

**Any progress from
the consumers' perspective
from EFSA's Draft
Risk Assessment
on Acrylamide in Food?**

Background

1994: IARC's Risk Assessment (RA): AA is „probably carcinogenic“ to humans
→ „There is **sufficient evidence in experimental animals** for the **carcinogenicity** of acrylamide“ (p. 425)

2002: Acrylamide (AA) was found in food

5 RAs of International Bodies

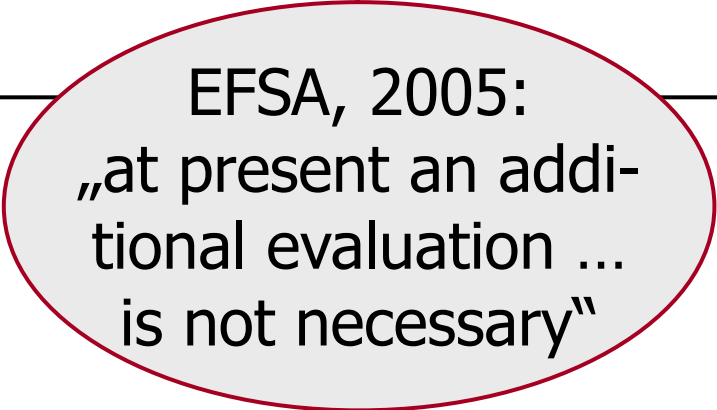
WHO	2002	Health implications of acrylamide in food. Joint FAO/WHO consultation, Geneva, Switzerland, 25 - 27 June 2002
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SCF	2002	Opinion of the Scientific Committee on Food on new findings regarding the presence of acrylamide in food, expressed on 3 July 2002
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JECFA	2005	WHO, 2006: Evaluation of certain food contaminants. Sixty-fourth report of the Joint FAO/WHO Expert Committee on Food Additives. WHO Technical Report Series: 930. Geneva
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JECFA	2010	WHO/FAO, 2011: Safety evaluation of certain contaminants in food. WHO Additives Series: 63. Geneva
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EFSA	2014	Draft Scientific Opinion on Acrylamide in Food
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EFSA, 2005:
„at present an additional evaluation ...
is not necessary“

Risk Assessments: Toxicology

WHO 2002 „AA has a carcinogenic potency in rats that is similar to that of other carcinogens in food, but the intake levels for AA are likely to be higher “ (p. 1)

SCF 2002 „AA is considered to be both genotoxic in vivo and carcinogenic in experimental animals“ (p. 12)

JECFA 2005 „a compound that is genotoxic and carcinogenic“ (p. 25)

JECFA 2010 „a compound that is genotoxic and carcinogenic“ (p. 139)

EFSA 2014 „AA is carcinogenic in multiple tissues in both male and female mice and rats“ (p. 4)

„carcinogenic“ in animals

Toxicology: Development

- ☞ 2002 – 2009: > 47 studies on rats and mice
- ☞ PubMed „acrylamide“ + „toxicology“: 145 publications since 2002

Toxicology: Conclusion

**No progress after 5 RAs
since 2002 (1994)!**

Risk Assessments: Epidemiology

WHO	2002	„Only limited human population data are available for acrylamide ... from occupational exposure“ (p. 1)
SCF	2002	„The potential carcinogenicity ... has not been sufficiently investigated in humans“ (p. 9)
JECFA	2005	„The available results from epidemiological studies that estimated oral exposure to acrylamide were not suitable for use in risk assessment“ (p. 15)
JECFA	2010	„the available epidemiological data were not suitable for a dose-response analysis“ (p. 138)
EFSA	2014	„The data from human studies were not adequate for dose-response assessment“ (p. 5)

**Data „not existing“
„not adequate“**

Epidemiology: Development

JECFA, 2010

study base
= 8'819'539

JECFA, 2005

study base
= 2'322'144

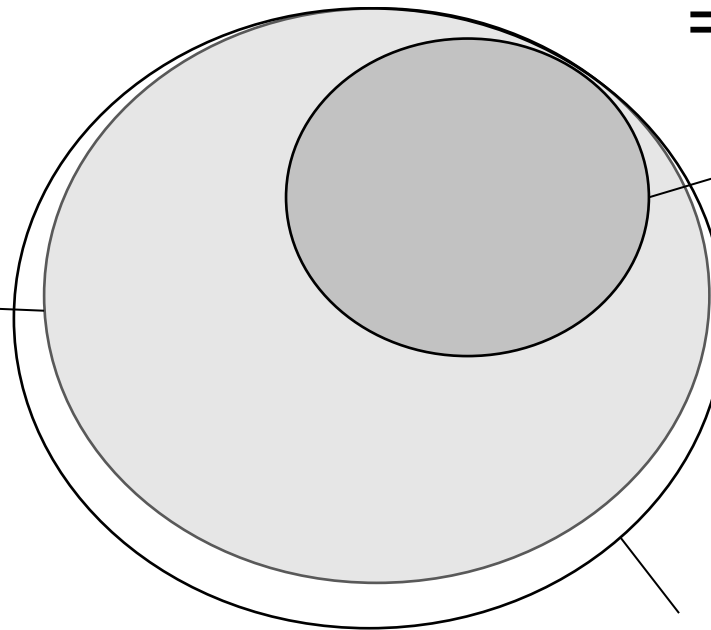
380 %
„not suitable“

100 %
„not suitable“

411 %
„not adequate“

EFSA, 2014

study base
= 9'532'512



Epidemiology: Conclusion

No progress since 2002!

Risk Characterization: MOE

EFSA, 2005: „an MOE of 10,000 or higher ... would be of low concern from a public health point of view“

JECFA,
2005

JECFA,
2010

EFSA,
2014

MOE

300

180

283-425

„concern“

„concern“

„concern“

Comment on the MOE

- ☞ The MOE of AA is less than 1 % of EFSA's low concern-level (10'000), even on the basis of a low calculated exposure. EFSA does not give any recommendation in this situation. Why not? Is this according to the sense of the MOE?
- ☞ EFSA states that the European monitoring data is insufficient. But EFSA takes this data to calculate an MOE which is higher than JECFA's MOE

General Aspects

- ➡ Advancement of Hazard Characterization is not possible as long as better methods are not available. EFSA did not comment on the fact that the research did not bring any progress.
- ➡ Why was a Risk Assessment performed in 2014 but not in 2005?

Conclusion: Summary

The main conclusion from the Draft is that nothing happened (no development) and nothing needs to happen (no recommendation) in spite of the fact that AA is a „concern“

→ No progress from the consumers' perspective!

Thank you!

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