

From Internal exposure to PDI and comparison to ADI

Feedback from HBM4EU

Vol. 1, 2.6.5.2. Comparison with the CLP criteria regarding carcinogenicity, p. 311.

Vol. 1, 2.6.9. Summary of medical data and information, p. 473-474.

Vol. 1, 2.6.10.1. Toxicological end point for assessment of risk following long-term dietary exposure – ADI (acceptable daily intake), p.487.

Vol. 3, B.6.1. Absorption, distribution, metabolism and excretion in mammals.

Vol. 3, B.6.9.8. Literature data – medical data / treatment / poisoning / exposure, p. 645-707.

Recent papers on human biomonitoring (HBM) as well as volunteer studies (GLY) have not been included in **Vol. 3, B.6.9.8** and **Vol. 3, B.6.1**, respectively, and so are missing from the risk assessment (RA) (**Vol. 1, 2.6.10**). Also, the project HBM4EU has determined GLY in urine collected from four studies.

Results indicate widespread GLY exposure, also in children (concentrations >0.1 µg/L in 8-54% of the samples). High detection rates advocate use of internal exposure levels in RA by using reverse dosimetry calculations to predict daily intake (PDI) and then use PDI to verify RA based on TMDI. In addition, they support inclusion of HBM in epidemiological studies as indicated in **Vol. 1, 2.6.5.2, p. 311**.

Using urinary P95 values (reasonable worst case) we have calculated PDI values and compared to the ADI. Then the **proposed ADI of 0.1 mg/kg bw/d (Vol. 1, 2.6.10.1, p.487)** is not exceeded but **actual exposure is certainly not two orders of magnitude below ADI** (suggested in **Vol. 1, 2.6.9, p. 473**). Considering that workers were not specifically included in the HBM studies and that worker exposure is in general higher than the exposure of the 'general population', the margins for safety might even be smaller for that subpopulation.

It is suggested taking internal exposure information into account in the RA for consumers including children as well as workers and bystanders.

References, calculations and assumptions in the attached file.