Kütting, Pischetsrieder, & Angerer, 2005; Fuhr, Boettcher, Kinzig-Schippers, et al., 2006; Hashimoto & Aldridge, 1970). Both compounds are reactive and form adducts with proteins. Mercapturic acid in urine and adducts of haemoglobin have both been used as biomarkers to estimate AA intake in research (Bergmark, Calleman, He, & Costa, 1993; Boettcher & Angerer, 2005; Kommission Human-Biomonitoring des Umweltbundesamtes, 2008).

In 2002, the Swedish National Food Administration alerted the world to the health risks of AA exposure resulting from eating fried and baked foods, based on analyses that detected foods containing up to 500 times the levels permitted in potable water by the WHO (Lofstedt, 2003). Previous studies have demonstrated neurological effects in humans exposed to environmental AA (Calleman, Wu, He, et al., 1994) and experimental animals exposed to AA administered orally suffered carcinogenic effects such as tumours of mammary glands in female rats, testicular tumours in male rats, and increased rates of tumours of the thyroid gland, central nervous system, uterus, clitoral gland and oral tissues (Friedman, Dulak, & Stedham, 1995; Johnson, Gorzinski, Bodner, et al., 1986). However, reproducibility in humans has so far been inconsistent, and the majority of available data on humans comes from prospective population-based epidemiological studies, which have primarily measured AA consumption on the basis of food frequency questionnaires (FFQ) and conversion tables, although some have used the formation of adducts or both methods together. (Hogervorst et al., 2010; Pelucchi, La Vecchia, Bosetti, Boyle, & Boffetta, 2011).

In view of the above, the objective of this study is to conduct a systematic review of the literature on methods for assessing dietary AA exposure and its effects on health in humans.

2. Materials and methods

2.1. Search strategy

Two independent reviewers (B.P.R. and A.M.V.) conducted searches of electronic databases (PubMed, Scopus, Scielo and ISI Web of Knowledge) using publication dates from 2000 to 2013 and humans as limits, with the following keywords and combinations: (Acrylamides) OR Acrylamide AND (((Food) OR Diet) OR Diets) OR Cooking AND (Acrylamides) OR Acrylamide AND Intake AND "Risk". After we selected the manuscripts of interest, we conduct an active search for possible articles that did not appear in the original search by looking the references of the selected articles, as well as revision articles that were excluded from the revision process. In the active search we found more 15 articles that were included in the revision. The Kappa index was calculated for agreement between the two independent reviewers (B.P.R. and A.M.V.). Disagreements were assessed by a third reviewer (J.D.M).

2.2. Inclusion and exclusion criteria

Articles were considered eligible if they investigated either the risks of dietary exposure to human health; or if they assessed the outcomes dietary AA intake.

Articles written in languages other than English, German or Spanish were excluded. Additionally, articles describing animal experiments, literature reviews or bromatology studies, articles dealing with synthetic AA, opinion statements by authors or institutions, studies exclusively describing comparisons between adduct assay methods or comparisons of AA adduct levels between smokers and non-smokers, just evaluated consumption of AA, and articles solely related to polymorphisms or transplacental or environmental exposure were all excluded.

2.3. Data extraction

Data were extracted by 2 reviewers (B.P.R. and A.M.V.) and verified by a third reviewer (J.D.M) using a predefined spreadsheet designed by the authors. The data harvested included author names, publication dates, sample characteristics where applicable and the studies' objectives, methods and outcomes.

3. Results

3.1. Selection and classification of the studies reviewed

The system used to select publications for the review is illustrated in Fig. 1. The original search returned 1743 studies, 1717 of which were excluded as unrelated to the subject on the basis of their titles or abstracts. The degree of agreement between reviewers was rated at $\kappa = 0.824$ ($p < 0.001$). After that, with de active search in the references, we add 15 articles. The remaining 41 articles were read in their entirety.

The articles reviewed were summarized in two tables. In Table 1 we presented the main information about the studies including authors, year of publication, population, methods to assess AA intake and analysis, the main results with the risks for human health. In Table 2, we organized the articles in relation to the method used to assess dietary AA intake in relation to the type of study performed and the risks associated.

The bulk of the studies were conducted with samples from Sweden and Holland, although there were also studies conducted in Norway, the United States, Denmark, Belgium, the United Kingdom, Italy, Finland, Vietnam, China, and Europe.

3.2. Methods for determination of acrylamide intake

Studies evaluating the relative risks of dietary AA intake for health predominantly employed dietary surveys such as FFQ (Table 2) and the majority of these were longitudinal population