

Introduction and Background

In this guide

[In this guide](#)

1. [The effects of mercury on maternal health - Introduction and Background](#)
2. [The effects of mercury on maternal health - Previous evaluations](#)
3. [The effects of mercury on maternal health - Hazard Identification](#)
4. [The effects of mercury on maternal health - Toxicity](#)
5. [The effects of mercury on maternal health - Reproductive toxicology](#)
6. [The effects of mercury on maternal health - Pregnancy outcomes](#)
7. [The effects of mercury on maternal health - Effects on maternal health](#)
8. [The effects of mercury on maternal health - Biomarkers of mercury exposure](#)
9. [The effects of mercury on maternal health - Epigenetic alterations via mercury exposure](#)
10. [Studies published on the Seychelles and Faroe Islands cohorts since the 2018 COT statement](#)
11. [The effects of mercury on maternal health - Hazard Characterisation](#)
12. [The effects of mercury on maternal health - Exposure assessment](#)
13. [The effects of mercury on maternal health - Aggregate exposure](#)
14. [The effects of mercury on maternal health - Conclusions](#)
15. [The effects of mercury on maternal health - Questions for the Committee](#)
16. [The effects of mercury on maternal health - List of Abbreviations and Technical terms](#)
17. [The effects of mercury on maternal health - Search terms](#)
18. [The effects of mercury on maternal health - References](#)

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Introduction

1. The Scientific Advisory Committee on Nutrition (SACN) last considered maternal diet and nutrition in relation to offspring health in its reports on 'The influence of maternal, foetal and child nutrition on the development of chronic disease in later life' (SACN, 2011) and on 'Feeding in the first year of life' (SACN, 2018). In the latter report, the impact of breastfeeding on maternal health was also considered.
2. In 2019, SACN agreed to conduct a risk assessment on nutrition and maternal health focusing on maternal outcomes during pregnancy, childbirth and up to 24 months after delivery; this would include the effects of chemical contaminants and excess nutrients in the diet.
3. SACN agreed that, where appropriate, other expert Committees would be consulted and asked to complete relevant risk assessments e.g., in the area of food safety advice. This subject was initially discussed by COT during the horizon scanning item at the January 2020 meeting with a scoping paper being presented to the Committee in July 2020. This included background information on a provisional list of chemicals proposed by SACN. It was noted that the provisional list of chemicals was subject to change following discussion by COT who would be guiding the toxicological risk assessment process: candidate chemicals or chemical classes can be added or removed as the COT considered appropriate. The list was brought back to the COT with additional information in September 2020. Following a discussion at the COT meeting in September 2020, it was agreed that papers on a number of components should be prioritised and to this end, papers on iodine, vitamin D and dietary supplements have been or will be presented to the Committee. The remaining list of compounds were to be triaged on the basis of toxicity and exposure.
4. Following discussion of the first prioritisation paper on substances to be considered for risk assessment by the COT, the Committee decided that each of the heavy metals (lead, mercury, cadmium, and arsenic) should be considered in separate papers. The following paper discusses the risks posed to maternal health by mercury in the diet and the environment.

Background

5. Mercury (Hg) is a d-block element in the periodic table and is the only metallic element known to be liquid at standard temperature and pressure. It is also known as quicksilver and was formerly named hydrargyrum. It is a group 12 metal, with atomic number 80, a relative atomic mass of 200.592 and its most

abundant isotope is ^{202}Hg with atomic mass 201.970 (Laeter et al, 2003). Mercury occurs naturally in the earth's crust at an abundance of 0.0000085%, chiefly as mercury (II) sulfide, also known as cinnabar, cinnabarite or mercurblende (Haynes, Lide and Bruno, 2016). Mercury has been used in thermometers, barometers, manometers, sphygmomanometers, float valves, mercury switches, mercury relays, fluorescent lamps, and other devices. However, the element's toxicity has led to phasing out of such mercury containing instruments. It remains in use for scientific research purposes, fluorescent lighting and in amalgam for dental restoration.

6. The three chemical forms of mercury are (i) elemental or metallic mercury (Hg^0), (ii) inorganic mercury (mercurous (Hg^{2+}) and mercuric (Hg^{2+}) cations) and (iii) organic mercury.

7. Inorganic mercury compounds are salts of Hg^+ and Hg^{2+} , which are used in several industrial processes and can be found in batteries, fungicides, antiseptics, or disinfectants (EFSA, 2008).

8. Organic mercury compounds have at least one carbon atom covalently bound to the mercury atom (FAO/WHO, 2011). Methylmercury (MeHg) is by far the most common form in the food chain (FAO/WHO, 2011). Other organic mercury compounds like phenylmercury, thiomersal and merbromin (also known as Mercurochrome) have been used as fungicides and in pharmaceutical products (EFSA, 2008).

9. Mercury is a metal that is released into the environment from both natural and anthropogenic sources. After release into the environment, it undergoes complex transformations and cycles between atmosphere, land, and aquatic systems. It ultimately settles in the sediment of lakes, rivers or bays, where it is transformed into MeHg , absorbed by phytoplankton, ingested by zooplankton and fish, and accumulates especially in long-lived predatory species, such as sharks, and swordfish, and tuna in the ocean and trout, pike, walleye, and bass in freshwater systems (WHO/IPCS, 1990). Populations that predominately depend on foods derived from fish or other aquatic environment are more vulnerable to Hg exposure.

10. Food sources other than fish and seafood products may contain mercury, but mostly in the form of inorganic mercury. Based on the available data the contribution to MeHg exposure from non-seafood sources is insignificant (EFSA, 2012).

11. After oral intake in humans, MeHg is much more extensively and rapidly absorbed than inorganic mercury (EFSA, 2012; FAO/WHO, 2011). Following absorption, it is able to enter the hair follicle, and to cross the placenta as well as the blood-brain and blood-cerebrospinal fluid barriers, allowing accumulation in hair, the fetus and the brain, respectively (EFSA, 2012).

12. The main adverse effect associated with MeHg exposure is toxicity to the central and peripheral nervous systems (WHO, 2017). Due to its ability to cross the placenta and the blood-brain barrier, MeHg exposure is of particular concern during embryonic neurodevelopment and in young children (COT, 2004). Thus, pregnant and breastfeeding women are sensitive sub- populations since maternal exposure can lead to exposure of the unborn child either via the placenta or breast milk. The bio accumulative properties of MeHg in combination with its long half-life, mean that the blood concentration of MeHg at the time of becoming pregnant depends on the exposure to MeHg during the preceding year. MeHg can also affect the kidneys. Acute neuro- and nephrotoxicity have been reported in cases of human MeHg poisoning, whereas neurotoxicity is usually associated with lower-level chronic exposures, especially in the developing fetus (COT, 2004).

13. The critical target for inorganic mercury toxicity is the kidney but other targets include the liver, nervous system, immune system, reproductive and developmental systems (EFSA, 2012). Inorganic mercury in food is considerably less toxic than MeHg (EFSA, 2004). This is attributed to the lower absorption of inorganic mercury and due to its low lipophilicity, mercuric mercury does not readily cross the placental, the blood-brain or the blood- cerebrospinal fluid barriers (EFSA, 2012). Mercuric mercury in the brain is generally the result of either in situ demethylation of organic mercury species or oxidation of elemental mercury (EFSA, 2012).