Synergy in the Autoxidation of S(IV) Inhibited by Phenolic Compounds

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The effects of phenol (C_6H_5OH), catechin [$C_6H_4(OH)_2$], and gallic acid [$HOOCC_6H_2(OH)_3$] on the kinetics of aqueous SO_2 oxidation by molecular oxygen have been investigated for dilute systems, as pertain to the chemistry of troposphere and surface waters. In experiments performed under batch (homogeneous oxidation) or semibatch (heterogeneous oxidation) conditions, we measured the decay of oxygen with a Yellow Spring Instruments probe or the increase in S(VI) concentration with an Orion conductivity cell. All of the studied phenolic compounds caused inhibition of the uncatalyzed autoxidation of S(IV), but the inhibiting effectiveness of phenol was much lower than that of catechin and gallic acid. The dual role of phenol in not only inhibiting but also promoting action was demonstrated in experiments carried out in the presence of Co or Mn catalyst when the synergy effect was evident. Another extraordinary behavior was observed in the case of gallic acid at prolonged experiments leading to oscillations in the rate of S(VI) formation. The results are discussed in terms of a radical chain reaction with termination by a step first order in $SO_4^{\bullet -}$ radicals and in a phenolic compound, with initiation supplemented by a step first order in phenoxyl radicals and in aqueous S(IV) species. From such a model of the inhibited autoxidation of S(IV), we determined the following rate constants for $SO_4^{\bullet}$ scavenging: $4.3 \times 10^9\ M^{-1}\ s^{-1}$ for catechin and $2.6 \times 10^9\ M^{-1}\ s^{-1}$ for gallic acid. The consequences of this inhibition in the environment are discussed.

Introduction

Phenolic compounds are known as effective inhibitors for the autoxidation of both organic and inorganic substances. They function as chain breakers by receiving excess energy from a "hot chain molecule",[1] as termed almost a century ago, or by scavenging free radicals, transients in a chain reaction,[2] as understood presently. Since the beginning of studies on inhibition by phenolics, it has been believed that this process is followed by oxidative transformations of the inhibitors.[3] The period in which an inhibitor undergoes slow oxidation corresponds to that of its effective inhibiting action. The agents causing this effect are usually oxidizing radicals, e.g., Br_2^- and sulfoxy radicals,[4] or organic molecules undergoing photoinduced electron transfer.[5]

Since the early 1930s, it has been a common conception that the inhibiting activity of phenols depends on the relative positions of their hydroxyl groups.[6] The first observations indicated that, to be effective, the groups should be attached directly to the benzene ring in an ortho or para configuration. The actual agreement is that the oxidation of phenolic compounds in aqueous solutions occurs via phenoxyl radicals.[2,5,7−11] Depending on the specificity of the oxidizing radicals, the phenoxyl radicals can be generated by routes common for radical−molecule interactions: hydrogen-atom abstraction, electron transfer, and addition to a double bond, the latter followed by rapid fragmentation of the transition state adduct.[4,12,13] Yet another route is through photoinduced electron transfer followed by rapid deprotonation of the transient radical cation.[5] The stability of phenoxyl radicals, both in the self-reaction leading to dimers and in the reaction with molecular oxygen leading to peroxides, greatly varies depending on substituents.[9] In the first case, bulky groups substituted in ortho and para positions generate steric hindrance to dimerization, whereas in the second case, the tendency to react with oxygen is reduced by substituents capable of withdrawing electrons.

The oxidation of phenolic compounds has been the focus of research attention because of the importance of this reaction in large-scale organic processes, such as the polymerization of vinyl acetate,[1] the treatment of edible fats,[6,14] the synthesis of organic chemicals, and the decontamination of wastes from petrochemical plants.[15] Another field where the oxidation of phenolic compounds plays a distinct role is biochemistry. Phenols are widespread in plants; they are also present in other living organisms, but in much smaller amounts.[16] Tannins[17] and various flavonoids[18,19] have been studied as effective biological antioxidants trapping free radicals. Recently, oxidative transformations of phenols has become an important aspect of atmospheric chemistry. The compounds in the atmosphere are mostly the result of human activity leading to the emission of volatile organic compounds (VOCs). Main sources of VOCs carrying phenols are industrial processes (e.g., oil refineries) and biomass burning. Phenols can also be formed by secondary transformations within the troposphere gas and liquid (aqueous) phases (clouds, fogs, raindrops, aerosols).[20] Their concentration in cloudwaters can even reach 5×10^{-5} M,[21] high enough to expect the role of these compounds in atmospheric aqueous-phase chemistry to be nonnegligible. Therefore, knowledge of the interaction between phenols and other atmospheric pollutants becomes essential for an improved understanding of the environmental consequences of pollution and for adequacy in atmospheric modeling.

In this paper, we describe laboratory experiments on the influence of simple phenol (C_6H_5OH), catechin [2-hydroxyphenol, $C_6H_4(OH)_2$], and gallic acid [2,3-dihydroxy-5-carboxyphenol, $HOOCC_6H_2(OH)_3$] on the kinetics of aqueous SO_2 autoxidation (oxidation by molecular oxygen at ambient temperatures). Attempts are also made to quantify this effect on the basis of measurements of the overall reaction rate under particularly

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selected conditions and a mechanistic model for the chain reaction at steady state.

S(IV) autoxidation markedly contributes to the chemistry of the atmosphere. Under urban conditions, this reaction path may be much more efficient than the oxidation of S(IV) by H_2O_2 and ozone.[22] This reaction pathway is known to involve sulfoxy radical anions (sulfite $^{\bullet}SO_3^-$, sulfate $SO_4^{\bullet-}$, and peroxymonosulfate $SO_5^{\bullet-}$ radicals) of different redox potentials.[23] The scavenging of sulfoxy radicals by phenolic compounds should negatively affect the kinetics of S(IV) autoxidation, i.e., lead to decreased chain lengths and thus lower oxidation efficiencies.

The inhibition of sulfite autoxidation by phenolic antioxidants has been studied by James and Weissberger,[24] Altwicker,[21,25] Altwicker and Sekulic,[26] Lim et al.,[27] and Sipos.[28] The first authors[24] were interested in the interaction between hydroquinones and sulfite, with an emphasis on the inhibitory action of sulfite in the autoxidation of hydroquinone and its derivatives. They explained the observed inhibition as resulting from the removal of quinone, the reaction transient thought to act as a catalyst, which transformed into sulfonates in reaction with sulfite. In his papers, Altwicker[21,25,26] considered some mechanistic aspects of the phenolic antioxidant inhibition of sulfur dioxide oxidation in relevance to control processes for the desulfurization of stack gases and atmospheric processes leading to acid rain. This author discussed the phenomenon of inhibition in terms of a radical-chain mechanism assuming interference of inhibitors such as hydroquinone, 4-methoxyphenol, and 2-*tert*-butyl-4-methoxyphenol with chain-propagating radicals $^{\bullet}SO_3^-$, $SO_4^{\bullet-}$, or $SO_5^{\bullet-}$. No differentiation was made among the radicals regarding their role in reaction steps involving the inhibitors. A complex mode of hydroquinone inhibition was concluded from the influence of this compound both on the induction period and the oxidation rate, the latter observed at higher concentrations of hydroquinone. Lim et al.[27] studied experimentally the inhibiting effects of phenol, hydroquinone, resorcinol, catechin, phloroglucinol, and pyrogallol. These authors interpreted their results assuming that the chain-breaking reaction involving $^{\bullet}SO_3^-$ is responsible for the inhibition of S(IV) autoxidation. According to these authors, there are two parallel mechanisms of stepwise conversion of hydroquinone into inerts in the course of S(IV) autoxidation: oxidation to quinone by $^{\bullet}SO_3^-$ or oxygen and reaction with sulfite leading to 1,4-hydroquinone-2,5-disulfonate. The latter transformation was deduced from the changing UV spectra. The possible reversibility of inhibitor oxidation restoring the original inhibiting compound was suggested. Recently, Sipos[28] confirmed the inhibition of S(IV) autoxidation by phenols using a Clark oxygen sensor to measure the decrease of the dissolved oxygen content in the course of reaction. He explained that phenols break the chain by reacting with sulfoxy radicals, not specifying the sulfoxy radical type nor the fate of the resulting phenoxyl radicals.

Materials and Methods

The autoxidation of S(IV) was studied in a glass reactor of 0.150 dm^3 liquid volume, and the conductometry technique was applied to follow the reaction progress. The increase in S(VI) concentration was determined using an Orion conductivity meter, model 170 and cell no. 018014. The reactor was open to the atmosphere and had the bulk of the contained liquid stirred at 700 rpm. The gas−liquid interface was maintained practically undisturbed through the use of immersed baffles counteracting the planar surface distortion. A scheme of the reactor is shown elsewhere.[29] To ensure a low but constant concentration of S(IV) in the course of an experiment, a portion of powdered $CaSO_3 \cdot {}^1/_2H_2O$ (load 13.5 g/dm^3, main particle size fraction 0.7−4 μm) was added to the liquid (aqueous phase). Because of solubility equilibrium, the concentration of S(IV) anions was kept at the 5.6×10^{-4} M level.[30] This literature value was confirmed with the ionic chromatography method in our laboratory. The pH values established in the reacting solutions were 7.3−7.5.

The autoxidation of phenol was carried out in a glass minireactor of 0.004 dm^3 volume, closed with a Lucite plunger [biological oxygen monitor YSI (Yellow Spring Instruments), model 5300]. The extent of reaction could be followed by measuring the decay of oxygen (oxygen probe YSI model 5331). To maintain conditions comparable to those in the presence of S(IV), it was necessary to adjust solution pH with NaOH (p.a., Merck).

In the minireactor, the reaction is of a homogeneous type, conducted in a closed system with oxygen predissolved. This reactor operated batchwise allows the intrinsic reaction rate to be determined directly from the measurements of reagent consumption with time. In the case of an open system, such as when a semibatch reactor with a planar gas−liquid interface is applied, the rate of reaction can be influenced by gas−liquid mass transfer. The intrinsic reaction rate was calculated directly from the measurements of product formation as a function of time only at relatively slow oxygen fluxes. When the fluxes increased sufficiently, this rate was found from the kinetic equation for oxygen absorption accompanied by the fast reaction zero order in oxygen.[31]

The p.a.-grade phenolic compounds phenol, catechin, and gallic acid were obtained, respectively, from Merck, Fluka, and Chemifarm. Solid $CaSO_3 \cdot {}^1/_2H_2O$ was prepared from $Ca(NO_3)_2 \cdot 3H_2O$ and anhydrous Na_2SO_3, both p.a. reagents manufactured by Merck. The reagents predissolved in water were mixed, and the resulting precipitate was filtered, washed, and dried. To reduce the oxidation of S(IV) to S(VI) during treatment, an Ar blanket was applied. As water-soluble catalysts $CoSO_4 \cdot 7H_2O$ (p.a., Merck), $MnSO_4$ (p.a., POCh), and $Fe_2(SO_4)_3 \cdot 9H_2O$ (p.a., POCh) were used. We performed all experiments at room temperature (23−25 °C) with Milli-Q water as the solvent.

Results and Discussion

Influence of Phenolic Compounds on the Autoxidation of S(IV). The S(IV) autoxidation intermediates, chain carriers $^{\bullet}SO_3^-$, $SO_4^{\bullet-}$, and $SO_5^{\bullet-}$, are known to attack phenolic compounds to produce phenoxyl radicals with an effectiveness that varies with the type of the oxidizing radical. Starting from the mid-1980s, information on the scavenging abilities of phenolic compounds with respect to sulfoxy radicals became available as a result of a series of works on elementary reactions.[2,4,11,13,32] It was shown that sulfite radicals $^{\bullet}SO_3^-$ behave as mild one-electron oxidants that react with hydroquinone, catechol, resorcinol, and hydroquinone sulfonates at rates that are strongly dependent on pH.[2,4] With increasing pH, the reactions between $^{\bullet}SO_3^-$ and the hydroxyphenols become faster, exhibiting rate constants that increase from about 1×10^7 M^{-1} s^{-1} at pH 9 to a value almost 10 times greater at pH 13.[2,4] This is related to the fact that, in alkaline solutions, phenolic compounds deprotonate, and the resulting anions are much more reactive with respect to $^{\bullet}SO_3^-$ than neutral species. The respective reactions written for the deprotonated hydroxyphenol are[2]

$$^{\bullet}SO_3^- + HOC_6H_4O^- \rightarrow SO_3^{2-} + {}^-OC_6H_4O^{\bullet} + H^+ \quad (1a)$$

$$^{\bullet}SO_3^- + {}^-OC_6H_4O^- \rightarrow SO_3^{2-} + {}^-OC_6H_4O^{\bullet} \quad (1b)$$

Reactions 1a and 1b are reversible. Such reversibility is also claimed for phenol.[2] In the latter case, the reverse reaction rate constant is 1×10^7 M^{-1} s^{-1} at pH 11.

To avoid the possible influence of scavenging $^{\bullet}SO_3^-$ radical anions in the experiments on the autoxidation of S(IV) inhibited by phenolic compounds, we carried out the reaction under near-neutral conditions. As demonstrated by others,[2] hydroxybenzenes are unreactive in neutral solutions with respect to sulfite radicals.

Peroxymonosulfate radical anions, $SO_5^{\bullet-}$, are known to react not only by one-electron transfer but also by oxygen-atom transfer. They are stronger oxidants of phenolic compounds than $^{\bullet}SO_3^-$.[2,4,13] At pH 7, the electron-transfer reaction of these radicals is favorable. Similarly to the case of $^{\bullet}SO_3^-$, the rate constants for the reactions between $SO_5^{\bullet-}$ and phenolic compounds are lower at pH 7 than at alkaline conditions. For hydroquinone, their values are 2.0×10^7 M^{-1} s^{-1} at pH 9.5 but only 2.7×10^6 M^{-1} s^{-1} at pH 6.7. The rate constant for catechin is also 2.7×10^6 M^{-1} s^{-1} at pH 6.7.

Sulfate radical anions, $SO_4^{\bullet-}$, react with many inorganic anions following the same pathway as is found with $^{\bullet}OH$.[33] They are also claimed, similarly to hydroxyl radicals,[34] to be very reactive toward organic compounds.[4] Both $SO_4^{\bullet-}$and $^{\bullet}OH$ can react with organic compounds by electron transfer, hydrogen abstraction, and addition to a double bond.[23] The latter reactions are very fast. In his detailed review of free radicals in the atmospheric aqueous phase, Huie[23] showed that the reactions of $SO_4^{\bullet-}$ with double bonds generally exhibit rate constants above 5×10^8 M^{-1} s^{-1}. Hydroxyl radicals react with phenolic compounds with diffusion-controlled rate constants of the order of 10^9 M^{-1} s^{-1} [34] or even 10^{10} M^{-1} s^{-1}.[2] Initially, $^{\bullet}OH$ adducts are formed, which then undergo fragmentation to yield phenoxyl radicals. The reaction with phenol proceeds via a dihydroxycyclohexadienyl radical[35]

$$^{\bullet}OH + C_6H_5OH \rightarrow HOC^{\bullet}{}_6H_5OH \rightarrow C_6H_5O^{\bullet} + H_2O \quad (2)$$

$SO_4^{\bullet-}$ radicals also react with phenol to form phenoxyl radicals (e.g., ref 32), and the reaction can be written, by analogy to reaction 2, as

$$SO_4^{\bullet-} + C_6H_5OH \rightarrow {}^-O_3SOC^{\bullet}{}_6H_5OH \rightarrow C_6H_5O^{\bullet} + HSO_4^- \quad (3)$$

The exact value for the corresponding rate constant has not been reported. Attempts to measure this constant for phenol have led to the values 1.5×10^{10} M^{-1} s^{-1} at pH ~2.5[36] or 1.4×10^{10} M^{-1} s^{-1} at pH 9.18,[37] evidently controlled by the encounter rate of the reacting solutes. In other experiments,[4] interference of the reaction between $S_2O_8^{2-}$ (precursor of $SO_4^{\bullet-}$ in pulse radiolysis) and phenolic compounds made it possible to find only that this rate constant is $\geq 10^9$ M^{-1} s^{-1}.

In this light, it seems justified to interpret the inhibiting effect measured under conditions of our experiments for phenol, catechin, or gallic acid as caused mainly by scavenging sulfate radicals (AH denotes a phenolic compound)

$$\text{primary inhibition} \qquad AH + SO_4^{\bullet-} \rightarrow A^{\bullet} + HSO_4^- \quad (4)$$

Figures 1−3 show plots of the kinetic data for the phenol, catechin, and gallic acid inhibitions, respectively, of the uncatalyzed autoxidation of S(IV). The data are given for various inhibitor concentrations and compared with the results obtained in the absence of an inhibitor. In the latter case, the liquid-side

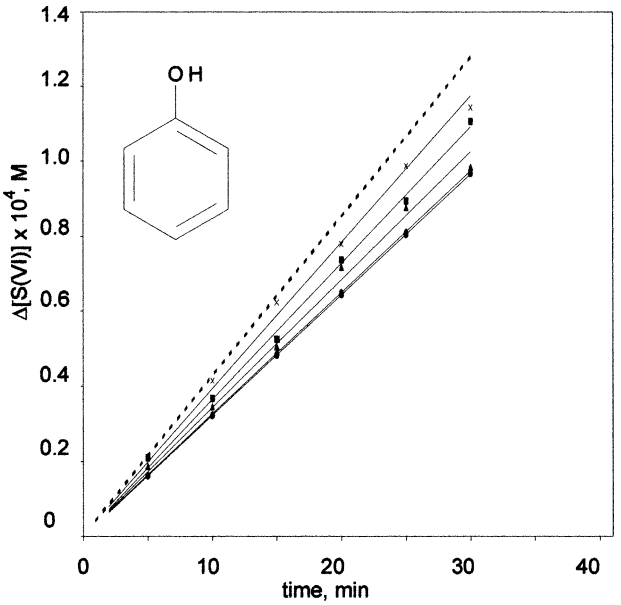

Figure 1. Effect of the phenol concentration on the uncatalyzed autoxidaton of S(IV): (×) 7.6×10^{-6} M, (■) 1.5×10^{-5} M, (▲) 2.3×10^{-5} M, (●) 3.1×10^{-5} M, (◆) 5.3×10^{-5} M; S(IV) 5.6×10^{-4} M, pH 7.5, 23 °C. (The dashed line represents the diffusional regime of oxygen absorption.)

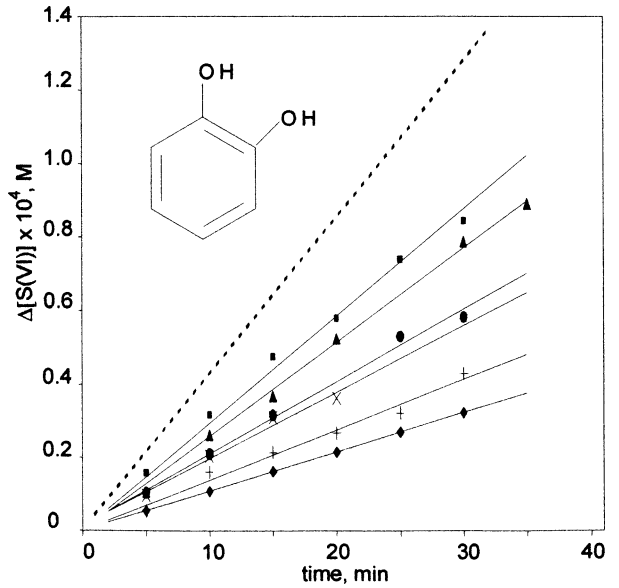

Figure 2. Effect of the catechin concentration on the uncatalyzed autoxidaton of S(IV): (■) 1.2×10^{-7} M, (▲) 1.8×10^{-7} M, (●) 2.3×10^{-7} M, (×) 2.8×10^{-7} M, (+) 3.5×10^{-7} M, (◆) 7.0×10^{-7} M; S(IV) 5.6×10^{-4} M, pH 7.5, 23 °C. (The dashed line represents the diffusional regime of oxygen absorption.)

limitation for oxygen mass transfer is attained, characteristic for the particular reactor used. This situation corresponds, in the sense of Astarita,[31] to the diffusional regime of a gas−liquid reaction (in the diffusional regime, the kinetics of an intrinsic reaction cannot be determined directly). As results from the figures, phenol is much less effective than the two other inhibitors tested. To slow the overall reaction to the same extent, one needs a concentration of phenol exceeding that of catechin or gallic acid by orders of magnitude. This observation is in accord with the weak effectiveness of phenol reported by Lim et al.[27] for the uncatalyzed and Cu(II)-catalyzed oxidation of sulfite at pH 9.4 and for the Mn(II)- and Fe(II)-catalyzed reaction at pH 1.0.[25] On the other hand, the values of a rate constant

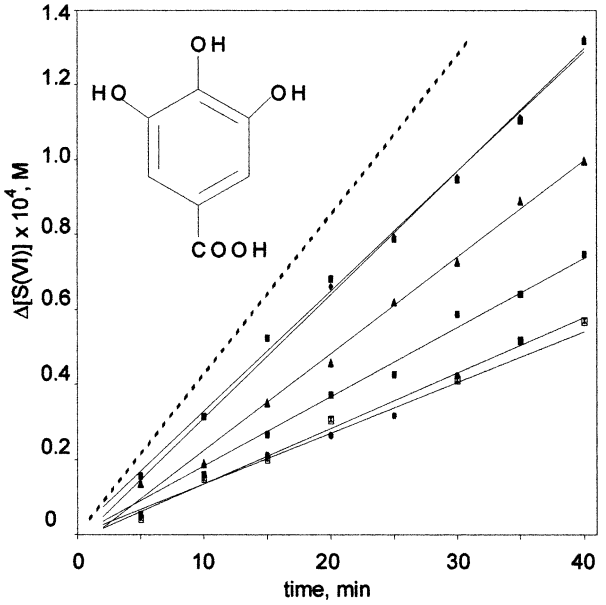

Figure 3. Effect of the gallic acid concentration on the uncatalyzed autoxidaton of S(IV): (◆) 1.9×10^{-7} M, (■) 2.3×10^{-7} M, (▲) 3.9×10^{-7} M, (×) 4.7×10^{-7} M, (+) 1.56×10^{-6} M, (●) 3.12×10^{-6} M; S(IV) 5.6×10^{-4} M, pH 7.5, 23 °C. (The dashed line represents the diffusional regime of oxygen absorption.)

reported for the reaction of phenol with $SO_4^{\bullet-}$ are much higher than those reported for other phenolic compounds (see above). This suggests that the inhibition caused by phenol should be much more effective than that by catechin and gallic acid. This apparent inconsistency can be explained by assuming that phenol exerts not only an inhibiting effect but also a promoting action on S(IV) autoxidation. Which of these two effects prevails should depend on experimental conditions.

To shed more light on the promoting role of phenol, we took into account the possibility that the oxidation products into which the compound AH can transform during the reaction studied do not necessarily behave as inerts. Phenoxyl radicals, $A^{\bullet}$ (e.g., $C_6H_5O^{\bullet}$), are known to be capable of oxidizing sulfite/bisulfite to sulfite radicals at a rather high rate; for phenol radicals, the rate constant is 1×10^7 M^{-1} s^{-1} at pH 11.[2] The variation in inhibition observed for particular phenolic compounds is considered as possibly arising from differences in the rates of the reaction of the phenoxyl radicals with sulfite.[2] Therefore, under appropriately adjusted experimental conditions, one should expect the contribution of phenoxyl radicals to the initiation of the S(IV) autoxidation chain, e.g., by transition metal ions of higher valency

main initiation

$$M(III) + SO_3^{2-}/HSO_3^- \rightarrow M(II) + {}^{\bullet}SO_3^-/{}^{\bullet}SO_3^- + H^+ \quad (5)$$

additional initiation

$$M(III) + AH \rightarrow M(II) + A^{\bullet} + H^+ \quad (6a)$$

$$A^{\bullet} + SO_3^{2-}/HSO_3^- \rightarrow A^-/AH + {}^{\bullet}SO_3^- \quad (6b)$$

Reaction 6b is reversible, and its direction is here forced by the next rapid and irreversible step removing sulfite radicals in reaction with molecular oxygen (first propagation step). This case can be compared to the well-known chain initiation discussed by Huie[23]

$$Fe^{3+} + HSO_3^- \rightarrow Fe^{2+} + {}^{\bullet}SO_3^- + H^+ \quad (7)$$

This author writes: "although this reaction is endothermic, the rapid reaction of $^{\bullet}SO_3^-$ with O_2 drives it to the right." Analogously to reaction 6b, the reactions of phenoxyl radicals with hydrocarbons

$$A^{\bullet} + RH \rightarrow AH + R^{\bullet} \quad (8)$$

are known to play a role in efficiently restarting autoxidation chains.[38]

To gain some idea of the importance of reaction 6b in counteracting the phenol inhibition caused by scavenging sulfoxy radicals (reaction 1), we performed experiments with the transition metal salts $CoSO_4$ or $MnSO_4$ introduced into the reacting system. These catalysts produce the autoxidation initiators Co(III) or Mn(III), respectively, so in their presence, an independent source of phenoxyl radicals starts to work.

The plot of S(VI) formation versus concentration of phenol shown in Figure 4 demonstrates the dual role of phenol. In one concentration region, this compound evidently promotes S(IV) autoxidation, whereas in the other, it causes the reaction to be retarded. Phenoxyl radicals can be multiplied in propagation steps of the catalyzed autoxidation of phenol. To investigate the possibility of this reaction under our experimental conditions, we tried to measure the decay of oxygen in aqueous solutions containing predissolved oxygen, phenol, and a catalyst, but not sulfite/bisulfite. The results of the measurements are displayed in Figure 5, showing no reaction consuming measurable amounts of oxygen in the case of gallic acid and traces of oxygen consumed in the case of phenol. This indicates that the contribution of phenol autoxidation steps in the multiplication of phenoxyl radicals is possible.

Another situation when organic radicals actively enter a chain scheme of S(IV) autoxidation manifests itself as reaction rate oscillations observed in prolonged experiments. Such oscillations in the case of the uncatalyzed autoxidation of S(IV) inhibited by gallic acid are given in Figure 6. They can be explained by including the following steps

dimeric radical formation

$$A^{\bullet} + AH \rightarrow (AAH)^{\bullet} \quad (9)$$

secondary inhibition

$$(AAH)^{\bullet} + {}^{\bullet}SO_3^- \rightarrow \text{inerts} \quad (10)$$

self-reaction

$$2(AAH)^{\bullet} \rightarrow \text{inerts} \quad (11)$$

Unlike phenoxyl radicals, dimeric radicals are not capable of oxidizing sulfite/bisulfite. Radical substitution by phenoxyl radicals on a phenol molecule (reaction 9) is a plausible route leading to dimeric products.[9] The dimeric radicals, unlike the parent phenoxyl radicals, have a spin density decreased at the radical center, and therefore, their tendency to oxidize sulfite/bisulfite should be diminished. Rolko et al.[32] found chromatographically products of reaction 9 heavier than the initial phenol.

Step 9 produces the new radical $(AAH)^{\bullet}$, which is capable of removing $^{\bullet}SO_3^-$ radicals in step 10. The latter reaction causes the oxidation of S(IV) to S(VI) to become halted, and this precludes the formation both of phenoxyl radicals $A^{\bullet}$ and of dimeric radicals $(AAH)^{\bullet}$: Under these circumstances, the concentration of $(AAH)^{\bullet}$ decreases through self-reaction,[11] so that the oxidation of S(IV) can restart.

Determination of Rate Constants for $SO_4^{\bullet-}$ Scavenging by Phenolics. The linearity of the plot of the rate of S(VI) formation versus 1/[AH], as shown in Figure 7 for catechin and gallic acid, is a helpful guide in a search for a rate expression

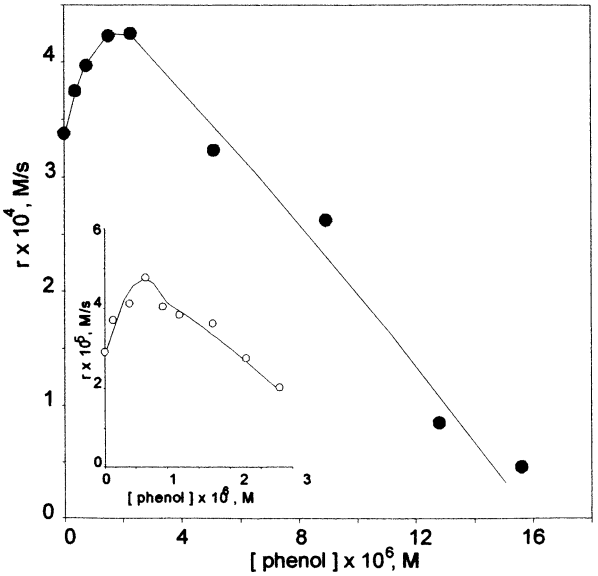

Figure 4. Synergy effect in the transition metal catalyzed autoxidaton of S(IV) caused by phenol: S(IV) 5.6×10^{-4} M, pH 7.5, 23 °C; (●) $CoSO_4$ 1.33×10^{-6} M and (○) $MnSO_4$ 1.33×10^{-6} M (reactor with a planar gas−liquid interface).

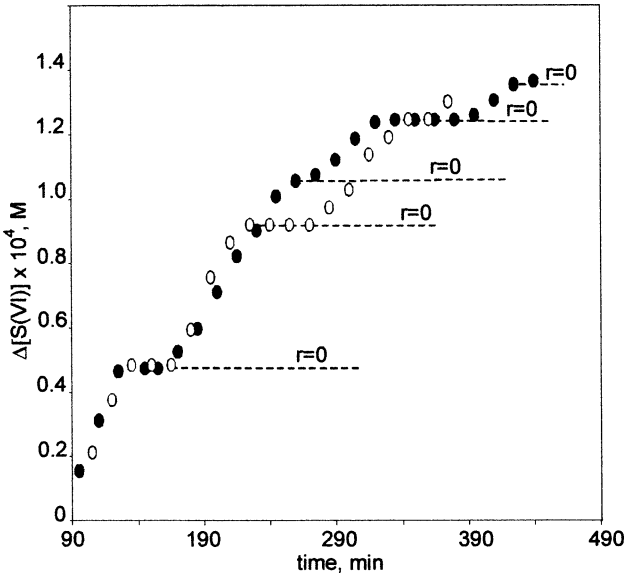

Figure 6. Nonlinear effect in the uncatalyzed autoxidation of S(IV) at an increased concentration of gallic acid: S(IV) 5.6×10^{-4} M, pH 7.5, 23 °C, gallic acid 2.10×10^{-3} M (two runs showing the reproducibility of oscillations).

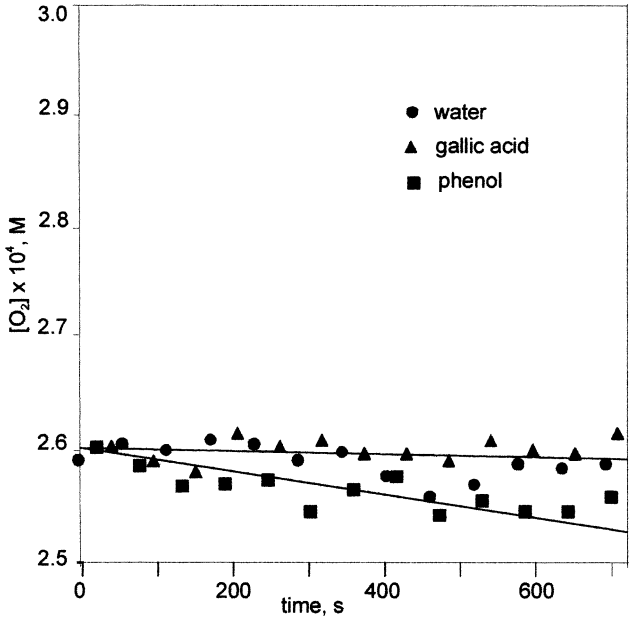

Figure 5. Decay of oxygen in the absence of sulfite/bisulfite: negligible in the case of (●) water and (▲) 2.0×10^{-4} M gallic acid and 5.0×10^{-4} M $Fe_2(SO_4)_3$, hardly visible in the case of (■) 2.0×10^{-4} M phenol and 1.4×10^{-6} M $CoSO_4$; pH 7.3, 25 °C (oxygen monitor YSI).

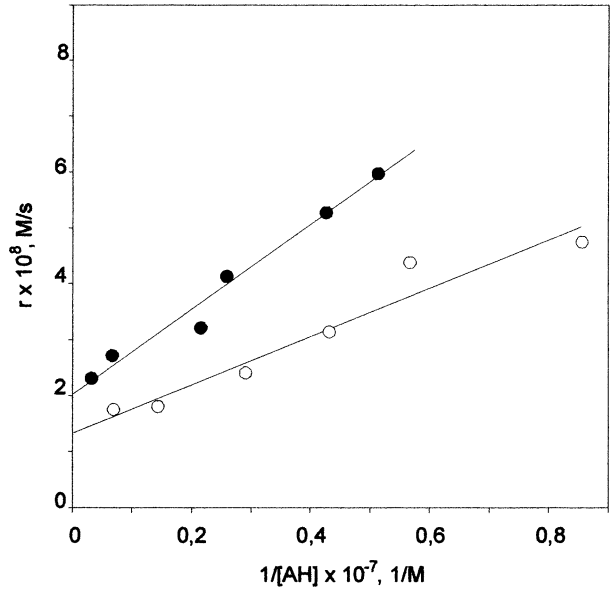

Figure 7. Determination of k_{in} from slopes of the plots of the S(VI) formation rate versus the reciprocal of the inhibitor concentration for (●) gallic acid and (○) catechin.

related to the inhibition mechanism. Figure 7 gives kinetic data for the quasi-steady-state period of the inhibited autoxidation of S(IV) as in Figures 2 and 3. In that case, the following restrictions are assumed to be valid: reaction 4 becomes the dominating termination of the S(IV) autoxidation chain, the steady-state approximation for a chain reaction applies, the influence of an inhibitor on the ratio $\alpha = [SO_5^{\bullet-}]/[SO_4^{\bullet-}]$ is negligible, and the initiation is equal to that of the uninhibited reaction increased by the contribution of reaction 6b (controlled by reaction 6a). The latter assumption means that the concentration of the metal ion initiator is established in a preequilibrium regardless of whether the S(IV) oxidation is inhibited.

A set of equations used to derive the kinetic expression for the rate of S(VI) formation under the above conditions is given in Table 1. They yield a straight-line equation that is in accord with the experimental results shown in Figure 7. Therefore, taking b in the final rate equation for the inhibited autoxidation of S(IV) (see Table 1) as equal to the slope of such a straight line, one can easily find a value of k_{in}

$$k_{in} = k_t \alpha r_0^2/(2k_p[\mathrm{S(IV)}]\mathrm{slope}) \qquad (12)$$

The composite rate constant k_p depends on pH and must be calculated from component rate constants as shown previously.[39] As results from our comparative calculations the numerical value of a sum of rate constants for the steps involving $SO_5^{\bullet-}$ and sulfite is the most important component of k_p under the conditions of our experiments, whereas the sensitivity of k_p to the values of other component constants is almost negligible. There are only two values of this sum reported in the literature: 1.3×10^7 1/(M s) at ionic strength 0.5 M[40] and 7.6

TABLE 1: Set of Rate Equations and Constants Used in Calculations of k_{in}

	rate expression	constant value taken for calculations
steady-state approximation	$r_{i,0} = r_{t,0}$,	$k_t = 2(4.8 \times 10^7)$ M^{-1} s^{-1} [41]
for the uninhibited reaction	where $r_{t,0} = k_t[SO_5^{\bullet-}]_0^2$	
steady-state approximation	$r_{i,0} + r_i = r_t$,	k_i = ?
for the inhibited reaction	where $r_i = k_i$[M(III)][AH]	k_{in} = ?
	and $r_t = k_{in}[SO_4^{\bullet-}]$[AH]	
rate of the overall reaction,	$r_0 = d[S(VI)]_0/dt$	$k_p = 5.1 \times 10^6$ M^{-1} s^{-1} [a]
no inhibition	$= 2k_p[SO_5^{\bullet-}]_0$[S(IV)]	$r_0 = 3.2 \times 10^{-6}$ M s^{-1} [b]
rate of the overall reaction	$r = d[S(VI)]/dt$	
with inhibition	$= 2k_p[SO_5^{\bullet-}]$[S(IV)]	
rate of the overall reaction	$r = a + b/$[AH],	$\alpha = 1 \times 10^2$ [a]
with inhibition (final expression)	where $a = 2k_p k_{i\alpha}$[M(III)][S(IV)]$/k_{in}$	
	and $b = k_{t\alpha r0}^2/2k_p k_{in}$[S(IV)]	

[a] Calculated as described previously[39] for component rate constants corrected for the ionic strength in this work. [b] Determined experimentally in a homogeneous reactor.

$\times 10^5$ 1/(M s) at ionic strength about 1.5×10^{-2} M[41] as reviewed by Huie and Sieck.[13] For the second of these values, we obtained unrealistic results: k_{in} of the order of 10^{11} 1/(M s) (greatly exceeding diffusional limitation) and a chain length of the order of tens (whereas the kinetics of our system obeys the equation derived under the assumption that the chain is long). The rate equation given in Table 1 is more general and also describes inhibition by sobrerol (nonaromatic C_6 ringed compound with the OH function at a ring).[42] We accepted the results obtained for the first value of a sum of rate constants for the steps involving $SO_5^{\bullet-}$ and sulfite, recalculated for the ionic strength 2.2×10^{-3} M as in our experiments, i.e., 8.5×10^6 M/s. The values for catechin and gallic acid found by this method are, respectively, 4.3×10^9 and 2.6×10^9 M^{-1} s^{-1}. They agree with the results of Neta and Huie,[4] who were able to determine only a lower limit for these rate constants as $\geq 10^9$ M^{-1} s^{-1} (see above). In the case of phenol, it was not possible to fulfill the conditions for the validity of eq 12, because, at low concentrations of phenol, comparable to those of catechin or gallic acid, the rate-controlling phenomenon is diffusion. The intercept a provides information on the promoting activity of a phenolic compound expressed as k_i. At the same reaction parameters, the ratio of intercepts for individual inhibitors is equal to the ratio of the appropriate values of k_i. Thus, the determined order of promotion for the autoxidation of S(IV) is phenol > gallic acid > catechin.

Conclusions

The results reported in this work are important to integrate knowledge on the individual steps in S(IV) autoxidation, so as to better understand the inhibition by phenolic compounds and quantify this process at high dilution as pertaining to troposphere chemistry and chemistry of surface waters. The mechanistic−kinetic analysis of the autoxidation of S(IV) in the presence of phenolic compounds indicates that: (i) under the conditions of our experiments, phenoxyl radicals are formed; (ii) in the absence of an external catalyst, traces of M(III) (below the detection limit) give rise to the additional initiation of the S(IV) autoxidation chain via the radicals (see reactions 6a and 6b); (iii) in the presence of an external catalyst, the autoxidation of phenol becomes a source of extra phenoxyl radicals generated in propagation steps; and (iv) the contribution of the radicals to the initiation of the S(IV) autoxidation chain leads to synergy effects.

The latter conclusion implies that the formation of troposphere and surface water acidity by the chain mechanism can take place also in the absence of transition metal catalysts. Then, the chain can be initiated by phenoxyl radicals formed in reactions of phenolic compounds with other oxidants, such as ubiquitous hydroxyl radicals.

Rate constants, k_{in}, calculated from the slopes of the straight lines shown in Figure 7 are in reasonable agreement with those expected by others.[4] The order of promotion for the autoxidation of S(IV) is phenol > gallic acid > catechin, which is in fact, contrary to the order of inhibition reported by Huie and Neta after Darkyina.[2]

The net result of phenolic inhibition in the environment, primary or under conditions of rate oscillations, is depression of the local concentration of noxious sulfoxy radicals and transport of the unreacted S(IV) to remote areas. However, when the conditions favor the development of the autoxidation of a phenolic compound, the effect of synergy should be expected.

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