Tellurium: Its Influence on Organisms

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Contributor: Stanislav Stuchlík

Tellurium (Te) is a member of the chalcogen group, which includes oxygen, sulphur, selenium (Se) and polonium . The first three members of the chalcogen group have crucial functions in biochemistry, biology and medicine, whereas Te is a strange element with no apparent role in biological systems. Moreover, it belongs to the group of very few elements in the Periodic Table that have been almost completely ignored.

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1. Tellurium and Its Occurrence

The first three members of the chalcogen group have crucial functions in biochemistry, biology and medicine, whereas Te is a strange element with no apparent role in biological systems. Moreover, it belongs to the group of very few elements in the Periodic Table that have been almost completely ignored. Its chemistry is similar to that of Se in many ways [1]. There are many not yet fully understood mechanisms of Te compounds in organisms but Se, with many similarities in properties, can provide a considerable example.

Information on environmental concentrations of Te is scarce and often misleading due to inadequate analytical methods. There are unknown data about elemental Te concentrations in crust [2], and values for soil concentrations differ with a broad range from 0.008 mg/kg to 0.03 mg/kg [2], and in some localities that are naturally anthropologically enriched with Te, compounds can reach from 0.166 mg/kg [3] even up to 0.5 mg/kg [4]. In areas adjacent to gold mines, the concentration of Te reaches extreme values of up to 14.8 ppm [5]. Te forms alloys with other metals, mainly copper, gold and silver [6], and can be incorporated into stainless steel, lead and bronze to improve their machinability and to make them more resistant to corrosion.

Inorganic Te compounds occur be in various oxidation states ranging from -II to +VI, namely -II (H_2Te, hydrogen telluride), 0 These oxides form tellurous acid (H_2TeO_3) and telluric acid (H_2TeO_4) and their salts are known as tellurites TeO_3^{2-} and tellurates TeO_4^{2-}. Ligand changes are responsible for the majority of the biological activities of various cysteine proteases [7]. Organic Te agents have GPx-like activity, as shown by tests in vitro in which organotellurides have been used as mimics of the antioxidant GPx (glutathione peroxidase), because of their resemblance to the human Se-containing enzyme GPx [8][9].

2. Effects of Tellurium and Its Compounds on Selected Prokaryotic Systems

Tellurites are highly toxic, even at micromolar levels (1 µg/mL) The toxicity of potassium tellurite (K_2TeO_3) was recorded for the first time by Sir Alexander Fleming in 1932 [10]. These compounds were used for the treatment of some medical conditions such as syphilis, tuberculosis, cystitis, dermatitis, eye infections and leprosy. Therefore, Te-containing soluble salts were historically used as antimicrobial and therapeutical agents before the era of antibiotics [11].

Te probably exploits the metabolic machinery of Se, and tellurodiglutathione (GSTeSG), which is an analogue of selenodiglutathione (GSSeSG), is produced during H_2Te formation. The toxicity mechanism of thiol-binding metal(loid)s is based on the interaction and subsequent inhibition of essential thiol groups of enzymes and proteins [12]. The similar physical and electrochemical features of Te to Se and sulphur lead to its substitution of them in proteins [13]. The erroneous incorporation of the resulting tellurocysteine and/or telluromethionine into protein structure leads to changes in protein activity or protein inactivity.

This effect is accompanied by the inhibition of ATP synthesis, resulting in the depletion of intracellular ATP stores during aerobic growth [14]. The same detrimental effect of Te compounds has been found in protein synthesis with regard to proteins containing amino acids with reduced thiol groups [15] of both low-level and high-level resistant microbes. Two high-resistant bacteria, namely *Erythromonas ursincola* strain KR99 and *Erythromicrobium ramosum* E5, are distinct from

other microbes, both showing an increase in protein and ATP synthesis in the presence of Te compounds [16]. The mechanism of this increase is uncertain, but it was implied [16][17][18][19] that reduction of such oxyanions could help with retaining optimal redox balance.

The reduction of Te compounds in modified cell culture media is accompanied by black colony formation. This chromogenic-based selective cultivation has been used for the diagnosis of the presence of antibiotic-resistant pathogenic bacteria for decades and, despite the emergence of genomic approaches, remains widely used [20][21][22].

Three main types of resistance exist: intrinsic, acquired and adaptive.

The mechanisms of bacterial resistance to toxicants, in general, are extraordinarily diverse. They can be specific, whereby the main role of the cell is to resist the action of a toxic compound, or nonspecific, in which the resistance is based on a component that is involved in other cellular functions but that also exerts a protective effect against the toxicant. Even if the toxicant enters the intracellular milieu and target affinity does not change, bacteria can enhance their resistance by actively expelling the toxicant from their cells by efflux. In the case of no permeability changes, the generally reduced uptake of toxicant through the semipermeable outer membrane acts in synergy with other resistance mechanisms such as enzymatic degradation and efflux [23], for instance, the possession of a semipermeable outer membrane with low permeability, as is the case for the Gram-negative pathogens *Pseudomonas aeruginosa* and *Acinetobacter baumannii*, or the constitutive efflux pumps observed in many bacteria.

Acquired resistance occurs when an originally sensitive microbe becomes resistant by incorporating new genetic material (e.g., plasmids, transposons, integrons and naked DNA), by lateral gene transfer, or as a result of mutation. These mutational events may lead to a large increase in the minimal inhibitory concentration [23][24][25].

Adaptive resistance involves a temporary increase in the ability of a bacterium to survive in the presence of a toxicant by alterations in gene and/or protein expression as a result of exposure to an environmental trigger, e.g., stress, nutrient conditions, growth state and sub-inhibitory levels of the toxicant or antibiotics themselves.

Intrinsic and acquired resistance mechanisms are stable and can be transmitted vertically to subsequent generations. Adaptive resistance has a transient character and usually reverts upon the removal of the inducing condition [23].

Several microorganisms that demonstrate tolerance/resistance to Te compounds have been isolated from various environments [26]. The first group of organisms horizontally obtained genetic equipment harboured in mobile genetic elements, the most commonly being plasmids, or integrated such genes (e.g., *ter* genes in *E. coli*) into their chromosomes and organized them into genomic islands consisting of gene clusters or operons with various gene compositions [27]. The second group of organisms evolved other mechanisms enabling cellular enzymes to be used for the reduction of the noxious effects of toxicants. In the first-mentioned group, gene clusters enable resistance against toxicants, whereas the latter triggers the ability to tolerate/adapt to life in a toxic environment.

The mechanism of such a type of resistance is based on either a chromosomal mutation or on the acquisition of a TeO_3^{2-} resistance plasmid that leads to the inability to transport TeO_3^{2-}, the acquisition of an efflux mechanism or the detoxification of the inhibitor [28]. The direct efflux mechanism evolved to prevent the intracellular accumulation of toxic compounds and involves energy-dependent systems that pump such molecules out of the cell in a process that does not alter or degrade the toxicants [23]. A decreased influx and increased efflux of TeO_3^{2-} is not responsible for the TeO_3^{2-} resistance of *E. coli* [29]. [30] revealed that a decreased uptake by an acetate transport system, based on tellurite-acetate competition, is responsible for TeO_3^{2-} uptake and resistance in *Rhodobacter capsulatus* [31].

One of the mechanisms of TeO_3^{2-} toxicity is based on its high oxidizing ability. The selection of resistance to TeO_3^{2-} and of the ability of some bacteria to reduce TeO_3^{2-} to less toxic elemental Te has been carried out for decades in specific media containing TeO_3^{2-} in various concentrations. As mentioned above, the reduction of Te/Se compounds in such modified cell media is accompanied by black/red colony formation, respectively. Despite the emergence of genomic approaches, this chromogenic-based selection continues to be extensively used [20][21].

Several key cellular enzymes are reported to be involved in the defence against ROS generated during TeO_3^{2-} reduction [32]. Regardless of the concentration of TeO_3^{2-}, this results in the generation of black or grey deposits of metallic Te^0 inside the cell [33]. [34] who also pointed out that TeO_3^{2-} reduction accompanied by black/grey colony formation and metallic Te crystal formation in obligate aerobic photosynthetic bacteria was not essential for the maintenance of high TeO_3^{2-} resistance. White-colony formation without TeO_3^{2-} reduction has also been described in highly tellurite-resistant *E. coli* strains [35].

Bonificio and Clarke [36] have proposed a steady-state system of Te compounds in *Pseudoalteromonas spp*. They have demonstrated that, even though many Te compounds are considered insoluble, they can nonetheless be transformed and suggest that a steady-state soluble Te concentration exists during Te transformation. This system is based on solid Te sources (tellurium dioxide, autoclave slime, cadmium telluride, bismuth telluride) being able to dissolve to yield soluble TeO_3^{2-}. The production of organic Te compounds, such as dimethyltelluride, is considered to be one of the detoxification mechanisms of bacteria [37][38].

harbouring additional *ter* genes was found in the opportunistic pathogen *Serratia marcescens* [39]. Several different *Escherichia coli* pathogens also possess *ter* gene clusters. have *ter* gene clusters incorporated into large plasmids. They function in bacteria, mitochondria and chloroplasts as membrane protein insertases.

A summary of all TeO_3^{2-} resistance mechanisms proposed to date is provided in Table 1. However, some of the mechanisms are not as yet supported by experimental evidence.

3. Tellurium Toxicity vs Potential Benefits for Prokaryotes and Eukaryotes

When Te compounds enter into cells, they can induce (I) changes in the integrity of cellular membrane structures [40], (II) changes in glutathione metabolism, (III) substitution of metal in enzymes and (IV) oxidative stress [41]. The common feature of these metal(loid)s is their chemical affinity to proteins and to non-protein thiols and their ability to generate cellular oxidative stress by the Fenton reaction. Te oxyanions (TeO_3^{2-}) are involved in the thiol: redox system of the cell and interfere with thiol: redox enzymes (glutathione reductase and thioredoxin reductase) and with their metabolites (glutathione, glutaredoxin and thioredoxin) [29]. The key target for SeO_3^{2-} and TeO_3^{2-} cellular processing is glutathione, which participates in TeO_3^{2-} to Te (Te^0) reduction [42] accompanied by reactive oxygen species (ROS) formation [43].

Glutathione is an endogenous tripeptide consisting of cysteine, glutamate and glycine and has antioxidative and other metabolic functions [44]. They can additionally be used in the treatment of a wide range of other disorders including poisoning with heavy metals and other compounds and even in COVID-19 disease therapy [45]. [46] have demonstrated, in their review, that the metabolic pathway of glutathione biosynthesis consists of two sequential ATP-dependent reactions allowing the synthesis of γ-glutamylcysteine from L-glutamate and L-cysteine, followed by the formation of glutathione by the addition of glycine to the C-terminal end of γ-glutamylcysteine [47]. These reactions are catalysed by γ-glutamylcysteine synthetase and glutathione synthetase.

The clinical manifestation of the ingestion of metal-oxidizing solutions containing substantial concentrations of Te include vomiting, nausea, metallic taste, black discoloration of the oral mucosa and skin, corrosive gastrointestinal tract injury and a characteristic garlic-like odour of the breath [48]. When exposure is limited, the mucous membrane and pulmonary system might become irritated. In animals, extensive exposure to hydrogen telluride have resulted in haemolysis, haemoglobinuria, anuria, jaundice and pulmonary oedema, symptoms similar to toxicity from the inhalation of arsine or the poisonous gas stibine [49]. The toxicity of Te, Se and As resides is associated with analogical secondary metabolite production, e.g., dimethyl telluride ($(CH3)_2Te$), dimethyl selenide ($(CH3)_2Se$) and monomethyl ($CH_3AsO(OH)_2$) and dimethyl arsenic acid ($(CH3)_2AsO_2H$), all of which have a characteristic garlic odour.

Experiments on rats and mice have revealed that, after TeO_3^{2-} ingestion, the peripheral nerves become transiently demyelinated because of the inhibition of squalene epoxidase (squalene monooxygenase) [50]. The mechanism of squalene epoxidase inhibition resides in the binding of methyltellurium compounds or TeO_3^{2-} itself to the thiol groups of the cysteine residues at the active site [51]. Another group of enzymes affected by organotellurium compounds toxicity belongs to the glutamatergic system. Neurotoxic diphenyl ditelluride is able to alter enzymes of the glutamatergic system because of its interaction with the thiol groups of cysteine-containing enzymes and proteins [52].

The human body is known to metabolize and excrete Te, even if the exact metabolic pathways are not understood. TeO_3^{2-} is methylated resulting in dimethyltellurium ($(CH3)_2Te$) and finally trimethyltellurium ($(CH3)_3Te^+$). [1] suggest that these methylated species are the most abundant forms of Te in the human body; they are found in the kidney, in the spleen and in the lungs. Finally, Te leaves the human body via urine and via breath as volatile $(CH3)_2Te$, which is responsible for the garlic-like odour.

The chemical elements Te and Se are members of the chalcogen group, with Se forming part of the amino acid selenocysteine. The selenocysteine-containing biomolecules, are essential for the cell to resist oxidative stress condition [53]. Hence, Se is an essential trace element for both prokaryotic and eukaryotic biological systems at low concentrations but is toxic at higher levels [54].

The importance of naturally present trace elements in human food is undisputed. Mineral trace elements such as Se, Zn and Cu are members of the group of micronutrients with antioxidative ability. Organic Se in food is found in selenomethionine and selenocysteine, whereas inorganic Se occurs as SeO_3^{2-} (more toxic form) and selenate (SeO_4^{2-}, less toxic form). The bioavailability of these compounds allows them to be easily absorbed by plants and animals from Se-rich soil or water.

Duganella violacienigra can reduce both Se and Te oxyanions and so it can be exploited in bioremediation and in eco-friendly approaches to produce rare element nanoparticles, rather than synthesising them by chemical means [55]. Se, Te and other elements such as cadmium (Cd) and sulphur (S) are also used in the production of semiconductor nanoparticles (NPs) or quantum dots (QDs) with unique fluorescent properties and great technological potential [56][57]. Te-containing nanoparticles also exhibit antibacterial properties against *E. coli*, with no apparent cytotoxicity against eukaryotic cells [58]. In connection with Te, plant accumulation from the soil is preferred and the further harvesting and absorption of Te from polluted water via plant roots for further decontamination holds promise [59].

Microorganisms, in general, play a crucial role in the biological transformation of SeO_3^{2-} and SeO_4^{2-} via metabolic reactions. The reduction of both forms, SeO_3^{2-} and SeO_4^{2-}, to Se^0 has been identified as an ideal strategy for Se detoxification and Se recovery in contaminated water, soil and industrial effluent [60]. A variety of microorganisms are used as microbial factories for the bioproduction of Se nanoparticles, e.g., *Enterobacter cloacae* [61], *Bacillus cereus* [62], *Duganella sp*., *Agrobacterium sp.* [63], *Bacillus mycoides* [64],

The concentration of Se in food depends on environmental conditions and thus its concentration in soil [65]. The food intake of nutrient antioxidants is therefore considerably reduced in comparison with recommendations in Europe. The best way to supplement food lacking antioxidants is their addition in an inorganic form or rather as selenoproteins. The yeasts *Saccharomyces cerevisiae* and bacterial strains *Escherichia coli* and *Lactobacillus spp*.

Since the chemistry of Te slightly resembles that of sulphur, Te can be incorporated into amino acids such as cysteine and methionine and subsequently into proteins and enzymes, as mentioned above. Telluromethionine and tellurocysteine are suitable for highly sensitive fluorescent imaging methods as biomarkers. ^{123m}Te can be synthesized as compounds with fatty acids. Te-containing fatty acids, Te-amino acids and many fluorescent

Compared with Se-containing compounds (e.g., ebselen and selenocystamine), some of the tellurides are significantly effective compounds. Some of the organotellurium compounds exhibit promising antioxidant activity in cell culture. [66] suggest that these compounds might lead to the development of a new class of water-soluble Te-based antioxidant and Zn-releasing drugs. telluride have potent antioxidative properties; bis(4-aminophenyl) telluride has higher glutathione peroxidase-like activity and protects against peroxynitrite-mediated oxidation more efficiently than its Se analogue or ebselen [67].

Inorganic salts of Te and a wide range of diverse organotellurium compounds show potential in diagnostics, pharmacology and therapy. They may provide the basis for innovative drug development in the future [1]. They are powerful agents in protein and enzyme inhibition, they can kill a wide range of microorganisms including bacteria and plasmodia and they are able to induce apoptosis of specific cancer cells.

Extreme environments are rarely distributed evenly over the Earth but they harbour a relatively high proportion of microorganisms considered valuable to science and technology [68], such as bacteria producing antibiotics [69] and bioactive molecules and bacteria useful in the biodegradation of pollutants [70]. Such environments provide habitats for representatives of various genera that possess the ability to resist and reduce elevated levels of toxic metal(loid)s compounds, specifically, Te compounds. These extreme environments are rich in heavy metals and metalloids and present excellent sites for the isolation of metal-resistant microorganisms.

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