Risk Characterisation

In this guide

59. EAs can act as both agonists and antagonists at noradrenaline, dopamine and serotonin receptors and produce peripheral effects (including uterotonic action and vasoconstriction) and CNS effects (including induction of hypothermia and emesis, and effects on the secretion of pituitary hormones).

60. EAs are not considered carcinogenic but have not been assessed by the IARC. The data on the genotoxic and mutagenic effects of EAs are somewhat limited and at times contradictory. EFSA (2012) considered the available genotoxicity studies to be insufficient, with the exception of those for ergotamine, which indicated that it did not cause bacterial or mammalian cell mutation. JECFA (2023) concluded that overall, naturally occurring EAs do not raise concerns for genotoxicity. The COT is of a similar view.

61. Exposure to EAs has been associate with pregnancy hindrance by interfering with egg implantation and embryotoxicity in rodents, negative effects on maternal blood supply to the placenta in ewes and possibly sirenomelia associated with *in utero* exposure in humans. EAs can negatively affect lactation due to their hormone mimicking activity, in particular on LH/FSH balance and prolactin levels (Della-Giustina eta al., 2003).

62. EFSA (2012) established an ARfD of 1 µg/kg bw and a TDI of 0.6 µg/kg bw per day for EAs. JECFA established a group TDI for the sum of total EAs in the diet at the same value as the group ARfD of 0.4 µg/kg bw. The COT considered the JECFA ARfD and TDI of 0.4 µg/kg bw more appropriate due to the more recent evaluation and its inclusion of human endpoints.

63. Using mycotoxin data from the TDS, mean and 97.5th percentile total estimated acute exposures were 52 - 57 ng/kg bw and 120 - 130 ng/kg bw respectively. Mean and 97.5th percentile total estimated chronic exposures were 31 - 35 ng/kg bw and 72 - 80 ng/kg bw respectively. All estimated exposures are below the respective ARfD and TDI established by JECFA and are therefore not a toxicological concern. The exposures here were also below any therapeutic doses that have shown adverse effects.

64. The food groups contributing most to the overall exposures were wholemeal and granary bread, white sliced bread and other bread. However, it should be noted that the dietary exposure estimates were based on a limited number of food groups and that data from ready-to-eat foods were scarce. A contribution to overall EAs exposure from other foods cannot therefore be excluded.

65. Total exposure was estimated by summing food consumption for each individual in the food survey and deriving distributions of consumption. The total mean, and 97.5th percentile were determined from an overall distribution of the consumption of any combination of the food categories included in the assessment, rather than by summation of the individual mean or 97.5th percentile

consumption values for each of the food categories. For food groups with non detects, exposure was calculated using the limit of detection (LOD)/LOQ as the upper bound and 0 as the lower bound occurrence value. This approach may produce a more conservative exposure estimate and increase the margin of safety (MoS).

66. The current assessment was based on consumption data from the NDNS for women of childbearing age and therefore may not be fully representative of maternal diet. The relatively small data set and limited number of EAs evaluated further add a level of uncertainty to the results.