Studies on the Degradation of Phenolic Lignin Units of the β-Aryl Ether Type with Oxygen in Alkaline Media*

JOSEF GIERER, FINN IMSGARD** and ISA NORÉN

Swedish Forest Products Research Laboratory, Chemistry Department,
Box 5604, S-114 86 Stockholm, Sweden

The degradation of p-hydroxy-arylglycerol-β-aryl ether structures in lignins by oxygen in alkaline media has been studied using appropriate model compounds. Three pathways (A, B, C, Schemes 1 − 3), explaining the formation of the various degradation products under different conditions, have been elucidated. Pathways A and B involve oxidative elimination of the side chain by oxygen and hydrogen peroxide, respectively. Hydrogen peroxide may be generated by autoxidation of certain lignin structures and intermediates. Pathway C entails the oxidative cleavage of the $C_\alpha - C_\beta$ bond by oxygen.
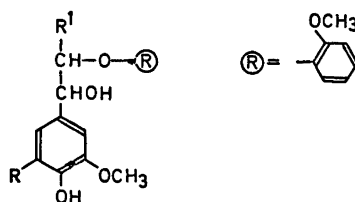
The primary oxidation products (aliphatic and aromatic aldehydes and p-quinones) undergo further oxidative-alkaline degradation to give ultimately aliphatic acids.

It is also shown that the oxidative-alkaline degradation of certain enolizable phenacyl aryl ether structures follows similar routes.

In recent years, the treatment of wood and pulps with oxygen in alkaline media has become an attractive alternative to conventional delignification processes.[1,2] Numerous studies have therefore been carried out to elucidate the breakdown of lignins by oxygen in alkali and plausible mechanisms have been proposed.[3–8] These studies deal primarily with the oxidative attack on phenolic lignin nuclei and do not account for the reactions involving also lignin side chains.

The main subject of the present work is the degradation of structural elements of the p-hydroxy-arylglycerol-β-aryl ether and related types with oxygen in alkali. The reactions involved were studied using model compounds $1 − 6$ representing "uncondensed"[9] (1 and 2) and "condensed"[9] (3 and 4) guaiacyl type units, as well as syringyl (5 and 6) type units.

	R	R¹
1	H	H
2	H	CH_2OH
3	t-butyl	H
4	t-butyl	CH_2OH
5	OCH_3	H
6	OCH_3	CH_2OH

Unless otherwise stated, the oxidative treatments were carried out under standard conditions. The term "standard conditions" refers here and in the following to an approximately 0.05 M concentration of the compound in 0.2 − 0.3 M sodium hydroxide, a reaction temperature of 40 °C and an oxygen pressure of 1 bar. The standard treatment was stopped when

3

1.0 – 1.5 mol of oxygen per mol of compound had been consumed. The mild conditions and the length of the treatment were chosen to facilitate the isolation of intermediates.

RESULTS AND DISCUSSION

The results from the treatments of the model compounds with oxygen in alkali are summarized in Table 1 (see EXPERIMENTAL).

Treatment of p-hydroxy-arylglycerol- and p-hydroxy-arylethyleneglycol-β-aryl ethers with oxygen in alkaline media

Compounds *1* and *2*, when autoxidized under standard conditions for 48 h, were essentially stable and gave only small amounts of guaiacol. No vanillin was detected in the reaction mixtures. However, when the oxidative treatment of *2* was carried out at 100 °C using an oxygen

Table 1. Autoxidation of model compounds. Temperature: 40 °C. Alkalinity: 0.2 – 0.3 M NaOH. Oxygen consumption: 1 – 1.5 mol O_2/mol compound.

Compound	Reaction time, h	Reaction products isolated and/or identified (Yield %)	Starting material %
1	48	G(8)	80
1-arom. methyl ether	48		97
2	48	G(8)	81
2 [a]	1	G(3), V(5), M[b] acids: FA(4), AA(29.0), GA(15.0), OA(31.0), MA(4.2), SA (3.6), FuA(1)	9
2-triacetate	48	G(27), V(14), *21*(16), acids: VA(1), FA(64), other acids (5)	25
3	2	G(22), *11*(28)	38
4	1	G(5), *11*(15), *15*(3)	80
5-α-methyl ether	48		93
6	1	G(14), *15*(15) acids: FA(21), AA(2.7), GA(5)	35
6-triacetate	3	G(52), syringaldehyde(4), *15*(3) acids: FA(40), GA(15)	8
7	0.5	M(41) acids: FA(17), AA(2.8), GA < (0.1), OA(6.6), MA(1.6), SA(0.3), FuA(0.1)	0
8	2	*11* (96)	0
9	1.5	acids: FA(3.8), AA(3.5), OA(7.9), MA(1.7)	0
Vanillin	40	acids: FA(103), AA(12), OA(5), MA(2), FuA(0.5), SA(3)	44
14 (R¹ = H)	2	G(66) acidic products (20); not identified	0
Guaiacoxy-AA	18.5	G(0)	100
20 (R = H, *trans + cis*)	16	G(55), V(21)	6
22	28	G(55) acid: Ver A(53)	45
23	28	G(trace)	89
24	28	dimerization products [15]	0
25	28	*23*(100)	0
26	48	G(7) acids: VA(2), guaiacoxy-AA(2)	90
27	28	*28* (90)	0

[a] Reaction conditions: 100 °C, 7 bar. O_2. [b] Yield not determined.

AA = acetic acid, FA = formic acid, FuA = fumaric acid, G = guaiacol, GA = glycolic acid, M = methanol, MA = malonic acid, OA = oxalic acid, SA = succinic acid, SM = starting material, V = vanillin, VA = vanillic acid, VerA = veratric acid.

pressure of 7 bar, an extensive degradation took place within one hour. The reaction mixture contained small amounts of guaiacol and vanillin and a great number of aliphatic acids (GLC).

The oxidations of the *t*-butyl-substituted analogues *3* and *4* under standard conditions proceeded considerably faster than those of *1* and *2*. Compound *3* yielded considerable amounts of guaiacol and of the *p*-benzoquinone derivative *11* after treatment for only 2 h. Compound *4* gave, in addition to guaiacol and *11*, α-guaiacoxyacrolein (*15*) after autoxidation for 1 h. No 5-*t*-butylvanillin was detected in the reaction mixtures from *3* and *4* which contained large amounts of the starting compounds.

A still more rapid autoxidation under standard conditions was observed with the 2,6-dimethoxy-substituted phenols *5* and *6*. After a reaction period of 1 h, the reaction mixture from compound *6* contained appreciable amounts of guaiacol and α-guaiacoxyacrolein (*15*). No syringaldehyde was detected.

The different behaviour of the model compounds on treatment with oxygen in alkali and the formation, in varying yields, of the reaction products listed in Table 1 can be explained by suggesting three possible pathways of oxidative-alkaline degradation:

Pathway A involves elimination of the side chain by an alkali-promoted rearrangement of a cyclohexadienone hydroperoxide intermediate (*1a − 6a*) and a further oxidative degradation of the quinoid and aldehydic fragments (Scheme 1). The first two steps in Scheme 1 have previously been proposed[4] on the basis of results from the autoxidation of simple *p*-hydroxy-benzyl alcohols. These steps have now been confirmed by the isolation of the appropriate fragmentation products from *4*, viz. *11* and *14* ($R^1 =$ CH_2OH). The first product was characterized as the pyrazoline derivative *13* formed by reaction with diazomethane and the second as

Scheme 1. Pathway A. (1) oxygenations, (2) alkaline rearrangements with fragmentation, (3) alkaline demethoxylation, (4) further oxidative degradation. Compounds *1 − 6* and *10 − 12* are depicted as anions.

the dehydration product *15* which arises in a reaction competing with the oxidative degradation of *14*. Further support for the validity of oxidative side chain elimination according to Scheme 1 was supplied by the behaviour of methylated model compounds. Thus, the aromatic methyl ether of *1* and the α-methyl ether of *5* were found to be essentially stable towards treatment under standard conditions. Methylation of the phenolic hydroxyl group in *1* may be expected to block step (1) and methylation of the α-hydroxyl group in *5* should inhibit step (2). If it is assumed that the α-methyl ether of *5*, like the parent compound, is oxygenated, its recovery after the oxidative treatment would indicate that the oxygenation step (1) is reversible.[10]

Step (3) was shown by treating *8* under standard conditions and isolating *11* in high yield. The remarkable stability of *11* is obviously due to the *t*-butyl substituent and to hybridization between the *para*- and *ortho*-quinoid resonance structures of the anion. On prolonged treatment with alkali, the *ortho*-quinoid tautomer of *11* undergoes benzilic

acid rearrangement followed by decarboxylation and various oxidation and addition reactions.[11] When $R=H$ or $R=OCH_3$, the quinoid intermediates (*10* and *12*) could not be isolated. It was shown in a separate experiment that the authentic compound *7*, when exposed to standard conditions, gave among other products a mixture of aliphatic acids having the same qualitative composition as that of the mixture obtained from *2* after treatment under more drastic conditions (Table 1).

These results are in agreement with the assumption that *para*-quinones are intermediates in the oxidative degradation of phenolic nuclei in compounds of the β-aryl ether type (*e.g.* *2*) according to Scheme 1. The reaction mixture from *7* also contained hydrophilic products not extractable with organic solvents. This fraction has not been investigated.

The further oxidative-alkaline degradation of the aldehydic fragments (*14*, $R^1=H$ or CH_2OH) also includes oxygenation (1) and alkali-induced rearrangements (2) proceeding *via* hydroxy dioxetanes [8,12] (*cf.* also Scheme 4)
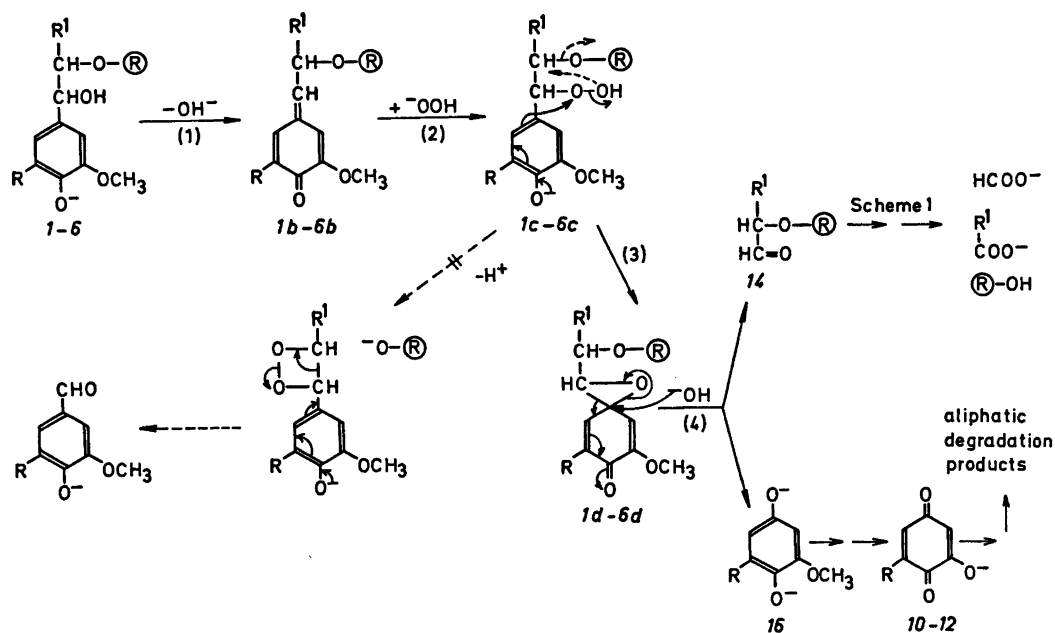
Scheme 2. Pathway B. (1) quinone methide formation, (2) addition of hydrogen peroxide, (3) alkaline rearrangement with formation of a spiro epoxide, (4) alkali-promoted fragmentation. Compounds *1 − 6*, *1c − 6c*, *10 − 12* and *16* are depicted as anions.

or *via* keto hydroperoxides in their hydrated or hemiacetal forms.[8,12] The formation of the appropriate fragmentation products, guaiacol, formic acid and glycolic acid, was demonstrated by autoxidation of *2* and *6*. The facile oxidative-alkaline degradation of α-aroxy aldehydes was confirmed by reacting guaiacoxy acetaldehyde (*14*, R[1]=H) under standard conditions for 2 h and isolating guaiacol in 66 % yield. Alkaline treatment of *14* (R[1]=H) in a nitrogen atmosphere under otherwise identical conditions did not afford any detectable amount of guaiacol which excludes purely alkaline degradation. As expected, guaiacoxyacetic acid, which does not enolize, proved stable when exposed to standard autoxidation conditions.

Pathway B. Although pathway A (Scheme 1) satisfactorily explains the formation of the quinoid and aldehydic intermediates, a further route of formation of these fragments, involving hydrogen peroxide as oxidant, must be considered (Scheme 2).

The possibility of the oxidative and alkali-promoted elimination of side chains following pathway B was indicated by the behaviour of *1*-diacetate towards alkaline hydrogen peroxide (Table 2). The sequence was illustrated by treating the *p*-hydroxybenzyl alcohol *17* with the same oxidant at room temperature and isolating the corresponding cyclohexadienone spiro epoxide *18* [steps (1)−(3), Schemes 2 and 3]. The elimination of the side chain from this intermediate by the action of alkali [step (4)] was demonstrated by treating compound *18* with a solution of sodium hydroxide in aqueous methanol at 60 °C. The hydroquinone monomethyl ether *19* thus expected was isolated in high yield. Compound *19* was also obtained when *17* was oxidized with hydrogen peroxide in the same solvent (Table 2).

Instead of rearranging to give spiro epoxides (*1*d − *6*d), the intermediates of the *p*-hydroxybenzyl hydroperoxide type in β-aryl ether structures (*1*c − *6*c, Scheme 2) could also be thought to form dioxetanes by neighbouring group participation with concomitant elimination of the β-aroxy substituent. This reaction would be reminiscent of the cleavage of β-aryl ether bonds involving the formation of oxirane[13] or thiirane[14] intermediates. However, treatment of the diacetate of compound *1* with alkaline hydrogen peroxide in a nitrogen atmosphere yielded a large amount of guaiacol (Table 2) but no vanillin (TLC). In a separate experiment it was shown that vanillin survives a similar oxidative treatment. The absence of vanillin in the reaction mixture from *1*-diacetate is in accordance with steps (3) and (4) and excludes the other fragmentation alternative in Scheme 2.

The initial oxidative steps of the degradation following routes A and B are due to oxygen and hydrogen peroxide, respectively. Both routes involve cleavage of the Cα-aryl bond and lead to the same aldehydic and quinoid intermediates. The further degradation of these intermediates may be common for both pathways.

Pathway C, presented in Scheme 4, accounts for the formation of aromatic aldehydes by cleavage of the Cα − Cβ bond. Like pathway B, this route is initiated by alkaline conversion of the starting compound into the corresponding quinone methide which then gives the corresponding β-aroxystyrene intermediate.[13] In contrast to pathway B, however, the subsequent oxidative degradation is brought about by oxygen rather than by hydrogen peroxide.

The proposed oxidative-alkaline degradation of β-guaiacoxystyrene intermediates (*e.g.* *20*, R=H), formed from quinone methide intermediates (*1*b − *6*b), follows a route analogous to that for the degradation of the aldehydic fragments *14* (Scheme 1). This analogy in the
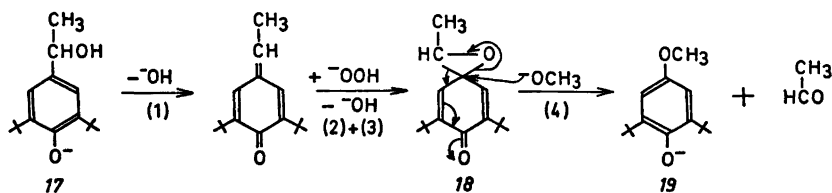
Scheme 3. (1) quinone methide formation, (2) addition of hydrogen peroxide, (3) formation of a spiro epoxide, (4) opening of the spiro epoxide ring with rearomatization and elimination of the side chain. Compounds *17* and *19* are depicted as anions.
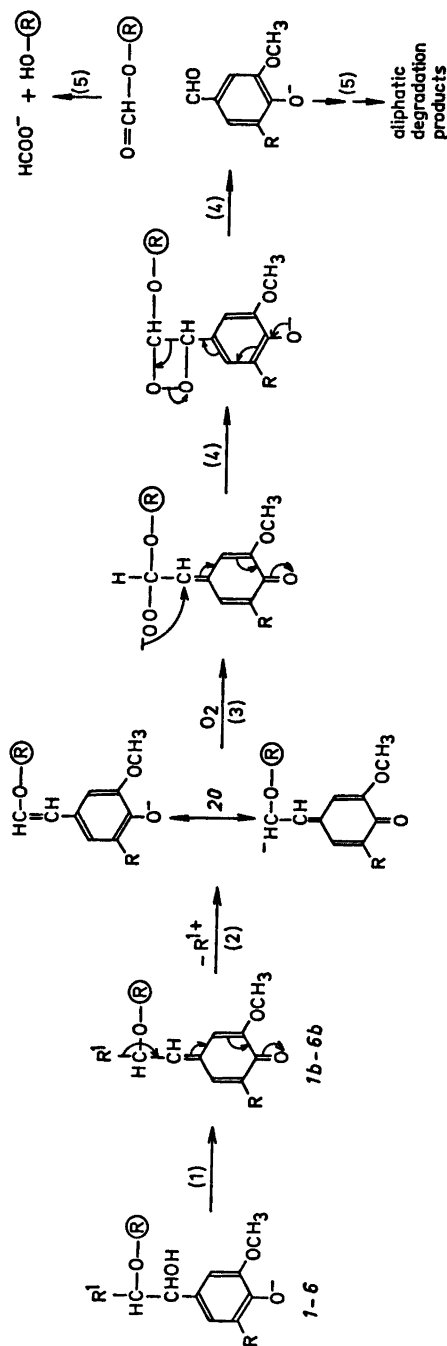
Scheme 4. Pathway C. (1) quinone methide formation, (2) β-aroxystyrene formation, (3) oxygenation, (4) alkaline rearrangement with fragmentation, (5) further alkaline and/or oxidative degradations. Compounds *1–6* and *20* are depicted as anions.
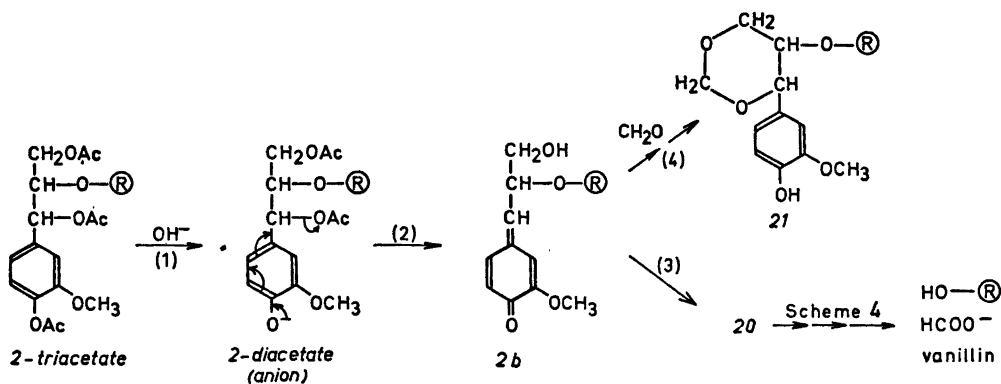
fragments *14.* As in the case of the aldehydic intermediates (*14*), the fragmentation of β-guaiacoxystyrenes could also proceed *via* addition of hydroxide ions, followed by rearrangement of the resulting α-hydroxy hydroperoxides.

The suggested formation of β-aroxystyrene derivatives prior to the oxidative degradation was supported by treating a mixture of the *trans-* and *cis*-forms of *20* (R = H) (approximate ratio 2:1) under standard autoxidation conditions for 16 h. Guaiacol and vanillin were isolated as main reaction products. The starting material recovered (6 %) was a mixture of the *trans-* and *cis*-forms in an approximate ratio of 1:3.

The more facile degradation of the β-guaiacoxystyrene derivative *20* (R = H) compared to that of the parent β-guaiacyl ethers *1* or *2* shows that the formation of either the quinone methide intermediates [step (1)] or the styrene intermediates [step (2)] is the rate-determining step in the over-all reaction. When *2*-triacetate was treated under standard conditions (Scheme 5), considerable amounts of guaiacol, vanillin and the 1,3-dioxane derivative *21* were formed (Table 1). The extensive reaction may be attributed to the acetoxy group in the α-position which constitutes a better leaving group than the α-hydroxyl group in *2*. In this way, the conversion into *2b*, *i.e.* quinone methide formation [step (2)] is facilitated. Compound *21* arises from *2b* by an alkali-promoted condensation with liberated formaldehyde (4).[15] Reaction (4) competes with reaction (3) (formation of the β-aroxystyrene intermediate *20*, R = H).

Further oxidation of the aromatic aldehydes (*e.g.* vanillin) gives, in addition to formic acid, a mixture of other aliphatic acids which were also obtained after a similar treatment of the corresponding methoxy-*p*-quinones (*e.g.* *7*). This result is consistent with the view that the oxidative-alkaline degradation of aromatic aldehydes proceeds *via* a Dakin type of reaction to give the appropriate *p*-quinones.[4]

Some of the products listed in Table 1 can be considered to indicate the actual degradation route for the side chain of a phenolic aryl-glycerol-β-guaiacyl ether model compound. Thus, glycolic acid and α-guaiacoxyacrolein (*15*) should be generated by pathway A or B

mechanism of degradation may be ascribed to the fact that styrene derivatives *20* in the carbanion form constitute conjugated analogues to the carbanions derived from the aldehydic

Scheme 5. (1) Alkaline hydrolysis, (2) quinone methide formation, (3) guaiacoxystyrene formation, (4) condensation with formaldehyde liberated in (3).

(Schemes 1 or 2) whereas aromatic aldehydes (*e.g.* vanillin) should only arise through pathway C. *p*-Quinones (*e.g.* 7−9), on the other hand, could be produced both directly (pathways A and B) and *via* aromatic aldehydes (pathway C). However, quantitative estimates of the significance of the different pathways based on the amounts of products isolated cannot be made because of the instability of some of these products under the conditions used. Furthermore, alkali-promoted non-oxidative reactions may contribute to the over-all degradation of phenolic *β*-aryl ether structures, provided that the formation of quinone methide intermediates is possible.[13,16]

Treatment of phenacylaryl ethers with oxygen in alkaline media

When compound **22** was treated under standard conditions, extensive oxidation took place. Guaiacol and veratric acid were isolated from the reaction mixture in high yields. The methyl derivative of **22** (**23**) was not oxidatively degraded to any appreciable extent and was recovered in high yield.

The hydroxy derivative of **23** (**24**) also resisted oxygenation during the standard treatment but underwent alkali-promoted dimerization.[16] Compound **25** lost one molecule of formaldehyde upon treatment under standard

22	R=H	R¹=H
23	R=CH₃	R¹=H
24	R=CH₂OH	R¹=H
25	R=CH₂OH	R¹=CH₃
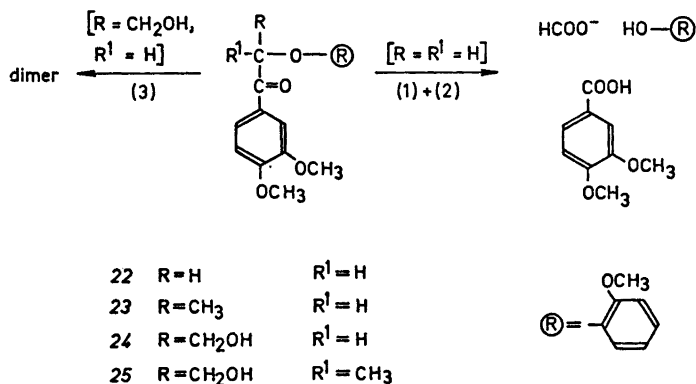
Scheme 6. Reactions of phenacyl aryl ethers: (1) oxygenation, (2) rearrangement with fragmentation, (3) dimerization.

26 R=H
27 R=CH2OH
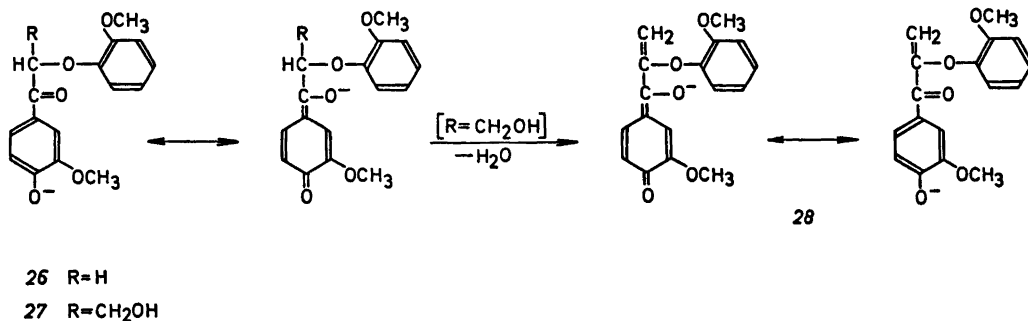
Scheme 7. Compounds *26 – 28* are depicted as anions.

conditions and was converted quantitatively into *23* (*cf.* also Ref. 15).

The phenolic counterparts of *22* and *24* (*26* and *27*, respectively) exhibited considerable stability towards oxygen under standard conditions. After a 48 h treatment of *26*, the starting material was thus recovered in high yield. Only small amounts of guaiacol, guaiacoxyacetic acid and vanillic acid were formed. Similar treatment of compound *27* afforded the unsaturated carbonyl compound *28* (*cf.* also formation of *15*, Scheme 1) in a high yield. No detectable amounts of dimerization or oxidation products were found.

The behaviour of compounds *22 – 28* towards oxygen-alkali can be interpreted as follows: *22* is degraded following a route [steps (1) and (2), Scheme 6] analogous to the degradations

of the aldehydic fragmentation products (Schemes 1 and 2) and of the aroxystyrene intermediates (Scheme 4). The slight oxygenation of *23* is due to a low degree of enolization while the stability of *25* towards oxygen is a result of its inability to enolize. Compound *24* does enolize but the carbanion formed is consumed by a competitive addition to an enone system [step (3)] arising from *24* by dehydration.[15] The phenolic phenacyl aryl ethers *26* and *27* have a greatly impaired ability to enolize due to the efficient stabilization of their phenolate anions by resonance. The small amounts of guaiacoxyacetic acid and vanillic acid obtained from *26* are indicative of pathway A (or B) and pathway C, respectively. Subsequent to enolization, *27* undergoes dehydration to give *28* rather than oxygenation
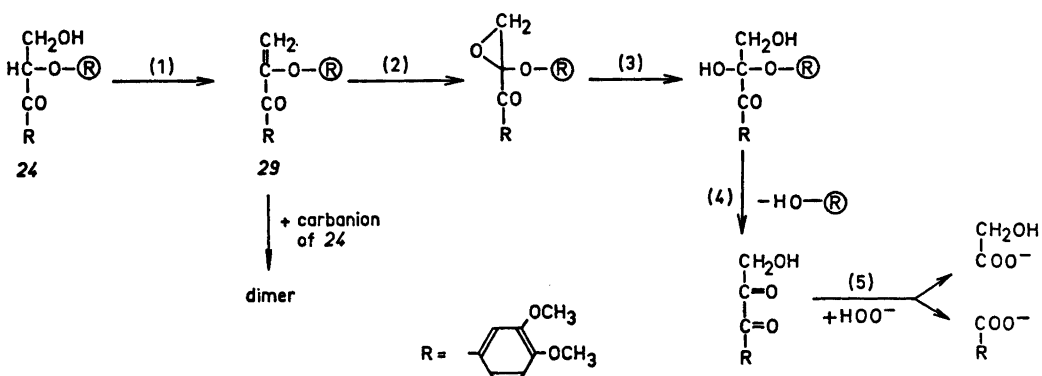
Scheme 8. Oxidation of phenacyl aryl ethers by hydrogen peroxide: (1) dehydration, (2) epoxidation by hydrogen peroxide, (3) alkaline opening of the epoxide ring, (4) cleavage of the hemiketal, (5) oxidative cleavage of the 1,2-diketone (cleavage of the original $C_\alpha - C_\beta$ bond).

or dimerization.[15] *28* remains unchanged when exposed to the standard autoxidation conditions.

In alkaline solution the unsaturated phenolic keto compound *28* exhibits strong absorption at 370 nm ($\varepsilon = 18.440$ l mol^{-1} cm^{-1}) due to the extended conjugation. This chromophoric system is reminiscent of the chromophore formed from coniferaldehyde in alkaline solutions.[17]

Phenacylaryl ethers, which give enones by an alkali-promoted reaction,[15] are extensively degraded when alkaline hydrogen peroxide is used as oxidant instead of oxygen (Scheme 8). Compound *24* yielded guaiacol and veratric

acid in varying yields depending on the amount of oxidant employed. Treatment with an equimolar amount afforded, in addition to guaiacol and veratric acid, the enone *29* and a dimerization product, both of which are formed merely by the action of alkali.[15]

The oxidative fragmentation of *24* by alkaline hydrogen peroxide may be explained by the reaction route outlined in Scheme 8.

CONCLUSIONS

The results listed in Table 1 suggest that *pathway A* is the dominant process under standard conditions. Phenols carrying sub-
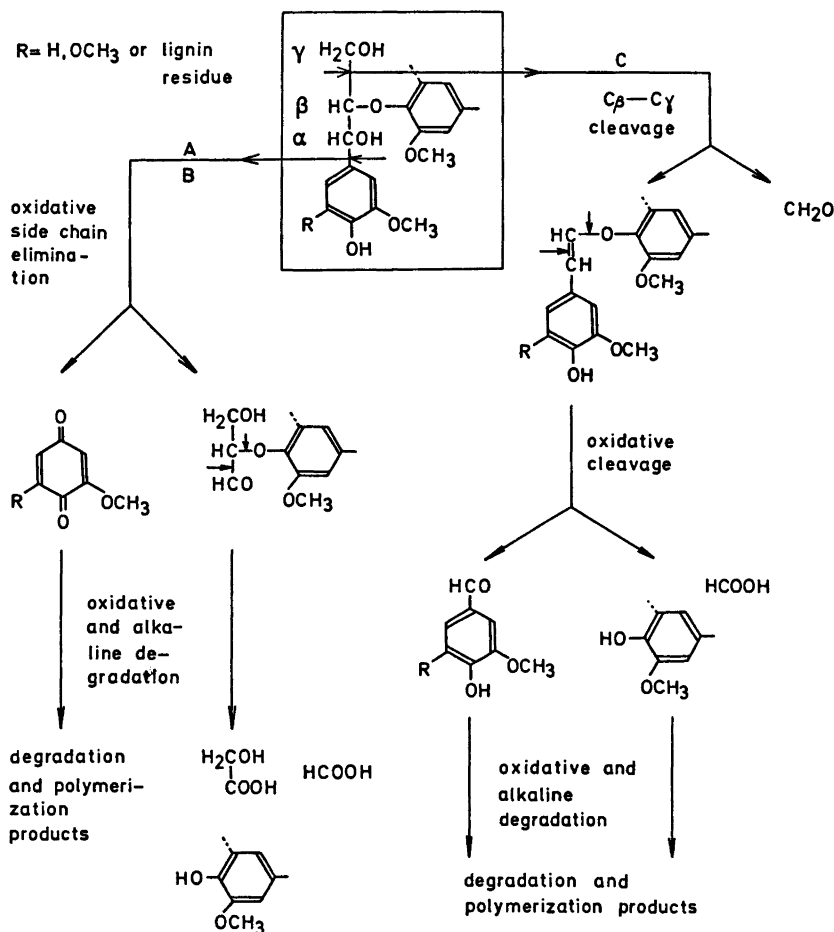
Scheme 9. Pathways of oxidative-alkaline degradation of *p*-hydroxy-arylglycerol *β*-aryl ether structures (Summary).

stituents in both the 2 and 6-positions (*e.g.* 3 − 6) react with particular ease probably due to their relatively low acidity. The inverse correlation of oxidizability with acidity seems reasonable since weak acids give relatively unstable carbanions which are more sensitive to the electrophilic attack by oxygen.[16]

Extensive oxidative degradation of mono-*o*-substituted phenols (*e.g.* *1* and *2*) requires more drastic conditions. Under such conditions, quinone methide intermediates may be formed which can initiate also *pathways B and C*. The competition between B and C will be primarily determined by the alkalinity of the solution and the availability of hydrogen peroxide during the oxidative treatment.

All three pathways, outlined in Schemes 1, 2 and 4 and summarized in Scheme 9, lead ultimately to the formation of aliphatic degradation products arising from eliminated side chains and to aliphatic acids and hydrophilic polymerization products arising from intermediates of the *para*-quinone and aromatic aldehyde types. The alkaline-oxidative degradations of *β*-aryl ether structures also result in the liberation of the *β*-aroxy substituent as a phenolate ion. The stability of the latter (*e.g.* the guaiacolate ion liberated from *1−6*) towards oxygen in alkali under the conditions used shows that the oxidative degradation of phenolic units containing side chain structures of the benzyl alcohol type (*e.g.* *1−6*) starts with the oxidative elimination (pathways A and B) or fragmentation (pathway C) of the side chain rather than with the oxidative cleavage of the phenolic nucleus.

Once the aroxy substituent is liberated by one of the routes A − C, the degradation process may be repeated, provided the liberated phenolic unit has the same or a similar type of *β*-aryl ether structure. The oxidative lignin fragmentation ("oxidative lignin peeling") may thus continue until it reaches a unit containing a side chain which does not meet the structural requirements for at least one of the three pathways.

The degradation of phenacyl-aryl ethers by oxygen-alkali (Scheme 6) exhibits many formal analogies to that of the aldehydic fragments (Scheme 1) and the *β*-quaiacoxystyrene intermediates (Scheme 4). The degradation is restricted to structures having an easily enolizable keto group and lacking the ability to form an enone system for conjugate addition.[15]

The oxidative-alkaline degradation processes are accompanied and, in part, counteracted by oxidative coupling reactions[3−8] and alkali-promoted conjugated additions (condensations and polymerizations).[15]

EXPERIMENTAL

Materials

Syntheses of model compounds. The following compounds were prepared as previously described: *1*,[13] *2*,[19] *5* (analogous to *1*), *7*,[20] *8*,[21] *9*,[22] *14* (R[1]=H),[23] *17*,[24] *20* (R=H, *cis* and *trans*),[15] *22*,[25] *23*,[26] *24*,[25] *25*,[27] *26* (analogous to *27*), *27*.[15]

The *β*-aryl ethers *3 − 6* were prepared from the appropriate acetophenones as described for *1* and *2*.

All analyses agree within ± 0.3 % units with the calculated values.

4-Acetoxy-5-t-butyl-3-methoxyacetophenone. 6-*t*-Butylguaiacol[28] (50 g) dissolved in acetyl chloride (100 ml) was added to a cooled solution (10 °C) of aluminium chloride (50 g) in acetyl chloride (200 ml). The temperature of the reaction mixture was kept below 15 °C. After the addition had been completed, the reaction mixture was stirred for 30 min, treated with cooled 0.5 M hydrochloric acid and extracted with chloroform. The excess of 6-*t*-butylguaiacol acetate was distilled off (142 − 146 °C/19 mmHg). Crystallization of the distillation residue from light petroleum yielded the pure compound (17 %), m.p. 83 − 85 °C. (Found: C 68.47; H 7.32; O 24.19. $C_{15}H_{20}O_4$ requires: C 68.18; H 7.58; O 24.24.)

3,5-Dimethoxy-4-hydroxyacetophenone. This compound was prepared using a modification of a method previously[29] described. The acetate of 1,3-dimethoxyphenol (39 g) was treated with aluminium chloride (32 g) in nitrobenzene (200 ml) at 50 °C for 1 h. After addition of cooled 0.5 M hydrochloric acid, the reaction mixture was extracted with 2 M sodium hydroxide. The aqueous layer was acidified and extracted with methylene chloride. Evaporation of this extract and crystallization of the residue from ethanol gave the pure compound (20 %), m.p. 123 − 126 °C (lit. 110 − 120 °C).

Analytical data of β-aryl ethers.

1-(4-Acetoxy-5-t-butyl-3-methoxyphenyl)-2-O-(2-methoxyphenyl)ethyleneglycol (3-acetate), m.p. 97 − 99 °C (ligroin). (Found: C 68.03; H 6.91; O 24.99. $C_{22}H_{28}O_6$ requires: C 68.02; H 7.26; O 24.71).

1-(4-Acetoxy-5-t-butyl-3-methoxyphenyl)-2-O-(2-methoxyphenyl)-1,3-O-diacetylglycerol (4-triacetate), amorphous. The ^{1}H NMR spectrum

Table 2. Oxidation of model compounds with alkaline hydrogen peroxide. Alkalinity: 0.2 − 0.3 M NaOH, nitrogen atmosphere. Temperature: 40 °C.

Compound	Solvent	Reaction time, h	Equiv. of H_2O_2	Reaction products isolated and/or identified (yield % of theoretical)	Starting material, %
1-Diacetate	d-w (2:3)	1	1	G (60)	0
Vanillin	d-w (2:1)	1	1.5		100
17	d-w (2:1)	0.5	1.5	*18* (25)	
17	m-w (3:1)	2.5	1.2	*19* (26)	
24	e-w (3:1)	2.5	1	*29* (19) G (35), Ver A (31), Dimeric product (12)[15]	0
24	e-w (3:1)	24	3	G (88), Ver A (80)	0
27	e-w (3:1)	1	1		90

d-w dioxane-water, m-w methanol-water, e-w ethanol-water, G = guaiacol, VerA = veratric acid.

indicated a 1:1 mixture of the *erythro* and *threo*-forms. (Found: 64.60; H 7.00; O 28.40. $C_{27}H_{34}O_9$ requires: C 64.52; H 6.81; O 28.66).

1-(3,5-Dimethoxy-4-hydroxyphenyl)-2-O-(2-methoxyphenyl)-glycerol (6), m.p. 162 − 164 °C (chloroform). The ^{1}H NMR spectrum of the synthetic compound indicated the *threo*-configuration. The coupling constant of the α and β-protons in the side chain was $J_{\alpha\beta} = 7.0$ cps.[30] Anal. $C_{18}H_{22}O_7$: C, H, O.

Methods

Autoxidation of model compounds. The reaction conditions and results are given in Table 1. Autoxidation with oxygen under pressure was carried out in a 1 l stainless steel autoclave. The yields of the low molecular weight acids were determined by an ion-pair benzylation or methylation[31] technique in combination with GC analysis. The work-up procedure and preparative separation of the reaction mixtures were carried out as previously described.[8]

Oxidation of model compounds with alkaline hydrogen peroxide. The reaction conditions and results are given in Table 2.

Treatment of guaiaoxyacetaldehyde with alkali. Guaiaoxyacetaldehyde (*14*, $R^1 = H$) (2 mmol) was treated with a 0.3 M sodium hydroxide solution in ethanol-water (4:1) at 40 °C for 2 h (nitrogen atmosphere). No guaiacol could be detected in the reaction mixture (TLC).

Treatment of spiro epoxide 18 with alkali. Compound *18* (0.18 mmol) was treated with a 0.2 M sodium hydroxide solution in methanol-water (4:1) (10 ml) at 60 °C for 6 h (nitrogen atmosphere). 2,6-Di-*t*-butyl-4-methoxyphenol (*19*) was isolated in 91 % yield.

Product identification

6-t-Butyl-2-hydroxy-p-quinone (11), yellow crystals from ethanol, m.p. 135 − 138 °C (Lit.[11] 138 − 139.5 °C).

^{1}H NMR (60 MHz, $(CD_3)_2SO$): 1.24 (s, 9 H, *t*-Bu), 5.84 (d, 1 H, ol, *J* 2.7 Hz), 6.37 (d, 1 H, ol, *J* 2.7 Hz).

7a-t-Butyl-6-methoxy-3a,7a-dihydro-3H-indazolquinone (4,7) (*13*), pale yellow crystals from ether. Decomposes between 155 and 162 °C. Anal. $C_{12}H_{16}O_3N_2$: C, H, O, N. IR (KBr): 1690 (s, C = O), 1650 (s, C = O), 1605 (s, C = C − C = C). UV [MeOH (ε)]: 268 (9150). ^{1}H NMR (60 MHz, $CDCl_3$): δ 1.10 (s, 9 H, *t*-Bu), 3.80 (s, 3 H, OMe), 5.03 (A) and 4.39 (B) [CH_2], 3.06 (X) [CH]; ABX; J_{AB} 18.3; J_{AX} 11.1; J_{BX} 7.5; 5.88 (s,1 H, ol).

α-(2-Methoxyphenoxy)-acrolein (15). GC-MS analysis (fragments having $m/e > 100$ and intensities > 10 % of base peak are given: 178 (M, 100), 149 (M − 29, 14), 135 (M − 43, 13), 134 (M − 44, 12), 123 (M − 55, 30), 121 (M − 57, 25), 109 (35), 108 (50). ^{1}H NMR (60 MHz, $CDCl_3$) of the crude fraction (90 % *15*) obtained by autoxidation of *6* and ether extraction of the alkaline reaction mixture: δ 3.79 (s, 3 H, OMe), 5.02 (d, 1 H, ol, J_{gem} 2.7 Hz), 5.22 (s, 3 H, ol, J_{gem} 2.7 Hz), 7.00 (m, 3 H, ar), 9.43 (s, 1 H, CHO).

5,7-Di-t-butyl-2-methyl-1-oxaspiro[2,5]octa-4,7-diene-6-one (18), white crystals, m.p. 95 − 97 °C (ether). Anal. $C_{16}H_{24}O_2$: C, H, O. UV [MeOH (ε)]: 258 (15 200). ^{1}H NMR (60 MHz, $CDCl_3$): 1.23 (s, 18 H, 2 × *t*-Bu), 1.47 (d, 3 H, CH_3, J_{CH,CH_3} 5.3 Hz), 3.44 (q, 1 H, CH, J_{CH,CH_3} 5.3 Hz), 6.08 (d, 1 H, ol, $J_{ol,ol}$ 2.7 Hz), 6.33 (d, 1 H, ol, $J_{ol,ol}$ 2.7 Hz).

2,6-Di-t-butyl-4-methoxyphenol (19), white crystals, m.p. 102 − 104 °C (light petroleum) (lit.[32] 105 − 106 °C). GC-MS analysis (fragments having $m/e > 100$ and relative intensities > 10 % of base peak are given): 236 (M, 32), 222 (M − 14, 15), 221 (M − 15, 100), 220 (M − 16, 10), 205 (M − 31, 10), 193 (M − 43, 10), 179 (M − 57, 17), 177 (20), 165 (10), 163 (10), 161 (11), 151 (10), 149 (13), 135 (10). ^{1}H NMR (60 MHz, $CDCl_3$): 1.40 (s, 18 H, 2 × *t*-Bu), 3.73 (s, 3 H, OMe), 4.70 (s, 1 H, OH), 6.74 (s, 2 H, ar).

Compounds *21* and *28* were identified as previously [15] described.

REFERENCES

1. Croon, I. and Andrews, D. *Tappi 54* (1971) 1893; Engström, H. E., Ivnäs, L. and Pettersson, B. *Tappi 54* (1971) 1899; Jamieson, A., Noréus, S. and Pettersson, B. *Tappi 54* (1971) 1903.
2. Abrahamsson, K. and Samuelson, O. *Sven. Papperstidn. 78* (1975) 417, and preceding 5 communications of the series "Oxygen-alkali cooking of wood meal".
3. Kratzl, K., Gratzl, J. and Claus, P. *Adv. Chem. Ser. 59* (1966) 157.
4. Kratzl, K., Schäfer, W., Claus, P., Gratzl, J. and Schilling, P. *Monatsh. Chem. 98* (1967) 891.
5. Kratzl, K., Claus, P., Lonsky, W. and Gratzl, J. *Wood Sci. Technol. 3* (1974) 35.
6. Eckert, R. C., Chang, H-M. and Tucker, W. P. *Tappi 56* (1973) 134.
7. Sundin, S., San Clemente, M. R. and Sarkanen, K. V. *167th ACS National Meeting*, Los Angeles, Cal., March 31 — April 5, 1974; Abstr. of Papers, Cell. 032.
8. Gierer, J. and Imsgard, F. *Acta Chem. Scand. B 31* (1977) 546.·
9. Leopold, B. *Sven. Kem. Tidskr. 64* (1952) 18; *Acta Chem. Scand. 6* (1952) 38; Adler, E. *Paperi Puu 43* (1961) 634.
10. Gersmann, H. R. and Bickel, A. F. *J. Chem. Soc. C* (1962) 2356.
11. Musso, H. and Bormann, D. *Chem. Ber. 98* (1965) 2774.
12. For analogous cleavage reactions, see Doering, W. E. von and Haines, R. M. *J. Am. Chem. Soc. 76* (1954) 482; Bordwell, F. G. and Knipe, X. *J. Am. Chem. Soc. 93* (1971) 3416.
13. Gierer, J. and Norén, I. *Acta Chem. Scand. 16* (1962) 1713.
14. Gierer, J. and Smedman, L. Å. *Acta Chem. Scand. 19* (1965) 1103.
15. Gierer, J., Imsgard, F. and Pettersson, I. *Applied Polymer Symposium* No. 28, Wiley, New York 1976, pp. 1195 – 1211.
16. Gierer, J. *Sven. Papperstidn. 73* (1970) 571.
17. Aulin-Erdtman, G. *Sven. Papperstidn. 56* (1953) 91; Aulin-Erdtman, G. and Hegbom, L. *Sven. Papperstidn. 61* (1958) 187.
18. Gersmann, H. R. and Bickel, A. F. *J. Chem. Soc. C* (1959) 2711.
19. Kratzl, K., Kisser, W., Gratzl, J. and Silbernagel, H. *Monatsh. Chem. 90* (1959) 771.
20. Erdtman, H. *Proc. R. Soc. London 43* (1933) 186.
21. Hewgill, F. R., Kennedy, B. R. and Kilpin, D. *J. Chem. Soc. C* (1965) 2904.
22. Teuber, H. J. and Rau, W. *Chem. Ber. 86* (1953) 1036.
23. Kirk, T. K., Harkin, J. M. and Cowling, E. B. *Biochim. Biophys. Acta 165* (1968) 145.
24. Volodkin, A., Portnykh, N. V. and Ershov, V. V. *Izv. Akad. Nauk SSSR, Ser. Khim.* (1968) 220; *Chem. Abstr. 69* (1968) 76799.
25. Adler, E., Lindgren, B. O. and Saedén, U. *Sven. Papperstidn. 55* (1952) 245.
26. Adler, E. and Marton, J. *Acta Chem. Scand. 15* (1961) 357.
27. Marton, J., Adler, E., Marton, T. and Falkehag, S. I. *Adv. Chem. Ser. 59* (1966) 125.
28. Rosenwald, R. H. *J. Am. Chem. Soc. 74* (1952) 4602.
29. Zambeletti, D. L. S.p.A. *Brit. Pat.* 1,188, 480; *Chem. Abstr. 73* (1970) 3660.
30. Johansson, B. and Miksche, G. *Acta Chem. Scand. 26* (1975) 289.
31. Bethge, P. O. and Lindström, K. *Analyst (London) 99* (1974) 137.
32. Cook, C. D., Inskeep, K. G., Rosenberg, A. S. and Curtis, Jr., E. C. *J. Am. Chem. Soc. 77* (1955) 1672.