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The antioxidant action of taurine, hypotaurine and their metabolic precursors

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It has been suggested that taurine, hypotaurine and their metabolic precursors (cysteic acid, cysteamine and cysteinesulphinic acid) might act as antioxidants *in vivo*. The rates of their reactions with the biologically important oxidants hydroxyl radical ($^{\cdot}OH$), superoxide radical ($O_2^{\cdot-}$), hydrogen peroxide (H_2O_2) and hypochlorous acid (HOCl) were studied. Their ability to inhibit iron-ion-dependent formation of $^{\cdot}OH$ from H_2O_2 by chelating iron ions was also tested. Taurine does not react rapidly with $O_2^{\cdot-}$, H_2O_2 or $^{\cdot}OH$, and the product of its reaction with HOCl is still sufficiently oxidizing to inactivate α_1-antiproteinase. Thus it seems unlikely that taurine functions as an antioxidant *in vivo*. Cysteic acid is also poorly reactive to the above oxidizing species. By contrast, hypotaurine is an excellent scavenger of $^{\cdot}OH$ and HOCl and can interfere with iron-ion-dependent formation of $^{\cdot}OH$, although no reaction with $O_2^{\cdot-}$ or H_2O_2 could be detected within the limits of our assay techniques. Cysteamine is an excellent scavenger of $^{\cdot}OH$ and HOCl; it also reacts with H_2O_2, but no reaction with $O_2^{\cdot-}$ could be measured within the limits of our assay techniques. It is concluded that cysteamine and hypotaurine are far more likely to act as antioxidants *in vivo* than is taurine, provided that they are present in sufficient concentration at sites of oxidant generation.

INTRODUCTION

Taurine (2-aminoethanesulphonic acid) is present at millimolar concentrations in many animal tissues, especially nervous tissue, retina and neutrophils [1,2]. Its precise metabolic function is unclear, but there have been several suggestions that taurine acts as an antioxidant *in vivo* [1–3]. Some doubt is cast on these suggestions by the observation that reaction of taurine with the powerful oxidant hypochlorous acid (HOCl), generated at sites of inflammation, yields a taurine chloroamine that is still sufficiently oxidizing to inactivate α_1-antiproteinase [2,4]. Hypotaurine, the metabolic precursor of taurine [1], has also been suggested to act as an antioxidant *in vivo* by scavenging highly reactive hydroxyl radicals ($^{\cdot}OH$) [5]. The metabolic precursors of hypotaurine, such as cysteamine, cysteinesulphinic acid and cysteic acid, have also been proposed to act as antioxidants in brain [3].

However, the mere demonstration that a compound is able to react with an oxidant *in vitro* does not prove that it acts as a scavenger *in vivo*. For example, almost all non-steroidal anti-inflammatory drugs are capable of reacting with $^{\cdot}OH$ [6,7] and with HOCl [8,9]. However, only in a few cases is the reaction fast enough for scavenging to be feasible at the concentrations of drug present *in vivo* during the therapeutic regimens usually employed [6,8,9].

In order to help evaluate the proposals that taurine [1,2], hypotaurine [3,5] and their metabolic precursors [3] might be able to act as oxidant scavengers *in vivo*, we investigated the rates of reaction of these molecules with four oxidants. One oxidant is the highly reactive $^{\cdot}OH$. Production of $^{\cdot}OH$ in biological systems usually involves metal-ion-dependent decomposition of hydrogen peroxide (H_2O_2); iron ions are the most likely catalyst of $^{\cdot}OH$ formation *in vivo* (reviewed in ref. [10]). Hence the ability of these 'scavengers' not only to react directly with $^{\cdot}OH$ but also to bind iron ions in ways that influence $^{\cdot}OH$ production from H_2O_2 was examined. Secondly, scavenging of the myeloperoxidase-derived oxidant HOCl was studied; HOCl is known to contribute to tissue damage at sites of inflammation [2,4,11]. Also, the ability of the compounds to react with superoxide radicals ($O_2^{\cdot-}$) and H_2O_2 was examined. Both $O_2^{\cdot-}$ and H_2O_2 are much less reactive than $^{\cdot}OH$ or HOCl, but they can still find targets within certain cells at which they can do direct damage [12,13]. $O_2^{\cdot-}$ and H_2O_2 can also cause formation of $^{\cdot}OH$ in the presence of suitable metal ion catalysts (reviewed in ref. [10]).

MATERIALS AND METHODS

Reagents were of the highest quality available from Sigma Chemical Co. or from BDH Chemicals. KCNS was carefully dried in an oven overnight before solutions were made up. α_1-Antiproteinase was Sigma type A9024. Elastase and α_1-antiproteinase were assayed essentially as described in ref. [14]; full details are given in the legend to Table 2. HOCl was produced immediately before use by adjusting NaOCl to pH 6.2 with dil. H_2SO_4 [14]. Deoxyribose degradation in the presence of ascorbate, H_2O_2 and $FeCl_3$ (with or without EDTA) was measured as described in ref. [15]. Pulse radiolysis was performed by using the Paterson Laboratories linear accelerator facility [16], with 10 mM-KH_2PO_4/KOH solutions at pH 7.0. All solutions to be evaluated for scavenging activity were made up immediately before use and, where necessary, the pH of solutions was adjusted to 7.4. Generation of $O_2^{\cdot-}$ by the hypoxanthine/xanthine oxidase system was carried out essentially as described in ref. [17]. Reaction mixtures contained, in a final volume of 3 ml, 0.1 ml of 30 mM-EDTA, 10 μl of 30 mM-hypo-

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xanthine in 50 mM-KOH, 100 μl of 3 mM-cytochrome *c* or 3 mM-Nitro Blue Tetrazolium, and 50 mM (final concentration)-KH_2PO_4/KOH buffer, pH 7.4. Reaction was started by adding 0.2 ml of xanthine oxidase (freshly diluted in the above phosphate buffer to give 1 unit of enzyme activity/ml) and the rate of Nitro Blue Tetrazolium or cytochrome *c* reduction was measured at 560 nm or 550 nm respectively in a recording spectrophotometer at 25 °C.

H_2O_2 was measured by the formation of a brown colour (recorded at 436 nm) in reaction mixtures containing, in a final volume of 1 ml, 0.15 M-KH_2PO_4/KOH buffer, pH 7.4, 50 μl of guaiacol solution (100 μl of pure liquid in 50 ml of water) and 10 μl of Sigma type IV horseradish peroxidase (5 mg/ml in the same phosphate buffer). The rate of absorbance change at 436 nm is proportional to the concentration of H_2O_2 added. Substances to be tested for their reaction with H_2O_2 were incubated at concentrations up to 14.40 mM with 3.53–10.00 mM-H_2O_2 for 30 min at 25 °C. Samples were then taken and assayed for remaining H_2O_2 by using the peroxidase system. For studies of the reaction of cysteamine with H_2O_2, they were incubated in a 5 ml reaction mixture containing 1.5 ml of 100 mM-KH_2PO_4/KOH buffer, pH 7.4, for various times at 25 °C. At intervals, 0.25 ml samples were added to another reaction mixture containing 2.5 ml of buffer and 0.25 ml of 6 mM-5,5′-dithiobis-(2-nitrobenzoic acid) (Ellman's reagent). The absorbance at 412 nm was measured after 5 min and the concentration of thiol was determined from a calibration curve. The A_{412} was proportional to cysteamine concentrations up to 3 mM in the 5,5′-dithiobis-(2-nitrobenzoic acid) assay mixture.

RESULTS

Scavenging of $^{\bullet}OH$ generated by pulse radiolysis

Radiolysis of a dilute (10 mM) aqueous phosphate-buffered (pH 7.0) solution saturated with N_2O produces $^{\bullet}OH$:

$$H_2O \text{—}\!\!\sim\!\!\rightarrow {}^{\bullet}OH, e^-_{aq.}, H^{\bullet}, H_2O_2, H_2 \quad (1)$$

$$e^-_{aq.} + N_2O + H_2O \rightarrow {}^{\bullet}OH + OH^- + N_2 \quad (2)$$

If KCNS is added to the solution, the $^{\bullet}OH$ radical reacts with CNS^- to give the radical anion $(SCN)_2^{\bullet-}$:

$$^{\bullet}OH + CNS^- \rightarrow CNS(OH)^- \quad (3)$$

$$CNS(OH)^- \rightarrow CNS^{\bullet} + OH^- \quad (4)$$

$$CNS^{\bullet} + CNS^- \rightarrow (CNS)_2^{\bullet-} \quad (5)$$

At pH 7 reactions (4) and (5) are non-rate-determining and so the production of $(CNS)_2^{\bullet-}$ from KCNS can be considered as a single oxidation step with a second-order rate constant of 1.1×10^{10} $M^{-1} \cdot s^{-1}$ [18]. The $(CNS)_2^{\bullet-}$ radical ion absorbs strongly in the visible region ($\epsilon = 7.1 \times 10^3$ $M^{-1} \cdot cm^{-1}$ at 500 nm).

By observing the ability of taurine, hypotaurine and their metabolic precursors to compete with CNS^- for $^{\bullet}OH$ and so decrease the absorbance changes observed, rate constants for their reaction with $^{\bullet}OH$ can be calculated. Fig. 1(*a*) shows that hypotaurine and cysteamine are excellent scavengers of $^{\bullet}OH$; rate constants (as the mean of three values that differed by no more than 10%) were 1.15×10^{10} and 5.9×10^9 $M^{-1} \cdot s^{-1}$ respectively. By contrast, taurine (k_2 2.42×10^6 $M^{-1} \cdot s^{-1}$) and L- or DL-cysteic acid (5.3×10^7 $M^{-1} \cdot s^{-1}$) were poor $^{\bullet}OH$ scavengers.

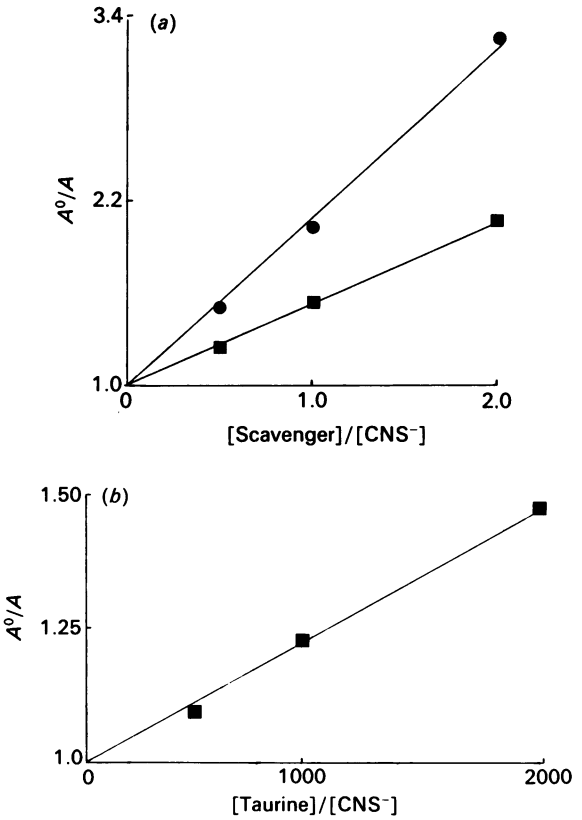

Fig. 1. Determination of rate constants for the reactions of taurine, hypotaurine and cysteamine with $^{\bullet}OH$ by competition with CNS^-

The absorbance at 500 nm of an irradiated solution of KCNS (100 μM) in 10 mM-phosphate buffer saturated with N_2O, pH 7.0, was determined in the presence (A) or in the absence (A^0) of scavenger. Absorbances are related by the equation:

$$\frac{A^0}{A} = 1 + \frac{k_c}{k_1} \cdot \frac{[\text{added scavenger}]}{[CNS^-]}$$

Thus plotting A^0/A against the [scavenger]/[KCNS] concentration ratio allows calculation of k_c, the rate constant for reaction of scavenger with $^{\bullet}OH$. k_1 is taken as 1.10×10^{10} $M^{-1} \cdot s^{-1}$ (see the text). (*a*) Scavenging by hypotaurine (●) and cysteamine (■); (*b*) scavenging by taurine.

Fig. 1(*b*) shows the results of a typical experiment on $^{\bullet}OH$ scavenging by taurine.

Scavenging of $O_2^{\bullet-}$

A mixture of hypoxanthine and xanthine oxidase at pH 7.4 generates $O_2^{\bullet-}$, which can be measured by its ability to reduce ferricytochrome *c* to ferrocytochrome *c*, measured as an increase in absorbance at 550 nm [19]. $O_2^{\bullet-}$ can also reduce Nitro Blue Tetrazolium (NBT), measured as an increase in absorbance at 560 nm [20]. Any added substance that is itself able to react with $O_2^{\bullet-}$ ion should decrease the rate of these absorbance changes. It was found that 1.5 mM final concentrations of taurine, hypotaurine, cysteamine or cysteinesulphinic acid did not decrease the rate of reduction of 100 μM-cytochrome *c* by the hypoxanthine/xanthine oxidase system (none of these substances themselves reduced

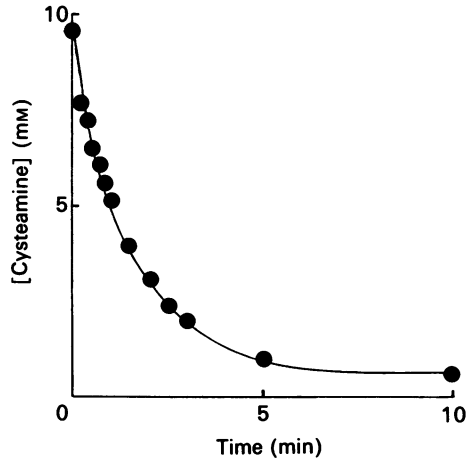

Fig. 2. Reaction of cysteamine with H_2O_2

H_2O_2 (10.6 mM) and cysteamine (9.6 mM) were incubated in a final volume of 5 ml at pH 7.4 at 25 °C. At intervals samples of the reaction mixture were removed and added to a separate reaction mixture containing 5,5′-dithiobis-(2-nitrobenzoic acid) (for full details see the Materials and methods section). The concentration of cysteamine remaining in the reaction mixture was calculated by using a calibration plot of A_{412} against cysteamine concentration.

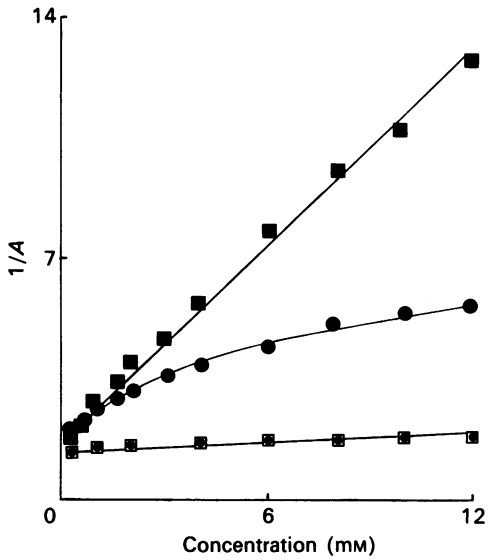

Fig. 3. Inhibition of deoxyribose degradation by ·OH in the presence of hypotaurine and the lack of effect of taurine

Reaction mixtures contained, in a final volume of 1.0 ml, the following reagents at the final concentrations stated [15]: deoxyribose (2.8 mM), KH_2PO_4/KOH buffer, pH 7.4 (20 mM), iron ion (10 μM-$FeCl_3$ or 10 μM-$FeCl_3$ plus 100 μM-EDTA where indicated), H_2O_2 (1 mM), scavenger (hypotaurine or taurine) and ascorbate (100 μM). Reaction mixtures were incubated at 25 °C for 1 h and colour developed by heating with thiobarbituric acid at low pH [15]. The A_{532} is a measure of the extent of ·OH attack on deoxyribose. Rate constants were calculated from the slope of the competition plots as described in ref. [15]. ■, Hypotaurine, EDTA present; ●, hypotaurine, no EDTA present; ◙, taurine, EDTA present.

cytochrome *c* or Nitro Blue Tetrazolium directly under our experimental conditions). Taurine and hypotaurine at 3 mM final concentrations had no effect on the rate of reduction of 100 μM-Nitro Blue Tetrazolium by $O_2^{\cdot-}$; 3 mM-cysteamine or -cysteinesulphinic acid decreased Nitro Blue Tetrazolium reduction by no more than 13–15%.

Under our reaction conditions cytochrome *c* reacts with $O_2^{\cdot-}$ with a second-order rate constant of 2.6×10^5 $M^{-1} \cdot s^{-1}$ [21], and Nitro Blue Tetrazolium reacts with a rate constant of about 6×10^4 $M^{-1} \cdot s^{-1}$ [22]. The inability of taurine, hypotaurine, cysteamine or cysteinesulphinic acid, at concentrations 15-fold greater than those of cytochrome *c* or 30 times greater than those of Nitro Blue Tetrazolium, to decrease significantly the rates of cytochrome *c* or Nitro Blue Tetrazolium reduction suggests that their reactions with $O_2^{\cdot-}$, if any, proceed with rate constants less than 10^3 $M^{-1} \cdot s^{-1}$.

Scavenging of H_2O_2

Incubation of taurine, hypotaurine, DL- or L-cysteic acid or cysteinesulphinic acid at concentrations up to 14.4 mM with 3.53–10 mM-H_2O_2 for 30 min at 25 °C or 37 °C caused no significant loss of H_2O_2, as measured by a peroxidase/guaiacol assay (described in the Materials and methods section). It thus seems unlikely that they could be effective scavengers of the much lower concentrations of H_2O_2 present under physiological conditions. Cysteamine was found to interfere with the peroxidase-based assay system for H_2O_2, probably because thiols are substrates for horseradish peroxidase [23]. Its rate of reaction with H_2O_2 was therefore monitored as loss of the thiol group, measured with 5,5′-dithiobis-(2-nitrobenzoic acid) [24]. Initial experiments showed that when cysteamine (final concentration 0.24–2.4 mM) was incubated with H_2O_2 (10.6 mM final concentration) at 25 °C the initial rate of loss of thiol was proportional to the concentration of cysteamine. A second-order rate constant of approx. 1.7 $M^{-1} \cdot s^{-1}$ was calculated from the data. Fig. 2 shows the extent of loss of cysteamine when a 9.6 mM final concentration was incubated with 10.6 mM-H_2O_2.

Hence cysteamine does react slowly with H_2O_2, but the low rate constant for this reaction suggests that it may not be significant at the very low concentrations of H_2O_2 likely to be present *in vivo*.

Scavenging of ·OH generated by ascorbate/iron ion/H_2O_2 systems

A mixture of ascorbic acid, H_2O_2 and a suitable iron complex generates ·OH at pH 7.4 [15]. This ·OH may be measured by its ability to degrade the sugar deoxyribose into fragments that, on heating with thiobarbituric acid at low pH, generate a pink chromogen. If iron is added to the reaction mixture as an $FeCl_3$–EDTA complex, ·OH is generated by the reactions:

$$Fe^{3+}\text{–EDTA} + \text{ascorbate} \rightarrow Fe^{2+}\text{–EDTA} + \text{oxidized ascorbate} \quad (6)$$

$$Fe^{2+}\text{–EDTA} + H_2O_2 \rightarrow Fe^{3+}\text{–EDTA} + {\cdot}OH + OH^- \quad (7)$$

Any ·OH that escapes scavenging by the EDTA is equally accessible to deoxyribose and to any added 'scavenger' of ·OH [15]. Thus the ability of a scavenger to inhibit deoxyribose degradation in this system depends only on its concentration relative to deoxyribose and on its rate constant for reaction with ·OH.

Fig. 3 shows the ability of hypotaurine to inhibit

Table 1. Rate constants obtained by the deoxyribose method: a comparison with pulse radiolysis

Cysteamine was not studied in the deoxyribose system because its ability to react with H_2O_2 (Fig. 2) interferes with the assay. Values given are the means of at least three observations that differed by no more than 10%. Abbreviation: N.D., not determined.

Compound	Second-order rate constant for reaction with ·OH ($M^{-1} \cdot s^{-1}$)	
	By pulse radiolysis	By deoxyribose method ($FeCl_3$–EDTA/H_2O_2/ascorbate)
Hypotaurine	1.15×10^{10}	5.00×10^9
Cysteamine	5.90×10^9	N.D.
Taurine	2.42×10^6	1.40×10^7
DL- or L-Cysteic acid	5.30×10^7	1.60×10^8
Cysteinesulphinic acid	N.D.	3.20×10^9

deoxyribose degradation in this system. From the slope of the competition plot obtained, a rate constant of 5×10^9 $M^{-1} \cdot s^{-1}$ was calculated, slightly lower than the definitive value obtained by pulse radiolysis, but broadly comparable (Table 1). In other experiments taurine and cysteic acid (DL- or L-form) were found to be poor scavengers of ·OH (Table 1). Fig. 3 shows the poor scavenging ability of taurine as compared with hypotaurine, whereas cysteinesulphinic acid was a good scavenger (Table 1).

If iron is added to the deoxyribose assay system as $FeCl_3$ (not complexed with EDTA), some of the iron ions bind to deoxyribose [27] and deoxyribose degradation becomes site-specific, i.e. ·OH is formed by iron ions bound to deoxyribose and immediately attacks the deoxyribose molecule [6,25–28]. Most ·OH scavengers fail to inhibit this deoxyribose degradation, presumably because they cannot interfere with site-specific ·OH attack [6,25–28]. Evidence suggests that the only molecules that can prevent deoxyribose degradation under these conditions are those that themselves complex iron ions, in forms poorly reactive in generating ·OH, and so remove iron ions from the deoxyribose [6,25–28]. Fig. 3 shows that hypotaurine is able to inhibit deoxyribose degradation in the absence of EDTA (although less well than in the presence of EDTA), suggesting that it is able to interfere with site-specific ·OH generation in this system. By contrast, taurine had no effect in the absence of EDTA, and cysteinesulphinic acid only a very slight inhibitory effect (results not shown).

Scavenging of hypochlorous acid: protection of α_1-antiproteinase

HOCl is produced by oxidation of Cl^- ions at sites of inflammation by the neutrophil enzyme myeloperoxidase [4,11]. One of the major extracellular targets of HOCl attacks is α_1-antiproteinase, the major circulating inhibitor of serine proteinases such as elastase [11,29]. α_1-Antiproteinase is rapidly inactivated by HOCl, losing its elastase-inhibitory capacity [4,11,14,29]. A good scavenger of HOCl should therefore be able to protect α_1-antiproteinase against inactivation.

Table 2. Inactivation of α_1-antiproteinase by HOCl: effect of scavengers

α_1-Antiproteinase (0.1 mg/ml), HOCl (60 μM) and scavenger (if any) were incubated in a final volume of 1.0 ml in phosphate-buffered saline, pH 7.4 (full details in ref. [14]), at 25 °C for 30 min. Then 2 ml of phosphate-buffered saline and 0.05 ml of elastase were added, followed by further incubation at 25 °C for 20 min. This allows any α_1-antiproteinase still active to inhibit elastase. (Any HOCl remaining is diluted out to the point at which it cannot affect elastase itself). The remaining elastase activity was then measured by adding elastase substrate [14], which is hydrolysed by elastase, resulting in an increase in A_{410} [14]. Concentrations of scavengers added were those present in the first (1.0 ml) reaction mixtures; scavengers and α_1-antiproteinase were mixed together before the addition of HOCl. Control experiments showed that none of the substances tested themselves affected elastase activity or interfered with the ability of α_1-antiproteinase to inhibit it.

Addition to first reaction mixture	Elastase activity in final reaction mixture (A_{410}/s)	Comment
Buffer only	3.10×10^{-3}	Activity of uninhibited elastase
α_1-Antiproteinase	0	α_1-Antiproteinase inhibits elastase: no activity detected
α_1-Antiproteinase + HOCl	3.18×10^{-3}	α_1-Antiproteinase inactivated by HOCl; no longer inhibits elastase
α_1-Antiproteinase + 240 μM-taurine + HOCl	2.9×10^{-3}	Taurine does not protect α_1-antiproteinase against inactivation
α_1-Antiproteinase + 2 mM-taurine + HOCl	2.95×10^{-3}	
α_1-Antiproteinase + 240 μM-L-cysteic acid + HOCl	2.8×10^{-3}	Cysteic acid only slightly protects α_1-antiproteinase against inactivation
α_1-Antiproteinase + 1.2 mM-L-cysteic acid + HOCl	2.2×10^{-3}	
α_1-Antiproteinase + 1.2 mM-DL-cysteic acid + HOCl	1.9×10^{-3}	
α_1-Antiproteinase + 240 μM-cysteamine + HOCl	0	Cysteamine, hypotaurine and cysteine sulphonic acid scavenge HOCl rapidly enough to protect α_1-antiproteinase
α_1-Antiproteinase + 240 μM-hypotaurine + HOCl	0	
α_1-Antiproteinase + 240 μM-cysteinesulphinic acid + HOCl	0	

Table 2 (second line) shows that α_1-antiproteinase inhibited the activity of pig pancreatic elastase *in vitro*; a concentration of α_1-antiproteinase able to inhibit elastase completely was used. Incubation of α_1-antiproteinase with HOCl led to loss of its elastase-inhibitory capacity (Table 2, third line). Inclusion of taurine at concentrations up to 2 mM during the incubation of HOCl with α_1-antiproteinase did not protect the protein against inactivation. This is presumably because the product of reaction of taurine with HOCl is itself capable of inactivating α_1-antiproteinase [4,11]. Similarly, cysteic acid (L- or DL-) was not able to protect α_1-antiproteinase unless high (> 1 mM) concentrations were used. By contrast, hypotaurine, cysteamine and cysteinesulphinic acid were able to protect α_1-antiproteinase completely against the effects of 60 μM-HOCl at only 240 μM concentrations (control experiments showed that none of these compounds was able to inhibit elastase directly, or to interfere with the ability of α_1-antiproteinase to inhibit elastase). Thus hypotaurine, cysteamine and cysteinesulphinic acid are powerful scavengers of HOCl, and any products of their reaction with HOCl do not themselves inactivate α_1-antiproteinase.

DISCUSSION

Although taurine has been observed to accumulate at high concentrations in some mammalian tissues [1–3], it seems unlikely to act as an antioxidant *in vivo*. It does not appear to react with H_2O_2 or with $O_2^{\cdot-}$ (within the limits of the assay procedures used), it scavenges $^{\cdot}OH$ about two orders of magnitude less well than such compounds as glucose or mannitol, it does not appear to bind iron ions in ways that affect $^{\cdot}OH$ generation, and the product of its reaction with HOCl is still sufficiently oxidizing to inactivate α_1-antiproteinase, the major extracellular target of HOCl attack *in vivo*.

Fellman & Roth [5] have argued that hypotaurine could act as a scavenger *in vivo*. Our experiments show that this compound is a good scavenger of HOCl and $^{\cdot}OH$ *in vitro*, appears to inhibit weakly iron-dependent 'site-specific' $^{\cdot}OH$ damage to the sugar deoxyribose (Fig. 3), but does not react with H_2O_2 or $O_2^{\cdot-}$ (within the limits of our assay methods). Of course, for hypotaurine to function as an antioxidant *in vivo* would require its presence, at sufficient concentration, at sites of oxidant generation. Whether this occurs remains to be established.

Cysteamine and cysteinesulphinic acid were also good scavengers of HOCl and $^{\cdot}OH$, but not of $O_2^{\cdot-}$. Cysteamine also reacts slowly with H_2O_2, but this reaction can lead to significant rates of H_2O_2 removal if high (millimolar) concentrations of cysteamine are present. Cysteamine is also a well-established radioprotective agent [30]. Thus the suggestion by Schurr & Rigor [3] that some metabolic precursors of taurine might act as antioxidants in brain is feasible, provided that they accumulate to significant concentrations. It must also be realized that products of reaction of such compounds with oxidants (e.g. peroxy radicals or thiyl radicals) might themselves exert deleterious biological effects under certain circumstances [7,31].

Thus it seems unlikely that the true function of taurine *in vivo* is to act as an antioxidant. Indeed, its metabolic formation seems to consume better antioxidants such as cysteamine, cysteinesulphinic acid and hypotaurine.

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