Methods for assessing genotoxicity

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7. The evaluation of data quality for hazard/risk assessment includes the evaluation of reliability and relevance (Klimisch et al., 1997; OECD, 2005; ECHA, 2011; EFSA Scientific Committee, 2017c; EFSA Scientific Committee, 2021).

8. In the assessment of genotoxicity studies, the data quality has been evaluated based on reliability and relevance. Reliability has been assessed using a scoring system based on criteria published by Klimisch et al. (1997).

9. In a second step, the relevance (high, limited, low) of the study results was assessed based on reliability of the study and other aspects, e.g. genetic endpoint, purity of test substance, route of administration and status of validation of the assay.

10. Genotoxicity studies evaluated as of high or limited relevance have been considered in a WoE approach as described in Annex A. Genotoxicity studies evaluated as of low relevance have not been further considered in the assessment.

The different steps of the evaluation of reliability and relevance are described in Annex A.

Method for uncertainty analysis for genotoxicity

11. Details on how the uncertainty analysis was carried out as well as the results discussion can be found in Annex A.

Genotoxicity studies considered for this assessment

12. Publication of 88 in vitro and in vivo studies were retrieved from the literature search:

• in vitro and in vivo studies (15 publications) considered in the Scientific opinion on the risks to public health related to the presence of BPA in foodstuffs (EFSA CEF Panel, 2015) (Annex A).

13. In vitro and in vivo studies were grouped based on the genotoxicity endpoint investigated:

• gene mutations (e.g. bacterial reverse mutation assay);

• chromosomal damage (CA and micronucleus assays);

• DNA damage (comet assay).

14. These studies were summarized in synoptic tables (Annex A), evaluated for reliability and relevance and grouped into lines of evidence in a WoE approach (Annex A).

15. Studies not investigating classical genotoxicity endpoints (e.g. γH2AX, oxidative DNA damage, DNA binding, ROS generation) and studies in humans are considered in the Mode of Action MoA and as supportive evidence.