On the Mechanism of the Thermal Decomposition of the Peroxodisulphate Ion in Aqueous Solution

One of the mechanisms proposed for the thermal decomposition of potassium peroxodisulphate[1] in weakly acidic to alkaline aqueous solutions is that of FRONAEUS and OSTMAN[2] (hereafter referred to as mechanism A),

$$S_2O_8^{2-} + H_2O \longrightarrow SO^{\cdot-}_4 + HSO_4^- + HO^{\cdot} \quad (1)$$

$$SO^{\cdot-}_4 + H_2O \longrightarrow HSO_4^- + HO^{\cdot} \quad (2)$$

$$2HO^{\cdot} \longrightarrow H_2O + {}^1/_2O_2. \quad (3)$$

This mechanism differs from that of BARTLETT and COTMAN[3], the one generally accepted as representing the reaction sequences involved, only in the first step, which proposes instead a comparitively slow homolytic scission of the peroxide bond (mechanism B),

$$S_2O_8^{2-} \longrightarrow 2SO^{\cdot-}_4, \quad (4)$$

followed by reactions (2) and (3). Both reaction mechanisms are quite compatible with most of the experimental evidence presented to date. However, there is some evidence that indicates that mechanism A may not be operative. SMITH[4] and KOLTHOFF et al.[5] have measured the rate of capture of sulphate radical-ions by styrene monomer, during its emulsion polymerization, and compared it with the rate of decomposition of the peroxodisulphate ion. Their joint results[6] indicate that 2 sulphate radical-ions are produced for every peroxodisulphate ion decomposed. It is patent that mechanism A, which predicts that only one sulphate radical-ion is produced for every peroxodisulphate ion decomposed, cannot account for these experimental results. However, as pointed out by KOLTHOFF et al.[5], such experiments are difficult to perform and are subject to rather large error. The present communication describes a test of mechanisms A and B that is experimentally simpler and more accurate than the radical-capture experiments described above.

If peroxodisulphate is decomposed in the presence of a free-radical scavenger, then mechanisms A and B differ in their predictions of the rate of production of acid, relative to its rate in the absence of the scavenger. Mechanism A predicts that even if the scavenger is completely effective in removing $SO^{\cdot-}_4$ and $HO^{\cdot}$ as they are formed, then as a minimum, the rate of production of acid should be equal to half the rate in the absence of scavenger. Mechanism B predicts that no acid is formed if the scavenger is completely effective.

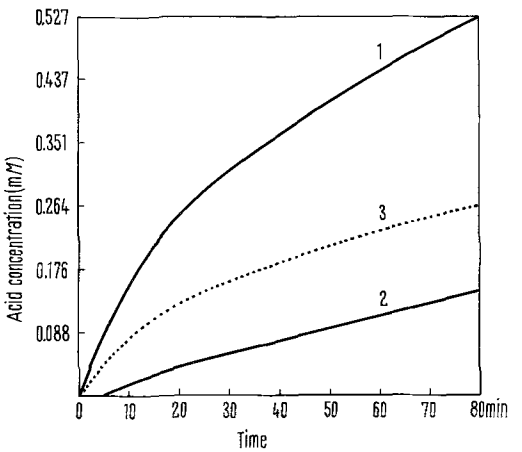
Accordingly, the production of acid from peroxodisulphate decomposition in aqueous solution was followed by a pH-stat continuous potentiometric technique. The radical scavenger was styrene monomer, solubilized with sodium tetradecysulphate[7] (a radical-scavenging system that does not affect the rate of decomposition of potassium peroxodisulphate[5]).

Experimental. The potassium peroxodisulphate was B.D.H.A.R. quality and was recrystallized 3 times from water ($< 50\,°C$) and dried under vacuum (final purity: $< 99.8\%$, by iodometric assay). The sodium hydroxide was a Merck guaranteed reagent ($< 99\%$). The sodium sulphate was of A.R. purity. The styrene was Fluka purum grade ($> 99.5\%$) and was vacuum distilled before use. The sodium tetradecylsulphate was prepared by the method of DREGER et al.[8].

The experiments were performed with a Radiometer PHM 26c pH meter with a G202C glass electrode and a K401 calomel reference electrode, a TTT 11b titrator, an SBR 2c titrigraph, and an ABU 1b autoburette (2.5 ml) with a modified TTA 3 titration assembly. The ionic strength of the solution in the burette was adjusted to that of the solution in the reaction vessel by the addition of sodium sulphate. Titrations were performed at $60 \pm 0.05\,°C$ in a thermostated vessel and at pH 8.00 ± 0.05. Purified nitrogen gas was passed through the water used for 35–40 min and a concentrated peroxodisulphate solution was injected into the reaction system from a hypodermic syringe. Purified nitrogen (previously saturated with water) was continuously passed through the reaction vessel[9].

Results and discussion. The Figure shows the acid concentration during the thermal decomposition of initially $5 \times 10^{-3} M$ potassium peroxodisulphate in the presence and absence of free-radical scavenger. Also included is the curve (dotted) showing the minimum acid concentration predicted by mechanism A for the decomposition in the presence of scavenger.

The first order rate coefficient for the decomposition in the absence of scavenger was found to be $4.7 \pm 0.3 \times 10^{-4}$ min^{-1}. This value is in agreement with those reported in the literature[10] that, for the most part, range from $4–5 \times 10^{-4}$ min^{-1}.



Acid concentration during the decomposition of $5 \times 10^{-3} M$ $K_2S_2O_8$; temperature $60 \pm 0.05\,°C$; pH 8 ± 0.05. Curve 1, no additive; Curve 2, in the presence of scavenger-styrene (5.0 ml/25.0 ml aqueous phase) and $0.02 M$ sodium tetradecylsulphate; Curve 3, minimum acid concentration expected if mechanism A were operative.

[1] The mechanisms of peroxodisulphate decomposition are excellently reviewed in the following: (a) D. A. HOUSE, Chem. Rev. *62*, 185 (1962). (b) W. K. WILMARTH and A. HAIM, in *Peroxide Reaction Mechanisms* (Ed. J. O. EDWARDS; Interscience, New York 1962), p. 175.

[2] S. FRONAEUS and C. O. OSTMAN, Acta chem. scand. *9*, 902 (1955); *22*, 2827 (1968).

[3] P. D. BARTLETT and J. D. COTMAN JR., J. Am. chem. Soc. *71*, 1419 (1949).

[4] W. V. SMITH, J. Am. chem. Soc. *71*, 4077 (1949).

[5] I. M. KOLTHOFF, P. R. O'CONNOR and J. L. HANSEN, J. Polym. Sci. *15*, 459 (1955).

[6] For a discussion of the results of references [4] and [5] see: F. A. BOVEY, I. M. KOLTHOFF, A. I. MEDALIA and E. J. MEEHAN, in *High Polymers* (Interscience, New York 1955), vol. IX, p. 69.

[7] Acid from the hydrolysis of the sodium tetradecylsulphate is quite insignificant at the experimental conditions used in this study.

[8] E. E. DREGER, G. I. KEIM, G. D. MILES, L. SHEDLOVSKY and J. Ross, Ind. Engng. Chem. *36*, 610 (1944).

[9] B. BALLANTYNE, Experientia *24*, 329 (1968).

[10] For compilations of rate coefficients see: E. HAKOILA, Ann. Univ. Turku Ser. A66 (1963), and reference [1](a).

In the presence of scavenger, the initial rate of acid formation is zero. As the efficiency of the radical trap falls off – due to the incorporation of monomer into the growing polymer radicals – the acidity of the system correspondingly increases. The initial zero rate of acid production emphasizes however that such an increase in acidity is due to reaction (2) and not to any significant contribution of reaction (1) to the production of acid.

It is apparent that the mechanism of FRONAEUS and OSTMAN[2] does not account for these experimental findings which strongly support the view that reaction (4) is the first step of the decomposition sequence[11].

Zusammenfassung. Säurebildung als Zerfallsprodukt des Peroxodisulfations wurde in Gegenwart eines Radikalsammler mittels pH-stat-Technik untersucht: Die Säurebildung ist zu Beginn null. Dieses Ergebnis stützt die Theorie, wonach $S_2O_8^{2-} \rightarrow 2SO\cdot_4^-$ die Anfangsstufe der Zerfallsfolge ist.

E. P. CREMATY

Department of Physical Chemistry,
University of Sydney,
Sydney (N.S.W. 2006, Australia), 25 August 1969.

[11] Acknowledgement. The author is deeply indebted to Prof. A. E. ALEXANDER for his guidance in this work and for his valuable advice during the preparation of the manuscript.

Retardation of Protein Synthesis in Rat Tumours on Inhibiting Histamine Formation

The discovery of high histidine decarboxylase activity in foetal rat tissues was the starting point for a new field of study: the association between high histamine forming capacity (HFC) and rapid growth[1]. High HFC has been found in various normal and malignant rapidly growing tissues. In healing rat skin wounds collagen formation and tensile strength of the wound could be reduced or enhanced by respectively inhibiting or elevating tissue HFC. Histidine decarboxylase (EC 4.1.1.22) could be specifically and strongly inhibited by α-methyl histidine in vitro and in vivo[2]. The relevant literature has been reviewed by KAHLSON and ROSENGREN[3].

In the rat foetus histidine decarboxylase activity of the liver exceeds that of the young or adult by about 1000 times. Under the influence of α-methyl histidine the rate of protein synthesis, as measured by the incorporation of ^{14}C-leucine, has been shown to be substantially diminished in foetal liver but not in that of the young[4]. The retarded leucine incorporation could not be restored by adding histamine to foetal liver tissue, an instance, among others already known, where extracellular histamine lacks the function of 'nascent histamine', a term coined for the kind of intracellular histamine believed to be associated with rapid growth.

The present study extends the earlier investigation[4] to include rapidly growing malignant tissues, the Walker 256 mammary carcinosarcoma and the Rous virus sarcoma. These rat tumours form histamine at relatively high rates, and the enzyme concerned, histidine decarboxylase, is specifically inhibited by α-methyl histidine[5,6]. The methods employed in the present report are the same as in the previous study[4]. In some experiments, besides α-methyl histidine, the compound 4-bromo-3-hydroxy benzyloxyamine (NSD-1055) was used. This compound inhibits histidine decarboxlase strongly in vitro[7–9].

Both tumours were transplanted subcutaneously to female Sprague-Dawley rats. The Walker 256 tumour was allowed to grow for 5–7 days and the Rous sarcoma for 10–13 days. In each experiment pooled tissue from 2 or 3 animals was used. The tumours were rapidly excised and freed of necrotic parts before mincing. For HFC determinations[2], minced tissue samples of about 200 mg were incubated for 90 min with or without inhibitor. To determine the rate of protein synthesis[4], about 100 mg of minced tumour tissue was preincubated for 20 min at 37 °C with or without inhibitor, 0.05 μmoles of 1-^{14}C-L-leucine (25 mCi/mmole) was added and the samples were incubated for 90 min. Incorporation rates of leucine were estimated in 2 fractions, referred to as soluble and insoluble protein fractions, obtained by the centrifugation of the homogenized samples at 30,000 *g* for 30 min. Results are detailed in Tables I and II.

The rate of histamine formation was higher in the Rous virus sarcoma than in the Walker 256 tumour. In the former tumour 2.5 m*M* DL-α-methyl histidine depressed histamine formation by about 80% (Table I). In the latter tumour (Walker 256), however, inhibition by α-methyl histidine was inconsistent, and in 2 cases it had no effect. The inhibitor NSD-1055 was employed in the Rous sarcoma only, in which 0.5 m*M* concentration strongly depressed histamine formation (Table I).

The rate of leucine incorporation was about the same in the 2 protein fractions in both tumours (Table II). This result differs from the situation in the rat liver in which the incorporation rate into the insoluble protein fraction was only 66% of that in the soluble fraction[4]. In the absence of inhibitor, the rate of incorporation was nearly the same in both tumours. In the Walker 256 tumour, leucine incorporation under the influence of α-methyl histidine (2.5 m*M*) was about 80% of the control values in both protein fractions, and in 2 instances there was no effect (Table II). It would thus appear that this tumour to a minor extent, or not at all, depends on nascent histamine for its growth. In the Rous tumour, by contrast, either of the inhibitors at the concentration indicated, depressed leucine incorporation into both protein frac-

[1] G. KAHLSON, Lancet *1*, 67 (1960).
[2] G. KAHLSON, E. ROSENGREN and R. THUNBERG, J. Physiol. *169*, 467 (1963).
[3] G. KAHLSON and E. ROSENGREN, Physiol. Rev. *48*, 155 (1968).
[4] B. GRAHN, R. HUGHES, G. KAHLSON and E. ROSENGREN, J. Physiol. *200*, 677 (1969).
[5] M. JOHNSTON, Experientia *23*, 152 (1967).
[6] C. G. AHLSTRÖM, M. JOHNSTON and G. KAHLSON, Life Sci. *5*, 1633 (1966).
[7] R. J. LEVINE, T. L. SATO and A. SJOERDSMA, Biochem. Pharmac. *14*, 139 (1965).
[8] M. JOHNSTON and G. KAHLSON, Br. J. Pharmac. Chemother. *30*, 274 (1967).
[9] L. ELLENBOGEN, E. MARKLEY and R. J. TAYLOR JR., Biochem. Pharmac. *18*, 683 (1969).