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COMMENTARY

The ETA–ESE statement on the European Chemicals Agency opinion on iodine as an endocrine disruptor

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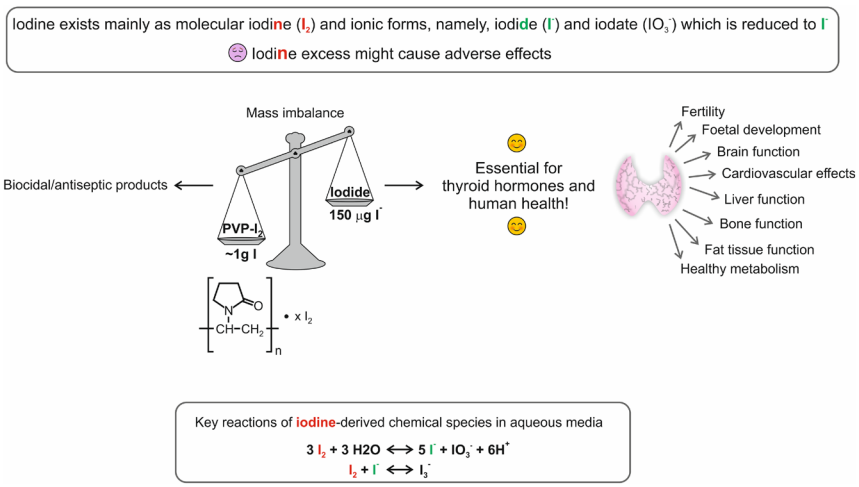
Graphical abstract



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Abstract

In 2022, the European Chemicals Agency (ECHA) made a statement concluding that iodine is an endocrine disruptor (ED). We stress the fact that the ECHA opinion ECHA/BPC/357/2022 is based on their misguidedly zooming in on exclusively the biocidal products (e.g. hand disinfectants, disinfection of animals' teats/udder, embalming fluids before cremation) that contain molecular iodine (I_2), entirely neglecting the 2013 ECHA Regulation (EU) no. 528/2012 describing iodine as being of 'great importance for human health'. Clearly, the current sweeping and erroneous classification of 'iodine' as an endocrine disruptor is ill-advised. We moreover call upon the scientific and medical community at large to use the accurate scientific nomenclature, i.e. iodide or iodate instead of 'iodine' when referring to iodized salts and food prepared there with. Drugs, diagnostic agents, and synthetic chemicals containing the element iodine in the form of covalent bonds must be correctly labeled 'iodinated', if possible, using each time their distinctive and accurate chemical or pharmacological name.

Keywords: ETA-ESE; iodine statement; iodine; ECHA

Introduction

Why is European Chemicals Agency's (ECHA) statement not considered scientifically accurate by the thyroid community?

The ECHA statement made in September 2022 (1) concluding that iodine is an endocrine disruptor (ED) and the adoption of this decision by the European Commission (EC) compels the European Thyroid Association Public Health Board (ETA-PHB), backed by the European Society of Endocrinology (ESE), to react and clarify a misunderstanding evidently due to lack of accurate scientific knowledge among the members of the ECHA and the EC regarding this particular issue. What is more, ECHA's recent statement contradicts the 2013 ECHA Regulation (EU) no. 528/2012 declaring iodine to be highly beneficial, indeed vital to human health, specifically iodine forms (iodide, iodate) which are present in iodized salts, food supplements, and nutrients (2).

Iodine, which has the chemical symbol I, is an essential trace element that occurs naturally in seawater, seaweed, fish, and shellfish. The thyroid gland traps iodide (I^-), which is then oxidized and bound to thyroglobulin. This reduced anionic chemical species of the element iodine is crucial to produce thyroid hormones, which are composed of covalently bound iodine. Today, despite increased salt iodization programs on a global scale, approximately 2 billion people worldwide are classified as having 'iodine' deficiency and about 50 million develop clinical symptoms (3). Changing dietary habits worldwide are partly to blame for this deficiency, with, for example, people cutting down for health reasons on salt consumption or else switching to sea salt, which is low in natural iodide. The result is that ever more people have inadequate levels of iodide intake, which can lead to or worsen thyroid disease as well as cause other significant health problems. Therefore, having well-grounded knowledge of the complex metabolism of iodine-derived chemical species (such as I_2, iodide, or iodate), and molecules containing covalently bound iodine (such as thyroid hormones or synthetic iodine-containing pharmaceuticals and diagnostics), its molecular forms relevant in human and animal physiology is of utmost importance. Yet, unfortunately, this has not been realized and is hence absent from the published ECHA opinion. The ECHA opinion focuses on the topically applied iodine-containing antiseptic used for skin disinfection in healthcare. The antiseptic agent consists of a stable chemical complex formed between very high concentrations of molecular iodine (I_2) with the carrier molecule polyvinylpyrrolidone (povidone) bound in a non-covalent way. The use of these iodine-containing antiseptics for long periods can release high amounts of iodine that may transiently impair thyroid function.

In the body, iodine species in all oxidation states can be formed, depending on the pH, temperature, redox conditions and presence of fatty acid and protein reaction partners. While regular antiseptic dermal application of high concentrations (1–10% w/v povidone–iodine) of the stable triiodide I_3^- complex iodophors does not result in major 'iodine' absorption by the adult skin and may be considered safe, a mucosal or internal triiodide I_3^- application leads to its rapid absorption followed by immediate reduction of triiodide I_3^- to iodide, thus resulting in unwanted very high iodide exposure doses as well as mother's blood and breast milk iodide concentrations (4).

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Notably, acute exposition during the caesarian section may also have similar effects on the thyroid function of newborns sensitive to the iodine status of the pregnant woman (5).

Iodine excess (I_2 or I^-) indeed disrupts thyroid function. Unfortunately, in the ECHA's opinion the importance of the high amount of iodine exposure associated with the chronic use of iodine-containing antiseptics is neglected.

The misleading wording and contextualization of the ECHA opinion are regrettable, as salt iodization was and is one of the most successful WHO supported public health measures to prevent the devastating consequences of 'iodine' deficiency worldwide. To avoid inaccuracies, the terminology of iodine used for health purposes urgently needs clarification.

Iodine, iodide, and biologically relevant iodine-derived chemical species

Iodine is an essential trace element crucial for biosynthesis of thyroid hormones. There are several chemical forms of iodine important to human and animal life (Fact Box 1), namely, molecular diatomic iodine (I_2) and the ionic forms, iodide (I^-) and iodate (IO_3^-). In the environment, iodine is mainly present in the anionic forms in water, while in the air iodine is found in the form of I_2 associated with particles or volatile species, aerosol iodide, and IO_3^- (6). Vertebrates, via the food they consume, absorb I^- or IO_3^- which, following ingestion, is rapidly reduced to I^- and transported to the thyroid gland where it serves as the key substrate for thyroid hormone synthesis. In some medical products, such as povidone–iodine or Lugol's solution, the anionic complex triiodide (I_3^-) is used, which forms by non-covalent association between molecular iodine I_2 and the iodide anion I^-.

Unfortunately, in many scientific articles, the term 'iodine', which in food and iodized salt is known as I_2, is broadly used as a generic name for all chemical forms of 'iodine', including its anionic forms, particularly IO_3^- or I^- (7). For experts in the field, this nomenclature inaccuracy usually does not lead to major misunderstandings since the context of scientific papers clearly indicates that they in fact refer to IO_3^- or I^-, despite the use of the generic term 'iodine'. This is particularly evident in the case of the term 'iodine' deficiency or 'iodine' fortification, which refers to the supplementation of salt and food with sodium (or potassium) IO_3^- (or I^- in some cases) (8). This erroneous nomenclature has been adopted by the non-scientific media and has resulted in the widespread and inaccurate use of the term 'iodine'.

This widespread inaccuracy of the nomenclature has recently led ECHA, in its publication on 'iodine', to classify generic 'iodine' as an endocrine disruptor. This latter decision will regrettably raise serious concerns about the scientific/medical need for 'iodine' fortification and the legitimate and legal use of iodized salt. Moreover, this all-around misunderstanding may in turn result in political decisions on the limitation or even prohibition of the use of iodized salt in some countries also in response to demands made by the under-informed or misinformed public. This is a real threat, particularly when we consider the present-day worldwide circulation of fake news and conspiracy theories affecting health-related issues (9). The use of iodized salt has already several times in the past been eroded by misinformation, for instance in Pakistan, where loudly protesting activist groups oppose the consumption of 'iodized table salt' and food supplements, thus leading to iodine deficiency in this country (10).

Clinical significance of iodine and iodine deficiency

As mentioned above, the trace element iodine is essential for the synthesis of thyroid hormones which regulate all mammalian metabolic processes, the latter being crucial for the development and maintenance of optimal health. Iodine covalently bound to thyroglobulin is present in the thyroid gland and physiologically has a vital endocrine effect on the production of thyroid hormones themselves. The most prominent sign of moderate to severe iodine deficiency in a population is a high prevalence of enlarged thyroid glands – previously known as 'endemic goiter'. In the early 20th century, it was discovered that iodide or iodate supplementation was able to prevent endemic goiters. Salt iodization was first used in Switzerland and the USA in the 1920s and has subsequently become the mainstay of prevention efforts against iodine deficiency, as summarized in a recent landmark paper on the topic (3). During the following decades, it was demonstrated that severe iodine deficiency not only caused goiter but also a spectrum of diseases, such as cretinism, intellectual impairment, poor growth, and development, as well as adverse obstetric outcomes (Table 1).

Within a few years after the introduction of iodized salt in Switzerland (10), goiter in newborns and most childhood goiters had disappeared and no more infants were born with cretinism. The dramatic reduction in the global prevalence of 'iodine' deficiency disorders (IDD) over the past 30 years represents an outstanding and underrecognized public health achievement.

Both iodine deficiency and iodine excess impair optimal thyroid function, but iodine deficiency is a global public health problem (11). Despite routine iodization of salt to prevent the severe consequences of 'iodine' deficiency, e.g. endemic goiter and intellectual impairment, which are still observed in several EU countries affected by severe iodine deficiency, mild

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Fact Box 1 Summary of the chemical compounds and molecular species derived from elemental iodine with relevance to medicine, nutrition, and prevention.

Chemical designation	Properties	Chemical formula	Concentration range of iodine species in product	Presence in the environment, food, nutrients, and medical products
Iodine	Chemical element; molecular iodine; neutral, non-charged; non-metallic; solid and volatile	I_2		Air (associated with particles or volatile species); topically applied antiseptics (e.g. povidone–iodine)
Iodide	Reduced form of iodine; anion; component of water, soluble salts (e.g. sodium iodide, potassium iodide)	I^-	~2 ppm iodine (parts per million)	Air aerosol, water, plants and animals; rock salt; mineral salts; sea salt; (iodide is not present in 'Himalaya salt'); some nutritional supplements (potassium iodide) for humans and livestock
Triiodide	Anionic non-covalent complex between molecular iodine I_2 and iodide I^- anion	I_3^- $I_2 :: I^-$	2.5–25 mg iodine/100 mL 1 g iodine/100 mL (1% total iodine)	Contained in aqueous solutions like: Lugol's solution or povidone–iodine, used for medical purposes
Iodate	Oxidized form of iodine; anion; component of sea water, soluble salts (e.g. sodium iodate, potassium iodate)	IO_3^-	20–40 mg iodine/kg table salt	Air aerosol, water; chemically stable; iodized table salt (used as 'iodine' fortification in preventive 'iodization' programs)
Periodate	Oxidized form of iodine; anion; similar to perchlorate	IO_4^-		Powerful oxidizing agent; not present in food and nutrients
Iodinated compounds	Molecules in which iodine is covalently bound (e.g. drugs)	X-I	2–200 µg iodine per tablet; 240 to 400 mg iodine/mL 75 mg iodine/200 mg tablet	Levothyroxine, Liothyronine; Synthetic iodinated contrast media (e.g. iopanoic acid); Iodinated antiarrhythmic drug amiodarone
Iodine-containing complexes, 'iodophores'	Polymeric structures forming non-covalently bound complexes with iodine containing compounds (e.g. povidone–iodine; starch)	$[X :: I_n]_y$	1 g iodine/100 mL (1% total iodine)	Topically applied antiseptics (e.g. 'povidone–iodine': $PVP^+ :: I_3^-$); not present in food and nutrients
'iodized salt'	Minute amounts of various salts of the element iodine added to table salt	$K^+ IO_3^-$ $Na^+ IO_3^-$	20–40 mg iodine/kg table salt (EU regulation)	Table salt containing small amounts of potassium (or sodium) iodate (IO_3^-, stable) or potassium iodide (I^-, instable)

iodine deficiency remains a health problem of great impact. Mild iodine deficiency represents an economic and health burden in this region. Pregnant women and their offspring are at particularly high risk. However, both the adult population and adolescents in Europe are at worryingly high risk for mild "iodine" deficiency given that longstanding mild iodine deficiency exposes the European population to a higher risk of nodular thyroid diseases. These may develop into hyperthyroidism, causing increased morbidity and mortality in the population above middle age (12). Furthermore, this heightened risk is particularly relevant and can potentially be devastating for patients exposed to high iodine doses in the range of several grams per dose by frequent diagnostic medical use of, for example, contrast media for angiography of cardiac, vascular, and other diseases (Fact Box 1). Similarly, synthetic iodinated contrast media, containing covalently bound iodine, are routinely used for computer tomography scans in medical imaging procedures for benign, malignant, acute, and chronic diseases (13). Finally, many patients are dependent on the iodinated drug amiodarone, also containing covalently bound iodine, to control their malignant cardiac arrhythmia. A further serious risk of iodine deficiency at any age translates to an increased susceptibility to irradiation

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Table 1 Iodine deficiency in age groups.

Age group	Effects of iodine deficiency
Fetuses	Spontaneous abortions, stillbirths, congenital anomalies, perinatal mortality
	Increased susceptibility of the thyroid gland to nuclear radiation
Neonates	Neonatal hypothyroidism, endemic cretinism
	Increased susceptibility of the thyroid gland to nuclear radiation
Children and adolescents	Goiter, hypothyroidism, or hyperthyroidism
	Impaired mental function, delayed growth, and puberty
	Increased susceptibility of the thyroid gland to nuclear radiation
Adults	Goiter with its complications, hypothyroidism
	Infertility, impaired mental function
	Decreased work capability
	Spontaneous hyperthyroidism in the elderly
	Iodine-induced hyperthyroidism
	Higher ratio of the more aggressive follicular to papillary thyroid cancers
	Increased susceptibility of the thyroid gland to nuclear radiation

Adapted from: Zimmermann, M.B. Iodine Deficiency. *Endocr. Rev.* 2009, 30, 376–408 (12).

damage of the thyroid gland in the event of a nuclear accident as well as a documented shift toward more aggressive forms of thyroid follicular and anaplastic cancers compared to the more benign papillary ones in iodine-sufficient areas (14) (Table 1).

Unlike iodine deficiency, the effect of iodine excess on human health is less harmful, affecting relatively few individuals exposed to compounds containing high amounts of or iodine-containing agents, respectively, for a short period. This exposure is reversible. Thus, iodine excess does not expose the European population to a meaningful health risk. Iodine deficiency, by contrast, affects the entire population.

A recent analysis using a regression model estimated the potential health and economic benefits gained between 1993 and 2019 by the global program to reduce iodine deficiency and goiter (15). Based on this approach, the global prevalence of clinical IDD (as assessed by total goiter rate) fell from 13.1 to 3.2%. Universal salt iodization has significantly reduced the number of newborns affected by IDD, with 20.5 million cases prevented annually (15). The resulting projected improvements in cognitive development and future earnings suggest a potential global economic benefit of nearly $33 billion. The estimated annual economic benefit coming from the achievement of salt iodization in European countries is as much as €3.5 billion annually. In 2019, there were 0.599 million newborns suffering from IDD in Europe whose expected lifelong productivity losses would hit €4.4 billion. To this can be added a yet unknown economic burden of all other health-related iodine deficiency costs (16, 17) (Table 1).

Progress towards optimal iodine nutrition in Europe needs continuing efforts, constant reinforcement, monitoring, and commitment by all member states, and the same applies globally. In some regions there has been backsliding, and, once "iodine" sufficiency has been achieved resources are diverted to other public health priorities and gains are not sustained. Since the introduction of universal salt iodization in most countries, mild iodine deficiency remains prevalent, especially in pregnant women. The acknowledgement of mild iodine deficiency as a health problem is the cornerstone for optimization and maintenance of adequate iodine intake for the most vulnerable population groups. It would be extremely unfortunate if one of the most globally successful – yet forgotten even by health authorities' public health-promoting actions of eradicating severe 'iodine' deficiency were to develop into a total long-term failure with devastating consequences for the coming generations. Work needs to be done!

The ETA and the ESE are very concerned that labeling of iodine as an ED without any contextualization and clear definition of the term iodine will render optimization of iodide intake in Europe a more difficult or even an impossible task. In brief, the consequences of adopting the ECHA opinion as it now stands may well have a very negative impact on the health of the European population – and beyond.

Concluding remarks

- We stress the fact that the ECHA opinion ECHA/BPC/357/2022 erroneously focuses on the biocidal products that contain molecular iodine (I_2). Hence, the classification of iodine as an endocrine disruptor does not apply to the anionic iodine forms (iodide, iodate) that are present in physiological amounts in iodized salts, food supplements, and nutrients as well as in chemical and pharmaceutical compounds.
- We call upon the scientific and medical community at large to use the accurate scientific nomenclature,

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i.e. iodide or iodate instead of 'iodine' when referring to iodized salts and food prepared therewith. Drugs, diagnostic agents, and synthetic chemicals containing the element iodine in the form of covalent bonds must be correctly labeled 'iodinated', if possible, using their proper chemical or pharmacological name. This is in stark contrast to PVP–iodine, which contains molecular diatomic iodine (I_2) in a non-covalently bound way, and which is the specific molecular iodine form I_2, to which the misleading EU commission and ECHA statement incorrectly referred.

- We call upon the ECHA and EC to include and consult the scientific medical community and endocrine specialists who are engaged in the process of evaluation of potential endocrine disruptors, particularly when they are dealing with essential micronutrients that physiologically interact with the endocrine organ and the hormone system under discussion.

Declaration of interest

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the study reported.

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