Excited Complexes in Photosensitized Processes. Kinetic Characteristics

Chemical reactions induced by light in the presence of luminescent energy carriers (photosensitizers), in the vapour phase or in solution, show certain characteristic features. The kinetics of these systems in particular often appear to conform to relations of the LANGMUIR isotherm type. This paper outlines a general kinetic treatment of these cases, which assumes the formation of a steady state excited complex between photosensitizer and reactant. The concept provides a useful approach to problems of energy transfer in photoexcited systems.

Consider the typical system:

$$A^* + B \underset{k_2}{\overset{k_1}{\rightleftarrows}} A^*B \xrightarrow[k_3]{} C$$

where A^* ≡ excited photosensitizer; B ≡ ground state reactant; A^*B ≡ steady state complex and C ≡ products of reaction.

In the steady state:

$$k_1 [A^*] [B] - k_2 [A^*B] - k_3 [A^*B] = 0$$

We may write $[A_0^*] \simeq [A^*] + [A^*B]$ (for negligible product formation)

where $[A_0^*]$ is the total concentration of excited species whence, by elimination of $[A^*]$ we get:

$$[A^*B] = \frac{k_1 [A_0^*] [B]}{k_1 [B] + (k_2 + k_3)}$$

if v ≡ measured rate of reaction:

$$v = k_3 [A^*B] = \frac{k_3 [A_0^*] [B]}{[B] + K_s}$$

where k_s ≡ steady state dissociation constant $= (k_2 + k_3)/k_1$ $[A_0^*]$ is related to the light absorption of the system by:

$$[A_0^*] = \tau_0 I_\alpha$$

where τ_0(sec) ≡ observed lifetime of A^* in the absence of B, I_α (einsteins l^{-1} sec^{-1}) ≡ light absorbed by $[A_0]$.

Since v is a function of the type $y = ax/(bx + c)$, a plot of rate against reactant concentration $[B]$ will give a hyperbolic curve (Figure 1a). The limiting rate which the function approaches as $[B]$ increases is given by $v_{max} = k_3 [A_0^*]$.

The hyperbolic dependence of rate on reactant concentration is a familiar feature of heterogeneous catalytic reactions which follow the simple LANGMUIR isotherm. (A similar kinetic form is, in fact derivable from LINDEMANN's theory[1,2] in the special case of a unimolecular reaction.) The present treatment shows that homogeneous photosensitized reactions which involve a steady state complex may conform to similar kinetics. In the simplest case the reaction rate should rise to a limiting value as the reactant concentration is increased. A hyperbolic form of the rate function indicates an initial first order process which approaches zero order kinetics at higher reactant concentrations. Certain homogeneous gas phase reactions photosensitized by mercury vapour behave in this way[3–6]. Experimental evidence supports our theory that, in these cases, excited photosensitizer and reactant species are in equilibrium with a stationary complex[7–9].

Similar kinetic characteristics have been found in certain photosensitized reactions in solution. Typical of these are the photochemical decomposition of iodine in the presence of diazoacetic ester[10], and the photosensitized oxidation of amino acids by excited flavines[11,12]. In the case of excited flavine systems, evidence indicates that flavine complexes are probably involved[13].

Fluorescence quenching. For fluorescence quenching of A^* by the quenching species B we have:

$$A^* + B \underset{k_2}{\overset{k_1}{\rightleftarrows}} A^*B \xrightarrow[k_3]{} A + B^*$$

A^* ≡ excited (singlet) A, A^*B ≡ excited complex, B^* ≡ excited B. As before, for steady state conditions:

$$[A^*B] = \frac{[A_0^*] [B]}{[B] + (k_2 + k_3)/k_1}$$

The experimentally observed overall rate constant k_e in the steady state is

$$k_e = k_3 k_1/(k_2 + k_3)$$

If the STERN-VOLMER equation is followed:

$$(I_0/I) - 1 = K [B] = k_e \tau_0 [B]$$

I_0 ≡ fluorescence intensity in absence of quencher, I ≡ fluorescence intensity in presence of quencher, K ≡ STERN-VOLMER constant, τ_0 ≡ mean fluorescent lifetime of A^*, we may then write

$$\frac{I_0}{I} - 1 = \frac{k_3 k_1}{k_2 + k_3} \tau_0 [B]$$

If thermal energy E is needed for dissociation of the complex into A^* and B (for example by triplet to singlet activation) we get[14]:

$$\frac{I_0}{I} - 1 = \frac{k_3 k_1 \tau_0 [B]}{k_2 \exp(-E/RT) + k_3}$$

The quenching constant will therefore tend to decrease with rise in temperature, in those cases where the E of k_2 is predominant[14].

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When the quenching process is diffusion controlled ($k_3 \gg k_2$)

$$\frac{I_0}{I} - 1 \simeq k_1 \tau_0 [B]$$

and the magnitude of k_1 may be calculated from the diffusion equation[15–22]:

$$k_1 = 4\pi \, \mathrm{DRNP} \times 10^{-3}$$

where $R \equiv$ sum of radii of A and B, $D \equiv$ the diffusional constant $= kT/(6\pi\eta)$ $(1/\gamma_A + 1/\gamma_B)$, $\eta \equiv$ viscosity, $P \equiv$ probability factor and $\gamma_A \gamma_B$ are radii of A and B. If however $k_3 \ll k_2$

$$\frac{I_0}{I} - 1 = \frac{k_3 k_1}{k_2} \tau_0 [B] = k_3 K^*_{AB} \tau_0 [B]$$

where $K^*_{AB} = [\overset{*}{A}B]/([\overset{*}{A}]\,[B])$.

Inhibition of photosensitized reaction. Consider a photosensitized process in which an unreactive ternary complex AB_2 is formed:

$$\overset{*}{A} + B \rightleftarrows \overset{*}{A}B \xrightarrow{k_3} C$$
$$\overset{*}{A}B + B \rightleftarrows AB_2$$

Steady state treatment gives:

$$[\overset{*}{A}B] = \frac{K_I [\overset{*}{A}_0] [B]}{[B]^2 + K_I [B] + K_I K_D}$$

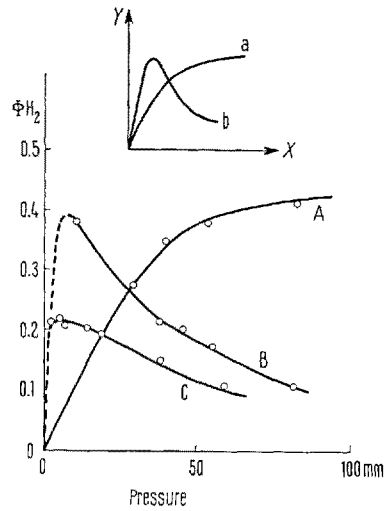
where K_D, K_I are respective steady state dissociation constants of $\overset{*}{A}B$ and AB_2.
Then v (measured rate of reaction)

$$= k_3 K_I [\overset{*}{A}_0] [B]/([B]^2 + K_I [B] + K_I K_D)$$

since this function has the form $y = ax/(x^2 + bx + c)$ a plot of rate against reactant concentration will pass through a characteristic maximum (Figure). This is analogous to the HINSHELWOOD-LANGMUIR case in heterogeneous catalysis. Homogeneous photosensitized reactions in which ternary complexes are formed should therefore conform to similar kinetics, with inhibition at increasing reactant concentrations. Several cases of this have been described[23–26], but no satisfactory explanation of the phenomenon has been available hitherto. Photosensitized reactions of unsaturated compounds in particular often display this behaviour[23–28]. The formation of dimers (B_2) occurs significantly in these circumstances[27–29].

Nature of the steady state complex. The expressions which we have derived for photosensitized processes are based on the hypothesis that an actual complex is formed between the species $\overset{*}{A}$ and B. We assume that the complex arises by interaction of an excited state of A with the ground state of B, probably to give a complex of the excimer type[30–33]. Formation of the excimer $\overset{*}{A}B$ would take place by diffusion-controlled collision with partial charge transfer from B to excited $\overset{*}{A}$. 'Static interaction' between ground states of A and B to give ground state complexes may be neglected in the examples considered here, since the absorption spectra of A and B in general are unaffected, and charge-transfer bands are absent at normal temperatures, in the dilute systems considered[34].

The concept of the steady state complex presented in this paper provides an alternative to 'collisional encounter' treatment[16]. It enables us to regard fluorescence quenching as an incipient electron transfer from B to $\overset{*}{A}$ which provides the first step necessary for photochemical reaction by a redox process[19,35]. This would tend to be facilitated by perturbed transitions to a common triplet



(A) Quantum yield of mercury photosensitized hydrogen production from n-C_4H_{10} at room temperature as a function of pressure (CVETANOVIC[6]). (B, C) Quantum yield of mercury photosensitized hydrogen production from C_2H_4 at 20 °C (B) and 0 °C (C) as a function of pressure (DARWENT[24]). Inset. (a) Characteristic form of the graph of the function $y = a\,x/(b\,x + c)$ (b) Characteristic form of $y = a\,x/(x^2 + b\,x + c)$.

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level within the complex, yielding the reactive triplet state of B by dissociation.

From a thermodynamic point of view it is certainly convenient to employ a steady state constant related to the constant of quenching, as an alternative to the more conventional use of collisional cross sections[36,37]. The transfer of excitation energy between different energy levels of the system is then capable of clearer definition[38]. In particular, the probability term P in the diffusion-encounter treatment may be correlated with transitions between available excited levels in A and B[21]. Finally, the concept provides an interesting analogy between photoexcited systems and enzyme systems where the hypothesis of the steady state 'MICHAELIS complex'[39] has proved so fruitful[40,41].

Zusammenfassung. Die Kinetik homogener Photoreaktionen, welche über einen Excimer-Komplex verlaufen, wird untersucht. Es ergibt sich, dass in Übereinstimmung mit zahlreichen Beobachtungen eine Konzentrationsabhängigkeit ähnlich der von LANGMUIR-HINSHELWOOD für die heterogene Katalyse abgeleiteten zu erwarten ist.

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Combined Action of Rous Sarcoma Virus and Chemical Carcinogen in Rats

The combined effect of oncogenic viruses and chemical carcinogens has been extensively studied in recent years (for review see DURAN-REYNALS[1], and SALAMAN and ROW[2]). The possibilities with the combined action can be summarized as: (1) enhancement of the oncogenic effect of the virus; (2) enhanced oncogenic effect of the chemical carcinogen; (3) simple additive effect of the 2 agents.

The first alternative was most often observed: viral tumours appeared earlier and/or in greater number, grew more rapidly or persisted for a longer time in carcinogen-treated animals than in the controls which had only been treated with virus. Enhanced effect of the chemical carcinogen by virus has rarely been observed.

Few investigations have been carried out on the effect of Rous sarcoma virus (RSV) in animals treated with chemical carcinogens. CARR[3] injected methylcholanthrene and RSV into chickens belonging to a strain of low viral susceptibility. Small tumours appeared in the breast muscles at the site of virus inoculation, whereas a large swelling developed in the methylcholanthrene-injected leg. The swelling slowly subsided and tumours subsequently appeared in various parts of the leg, which were histologically indistinguishable from those induced by the RSV. In young rabbits i.v. RSV has been shown to localize to the site of i.m. injected hydrocarbons, producing fibromatous nodules[4]. In addition many rabbits showed nodules in the lungs and liver, occasionally also in the spleen and kidneys. The nodules were larger and more numerous than in rabbits not treated with hydrocarbon.

The present experiments were carried out to investigate the effect of i.v. RSV in rats, which had been injected i.m. with a carcinogenic hydrocarbon.

The Rous virus was of the strain Schmidt-Ruppin (RSV-SR) which is able to induce tumours in a wide variety of mammals as well as in birds[5]. Pools of cell-free virus suspension were prepared from rapidly growing tumours induced in the chicken. To do this finely minced chicken sarcoma was suspended 1:5 in Hank's solution with antibiotics and homogenized for 5 min in an Ultra-thurrax homogenizer (24,000 rpm) in the cold; the suspension was then centrifuged for 30 min at 3000 *g*. The supernatant was pipetted off and stored at − 70 °C. Two pools were used in the experiments. The titer of the virus was $1.5–2.4 \times 10^6$ FFU/ml tested on monolayers of chick fibroblasts. The rats were white ones, kept as a closed colony for many years at the institute. 7,12-Dimethylbenz(*a*)anthracene (DMBA) dissolved in arachis oil or trioctanoine was used as a carcinogen.

One mg DMBA was injected i.m. into the left thigh of 41 rats, 2 weeks of age. 10–12 days later, 28 of the rats were given 1 ml of the virus pool i.v. via one of the tail veins. The same amount of virus was injected into 16 rats of the same age which had not been treated with the carcinogen. The rats were examined once a week for 4 months and then killed.

No tumours were observed in any of the rats which had been given the virus alone, nor did they show any hemorrhagic cysts in the lymph nodes which is a common finding when RSV-SR is inoculated into new-born rats.

Seven of the 13 rats which had been injected with DMBA and had not had any further treatment, developed a tumour at the site of injection. The first tumour appeared approximately 12 weeks after the injection and had often reached a considerable size by the time the rats were sacrificed. A few of the tumours had produced ulceration of the overlying skin. They had the histological appearance of various types of sarcomas: spindle cell sarcomas, polymorphous cell sarcomas, myosarcomas and anaplastic sarcomas. No metastases were seen in any of the internal organs or in the lymph nodes.

Fifteen of the 28 rats that had been exposed to the combined effect of DMBA and RSV-SR developed tumours in the left thigh at the site of the injected hydrocarbon. The tumours appeared at about the same time as in the rats treated with DMBA alone and were of the

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