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FEATURE ARTICLE

Critical review or scientific opinion paper: Arsenosugars—a class of benign arsenic species or justification for developing partly speciated arsenic fractionation in foodstuffs?

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Abstract In this opinion paper the toxicokinetic behaviour of arsenosugars is reviewed and compared with that of inorganic arsenic and arsenobetaine. It is concluded that the arsenosugars are similar to inorganic arsenic in terms of metabolite formation and tissue accumulation. As a pragmatic means of generating uniform data sets which adequately represent the toxicity of arsenic in food we recommend reporting partly speciated arsenic concentrations in food commodities in three fractions: i) toxic inorganic arsenic as arsenate (after oxidation); ii) arsenobetaine as established non-toxic arsenic; and iii) potentially toxic arsenic, which includes arsenosugars and other organoarsenicals.

Keywords arsenic · speciation · legislation · food analysis · metabolism · accumulation

The toxicity of arsenic depends on its molecular nature. The main arsenic compounds in water are inorganic arsenic species, arsenite (As^{III}) and arsenate (As^{V}), which are considered non-threshold class I carcinogens. Regulators and legislators have responded to this by setting very low maximum levels of 10 $\mu g L^{-1}$ for arsenic in drinking water in most countries worldwide [1]. Setting arsenic standards in food is, however, more complex, and so far regulators have been reluctant to establish maximum levels for arsenic in food commodities. The reason is that arsenic levels in food vary by more than five orders of magnitude, and its molecular forms are very diverse. Besides the (toxic) inorganic forms of arsenic, a multitude of compounds in which arsenic is bound to an organic moiety has been identified in foodstuffs. These are usually referred to as (non-toxic) organoarsenicals, which in most marine food, especially fish, make up the major proportion (>85%) of the total arsenic concentration.

The challenges for legislators wishing to regulate arsenic concentrations in food have been two-fold, one toxicological in nature, the other analytical, specifically:

1. to identify the fraction of arsenic species from which there is a risk of contracting cancer; and
2. to design proficiency testing in order to identify robust analytical methodology to monitor this fraction.

The need to legislate in relation to toxic arsenic in food and to generate more speciated arsenic data has been highlighted in a recent review from the European Food Safety Authority (EFSA) [2]. It was pointed out that in particular rice and rice-based products are a major risk, because of elevated levels of inorganic arsenic [3]. Although inorganic arsenic is often a major arsenic species in rice, the proportion of inorganic to organic arsenic species (here in the form of dimethylarsinic acid (DMA^{V}) and methylarsonic acid (MA^{V}), both toxicologically less potent) is variable [4]. Therefore, arsenic speciation analysis that quantifies the inorganic arsenic fraction is mandatory to adequately determine the risk associated with rice consumption. With the target species identified, current analytical methods have been introduced and have been

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shown in a recent proficiency test to be robust [5]. The authors state in their report that " ... an introduction of a maximum level of inorganic arsenic in food regulation of rice should not be delayed due to analytical concerns ... ", thus promising regulated threshold values of inorganic arsenic in rice to be implemented in the near future [5].

For marine food, the situation regarding arsenic concentration and speciation is much different. Fish and seafood can contain up to 100 times more arsenic than rice. However, it often contains only a small proportion of inorganic arsenic among a huge amount and variety of other organoarsenic compounds. Speciation analysis in this matrix requires different and complex analytical methods, and a robust, simple and affordable method is not yet envisaged.

Apart from this, the main organoarsenical in fish is arsenobetaine, a compound famous for its non-toxicity. This widely led to the assumption that organoarsenic compounds in general are benign, just because they are supposed to behave similar to arsenobetaine [6].

However, in a major proportion of other seafood, and among the variety of more than 30 organoarsenicals in marine organisms [7], one important group of arsenic species is the group of the arsenic-containing ribofuranosides or so-called arsenosugars. Structures of those arsenosugars with the nomenclature used here are shown in Fig. 1.

In contrast with inorganic arsenic and arsenobetaine, not much is known about the toxicity of arsenosugars. However they represent the main proportion of arsenic in, e.g., algae consumed with sushi (Fig. 2). Here in this opinion paper, we would like to focus on those, and elaborate whether the assumption is valid that arsenosugars in general are toxicologically as benign as arsenobetaine or whether the development of analytical methods for arsenosugars in environmental monitoring for risk assessment is justified.

Provided the first assumption is true, inorganic arsenic could be singled out as the most important target arsenic species, so analytical methods could be optimised for the determination of one species only (e.g. arsenate only after conversion of all inorganic arsenic to arsenate). In this case, regulatory bodies could readily proceed to establishing threshold values for inorganic arsenic in marine foodstuffs, as in the case of rice discussed earlier.

Arsenosugars

Arsenosugars, and there are more than 15 different molecular forms known, are not only the main arsenic species in seaweed, but also occur in large proportion in herbivorous molluscs and gastropods [8]. Most arsenosugars have a dimethylarsinoyl moiety, in which arsenic is pentavalent and binds to two methyl groups, to the sugar and to oxygen and they differ only in the side chain of the C1 position of the sugar backbone (Fig. 1). In the recently identified thio-arsenosugars the oxygen is replaced by sulfur [9, 10]. Arsenosugars can also occur in the form of trimethylarsonium compounds.

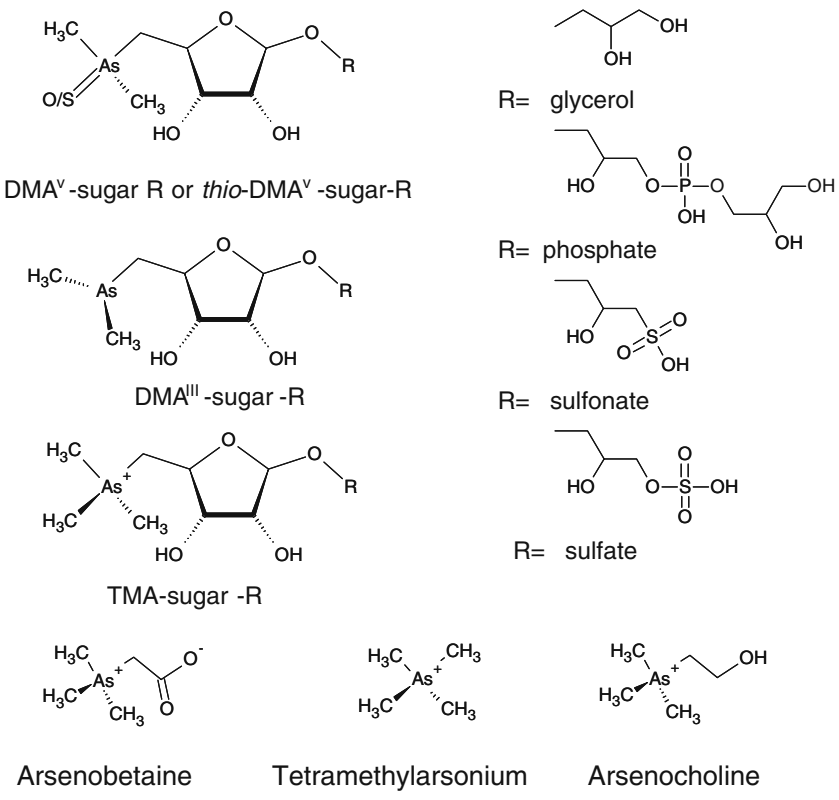

Fig. 1 General structures of arsenosugars compared to other organoarsenicals in which arsenic forms four carbon bonds

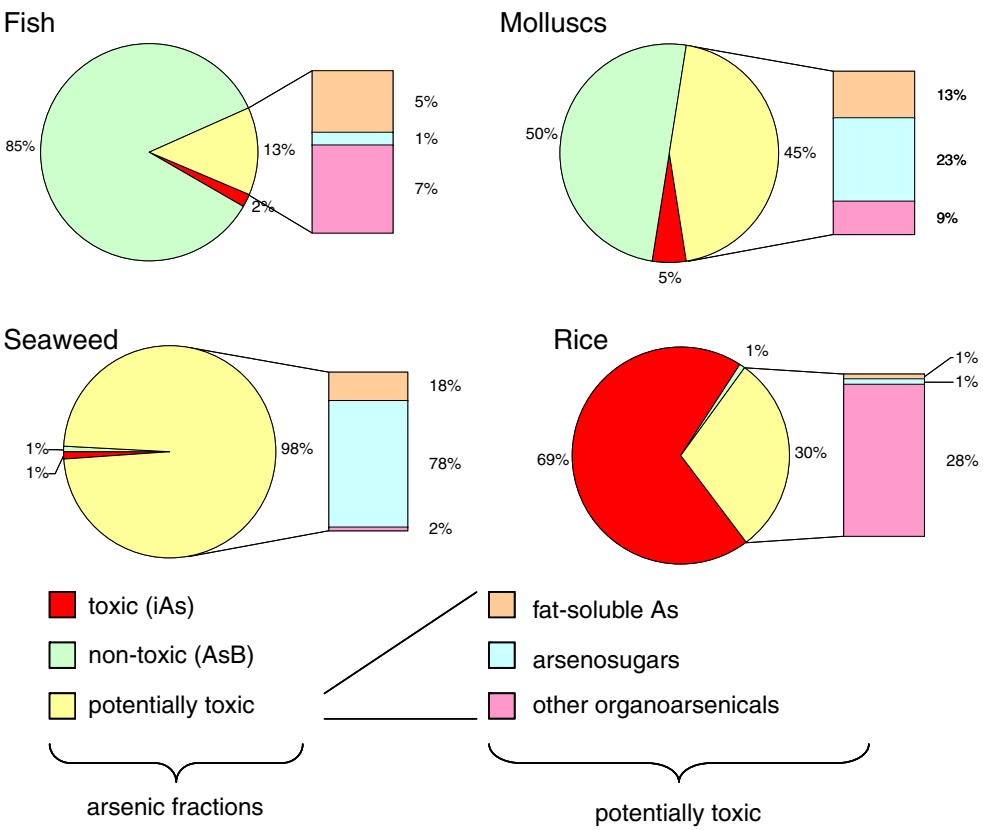

Fig. 2 Expected proportions of the three different arsenic fractions in three different marine food commodities, in comparison with rice, illustrating the importance of the potentially toxic fraction of which arsenosugars in seaweed and molluscs are the important species

These are structurally similar to arsenobetaine, but are found in traces only and thus not further discussed here [11].

To date only the cytotoxicity of a single arsenosugar (DMA^V–sugar–glycerol) has been evaluated using cell cultures [12, 13]. These tests showed no cytotoxicity at the micromolar level, suggesting that the arsenosugars as they occur in food have very limited toxicity and therefore are similar to arsenobetaine. However, the question remains whether we can draw immediate extrapolations that all arsenosugars are also as benign as arsenobetaine when ingested by humans? In order to answer this question the bioavailability, metabolic transformation, and the clearance from the body need to be investigated and compared with well known effects of inorganic arsenic and arsenobetaine.

Toxicokinetics of arsenicals

In arsenobetaine, arsenic is oxidised and has four stable carbon bonds, which are enzymatically or thermally hard to break [14], i.e. arsenobetaine is biochemically quasi-inert. This may be the reason why this compound, although readily available, does not transform into other metabolites when ingested by humans, and is rapidly excreted from the mammalian body unchanged [15]. There are however two studies which showed that small amounts of arsenobetaine may be retained in the body of rabbits [16] or humans [17]. Ingested inorganic arsenic is accumulated in hair, which therefore can be used to detect arsenic exposure, but this is not true for exposure to elevated levels of arsenobetaine, and in-vitro absorption experiments have shown that arsenobetaine has no affinity for hair, nor ever been identified in hair [18–20]. The hypothesis that the four stable As–C bonds are responsible for the benign character of arsenobetaine gains further support by the fact that tetramethylarsonium and arsenocholine, both of which share the same molecular feature, also show no sign of toxicity [21].

The toxicokinetics of arsenosugars seem however to be different. Arsenosugars are bio-accessible [22], readily taken up, and metabolised when ingested: no intact arsenosugar was identified in the urine of volunteers after quantitative urinary clearance of arsenic [23]. When seaweed (*Laminaria* spp.), containing mainly arsenosugars (AsSug-glycerol, phosphate, and sulfonate), was ingested by humans, no significant amounts of arsenosugars were found in the urine either [17, 24]. However, excretion was neither rapid (maximum of arsenic elimination in urine was between 20 and 26 h) nor quantitative. After 72 h, less than 50% of the arsenic was cleared through urine from the body [17]. A most interesting previously reported observation suggested that urinary elimination of arsenic after an arsenosugar-containing meal may differ greatly, even among members of the same family [25]. This study had however a limited cohort of volunteers. One recent study in which six

volunteers ingested a synthesised arsenosugar revealed enormous differences in urinary recovery within four days (4–95%) [26]. This suggests that there is an intrinsic difference in arsenosugar metabolism in humans, which is independent of the matrix in which the compound is taken up, but rather dependent on the individuals' metabolism, with so far no suggestion what may trigger these differences.

So, how can meaningful and generally applicable toxicity data be established when everybody has an individual metabolism for arsenosugars?

Metabolism of ingested arsenicals

At this point, it is worth having a closer look at what we know about the metabolism of arsenic in humans. When inorganic arsenic is ingested, it is methylated and excreted via the urine, mainly in the form of pentavalent DMA^V and MA^V (Fig. 3). These species are less toxic than the initially ingested inorganic arsenic. However, recently controversial discussions have arisen about possible intermediates in the form of the trivalent analogues (DMA^{III} and MA^{III}), which are extremely toxic, shedding doubt on the widely accepted opinion that the metabolic transformation is a detoxification mechanism for arsenic [27]. Now, arsenosugars are likewise metabolised, with DMA^V being the main metabolite among numerous other compounds [23–25]. All the other arsenic metabolites identified so far, for example dimethylarsinoyl-acetic acid ($DMAA^V$) or dimethylarsinoylethanol ($DMAE^V$) (Fig. 3), also have a dimethylarsinoyl moiety. Interestingly, some of those compounds also occurred in the urine as their thio-analogues e.g., *thio*-DMA^V, *thio*-DMAE, and *thio*-$DMAA^V$ [28–30]. Those species might occur even as trivalent species but have so far not been found.

Very little is known about the toxicity of those metabolites, but the main arsenical, DMA^V, is, even in its pentavalent form, more toxic than the ingested arsenosugars and is known to act as a co-carcinogen in rodents [31]. Additionally, the first toxicological studies of the thio analogues of the organoarsenicals (*thio*-DMA^V) suggest that this compound has enhanced cytotoxicity compared with the oxo-form of DMA^V [32]. As mentioned above, the arsenosugar might even be reduced or thiolated in the body before further degradation (Fig. 3). Trivalent arsenosugars, also not found in biota, presumably because of their reactivity, had a high cytotoxic activity when directly bound to plasmid DNA at the micromolar level [33]. This means that the toxicological potential of the initially ingested arsenosugar may increase dramatically during its metabolic pathway in the body. A reason for this may be the "soft" character of trivalent arsenic, which has higher affinity for the sulfhydryl groups found in peptides and proteins. Even thiolation of arsenic will soften the pentavalent arsenic in the dimethylarsinoyl moiety, and thus an interaction with sulfhydryl or thio-groups, as shown recently to take place in plants, can also be proposed [34]. All these reactions are not possible in arsenic species which cannot be reduced or thiolated without cleaving an As–C bond, for example arsenobetaine in which arsenic seems to be inert and fails to interact directly with biomolecules.

Bioaccumulation of arsenicals

A proxy to determine whether arsenosugars may pose a risk through long-term exposure, which might lead to cancer, is to look at arsenic accumulation in tissues and organs. No indications exists whether arsenic accumulates in human tissues after prolonged arsenosugar exposure, but conclusions may be drawn from animal studies, in this case from sheep. It has been shown that the arsenosugar metabolism in sheep is similar to that of humans, because the same arsenic metabolites were found in sheep urine after the sheep ingested large quantities of the arsenosugar-containing seaweed *Laminaria spp* [23, 35–37]. The woolly volunteers for this study were the seaweed-eating sheep from North Ronaldsay (Orkney Islands), who live entirely on a Laminaria based diet, which they devour directly from the beach. These algae contain between 50 and 100 $mg\,kg^{-1}$ arsenic, approximtely 80% of which is present as arsenosugars, with inorganic arsenic present in traces only.

Arsenic concentrations in the kidney, liver, and muscle of seaweed-eating sheep are two orders of magnitude higher than in sheep living on grass, indicating that seaweed eating results in similar bioconcentration as expected from inorganic arsenic ingestion [38, 39]. The arsenic speciation in the tissue was however not determined. Accumulation of arsenic in the wool and the horn of those sheep was also of the same order of magnitude [40, 41]. Here the arsenic was speciated. The main extractable arsenic in the wool and horn is DMA^V [40, 41]. This is in contrast with the bioaccumulation of arsenic after inorganic arsenic ingestion, which accumulated mainly as inorganic arsenic rather than DMA^V. Furthermore, it was shown that the arsenic metabolites generated after eating seaweed can be transported through the placenta and can subsequently accumulate in the horn of the unborn lamb. In-utero transport of arsenobetaine has never been shown, whereas it is well known to occur for inorganic arsenic [42].

However, the disadvantage of this kind of study is that our four-legged friends have a limited lifetime (4–6 years only), which is probably too short to assess long-term cancer risk, and can thus not be used as a proxy for human cancer risk. It should, however, be mentioned here that no specific health problems have ever been reported for those animals, and the organs or tissues of the tested animals did not reveal any obvious pathology related to arsenic.

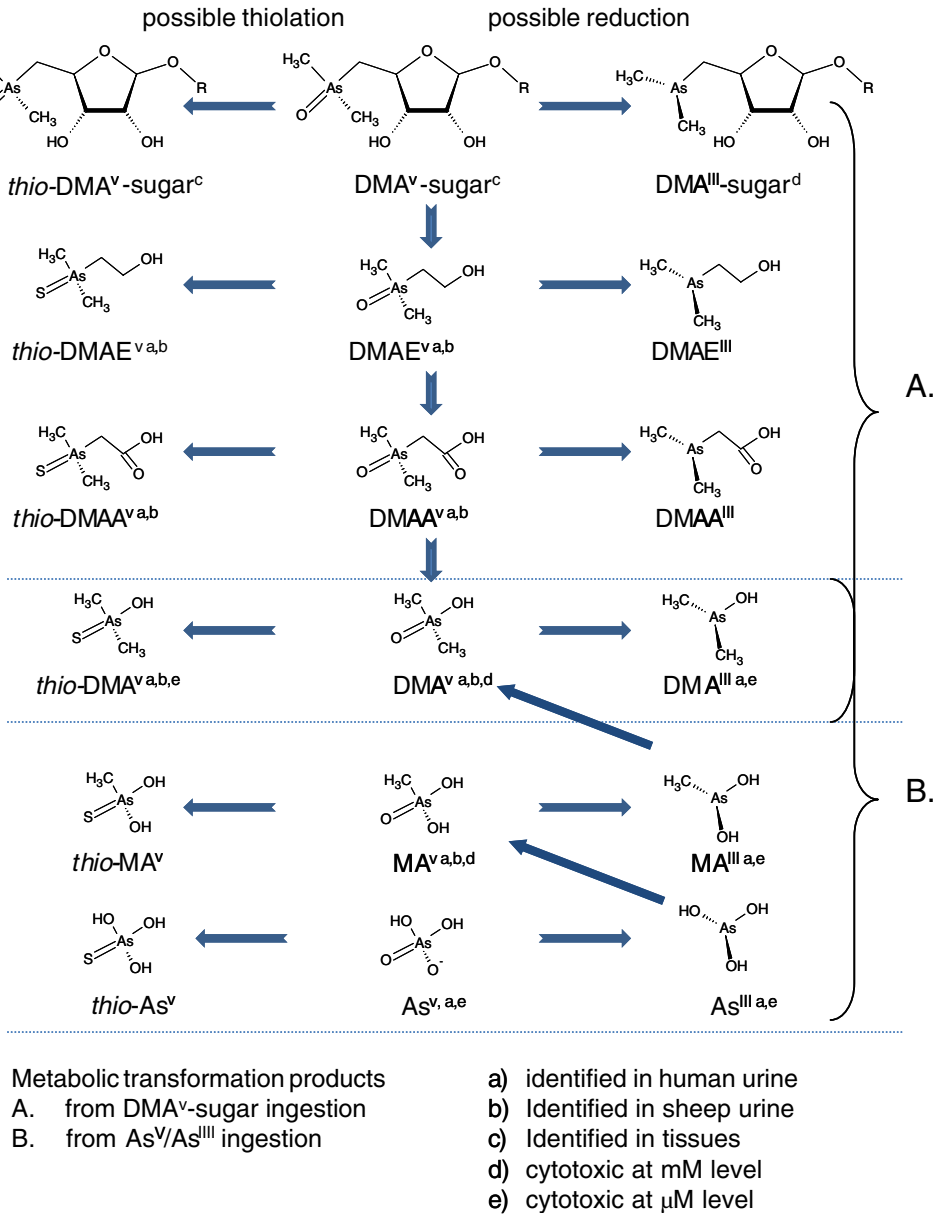

Fig. 3 Metabolic transformation of DMA^{V}-sugars and inorganic arsenic (As^{V}) to DMA^{V} with possible intermediates. While inorganic arsenic metabolism drawn here follows the Challenger mechanisms of consecutive reduction and oxidative methylation, the degradation pathway of the arsenosugars is unknown. All possible intermediates and end products from both arsenic species might be thiolated or even occur in their trivalent arsenic form

The study however emphasizes that some foodstuffs contain a major proportion of arsenic in a form which is metabolised, but whose toxicological potential has not been investigated thoroughly enough to reach sound conclusions. Another, maybe more worrying outcome of the arsenosugar metabolism studies cited above is the enormous diversity of metabolism of these compounds in different people. No hints so far are pointing to either genetic disposition or seafood matrix dependency; the findings rather suggest a great amount of general randomness.

Analytical considerations

It is evident that the total arsenic concentration as an indicator for risk assessment is not adequate [7], as fish and most seafood often contains high amounts of arsenic (~1 $mg\,kg^{-1}$) and would therefore be classified as a high-risk food, although more than 90% of the As content is harmless arsenobetaine (Fig. 2). Taking, however, solely the inorganic arsenic as a measure of toxic arsenic might underestimate the risk, because a major proportion of the arsenic might be present in a form with unknown toxicity (labelled as potentially toxic in Figs. 2 and 4). Therefore, it would not be in line with the precautionary principle of risk assessment to focus on inorganic arsenic solely, in particular for molluscs and seaweed (Fig. 4).

Here, the toxicity of arsenosugars and their metabolic products urgently needs to be tested which may be not a trivial task [43]. Because the toxicokinetic behaviour of arsenosugars has all the hallmarks of that of inorganic

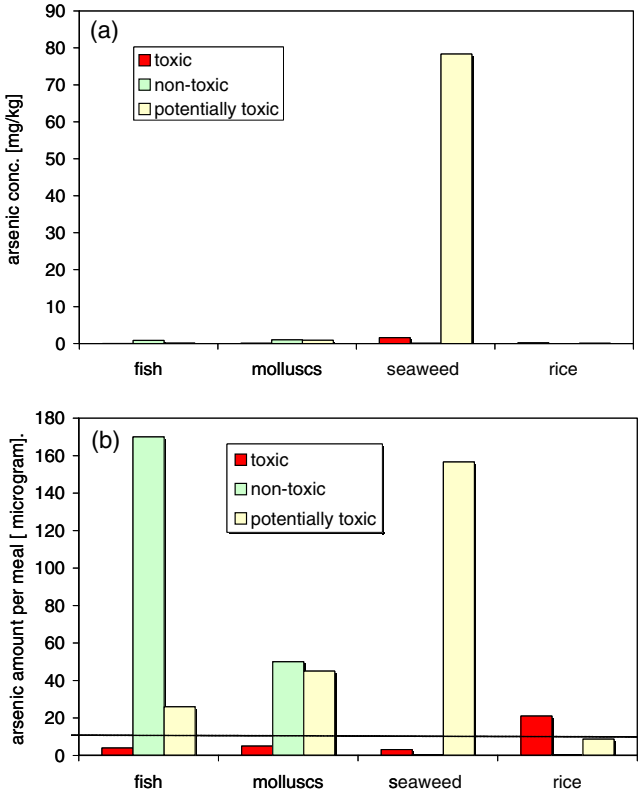

Fig. 4 Expected arsenic concentrations in the food commodities for the three different arsenic fractions (**a**) and intake of arsenic from an average meal containing these commodities (**b**) illustrating the importance of toxic arsenic in rice and the potentially toxic fraction in seaweed and molluscs. The *line* in (**b**) gives the level of 10 μg arsenic as a recommended maximum daily intake (of toxic inorganic arsenic) according to drinking water regulations

arsenic rather than that of arsenobetaine, it is justified to group them as potentially toxic arsenic species and it is therefore necessary to determine the arsenosugars in foodstuffs where this compound represents the main arsenic species (Fig. 4). This then needs to be reflected in the risk assessment and environmental monitoring program of such food commodities.

So, legislators and regulators face a serious dilemma. The variability and complexity of the task make determination of all the molecular forms of arsenic in all types of food an almost impossible objective, especially as routine and high-throughput monitoring is required [44], as is the case when threshold values in food are to be implemented. In order to produce reliable data on arsenic speciation, robust analytical methods have to be developed which reflect our toxicological knowledge of the different arsenic species [45]. A possible strategy for such a routine analytical approach could be to first classify the arsenic compounds into three fractions:

1. the toxic inorganic arsenic, determined as arsenate after oxidation;
2. arsenobetaine as established non-toxic arsenical; and
3. the leftover organoarsenical fraction, which may contain arsenosugars and other organoarsenicals, including non-water extractable, fat-soluble or lipophilic arsenic compounds which we know very little about [46, 47]; those would have to be reported as the sum of potentially toxic arsenicals. Whether this can be determined directly by generic determination of arsenosugars and arsenolipids or indirectly as a fraction resulting from a mass balance using total arsenic, inorganic arsenic, and arsenobetaine is a challenge for the analytical community interested in arsenic speciation.

Simple methods for the determination of these fractions should be an objective which should be achievable from an analytical perspective. This follows the precautionary principle and would provide the basis enabling generation of uniform data with partial speciation information. There is a lack of good quality speciation data on foodstuffs which report quantitatively the compounds which would fall into the potentially toxic arsenic fraction but which often are not conclusive; hence, a toxicological evaluation is impossible. An evaluation is also more challenging because the metabolism and long-term effects of organoarsenicals in the potentially toxic fraction cannot be studied by controlled dietary trials with human volunteers, as ethical approval for such a test would not easily be granted, due to the lack of body-clearance data; this renders such a study impossible. Possible indications might be obtained if epidemiological studies could be performed on seaweed-eating societies, for example Japan.

In conclusion, we feel that arsenosugars should not necessarily be speciated individually but that robust methods targeted at arsenic species such as inorganic arsenic and arsenobetaine in addition to a fully quantifiable non-speciated arsenic fraction is urgently needed and may lead to the generation of large data sets to push legislation forward and establish guideline standards for toxic arsenic in food of marine origin.

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