

COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

Overarching statement on the potential risks from contaminants in the diet of infants aged 0 to 12 months and children aged 1 to 5 years

Background

1. The Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) was asked to review the risk of toxicity of chemicals in the diets of infants and young children aged 0-5 years, in support of a review by the Scientific Advisory Committee on Nutrition (SACN) of Government recommendations on complementary and young child feeding. The reviews will identify new evidence that has emerged since the Government's recommendations were formulated and will appraise that evidence to determine whether the advice should be revised. The recommendations cover diet from birth to five years of age.

2. SACN is examining the nutritional basis of the advice and a first report covering infants aged 0 to 12 months has been published in 2018¹. A report considering the evidence on feeding young children aged 12 to 60 months is currently underway. SACN has asked that evidence on possible adverse effects of the diet should be considered by other advisory committees with relevant expertise.

3. The COT identified a number of dietary chemicals in 2015², which might pose a risk to infants and young children and for which advice might be needed. The following statement discusses the conclusions of the COT regarding a number of these chemicals. Chemicals identified for review and not included in this statement have been or will be subject to a full review or will be published in a subsequent addendum to the overarching statement at a later date. The remaining chemicals are listed in Annex 1.

4. The following reviews provide a brief overview of the chemical's characteristics but focus mainly on the exposure assessment (where applicable) and the risk characterisation and conclusions, for both infants and young children.

General information

5. Unless indicated otherwise, the sources of general background information were the most recent assessments by the COT or other risk assessment bodies, such as the European Food Safety Authority (EFSA), the Scientific Committee on Food (SCF), or the Expert Group on Vitamins and Minerals (EMV).

¹ <https://www.gov.uk/government/publications/feeding-in-the-first-year-of-life-sacn-report>

² <https://cot.food.gov.uk/sites/default/files/TOX2015-32%20Feeding%20Review%20Scoping%20Paper.pdf>

6. Exposure assessments are based on the most recent occurrence data available from food surveys conducted by the Food Standards Agency (FSA). For chemicals with no available in-house data, the exposure assessment and risk characterisation have been drawn from EFSA opinions, with emphasis on UK data.

7. Consumption data (on a body weight basis) for the estimated dietary exposure were from the Diet and Nutrition Survey of Infants and Young Children (DNSIYC) (DH, 2013) and from years 1-6 of the National Diet and Nutrition Survey (NDNS) (Bates et al., 2012 & 2014). Estimates of consumption of breastmilk and infant formula vary; in this statement average and high daily intake of 800 mL and 1200 mL, respectively, were used. This is in line with the approach taken by EFSA. Occurrence data in breastmilk were taken from the literature, preferably from the UK, where applicable.

8. Where possible, estimated exposures to chemicals were compared to health based guidance values (HBGVs) or (safe) upper limits (UL) established by the COT or other risk assessment bodies, with preference given to EFSA.

Assessment

Alcohol

9. Alcohol is widely consumed in the UK population; levels of alcohol in breastmilk are close to those in the mother's blood stream³. The government therefore advises that breastfeeding women should not drink more than 1 or 2 units of alcohol once or twice a week.

10. In line with the 2012 statement, the COT sees no reason to change the current advice to government regarding alcohol and breastfeeding, which it confirms.

11. As children aged 0 to 5 years would not be consuming alcohol directly, the current statement does not require any further assessment of alcohol in this age group.

12. The full COT statement (2012) can be found here:

<https://cot.food.gov.uk/sites/default/files/cot/cotstatementoverarch201203.pdf>

Caffeine

13. In 2008, the FSA advised pregnant women, based on a COT evaluation⁴, to consume not more than 200 mg per day of caffeine and provided guidance on how to achieve such intakes for different foods and beverages. In addition, the current government advice⁵ for pregnant and breastfeeding women is "to restrict their caffeine intake to less than 200 mg a day" and "to avoid energy drinks, which can be very high in caffeine".

³ <http://www.drinkaware.co.uk/alcohol-and-you/family/alcohol-and-breastfeeding>

⁴ <https://cot.food.gov.uk/sites/default/files/cot/cotstatementcaffeine200804.pdf>

⁵ <https://www.nhs.uk/common-health-questions/pregnancy/should-i-limit-caffeine-during-pregnancy/>

14. The COT previously concluded that breastfed infants can be exposed to caffeine through breastmilk. However, the scientific evidence does not demonstrate a health risk for infants from the levels of caffeine to which they would be exposed by this route. COT noted in their 2012 statement, that the basis for the current government advice to breastfeeding mothers on caffeine consumption was extrapolated from data on pregnant woman and the data available at the time did not allow for refinement.

15. The available information does not provide a basis to refine the current advice to Government regarding caffeine consumption by breastfeeding women, which the Committee confirms. As children aged 1 to 5 years would not be expected to be consuming high-caffeine beverages, the COT concluded that no further assessment of caffeine for this age group is required.

16. The full COT statement (2012) can be found here:

<https://cot.food.gov.uk/sites/default/files/cot/cotstatementoverarch201203.pdf>

Food Additives

17. Under EU law (Regulation (EU) No. 1169/2011⁶), manufacturers must provide information about any additives used in the foods they produce. Once the additive has been assessed for safety and approved it is allocated an E number and can be used in the UK and the rest of the EU.

18. The additives regulation applies to all foods produced, including foods specifically for infants and children. Therefore, the COT deemed it not necessary to assess food additives again in these age groups.

19. Previous evaluations for some food additives can be found here:

<https://cot.food.gov.uk/sites/default/files/cot/cotstatementadditives.pdf>

<https://www.food.gov.uk/safety-hygiene/food-additives>

<https://cot.food.gov.uk/sites/default/files/cot/colpreschil.pdf>

Legacy Chemicals

20. A number of chemicals, which were banned during the 1980s and 1990s, are still present in the environment and food chain today, because of their biopersistence. Although they are persistent in the environment, their levels have decreased since they ceased to be used.

⁶ On the provision of food information to the consumer (FIC)

<https://eur-lex.europa.eu/legal-content/EN/TXT/PDF/?uri=CELEX:32011R1169&from=EN>

<http://www.legislation.gov.uk/ukxi/2014/1855/contents/made>

Specific provisions on the labelling of food additives sold as such to the manufacturer or to the final consumer are contained in [Regulation \(EC\) No. 1333/2008](#).

21. Many of these legacy chemicals are on the list of EFSA's call for continuous collection of data⁷; the data are made publicly available through summary reports, the latest on contaminant occurrence data was from 2016⁸. The last European Union report on pesticide residues in food, including those now banned but designated persistent organic pollutants (POPs), was from 2015.

22. Results of the 2015 report showed that 97.2% of the samples analysed did not exceed the maximum residue levels (MRLs) permitted by EU legislation; POPs were the most frequently found at concentrations equal to or greater than the LOQ. DDT and hexachlorobenzene were the most frequently reported POPs, however levels have decreased since the 2012 report.

23. The COT assessed endosulfan, pentachlorobenzene (PeCB) and chlordecone in 2013 and in the absence of any more recent data or information and given the nature and use of these chemicals, the COT decided to refer to its previous statement for the current evaluation.

24. In brief, endosulfan has not been authorised as a pesticide in the European Union since 2005 and significant residues in food are not expected. No data on PeCB and chlordecone in food have been found. Even if both had been used previously in the UK, exposures would be expected to be decreasing, as they are no longer approved for use. Exposure is primarily from environmental contamination.

25. As the levels for legacy chemicals are expected to further decline, the COT confirmed the conclusions of its previous assessments, that there is no indication of concern for human health from the presence of these chemicals in the diet of infants and young children.

26. The 2015 European Union report can be found here:

<https://efsa.onlinelibrary.wiley.com/doi/pdf/10.2903/j.efsa.2017.4791>

27. The full COT overarching statement (2012) can be found here:

<https://cot.food.gov.uk/sites/default/files/cot/cotstatementoverarch201203.pdf>

28. The full COT statement (2013) on endosulfan, pentachlorobenzene and chlordecone can be found here:

<https://cot.food.gov.uk/sites/default/files/cot/tox201321.pdf>

Soya phytoestrogens

29. In the absence of any more recent data, the COT decided to refer to its previous statement on phytoestrogens and health (2003), soya phytoestrogens in the infant diet (2013) and effects of soya consumption on thyroid status (2014).

⁷ <https://www.efsa.europa.eu/en/consultations/call/180307>

⁸ <https://efsa.onlinelibrary.wiley.com/doi/pdf/10.2903/sp.efsa.2017.EN-1217>

30. In brief, phytoestrogens are naturally produced chemicals of plant origin, notably in soya. They are structurally similar to oestradiol and have been shown to influence biological processes through their ability to bind to estrogen receptors (ER) and interfere with natural hormonal responses in animals and humans.

31. Based on the data available at the time, the COT concluded that it was not possible to determine a dose-response relationship, nor to identify other risk factors with the exception of iodine deficiency. The COT therefore concluded that individuals with hypothyroidism would still be considered a subgroup of the general population and that there was a potential concern for their health. The COT however recommended that this population subgroup, as well as general practitioners and endocrinologists, should be made aware of the potential risk of an exacerbation of this condition from increased consumption of soya.

32. The main toxicological concern for infants arises from the oestrogen-like activity and the potential disruption of development and of the reproductive system. Other possible adverse effects include disruption of the immune and thyroid function. Due to limitations in the available data, it is not possible to establish HBGVs for soya isoflavones in infants.

33. The few critical epidemiological studies available do not suggest important impacts of soya-based formula on later reproductive health in humans. However, animal studies suggest some developmental and reproductive changes can be induced. Thus, there is some uncertainty about the safety of soya-based formula. The COT previously concluded that there is no substantive medical need for soya based infant formula, nor health benefits and they should therefore be used only in exceptional circumstances. The current government advice states soya formula to be suitable from > 6 months of age but only under medical supervision⁹.

34. Due to the lack of any new UK data, the COT based its conclusion on its previous evaluation, that there is no scientific basis for a change in the current advice to government.

35. The full COT statements can be found here:

<https://cot.food.gov.uk/sites/default/files/cot/phytoreport0503.pdf>

<https://cot.food.gov.uk/sites/default/files/cot/cotstaphytos.pdf>

https://cot.food.gov.uk/sites/default/files/TOX2014-41_0.pdf

Vitamin A

36. In 2017, the COT published an update to their 2013 statement on vitamin A which included updated exposures for children aged 1-5 years. No TULs could be established for the ages 12-60 months on the basis of the currently available data. Comparisons were therefore made with a conservative TUL based on teratogenic effects in adults.

⁹ <https://www.nhs.uk/conditions/pregnancy-and-baby/types-of-infant-formula/#soya-formula>

37. High-level consumers are approaching or exceeding levels of vitamin A reported in the literature as causing toxicity and the possibility of adverse effects from these levels cannot be excluded. However, if effects did occur they would only be in a small proportion of consumers. Though the data on liver consumption are limited, frequent consumption could be a cause for concern. The COT concluded in 2017 that the current Government recommendations that infants over six months old should not have more than one portion of liver per week is appropriate and found no scientific basis for a change in the current advice to government.

38. The addendum can be found here:

<https://cot.food.gov.uk/sites/default/files/statementaddendumvitamina.pdf>

Trans fatty acids

39. SACN are undertaking a review on saturated fat¹⁰, which will also include trend data on the intakes of trans fatty acids. No assessment of trans fatty acids by COT is currently required.

40. Public Health England (PHE) has provided the following information to be included in the COTs overarching statement: “While trans fats are associated with risk of heart disease, UK consumption is less than the recommended maximum intake. However, many still eat more saturated fat than recommended, which can lead to higher blood cholesterol and heart disease. Until all responses have been received, current advice (by PHE) is to consume no more than 10% of calories each day from saturated fat.”

Perchlorate

41. The data collected by the FSA on perchlorate has been submitted to, and is part of, the evaluation performed by EFSA. The COT therefore did not consider it necessary to undertake a full risk assessment itself. Thus, the following paragraphs provide an overview and assessment of EFSA’s evaluations in 2014 and 2017.

42. Perchlorate is a chemical contaminant which is released into the environment from both natural and anthropogenic sources. Perchlorate is further formed during the degradation of sodium hypochlorite, which is used for the disinfection of water and can contaminate the water supply. Water, soil and fertiliser are considered the most likely sources for perchlorate contamination of food. Perchlorate has been reported in a wide range of foods, including vegetables, fruit, milk and dairy products, juice, beer, wine and bottled water.

43. The main adverse effects of perchlorate are on the thyroid. It competitively inhibits the uptake of iodine via the sodium-iodide symporter (NIS) in humans and rodents and therefore can cause disruption of thyroid hormone synthesis and consequently may lead to the development of hypothyroid effects. In humans, severe iodine deficiency can lead to hypothyroidism; mild to moderate iodine deficiency can lead to the development of toxic multinodular goitre, which can subsequently result in hypothyroidism.

¹⁰ <https://www.gov.uk/government/consultations/saturated-fats-and-health-draft-sacn-report>

44. EFSA concluded in their evaluation in 2012, that prolonged 50% inhibition of thyroid iodine uptake by perchlorate may lead to goitre and multinodular goitre, even if short term exposure does not alter the thyroid function test. Therefore, using the BMDL₀₅ of 1.2 µg/kg bw from human dose-response data as reference point and applying an uncertainty factor (UF) of 4 to allow for inter-human differences in toxicokinetics, EFSA established a TDI of 0.3 µg/kg bw. EFSA considered a 5% inhibition of iodine uptake not to lead to adverse effects in any subgroup of the population and therefore did not apply any further UF for intraspecies differences in toxicodynamics.

45. No data are available on the acute toxicity of perchlorate in humans; data from rodent toxicological studies are of limited use for extrapolation to humans due to differences in thyroid hormone physiology. There has been no reported evidence of adverse effects following treatment with a single dose of potassium perchlorate at 10 mg perchlorate ion/kg bw (assuming a 70 kg adult) for diagnostic purposes.

46. Although acute effects in fetuses and infants have been suggested, EFSA concluded that an acute reference dose (ARfD) was not necessary on the basis that a single day acute exposure to perchlorate at concentrations found in food and drinking water is unlikely to cause an adverse effect in either healthy humans or more vulnerable groups. In fetuses, limitations in the reserve capacity are mitigated by the maternal supply of thyroid hormones. Neonates on the other hand rely on their own hormone synthesis and thus could be considered a more vulnerable population subgroup. However, iodine uptake from the diet can vary significantly from day to day and the thyroid system has a well-developed homeostatic mechanism to take account of this. In addition, controlled human studies showed that thyroid uptake was completely restored within 24 hours of the end of the exposure period to perchlorate. While the stores of iodine will generally be lower in individuals with mild to moderate iodine deficiency, the thyroidal iodine stores are considered to be sufficient for a one-day need. However, if the iodine inhibition continues, the situation could become critical in breast-fed infants and young children, within a week or two, especially in individuals with mild to moderate iodine deficiency. Therefore, EFSA included a short-term exposure assessment, to take into account possible adverse effects in vulnerable groups, if exposed to relatively high levels of perchlorate for a short period (two to three weeks).

47. For the total of European data, the upper bound (UB) mean and 95th percentile estimated short-term and chronic exposures exceeded the TDI of 0.3 µg/kg bw in all age groups. For UK data only, the UB mean and 95th percentile estimated short-term exposure exceed the TDI in all age groups. UB chronic estimated exposures exceed the TDI for infants and toddlers but are below the TDI in other children. Both the chronic and short-term exposures to perchlorate are therefore of potential concern, particularly for high consumers with mild to moderate iodine deficiency and/or low iodine intake.

48. No breast milk data for perchlorate were available for the UK or Europe. Based on data from the United States, the estimated exposures for breastfed infants exceeded the TDI for both average and high-level consumption of breastmilk. This could possibly be of concern for breastfed infants of mothers with low iodine intake,

however the relevance of the estimated exposures for Europe and the UK are uncertain.

49. Overall, the COT agreed with EFSA's approach and the establishment of the HBGVs. However, EFSA themselves considered the use of the lowest BMDL₀₅ measured in a human volunteer study conservative in establishing the TDI and furthermore noted that there is a degree of uncertainty on how long thyroid iodine uptake could be inhibited before the development of adverse effects. However, the COT noted that the BMDL used to establish the TDI was based on healthy individuals. The COT also noted that EFSA established an ARfD for chlorate due to the induction of methemoglobinemia. Members questioned whether the possibility of acute methaemoglobin formation by perchlorate should be considered, based on read across from chlorate, since the TDI for chlorate was based on read across from perchlorate, suggesting a similarity in the effects of the two compounds.

50. In agreement with EFSA, the COT concluded that while there are considerable uncertainties in the assessment, the chronic and short term estimated exposures for all age groups, are of potential concern, particularly in the case of a mild to moderate iodine deficiency.

51. The full EFSA evaluations can be found here:

<https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2014.3869>

<https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2017.5043>

Chlorate

52. The data collected by the FSA on chlorate has been submitted to, and forms part of, the evaluation performed by EFSA in 2015. Whilst FSA and the Expert Committee on Pesticide Residues in Food (PRiF) have undertaken further data collection, also to inform the discussion on possible future MRLs under the pesticide legislation, the data are unlikely to change the (UK) exposure assessment undertaken by EFSA or conclusions drawn therefrom. The COT therefore did not consider it necessary to undertake a full risk assessment itself. Thus, the following paragraphs provide an overview and assessment of the EFSA opinion on chlorate.

53. Chlorate is no longer permitted as a pesticide in the European Union (EU). Due to the lack of a maximum residue level (MRL), the default MRL of 0.01 mg/kg is applicable. No maximum level for chlorate in drinking water has been set by the EU, although the World Health Organisation (WHO) set a guideline level of 0.7 mg/L.

54. Chlorate can be formed as a by-product when using chlorine, chlorine dioxide or hypochlorite and residues in food have been shown to arise from the use of chlorinated water for food processing (for example washing) and the disinfection of surfaces and food processing equipment, although in many cases they may occur simply due to the presence of chlorate in potable water used for food production.

55. The primary targets of chlorate toxicity are the thyroid gland and haematological system and like perchlorate, chlorate acts as a competitive inhibitor of iodine uptake in the thyroid leading to chronic effects such as multinodular goitre,

especially in populations with mild to moderate iodine deficiency. Due to the lack of adequate epidemiological studies in humans, EFSA established a TDI of 3 µg/kg bw for chlorate based on a read across from perchlorate. Comparing the no observed adverse effect levels (NOAEL) and the lowest observed adverse effect levels (LOAEL) for thyroid follicular cell hypertrophy in rats, perchlorate is about 10 times more potent than chlorate. In vitro studies further showed perchlorate to be the more potent inhibitor of thyroid iodine transport. Hence, the TDI for perchlorate (0.3 µg/kg bw) was multiplied by a factor of 10 to account for the different potencies of chlorate and perchlorate in rats.

56. In contrast to perchlorate, EFSA found it appropriate to establish an ARfD for chlorate, based on the acute haematological and renal toxicity of chlorate in humans. EFSA identified the formation of methaemoglobin in a 12 week controlled clinical study as a critical acute effect of chlorate and established an ARfD of 36 µg/kg bw from the NOAEL of 36 µg/kg, which was the highest dose tested. No UF for more vulnerable individuals was applied in the establishment of the ARfD as EFSA concluded the difference between the critical NOAEL and the level in poisoning cases, without induction of methemoglobinemia, to be sufficiently large to cover individuals who are potentially more vulnerable. As with perchlorate, EFSA noted that a single acute exposure to chlorate at the concentrations found in food and drinking water would be unlikely to cause adverse effects on the thyroid, including in the more vulnerable population.

57. For the total of the European data, the TDI was exceeded for the UB 95th percentile estimated chronic exposure in all age groups; the UB mean estimated chronic exposure exceeded the TDI in infants and toddlers. The 95th percentile estimated chronic exposure for UK data only, exceeded the TDI in all age groups; all mean estimated chronic exposures were below the TDI, except for toddlers at the UB level. In all population groups exceeding the TDI, drinking water was the major contributor, comprising up to 40 to 60%.

58. Individuals with sufficient iodine intake are less likely to suffer adverse effects from exceedances of the TDI than fetuses, neonates and individuals with low iodine intake or individuals genetically predisposed to develop hypothyroidism. The chronic dietary exposure is therefore of potential concern for high consumers in these age groups with mild to moderate iodine deficiency.

59. No data for acute estimated exposures based only on UK data were available. The mean and 95th percentile estimated acute exposures using all European data in all age groups are below the ARfD. Single acute exposure to chlorate at levels found in food and drinking water are therefore unlikely to cause adverse effects, including in vulnerable individuals. However, if drinking water were to contain concentrations of 0.7 mg/L, as assumed in one of EFSA's extremely conservative additional scenarios, mean water consumption could lead to mean (infants) and 97.5th percentile (toddlers) estimated exposures similar to the ARfD, and high water consumption could lead to estimated exposures of up to three times the ARfD.

60. No data on concentrations of chlorate in human breastmilk were available. Some data were available on perchlorate concentrations in breastmilk from the United States and whilst it would be possible to extrapolate these data to chlorate concentrations based on the differences in potencies as reflected in their respective

TDIs, the limited toxicokinetic information would represent a major uncertainty. Using this approach, the exposure of infants to chlorate from breastmilk would be unlikely to be of toxicological concern but the relevance to the UK population cannot be determined.

61. While the COT agrees with the establishment of the HBGVs, they noted some uncertainties. No human studies on inhibition of iodine uptake by chlorate were available, the TDI by EFSA is therefore based on read across from perchlorate. The basis for the TDI of 0.3 µg/kg bw is human-dose response data, while the difference in potency is derived from animal data. EFSA therefore assumed the same potency difference of perchlorate and chlorate in humans and rats. Furthermore, different rat strains were used for tests of the two compounds, adding additional uncertainty.

62. An ARfD was established based on a NOAEL of 36 µg/kg bw per day in a human repeat dose study, the NOAEL being the highest dose tested, and it is unclear as to how much higher the dose would need to be before effects were seen. No UF was applied as the NOAEL was at least 300 times lower than the toxic concentration in a poisoning case without induction of methemoglobinemia. However, this difference was derived from a single poisoning case.

63. The COT agrees with the overall conclusion by EFSA. Chronic dietary exposure to chlorate is of potential concern for high consumers in all age groups, particularly to individuals with mild to moderate iodine deficiency. Drinking water was the major contributor, at up to 40 to 60%. Single acute exposures to chlorate at levels found in food and drinking water however, are unlikely to cause adverse effects, including in vulnerable individuals.

64. The full EFSA evaluation can be found here:

<https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2015.4135>

Furan

65. Furan and methylfurans (2-methylfuran, 3-methylfuran and 2,5-methylfuran) are volatile compounds that are formed in foods during thermal processing. Food characteristics, processing and cooking conditions, especially the preparation of the food at home, determines the final concentration of furan in foods as consumed. Furan can be found in a variety of foods, including coffee and canned and jarred goods.

66. In short term rodent studies (< 90 days), furan showed strong hepato- and nephrotoxicity; in long term studies, furan was associated with toxicity in the liver. Furan did not induce gene mutations in bacteria and results in mammalian cells in vitro were contradictory. Limited information is available on furan levels in humans. The studies available show a variety of inconsistencies and therefore do not allow for conclusions regarding the reported levels of blood and urinary furan and whether or not furan has an effect on the liver in exposed individuals.

67. The liver has been identified as the primary target for acute and short term (< 90 days) toxicity of methylfurans in rodents; 3-methylfuran also showed indications of nephrotoxicity after long term exposure. No information on the genotoxicity of 3-

methylfuran is available. 2-methylfuran and 2,5-dimethylfuran showed negative results in bacteria; some evidence however points to chromosome damage in mammalian cells in vitro.

68. The toxic potency of methylfurans was reported to be in the same order of magnitude as for furan.

69. EFSA (2017) found it not appropriate to establish a TDI because, whilst there was clear evidence of indirect mechanisms in the carcinogenic mode of action (MoA) of furan, there were some indications of a direct genotoxic mechanism. EFSA therefore used the margin of exposure (MOE) approach. Based on the available toxicity data and taking inter- and intraspecies variations into consideration, EFSA concluded that a MOE of 100 or greater was of low health concern for non-neoplastic effects. For substances that are both genotoxic and carcinogenic, EFSA concluded that a MOE of 10,000 or greater was of low health concern, if based on a BMDL₁₀ from an animal carcinogenicity study.

70. UK data from the FSA long-term surveillance programme (2014-2018) forms part of the 2017 EFSA opinion, however at the time of publication, the 2017 data were not yet available. The 2017 data have since been published in the final FSA report and have been used in the COT 2018 assessment forming part of this overarching statement. No data on breastmilk were available.

71. All MOEs for non-neoplastic effects of furan are greater than 100 and are therefore not of toxicological concern.

72. The mean and 97.5th percentile MOEs for neoplastic effects of furan for children ages 4 to 18 months and the 97.5th percentile MOEs for children aged 18 to 60 months, for both ready-to-eat meals and total exposure are less than 10,000. The MOEs at the 97.5th percentile in children aged 4 to 18 months are lower yet, with values of < 2500. These exposures are of potential toxicological concern. All other MOEs for neoplastic effects are greater than 10,000 and are therefore not of toxicological concern.

73. The 97.5th percentile MOEs for non-neoplastic effects of the sum of furan and the two methylfurans for children ages 4 to 18 month, for both ready-to-eat meals and total exposure, are at/below the MOE of 100. These combined exposures are therefore of potential toxicological concern.

74. The lack of occurrence data on 2,5-dimethylfuran and the lack of information regarding the contribution of 2-methylfuran and 3-methylfuran, add to the uncertainties around the summed exposure to furan and methylfurans and could therefore lead to an over- as well as underestimation of the risk.

75. There is a level of uncertainty concerning the carcinogenic MoA and whether or not furan is directly genotoxic. The Committee acknowledges that this is a worst-case assumption and that MOEs of less than 10,000 could potentially be of no concern.

76. The major contributor to the dietary exposure was ready-to-eat meals. Dietary exposure to furan of infants and young children in the UK is similar to that in other

European countries and therefore not dependent on particular aspects of UK dietary habits. There have been efforts to reduce concentrations of furan (and methylfurans) in food over recent years but the evidence so far is not sufficient to demonstrate whether there has been a decrease in dietary exposure. Therefore, efforts to reduce furan and methylfurans should continue, with respect to commercially produced food, and monitoring should be continued to allow for accurate risk assessments.

77. The full EFSA evaluation can be found here:

<https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2017.5005>

Chromium

78. Chromium (Cr) is a hard, highly lustrous metal that exists in various mineral forms and is present throughout the environment. The most prevalent natural ionic form of chromium is Cr(III). Some Cr(VI) is present in the environment, largely due to industrial activity. The general population is primarily exposed to chromium via food and drinking water. The IARC reviewed Cr(III) and Cr(VI) and their compounds. Cr(VI) and its compounds have been classified as human carcinogens that cause cancers of the lung, and paranasal sinuses after inhalation (IARC, 2012). However, the potency of the carcinogenic effect varies with the physicochemical properties of the compound. There is no consistent evidence to suggest that Cr(III) compounds cause cancer in humans at concentrations to which people are exposed in food or the wider environment.

79. Food is largely a reducing environment. EFSA (2014) consider the chromium in food to be entirely in the form of Cr(III) and established a TDI of 300 µg/kg bw. Drinking water, which is purified with oxidising agents, is a source of Cr(VI). Cr(VI) is largely reduced to Cr(III) in the acidic environment of the stomach (De Flora et al, 2016, Kirman et al 2016). EFSA (2014) concluded that the levels of Cr(VI) found in drinking water were safe for all average consumers but there might be a potential concern for 95th percentile consumers particularly in “Infants”, “Toddlers” and “Other children” groups, based on MOEs of <10,000. However, the CONTAM Panel concluded that the impact of the uncertainties on the risk assessment of exposure to Cr(VI) in drinking water was very large. This could have resulted in substantial over-estimation of risk. In the current assessment, therefore, only the intake of Cr(III) is considered.

80. Gastrointestinal absorption of Cr is low: The Agency for Toxic Substance and Disease Registry (ATSDR, 2012) estimates <1% for Cr(III) and 1 – 2% for Cr(VI). EFSA (2014) estimates 0.4 – 2.8% for Cr(III). WHO, (2013) estimates 2 – 8% for Cr(VI). Absorption depends largely on the solubility of the particular compound.

81. The range of concentrations in breastmilk were obtained from a search of published literature. No consumption data were available for exclusive breastfeeding in infants aged 0 to 6 months. Therefore, the default consumption values used by the COT in other evaluations of the infant diet of 800 and 1200 mL for average and high-level consumption have been used to estimate exposures to chromium from breastmilk.

82. Average- and high-level-consuming, exclusively breastfed, 0 to 6-month old infants had an intake of 0.11 to 0.87 and 0.21 to 1.3% of the EFSA TDI for Cr(III) respectively. Mean intakes of chromium for non-exclusively breast fed 4 to 18-month olds relative to the TDI were 0.026 to 0.60% and 97.5th percentile exposures were 0.05 to 1.0% of the TDI.

83. In 0 to 6-month olds, intakes of chromium from ready-to-feed formula were 0 to 0.14% of the TDI in average consumers, and 0 to 0.2% of the TDI in high level consumers. Mean and high-level exposure to chromium from infant formula reconstituted with water containing chromium up to 8 µg/L (the highest limit of detection (LOD)) were up to 0.53 and 0.83% of the TDI. Total mean intakes (excluding water) of chromium from infant formulae, commercial infant foods, and other foods, for 4 to 12-month olds were 0.11 and 0.43 % of the TDI and the 97.5th percentile intakes were 0.37 to 1.2% of the TDI.

84. Based on the Infant Metals Survey (FSA, 2016a), the ranges of total mean and 97.5th percentile intakes (excluding water) of chromium from infant formula, commercial infant foods and other foods were 0.17 to 0.37 and 0.32 to 0.74% of the TDI, respectively, for children aged 12 to 18 months.

85. Based on the TDS (FSA 2016b) the total mean and 97.5th percentile intakes of chromium from a combination of all food groups for children aged 12 to 18 months the estimated chromium intakes were. 0.47 to 1.1 and 1.1 to 1.8% of the TDI respectively. For children aged 18 months to 5 years, the mean and 97.5th percentile intakes were 0.60 to 1.2 and 1.1 to 2.0 % of the TDI respectively.

Soil, air and dust

86. The median and 90th percentile concentrations in 5,670 topsoil samples collected between 1978 and 1982 in England and Wales were 68 and 97 µg/g, respectively (Rawlins *et al.*, 2012). Harrison (1979) determined the levels of chromium in outside and domestic dust samples to be 11.8 ± 6.1 µg/g (Mean \pm SD, n = 4, range 5.0 – 20 µg/g). Data from 23 air sampling sites across the UK have been collected by Defra. For 2007 – 2016, the lowest and highest median values were 0.8 and 8.65 and lowest and highest 99th percentiles were 1.4 and 167 ng chromium/m³ across the sites.

87. Environmental exposure to Cr(III) was calculated to be at most 0.038, 0.15 and 0.036% of the EFSA TDI for dust, soil and air respectively.

88. Estimated dietary exposures for children aged 0 to < 12 months and 1 to < 5 years indicate that chromium intake, either from breastmilk or other foods is well below the TDI and is therefore considered not to be of toxicological concern.

89. The full EFSA evaluation can be found here:

<https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2014.3595>

Selenium

90. Selenium is a group VI metal with both metallic and non-metallic properties (EVM, 2003). It exists in four oxidation states (-2, 1, +2, +6) and forms compounds analogous to those formed by sulfur.

91. In food, selenium is mainly present in organic compounds, as L-selenomethionine and L-selenocysteine, with lower amounts in inorganic compounds, as selenate and selenite, and is an essential micronutrient to human health. It is present in a number of foodstuffs, notably nuts, offal, eggs and poultry, and mushrooms and in lower quantities in fruits and vegetables with the exception of members of Brassica genus (cabbage, cauliflower etc) which contain relatively high amounts of selenium (SCF, 2000; Kieniszek and Stanislaw, 2016).

92. Selenium compounds are readily absorbed in the small intestine. Selenium is widely distributed throughout the body and is found in breastmilk. It has also been reported to cross the placenta in animals. Selenium compounds are incorporated in selenoproteins, which play a role in a variety of biological functions including antioxidant defence, T-cell immunity, thyroid hormone metabolism, selenium homeostasis and transport, and skeletal and cardiac muscle metabolism.

93. Upon absorption, selenium compounds can bind to selenium binding proteins or, as a way of regulating selenium metabolism, form methylated metabolites in the liver. These compounds are excreted predominantly in the urine. Excretion of selenium can also occur to a smaller extent in the faeces or, for some volatile compounds, in the breath (EFSA, 2014; EVM, 2003).

94. Selenium deficiency interferes with the expression and function of selenoproteins. Although the clinical manifestations are poorly defined, signs and symptoms reported from epidemiological studies in populations with low selenium intake and patients receiving selenium-free total parenteral nutrition include skeletal myopathy, cardiomyopathy and muscle weakness. Selenium deficiency is also linked to the manifestation of the degenerative Keshan and Kashin-Beck disease (SCF, 2000; EFSA, 2014).

95. High exposure to selenium can lead to acute toxicity. This is characterised by hypersalivation, emesis and garlic aroma on the breath. Other signs and symptoms include severe vomiting and diarrhoea, hair loss, neurological disturbance and fatigue (EVM,2003).

96. Chronic toxicity, or selenosis, leads to hair and nail changes, skin lesions and clinical neurological effects such as peripheral hypoaesthesia, acroparasthaesia, pain and hyperreflexia; numbness, convulsions and paralysis may then develop (EVM, 2003).

97. The Scientific Committee on Food (SCF) established in 2000 an UL for selenium of 300 µg/day for adults, including pregnant and lactating women. This was based on a NOAEL of 850 µg/day for clinical selenosis (Yang et al., 1989) and application of an uncertainty factor of 3. The NOAEL was based on the absence of clinical signs in individuals with selenium levels below 1000 µg/L. In the absence of data to establish specific ULs for children, the SCF extrapolated the UL from adults to children based on reference body weights. The proposed UL values for children

and adolescents were 60, 90, 130, 200 and 250 µg/day for children aged 1-3, 4-6, 7-10, 11-14 and 15-17 years respectively.

98. In their evaluation, the EVM used the same data set as the SCF to derive an UL of 450 µg/day. They considered that there were discrepancies in NOAELs in the series of studies conducted by Yang et al. and therefore used the Lowest Observed Adverse Effect Level (LOAEL) of 900 µg/day and applied an uncertainty factor of 2 to extrapolate to a NOAEL. An uncertainty factor for inter-individual variation was not deemed necessary as they considered that the value was based on a population study.

99. Exposure estimates were calculated using consumption data from NDNS and concentrations of selenium measured in a FSA survey of metals and other elements in infant formula and foods. Mean dietary exposures to selenium for children aged 4 months to 5 years ranged from 7 µg/day (lowest lower-bound (LB)) to 46 µg/day (highest upper-bound (UB)). The corresponding 97.5th percentile exposures ranged from 20 to 76 µg/day.

100. Assuming a selenium concentration of 20.6 µg/L in mature breastmilk (Foster et al. 1996), estimated exposures for exclusively breastfed infants (0 to 6 months) for average and high-level consumption of breastmilk were 16 and 25 µg/day respectively.

101. The soil 90th percentile selenium concentration was reported to be 1.3 mg/kg (UKSO, 2017). This was used to estimate exposure from soil and also dust, given the absence of Se data specific to dust. The resulting exposures in infants and young children from soil and dust are at least two orders of magnitude below dietary exposures.

102. The ULs for children below 1 year of age were calculated on a body weight basis using the approach used by the SCF. These were 40 µg/day for children aged 4-12 months when considering dietary exposures. The extrapolated UL would be 26 µg/day for infants 0 to < 4 months and 34 µg/day for > 4 to < 6 months old infants.

103. Overall the COT concluded that estimated dietary exposures for children aged 0 to < 12 months and 1 to < 5 years were below the UL, either from breastmilk or other foods and are therefore unlikely to be of toxicological concern.

Zinc

104. Zinc is an essential trace element found in all plant and animal tissue. It plays a role in a wide range of biochemical and physiological functions; its key biochemical role is in the regulation of gene expression, including transcriptional and translational control/modulation. Zinc furthermore has catalytic/structural roles in enzymes and a regulatory role in signal transduction.

105. The first site of absorption of zinc is the stomach, the majority however is absorbed in the upper intestine. Gastrointestinal distress, vomiting and nausea are common effects of acute oral exposure to zinc. Excessive chronic high zinc intakes lead to biochemical and physiological effects, such as secondary copper deficiency which can result in severe neurological diseases, anaemia and bone abnormalities.

106. The SCF (2003; 2006; 2017) derived a tolerable upper intake level (UL) of 25 mg per day for adults based on a NOAEL of 50 mg per day for a wide range of indicators for copper status in epidemiological studies and the application of an UF of 2 to allow for the small number of subjects and relative short time period of exposure. In the absence of adequate data for children, the SCF chose to extrapolate from adults to children on a body weight basis, resulting in an UL of 7 mg per day for children age 1-3 years and 10 mg per day for children age 4-6 years. EVM and JECFA derived UL for adults only, while EFSA derived population reference intakes only.

107. The ULs derived by the SCF for children start at the age of 12 months and would therefore not be applicable to infants aged 4 to < 12 months. Using the same approach as SCF, the COT extrapolated an UL of 3.6 mg per day (based on a bodyweight of 9 kg for infants from DNSIYC) for infants aged 4 to < 12 months and an UL of 2.3 mg per day (based on a bodyweight of 5.9 kg for infants from DNSIYC) for infants aged 0 to < 4 months.

108. Exposure estimates were calculated using consumption data from NDNS and concentrations of zinc measured in a FSA survey of metals and other elements in infant formula and foods. For infants aged 0 to < 4, the mean and 97.5th percentile estimated exposures are at the UL of 2.3 mg per day and exceed the UL 1.6-fold, respectively. For infants aged 4 to < 12 months, the mean and 97.5th percentile estimated exposures exceed the UL of 3.6 mg per day marginally and approximately 2-fold, respectively. The mean estimated exposures for children aged 1 to < 4 years are below the UL of 7 mg per day set by SCF; the 97.5th percentile estimated exposures are at or marginally above the UL. Estimated mean and 97.5th percentile exposure for children aged 4 to < 5 years are below the UL of 10 mg per day set by SCF.

109. Assuming a zinc concentration of 3 mg/L (McCance and Widdowson, 2015) in breastmilk, estimated exposures for exclusively breastfed infants (0 to 6 months) are within or at the UL of 3.6 mg per day for infants.

110. The COT concluded, that overall, estimated dietary exposure for children aged 0 to < 12 months and 1 to < 5 years do not indicate excessive zinc intakes, either from breastmilk or other foods and are therefore unlikely to be of toxicological concern.

111. However, COT noted, that all HBGVs and UL for infants and children are derived from extrapolation from adults, based on epidemiological/clinical studies in adults. It is therefore difficult to identify a HBGV or UL that is applicable for all age groups of infants and children.

Conclusions

112. In the absence of any more recent information, the COT concluded that there is no basis to change its previous advice for alcohol, caffeine, soya phytoestrogens and vitamin A, which the Committee confirms.

113. Food additives are regulated under EU law and saturated fat, including trend data on intakes of trans fatty acids, are currently under assessment by SACN and are outside the remit of the COT. The levels for legacy chemicals are expected to further decline and the COT concluded, in line with the 2012 overarching statement, that there is no indication of concern about these for human health. The levels are furthermore regularly monitored and show a declining trend based on the 2015 EU report.

114. In the absence of any UK-specific data, COT assessed perchlorate and chlorate based on the evaluations by EFSA and while the COT identified a number of uncertainties in the evaluations, overall they agreed with EFSA's approach and the HBGVs established.

115. There is a level of uncertainty concerning the carcinogenic MoA of furan and whether it is directly genotoxic and the COT acknowledges that its assessment is based on worst case assumptions. There have been efforts to reduce concentrations of furan (and methylfurans) in food over recent years but the evidence so far is not sufficient to demonstrate whether there has been a decrease in dietary exposure. The exposures in this assessment are of potential toxicological concern and efforts to reduce furan and methylfurans should therefore continue.

116. Chromium is present in food and the environment largely as Cr(III). EFSA has established a TDI for Cr(III) of 300 µg/kg bw. Estimated dietary exposures for children aged 0 to < 12 months and 1 to < 5 years indicate chromium intake, either from breastmilk or other foods, well below the TDI and is therefore considered not to be of toxicological concern. Environmental exposure to Cr(III) from dust, soil and air was calculated to be at most 0.038, 0.15 and 0.036% of the EFSA TDI, respectively and is therefore considered not to be of toxicological concern.

117. Overall the COT concluded that estimated dietary exposures to selenium for children aged 4 to < 12 months and 1 to < 5 years were below the UL, either from breastmilk or other foods and are therefore unlikely to be of toxicological concern.

118. The COT concluded, that overall, estimated dietary exposures do not indicate excessive zinc intakes and are therefore unlikely to be of toxicological concern. However, the COT did note that all HBGVs and UL are derived from adults and it is therefore difficult to identify a HBGV or UL that is applicable to all age groups of infants and children.

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References

- ATSDR (Agency for Toxic Substance and Disease Registry 2012). Toxicological profile for chromium. US department for health and human services.
<https://www.atsdr.cdc.gov/toxprofiles/tp7.pdf>
- Bates B, Lennox A, Prentice A, Bates C, Page P, Nicholson S, Swan G (2014). National Diet and Nutrition Survey Results from Years 1, 2, 3 and 4 (combined) of the Rolling Programme (2008/2009 – 2011/2012)
https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/594360/NDNS_Y1_to_4_UK_report_executive_summary_revised_February_2017.pdf
- Bates B, Cox L, Nicholson S, Page P, Prentice A, Steer T Swan G (2016). National Diet and Nutrition Survey Results from Years 5 and 6 (combined) of the Rolling Programme (2012/2013 – 2013/2014)
https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/551352/NDNS_Y5_6_UK_Main_Text.pdf
- DH (Department of Health) (2013). Diet and Nutrition Survey of Infants and Young Children (DNSIYC), 2011. <http://transparency.dh.gov.uk/2013/03/13/dnsiyc-2011/>
- De Flora S, Camoirano A, Micale RT, La Maestra S, Savarino V, Zentilin P, Marabotto E, Suh M, Proctor DM (2016). Reduction of hexavalent chromium by fasted and fed human gastric fluid I. Chemical reduction and mitigation of mutagenicity. *Toxicology and Applied Pharmacology*, 306: 113 – 119.
- EFSA (2014). Scientific Opinion on Dietary Reference Values for zinc. *EFSA Journal*, 12(10): 3844.
<https://www.efsa.europa.eu/en/efsajournal/pub/3844>
- EFSA (2014) Scientific opinion on the risks to public health related to the presence of chromium in food and drinking water. *EFSA Journal*, 12(3): 3595.
<https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2014.3595>
- European Food Safety Authority (2014). Scientific Opinion on Dietary Reference Values for selenium. EFSA Panel on Dietetic Products, Nutrition and Allergies (NDA). *EFSA Journal*, 12(10):3846.
<https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2014.3846>
- EFSA (2017). Risks for public health related to the presence of furan and methylfurans in food. *EFSA Journal*, 15(10):5005.
<https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2017.5005>
- EVM (2003). Safe Upper Levels for Vitamins and Minerals.
<https://cot.food.gov.uk/sites/default/files/vitmin2003.pdf>
- Foster LH, Kondza B & Sumner S (1996). Selenium content of breast milk and infant formulae: an estimation of intakes in the UK. *Metal Ions in Biology and Medicine* 4, 505–507.

FSA (2016a) 'Survey of metals and other elements in infant foods' (to be published)

FSA (2016b) 'Metals and other elements in the 2014 Total Diet Study' (to be published)

FSA (2018). Survey of acrylamide and furan in UK retail products.

<https://www.food.gov.uk/print/pdf/node/757>

Harrison RM (1979). Toxic metals in street and household dust. Science of the total environment, 11(1): 89 – 97.

JECFA (). Zinc. WHO Food Additives Series 17.

<http://www.inchem.org/documents/jecfa/jecmono/v17je33.htm>

Kirman CR, Suh M, Hays SM, Gürleyuk, Gerads R, De Flora S, Parker W, Lin S, Haws LC, Harris MA, Proctor DM (2016). Reduction of hexavalent chromium by fasted and fed human gastric fluid II. Ex vivo gastric reduction modelling. Toxicology and Applied Pharmacology, 306: 120 – 133

Kieliszek M, Błazejak S (2016). Current Knowledge on the Importance of Selenium in Food for Living Organisms: A Review. Molecule, 21, 609

<https://www.mdpi.com/1420-3049/21/5/609>

Rawlins, BG.; McGrath, SP.; Scheib, AJ.; Breward, N.; Cave, M.; Lister, TR.; Ingham, M.; Gowing, C. and Carter, S. (2012) 'The Advanced Soil Geochemical Atlas of England and Wales'

<http://resources.bgs.ac.uk/ebooks/AdvancedSoilGeochemicalAtlasEbook/index.html#/1/>

SCF (2000). Opinion of the Scientific Committee on Food on the Tolerable Upper Intake Level of Selenium.

https://ec.europa.eu/food/sites/food/files/safety/docs/sci-com_scf_out80g_en.pdf

SCF (2003). Opinion of the Scientific Committee on Food on the Tolerable Intake Level of Zinc.

https://ec.europa.eu/food/sites/food/files/safety/docs/sci-com_scf_out177_en.pdf

SCF (2006). Tolerable Upper Intake Levels for Vitamins and Minerals.

http://www.efsa.europa.eu/sites/default/files/efsa_rep/blobserver_assets/ndatolerableuil.pdf

UK Soil Observatory (UKSO) (2017): NSI Topsoil Selenium

<http://www.ukso.org/nsi/Selenium.html>

Abbreviations

ADI	Acceptable daily intake
ARfD	Acute reference dose
ATSDR	Agency for Toxic Substance and Disease Registry
BMDL	Benchmark dose modelling
Bw	Body weight
COT	Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment
Cr	Chromium
DH	Department of Health
DNSIYC	Diet and Nutrition Survey of Infants and Young Children
EFSA	European Food Safety Authority
EMV	Expert Group on Vitamins and Minerals
ER	Estrogen receptor
EU	European Union
FSA	Food Standards Agency
HBGV	Health based guidance value
IARC	International Agency for Research on Cancer
JECFA	Joint FAO/WHO Expert Committee on Food Additives
LB	Lower bound
LOAEL	Lowest observed adverse effect level
LOD	Limit of detection
MoA	Mode of action
MOE	Margin of exposure
MRL	Maximum residue level
NDNS	National Diet and Nutrition Survey
NIS	Sodium-iodine symporter
NOAEL	No observed adverse effect level
PeCB	Pentachlorobenzene
PHE	Public Health England
POPs	Persistent organic pollutants
PRiF	Expert Committee on Pesticide Residues in Food
RfD	Reference dose

SACN	Scientific Advisory Committee on Nutrition
SCF	Scientific Committee on Food
TDI	Tolerable daily intake
UB	Upper bound
UF	Uncertainty factor
UKSO	UK Soil Observatory
UL	Upper limit
WHO	World Health Organisation

Bisphenol A

119. Bisphenol A is currently under re-evaluation by EFSA. The COT therefore decided to revisit their current advice following EFSA's updated evaluation.

Phthalates

120. The COT decided to assess phthalates in a separate statement after the upcoming evaluation by EFSA.

Dioxins and dioxin-like compounds

121. The COT has commented on the newest evaluation of dioxins and dioxin-like compounds by EFSA. The Committee's comments have been communicated to EFSA and the COT is awaiting publication of the "Report on the Information Session on the EFSA Opinion on PCDD/Fs and DL-PCBs in food and feed" held on the 13th of November 2018, prior to deciding if a full reevaluation of its current advice is required or if the COT's assessment can be covered in a later overarching statement.

Perfluorooctanesulfonic acid (PFOS) & Perfluorooctanoic acid (PFOA)

122. The COT has evaluated the information provided in EFSA's scientific opinion on PFOS and PFOA, published earlier in 2018, and will publish a statement later in 2019.

Monochloropropane diol (MCPD)

123. No in-house data is available for MCPD. The COT will therefore evaluate MCPD based on the most recent EFSA opinion, which includes UK data, and will be including its evaluation in a later overarching statement.

Tetrabromobisphenol (TBBPA)

124. TBBPA are currently under review by the COT. The most recent UK data are from 2004, however have not been included in the 2011 EFSA opinion. Based on the available in-house data and conclusion, the assessment will either be covered in a later overarching statement or published as a full review.

Sweeteners

125. No in-house data are available for sweeteners. The COT will therefore evaluate the main sweeteners (acesulfame K, aspartame, saccharin, sorbitol, sucralose, stevia, xylitol, NHS most commonly used) based on the most recent EFSA opinion and available literature and will be including its evaluation in a later overarching statement.

Mycotoxins

126. The remaining mycotoxins (diacetoxyscirpenol, nivalenol, aflatoxins, citrinin, ergot alkaloids, sterigmatocystin, zearalenone, cyclopiazonic acid, fumonisins, moniliformin, patulin, deoxynivalenol, fusarenon-x) are currently under evaluation by the COT. Based on the available in-house data and conclusions, the assessment will either be covered in a later overarching statement or published as a full review.

Polycyclic Aromatic Hydrocarbons (PAHs)

127. PAHs are currently under review by the COT. Based on the available in-house data and conclusion, the assessment will either be covered in a later overarching statement or published as a full review.

Hexachlorocyclohexane (HCH)

128. No in-house data are available for HCH; the COT is currently assessing the best approach to evaluating HCHs.