319. *Studies of the Autoxidation of Tetralin. Part III. An Interpretation of the Kinetics of Autoxidation.*

By Alan Robertson and William A. Waters.

The reactions involved in the decomposition of tetralin hydroperoxide can be used to elucidate the natures of the critical chain-starting and chain-stopping reactions of the " uncatalysed " autoxidation of tetralin. Free hydroxyl radicals are probably involved both as chain-starting and as chain-stopping agents.

The autoxidation of tetralin, like that of many other hydrocarbons, involves two consecutive, but inter-related phases, *viz.*, (I), the formation of a hydroperoxide, and (II) the breakdown of this hydroperoxide in the presence of both hydrocarbon and oxygen, to a series of other products. Now that the salient features of phase (II) of this process have been established experimentally (see Part II, preceding paper) it is possible to review their connections with phase (I) of the autoxidation process, and to interpret the course of the oxygen uptake process from the basis of a much wider range of experimental data than has been available hitherto.

The main features of the oxygen uptake process (I) have been described previously (Robertson and Waters, *Trans. Faraday Soc.*, 1946, **42**, 201; George and Robertson, *Proc. Roy. Soc.*, 1946, *A*, **185**, 309; Medvedev, *Acta Physicochim.*, *U.R.S.S.*, 1938, **9**, 395; Larsen, Thorpe, and Armfield, *Ind. Eng. Chem.*, 1942, **34**, 182). They consist of (*A*) a period of slow, but accelerating reaction in which tetralin hydroperoxide accumulates; (*B*) a period of reaction at a constant rate in which more peroxide is formed than is decomposed; and (*C*) a final period of decreasing oxygen uptake, when a very complex reaction mixture has been formed.

During the initial, autocatalytic, period (*A*), tetralin hydroperoxide, as it is formed, catalyses the reaction. This effect is the more noticeable at low temperatures. In these circumstances Medvedev found that the rate of oxygen uptake (his " partial rate " w_2) was a function of the peroxide content of the liquid, whilst George and Robertson (*loc. cit.*, p. 316) found that it was a function of the oxygen pressure. Our measurements, made at 76°, indicate that (i) initially over 99% of the oxygen uptake corresponds to hydroperoxide formation, and (ii) whilst the reaction is still accelerating, the rate of oxygen uptake is proportional to the *square root* of the peroxide content of the liquid (see Figure). In contrast to this observation with tetralin, Bolland (*Proc. Roy. Soc.*, 1946, *A*, **186**, 218) and Bolland and Gee (*Trans. Faraday Soc.*, 1946, **42**, 236) report that for the initial stage of the autoxidation of ethyl linoleate at 35—75° the rate of oxygen uptake is proportional to the first power of the peroxide content of the liquid, but is also a function of the oxygen pressure.

Kinetics of the initial stages of autoxidation of tetralin.
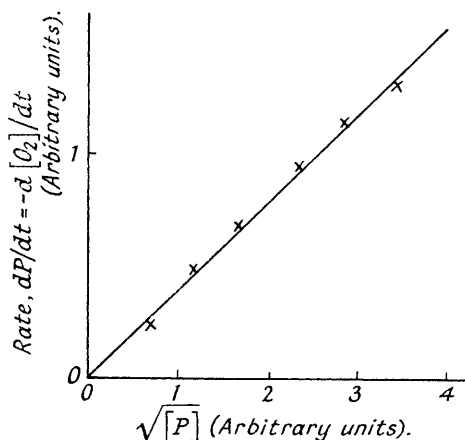
Most of the studies of the autoxidation of tetralin have, however, been concerned with period (*B*), during which the oxygen is absorbed at a steady rate. In these circumstances, George and Robertson, who used very small quantities of hydrocarbon, found (i) that under 90% of the oxygen uptake corresponded to additional peroxide formation, and (ii) that the rate of oxygen uptake was proportional to the *square of the hydrocarbon concentration*, and substantially independent both of the oxygen pressure and of the peroxide content of the liquid. Their equation :
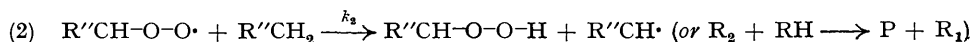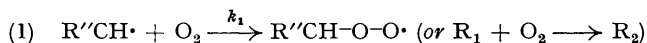
$$\text{Rate of } O_2 \text{ uptake} = k[\text{RH}]^2$$

corresponds to Medvedev's " primary process " with

$$\text{Rate } w_1 = a[\text{RH}]^2 \times f \text{ (tetralone present)}$$

Period (*B*) is also the principal reaction studied by Larsen Thorpe, and Armfield, who followed the reaction through to period (*C*), when, as pointed out by Medvedev, decomposition products of peroxides retard the reaction (compare Part I, this vol., p. 1574).

It is necessary to fit the whole of this reaction sequence into a comprehensive scheme, which must (*a*) account for the main products which are formed during both the synthesis and the breakdown of tetralin hydroperoxide, (*b*) require the postulation only of reactions involving substances (molecules, radicals, etc.) which are known from experimental evidence to be present in the system, and (*c*) take due account of the concentration and relative reactivity of each of the reacting entities throughout the successive stages of the whole autoxidation process. Thus it is essential to postulate the participation only of free radicals of types known to be present in the reacting system, and to take account only of their established chemical reactions. For instance, hypothetical reactions, such as $R\text{–}O\text{–}O\cdot + O_2 \longrightarrow R\text{–}O\cdot + O_3$ (George, *Trans. Faraday Soc.*, 1946, **42**, 217) must be excluded, since (i) there is no experimental evidence to support the view that free peroxide radicals can react with molecular oxygen, and (ii) ozone has not been detected as a reaction product : it is in fact an initiator of autoxidation (Briner, Demolis, and Paillard, *Helv. Chim. Acta*, 1932, **15**, 201) and not an end-product.

The Main Chain Reaction.—Chemical evidence, already set forth in detail (see Robertson and Waters, *loc. cit.*), indicates that the main oxygen uptake cycle is the *long chain* process :

(1) $R''CH\cdot + O_2 \xrightarrow{k_1} R''CH\text{–}O\text{–}O\cdot$ (*or* $R_1 + O_2 \longrightarrow R_2$)

(2) $R''CH\text{–}O\text{–}O\cdot + R''CH_2 \xrightarrow{k_2} R''CH\text{–}O\text{–}O\text{–}H + R''CH\cdot$ (*or* $R_2 + RH \longrightarrow P + R_1$)

since free hydrocarbon radicals (*e.g.*, phenyl) and free peroxide radicals (*e.g.*, CPh$_3$·O·O·) are both *immediate* catalysts for the autoxidation of tetralin and analogous substances. In accordance with this one may write :

$$- \mathrm{d}[O_2]/\mathrm{d}t = k_1[R_1][O_2] = + \mathrm{d}[P]/\mathrm{d}t \text{ (approx.)} = k_2[R_2][RH] \quad . \quad . \quad . \quad \textbf{(A)}$$

and must therefrom choose chain-starting and chain-stopping processes which lead to overall kinetic equations that satisfy the experimental observations.

The Chain-starting Process.—The autocatalytic nature of the primary stage of autoxidation, and the fact that tetralin hydroperoxide is an *immediate* catalyst, indicate that, in the absence of more active primary catalysts, such as dibenzoyl peroxide, tetralin hydroperoxide must be concerned in this chain-starting process in the " uncatalysed " reaction. Now the work detailed in Part II has shown that tetralin hydroperoxide generates some free radicals, probably both ·OH and C$_{10}$H$_{11}$·O· (R·O·), in the course of its thermal decomposition in solution. Some of these active radicals undoubtedly dehydrogenate surrounding molecules, giving water, α-tetralol, and possibly other compounds as their eventual stabilised products. The dehydrogenation of tetralin by these radicals is shown by the invariable production of some 1 : 2-dihydronaphthalene during the autoxidation (see Part I). Moreover, the facts that triphenylmethyl peroxide and *tert.*-butyl hydroperoxide are both immediate catalysts for tetralin autoxidation, together with recent studies of the properties of the free hydroxyl radical (compare Merz and Waters, Faraday Society Discussion, 1947, **2**, in the press), show that, in general R⁻O· radicals can attack most C–H bonds.

Neglecting for the moment any difference in reactivity between R″CH⁻O· and ·OH radicals, one may therefore conclude that the process

$$(3) \quad R_1\text{–}H + \cdot OH(\cdot OR) \xrightarrow{k_s} R_1 \cdot + H_2O \text{ (HOR)}$$
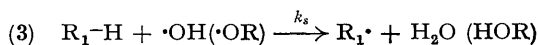
does occur under the reaction conditions appertaining to autoxidation; (3) is slightly exothermic (*ca.* 6 kcals.), and its activation energy must of course be primarily dependent on the bond strength of the C–H group attacked, so that the value of 26 kcals. reported both by Medvedev and by George and Robertson for the chain-starting process in the autoxidation of tetralin does not seem to be unduly high for it.

Stages (*A*) and (*B*) of the autoxidation of tetralin, when the concentration of R–H is high and the probability of encounter of two hydroperoxide molecules, or of radicals derived therefrom, is small, are particularly the circumstances in which (3) would be a more frequently occurring reaction than any other activation process which could conceivably operate as a chain-starter to the main oxygen uptake cycle. Chemical evidence supports the choice of (3) as the chain-starting process, since small amounts of water are always formed, even in the initial stages of the autoxidations of nearly all substances.

If one postulates (3) then it follows that

$$\text{Chain-starting rate} = k_s[OH][RH] \quad . \quad . \quad . \quad . \quad . \quad . \quad \textbf{(B)}$$

which accords with George and Robertson's findings that in the " uncatalysed " reaction the chain-starting process is of first order with respect to the hydrocarbon, and of zero order with respect to oxygen.

Chain-stopping Processes.—The choice of chain-stopping reactions is much more open, for there are several cogent possibilities; thus :

(i, *a*) *Dimerisation of* R$_1$. This should give ditetralyl, which has *not* been found as an autoxidation product, though its analogue is formed during the autoxidation of ethylbenzene (Sully, *Trans. Faraday Soc.*, 1946, **42**, 260).
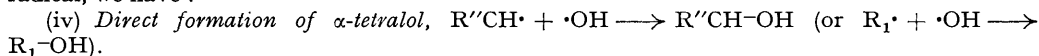
(i, *b*) *Disproportionation of* R$_1$. This would be kinetically indistinguishable from the above and does occur to a small extent (less than 2% of the whole reaction) since 1 : 2-dihydronaphthalene is an invariable autoxidation product of tetralin.

(ii) *Dimerisation of* R$_2$, *i.e.*, 2 R–O–O· ⟶ R–O–O–O–O–R. This is *most* improbable, though combination with loss of oxygen is somewhat more likely. Even this, however, does not correspond with the mode of formation of oxygen from hydrogen peroxide (which can be regarded as the prototype compound for peroxide reactions). The objections under (iii) are also cogent.

(iii) *Union of* R$_1$ *and* R$_2$, *i.e.*, R″CH· + R″CH–O–O· ⟶ R″CH–O–O–CHR″. This is possible, but could occur extensively only if R$_1$ and R$_2$ were present in roughly equivalent concentration, which is seldom the case. The reaction, however, would not lead to the formation

of a stable product, but to a fresh autoxidation catalyst (compare triphenylmethyl peroxide), and thus should be ruled out as a *main* chain-stopping process.

Of these three reactions therefore only (i) needs serious consideration. It is not necessary, however, to confine the choice of chain-stopping reactions to processes involving only the active radicals of the main oxygen uptake cycle (1—2). Serious consideration must be given to reactions involving radicals derived from the tetralin hydroperoxide. Thus, taking the hydroxyl radical, we have :

(iv) *Direct formation of* α-*tetralol,* $R''CH\cdot + \cdot OH \longrightarrow R''CH{-}OH$ (or $R_1\cdot + \cdot OH \longrightarrow R_1{-}OH$).

(v) *Formation of* α-*tetralol by elimination of oxygen from hydroxyl and peroxide radicals,* as in the " catalase " decomposition of hydrogen peroxide :

$$R''CH{-}O{-}O\cdot + \cdot OH \longrightarrow R''CH{-}OH + O_2 \; (or \; R_2 + \cdot OH \longrightarrow R_1{-}OH + O_2).$$
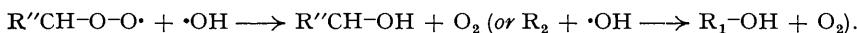
Similar equations can, of course, be derived for reactions involving alkoxy-radicals, $R{-}O\cdot$, but there is very little chemical evidence for them, since ethers, $R_1{-}O{-}R$, do not appear to be significant products of autoxidation of hydrocarbons. Much experimental evidence now indicates that alkoxy-radicals are often unstable, and so are destroyed in other ways (compare Walsh. *Trans. Faraday Soc.,* 1946, **42**, 269).
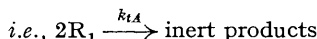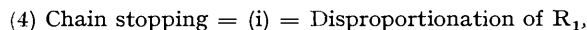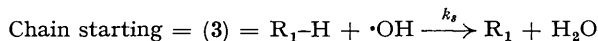
In special circumstances account should also be taken of surface reactions, or reactions with inhibitors (including molecular reaction products, see Part I, *loc. cit.*). These may for simplicity be represented as

(vi) $R_1 + I \longrightarrow$ inert products, and
(vii) $R_2 + I \longrightarrow$ inert products
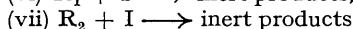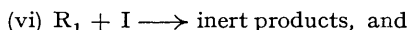
since they will be essentially of first order with respect to the radical which is removed from the system.

The following consideration of these reactions indicates (*a*) that (i) may be significant during the initial autocatalytic stage (*A*) of the autoxidation, (*b*) that reaction (v) replaces (i) as the main chain-stopping process when the hydroperoxide content of liquid has risen to a sufficient extent, and (*c*) that (vii) can account for the effects of many inhibitors. In these respects our conclusions differ from those of George and Robertson (*loc. cit.*), who based their kinetic analysis of the " thermal " autoxidation of tetralin only upon the steady second stage (*B*) of the whole process and, by showing that the chain-stopping reactions (i)—(iii) were not then concerned, attempted to discredit the whole free-radical chain mechanism for the autoxidation of tetralin in the absence of added catalysts. George and Robertson proposed instead a " thermal-chain " mechanism; this we would now suggest is basically equivalent to our view, *viz.*, that the promotion of the autoxidation of tetralin is essentially due to the thermal decomposition of minute quantities of tetralin hydroperoxide. The kinetic analysis of the chemical equations which we have already given leads to the following results.

Taking, first of all, the autocatalytic stage (*A*) of autoxidation, if, in addition to the oxygen-uptake cycle of reactions (1) and (2) (p. 1586), we write

$$\text{Chain starting} = (3) = R_1{-}H + \cdot OH \xrightarrow{k_s} R_1 + H_2O$$

(4) Chain stopping = (i) = Disproportionation of R_1,

$$\textit{i.e.,} \; 2R_1 \xrightarrow{k_{tA}} \text{inert products}$$

then we have the relationship

$$k_s[RH][OH] = k_{tA}[R_1]^2$$

whence

$$\frac{-d[O_2]}{dt} = k_1[R_1][O_2] = k_1[O_2]\left(\frac{k_s}{k_{tA}}[RH][OH]\right)^{\frac{1}{2}}$$

Now, when the peroxide content of our liquid is very small, and there are no oxidisable impurities in it, then the rate at which *free* hydroxyl radicals are produced depends upon the substantially first-order decomposition of tetralin hydroperoxide (see Part II), so that we may write $+ d[OH] = k_c[P]$, from which we see that so long as *all* the " free " hydroxyl radicals that emerge from the decomposition of individual tetralin hydroperoxide molecules are destroyed by reaction with the surrounding (tetralin) solvent

$$-\frac{d[O_2]}{dt} = k[P]^{\frac{1}{2}}[O_2]\left(\frac{k_s \cdot k_c}{k_{tA}}[RH]\right)^{\frac{1}{2}} \quad . \quad . \quad . \quad . \quad . \quad . \quad (C)$$

The complexity of the decomposition of tetralin hydroperoxide is such that the factor k_c may possibly be a function of [RH] and of any diluent liquid, for it may be difficult to discover one which is really " inert " to hydroxyl, but this consideration does not affect our argument. Equation (C) should also hold for the metal-ion catalysed reaction in its initial stages, provided that we substitute k'[P][M] for k_c[P], where [M] represents the agent which promotes the homolysis of the tetralin hydroperoxide.

Equation (C) agrees both with our own experimental demonstration that the initial rate of oxygen uptake varies with the square root of the peroxide content of the liquid, and with the fact that at first the rate of autoxidation is dependent upon the oxygen pressure.

Passing now to the second stage (B) of the autoxidation, we must remember that, as the peroxide content of the liquid increases, so too will increase the rate of production of " free " hydroxyl radicals. However, though reaction (3) thereby increases, the rate of the chain-stopping reaction (4) will not change, and soon some free hydroxyl radicals will become available for taking part in reactions such as (iv) and (v). Now, both reactions (iv) and (v) satisfy the conditions that the same catalyst is involved in both the chain-starting and the chain-stopping processes. As George, Robertson, and Rideal have pointed out (*Proc. Roy. Soc.*, 1946, *A*, **185**, 288; *Nature*, 1942, **149**, 601), this is an essential feature of all autoxidations which can proceed at a steady rate. Hence either (iv) or (v) may accord with the observed kinetics of stage (B) of tetralin autoxidation.
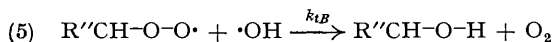
Taking (3) as the chain-starting reaction and (iv) as the chain-stopping reaction, we would find

$$- \frac{d[O_2]}{dt} = + d[P]/dt = k_1 \frac{k_s}{k_t} [RH][O_2] \quad . \quad . \quad . \quad . \quad . \quad (D)$$

whilst, taking (v) as the chain-stopping reaction, we would find

$$- \frac{d[O_2]}{dt} = + d[P]/dt = k_2 \frac{k_s}{k_{tB}} [RH]^2. \quad . \quad . \quad . \quad . \quad (E)$$

Chain-stopping reaction (v), *i.e.*,

$$(5) \quad R''CH-O-O\cdot + \cdot OH \xrightarrow{k_{tB}} R''CH-O-H + O_2$$

thus accords with the kinetics of the steady stage (stage B) in the thermal autoxidation of tetralin, as observed by George and Robertson (*Proc. Roy. Soc.*, *loc. cit.*), whilst chain-stopping by reaction (iv) corresponds to the kinetics of the metal-salt catalysed reaction at low oxygen pressures.

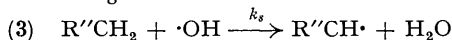Reaction (5) (= v) accords with the following significant experimental facts :

(*a*) Oxygen is liberated when tetralin hydroperoxide decomposes; it becomes approximately equivalent to the tetralol produced in the rapid, metal-salt catalysed, decomposition.

(*b*) When the autoxidation is studied by flow methods, the residual off-gases always contain some oxygen, even when the most active catalysts are used and there is prolonged contact between gas and liquid.
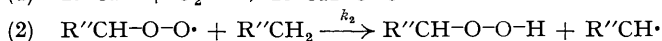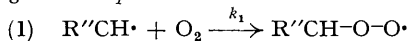
(*c*) α-Tetralol is always one of the *major* final reaction products, and is itself too stable to influence appreciably the rate of oxygen uptake by the reacting system (Robertson and Waters, *Trans. Faraday Soc.*, *loc. cit.*).

We therefore conclude that in the steady state (B) of the autoxidation the *main* reaction cycle can be represented adequately by the equations
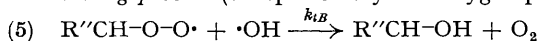
Chain starting.

$$(3) \quad R''CH_2 + \cdot OH \xrightarrow{k_s} R''CH\cdot + H_2O$$

Long chain sequence.

$$(1) \quad R''CH\cdot + O_2 \xrightarrow{k_1} R''CH-O-O\cdot$$

$$(2) \quad R''CH-O-O\cdot + R''CH_2 \xrightarrow{k_2} R''CH-O-O-H + R''CH\cdot$$

Chain-breaking process (except at very low oxygen pressures).

$$(5) \quad R''CH-O-O\cdot + \cdot OH \xrightarrow{k_{tB}} R''CH-OH + O_2$$

This sequence, and the kinetic expression (E) to which it leads, can hold so long as an adequate supply of hydroxyl radicals can be maintained by the decomposition of the tetralin hydroperoxide. The requisite condition for this is that there must be so much peroxide within

the system that the rate of production of free hydroxyl ($k_c[P]$) is *greater than* the combined rates of removal of hydroxyl radicals by both processes (3) and (5).

The excess of free hydroxyl radicals can, of course, be removed by subsidiary oxidation processes which do not affect the course of the main autoxidation cycle (see Part I), as, for example, by oxidations of tetralol or tetralone. Consequently, we find that as the autoxidation of tetralin proceeds in stage (*B*) the fraction of absorbed oxygen which is converted into tetralin hydroperoxide falls progressively. It may be noted that the velocity constant, k_s, of reaction (3) measures the mean life of free hydroxyl radicals in tetralin. The frequency of chain starting is thus limited by the extent to which bimolecular reactions of free hydroxyl, of which (5) is but one, limit its concentration. The final decline in the autoxidation rate [stage (*C*)] may in part be due to the accumulation in the liquid of secondary reaction products which remove hydroxyl radicals.

In the main, however, inhibition of autoxidation seems to be due to the removal of the radicals R″CH· and R″CH‒O‒O· which are concerned in the main oxygen-uptake cycle, since the partial removal of hydroxyl radicals would not seriously change the overall reaction velocity. Chain stopping by the removal of peroxide radicals, R″CH‒O‒O· (reaction vii), has now been established conclusively by Bolland and Ten Have (*Trans. Faraday Soc.*, 1947, **43**, 201; Faraday Society Discussion, 1947, **2**, in the press) for the autoxidation of ethyl linoleate in the presence of inhibitors such as quinol, and, for the sequence of reactions (3), (1), (2), (vii), we can derive the relationship

$$+ \frac{d[P]}{dt} = k_2 \frac{k_s}{2k_{tI}} [RH]^2 \frac{k_c[P]}{[I]} \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad . \quad \textbf{(F)}$$

where k_{tI} is the velocity constant for chain-stopping by the inhibitor; this still denotes reaction at a rate proportional to the square of the hydrocarbon content of the liquid, as found by George and Robertson in these circumstances.

The actual chain-stopping reaction in the presence of organic inhibitors can probably be represented as

$$\text{(vii, } a\text{)} \quad \text{R‒O‒O·} + \text{H‒X} \longrightarrow \text{R‒O‒O‒H} + \text{X·}$$
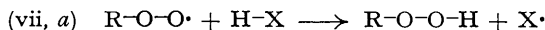
giving a hydroperoxide molecule and a resonance-stabilised radical such as a semi-quinone, particularly since most inhibitors do not destroy hydroperoxide molecules (compare Ivanov, *Compt. rend. Acad. Sci. U.R.S.S.*, 1939, **25**, 34). On the other hand, substances such as iodine, some metallic radicals, and surfaces would more probably act by promoting oxygen evolution, *e.g.*

$$\text{(vii, } b\text{)} \quad \text{R‒O‒O·} + \text{·I} \longrightarrow \text{R‒I} + \text{O}_2$$
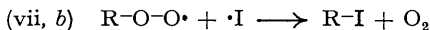
However, it may be noted that since the main oxygen uptake cycle (reactions 1 and 2) is a *long chain* process, it follows that, approximately, $[R_1]/[R_2] = k_2[RH]/k_1[O_2]$, and thus for a given substance (k_2/k_1 constant) the life of the free hydrocarbon radical will become relatively longer, and that of the peroxide radical R_2 relatively shorter, the lower the oxygen pressure. Consequently, at very low oxygen pressures chain-ending reactions of types (i), (iv), and (vi) will become prominent, and then the autoxidation rate will continue to be dependent upon the partial pressure of the oxygen, as in the initial autocatalytic stage of the reaction. If both the chain-starting and the chain-stopping reactions involve the hydrocarbon radicals, R_1, then the steady state equation (**D**) (p. 1589) may hold. An example of this is afforded by the catalysis of autoxidation by dibenzoyl peroxide for which we may write

Chain starting : Ph· + R″CH₂ ⟶ Ph‒H + R″CH·
Chain stopping : Ph· + R″CH· ⟶ Ph‒CHR″
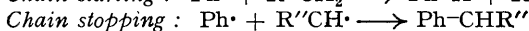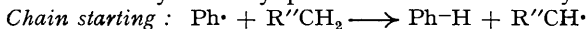
This review of the kinetics of oxygen uptake in the autoxidation of tetralin does not even yet give a complete picture of the subject, for we are still ignorant of the precise fate of the alloxy-radicals derived from the decomposition of the tetralin hydroperoxide and cannot be sure of the extent to which they affect the oxygen-uptake cycle. We do claim to have shown, however, that the free-radical reaction-chain theory will account for all the salient experimental facts, and would emphasize the point that the exact form of the reaction velocity equation seems to be much more dependent upon the nature of the chain-stopping process than upon the chain-starting process.