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EXCITATION POOLS IN THE FROG'S RETINA

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The pioneer work of Granit (1942) showed that a single ganglion cell in the frog's retina was not always connected to one type of receptor. The studies described in our three preceding papers (Donner & Rushton, 1959*a*, *b*; Donner, 1959) were undertaken to explore further the connexion between ganglion cells and the different types of receptor. It was clear that if in any given state only one type of photoreceptor was connected, stimulation by substitution would exhibit three characteristics. (*a*) A silent substitution could always be achieved if the intensity of the new light was correctly chosen, the correct intensity (in quanta/sec) for each wave-length varying inversely as the absorption at that wave-length by the visual pigment involved. (*b*) The Fechner fraction, that is the logarithmic range of intensities over which the substitution remained silent, must be constant whatever wave-length was involved. (*c*) Adaptation to any colour which did not alter the connexion between receptor and ganglion would leave the spectral sensitivity of silent substitution unchanged.

The results of our first paper (Donner & Rushton, 1959*a*) showed that some ganglion cells did not exhibit silent substitution, and hence must have been connected to more than one type of receptor at the same time. This was a complexity which we wished to avoid, and so we chose only those cells which did respond usually by silence when the substituted intensity was properly adjusted. Even these cells failed in a restricted range of intensity just below the level where the photopic dominator curve is obtained. But in the photopic range the results were consistent with the idea that ganglion cells are connected to a single class of receptors (cones) whose visual pigment absorbs according to Granit's (1942) photopic dominator curve: in full dark adaptation they are connected to other cells (rods) whose sensitivity corresponds to rhodopsin, and in changing from dark to mesopic adaptation there is a gradual change from the pure rhodopsin sensitivity to one with 'humps' in the green and in the blue, but with the characteristics (*a*, *b*, *c*) above still maintained. A formal explanation could be that some inert screening pigment

had been formed which changed the action spectrum of rhodopsin, but this rather unlikely suggestion was excluded by the investigations of our second paper (Donner & Rushton, 1959*b*).

In that paper we applied to the frog the beautiful analysis of Flamant & Stiles (1948), who showed that the sensitivity of human cones, but not rods, exhibited a retinal directional effect. We confirmed that in the frog too this sensitivity to the direction of incident light is shown only by cones. So when the green hump of the mesopic spectral sensitivity was found to be direction-sensitive, it was clear that cones were among the receptors involved. The blue hump on the contrary was not direction-sensitive, but it could be selectively depressed by adapting to bright blue light (though not to green). The hump therefore must be due to rods with blue-sensitivity—the 'grass-green' rods of Boll & Kühne which Denton & Wyllie (1955) have shown to be bleached by blue but not by green light.

It is therefore clear that the mesopic sensitivity is made up by at least blue-sensitive rods, green-sensitive rods and yellow-sensitive cones. And yet they are all organized in such a way that the ganglion cell exhibits the characteristics (*a*, *b*, *c*) above, though intense adaptation may alter the spectral sensitivity, probably by changing the connexions between the receptors and the ganglion.

What we need to know is by what arrangement can several types of receptors converge upon a ganglion cell and exhibit at the same time both these characteristics and the very different properties seen in Donner's beautiful family of kinked increment threshold curves (Donner, 1959, fig. 3). It is the purpose of this paper to set out a simplified and semi-quantitative hypothesis of retinal organization to embrace these observations.

Rods and cones do not have the same spectral sensitivity and so stimulation by substitution, e.g. a change from yellow to blue, involves a diminished relative excitation of cones and increased excitation of rods. The fact that this leads to 'silent substitution' for a critical intensity ratio means that the ganglion cell is exactly compensated for the loss of cone excitation by gain in rod excitation. So we are led to the idea of an 'Excitation Pool' whose level depends upon the streams of excitation from all the receptors connected to it. Only the rise (or fall) of the pool's level causes 'on' (or 'off') excitation. A change in the distribution of streams to this pool will not excite this ganglion cell, provided that the change is carefully adjusted so as to cause no alteration in the pool's level. Figure 1*A* shows a diagram of what is envisaged: steady changes are transmitted to the pool, but only alterations in level affect the ganglion.

Now the level of the pool is not at present a quantity that has been directly measured; it is inferred from a supposed relation to the intensity and colour of the light, and in terms of this inference the firing or non-firing of ganglion

cells is predicted, and so a verifiable structure of experiment is built which will relate light with ganglion discharges. The fundamental relation postulated between the light and the level of the excitation pool will now be stated.

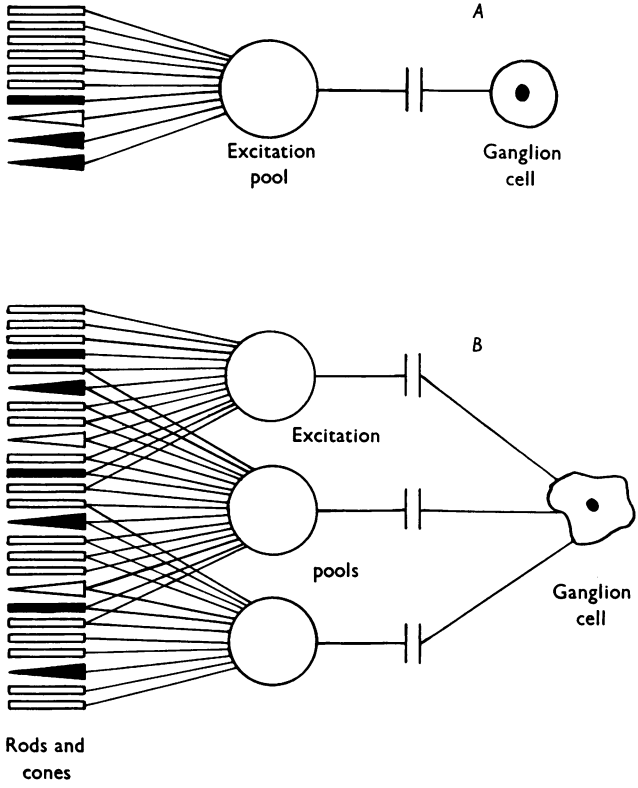

Fig. 1. Diagram of excitation pool. Steady excitation from various receptors is transmitted to the pool. Only changes in the pool's level are transmitted to the ganglion. *A*, the simplest model; *B*, the complication required by conditions of non-silent change.

Excitation pool postulates

(i) If any single receptor (1) absorbs light by its visual mechanism at an average rate J_1 quanta/sec it will generate in the pool an average excitation E_1 independent of the wave-length of the light.

(ii) The relation between E and $\log J$ is shown by the curve in Fig. 2. It is very nearly that given by the dotted line which meets the axis at $\log \theta$. That would express the relation that when J is less than its threshold value θ there is no excitation; when J is greater

$$E = \log J/\theta.$$

But the actual relation shows a small region of gradual transition from the

horizontal axis to the 45° slope, and there is a 'saturation' of the response at very high intensities. We may denote this function by ψ (log J/θ).

The basis of this relation is derived from the electroretinogram in vertebrates (Chaffee, Bovie & Hampson, 1923) and eccentric cell potentials in *Limulus* (Fuortes, 1959) which indicate that as soon as light is converted into an electric signal, that signal is related to log J in a way similar to E in Fig. 2. This relation is discussed further below.

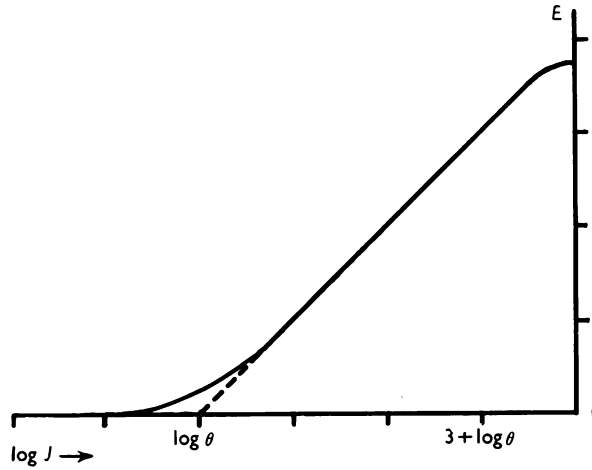

Fig. 2. Relation between J, the rate of absorption of quanta by a receptor, and E the excitation produced; $E = \psi \log J/\theta$.

(iii) If many receptors of the same type (1) are connected to the pool, the total level

$$E = b_1 \psi(\log J_1/\theta_1),$$

where b_1, the connexion coefficient, is a constant amounting to the sum of the excitations of all the receptors, taking into account their various attenuations between receptors and pool. All these receptors are assumed to have the same threshold θ_1.

(iv) If there are many types of receptors connected to the pool, the total level

$$E = b_1 \psi(\log J_1/\theta_1) + b_2 \psi(\log J_2/\theta_2) + \ldots.$$

(v) A ganglion discharge at 'on' (or 'off') will occur when E suddenly increases (or decreases) by a fixed amount ΔE, which is generally different for 'on' and for 'off' and is independent of the actual value of E, or of adaptation.

We have so little knowledge of the way in which the absorption of a quantum in a photoreceptor produces an electric signal that the function ψ which represents this change is purely empirical. But the sensitivity of rods is so great that the lower range of their ψ function corresponds to a rate of

quantum absorption of only 1 per rod per minute or less. So it is rather artificial to treat this average rate as a continuous variable, and a probability statistic of quantum events, though more complicated, would be more realistic. But the present analysis is making no attempt to explain the transduction from light to electricity and is concerned only with the way in which the conducted excitations, E, from various receptors interact. Therefore qualitatively we may accept a curve of the general form of ψ (Fig. 2), since this does appear to be the relation observed between the light and the electrical output. And quantitatively we take the exact shape shown in Fig. 2 because this does fit the increment threshold relation for a single receptor, e.g. rods alone, as will appear later. It will be observed that this hypothesis is very similar to that proposed by Willmer (1955) in his analysis of colour vision in the human fovea.

Our problem is to find how far this fairly plausible intensity function, ψ, together with the other postulates mentioned, will account for the rather extended set of experimental relations observed in the preceding papers. It is not to be expected that our limited and rigid assumptions will describe at all accurately the wonderful versatility of visual performance. In particular, adaptation, which has so profound an effect upon visual function, enters here in only two small features. Since different receptors have different thresholds, more receptors will contribute to the pools at high intensities than at low. But when the light is very bright the rhodopsin rods will reach their saturation point and give no contribution to vision, as Aguilar & Stiles (1945) have demonstrated in man.

The expectations of these restricted assumptions will be worked out first for the conditions of silent substitution, and will be found to accord well with observation, if all the receptors involved are adapted to a level well above their absolute thresholds. But there are serious difficulties with the blue receptors which do not satisfy this condition. Next the silent transition between the mesopic and photopic states is studied and it is found that not one but several excitation pools must be connected to the ganglion cell, as indicated in Fig. 1*B*. Finally we turn to Donner's (1959) experiments upon two-colour increment thresholds.

It might be thought that Donner's branched curves, which strongly suggest the transition from a lower rod portion to an upper cone portion, were incompatible with an arrangement where all the receptors pour their contributions into a common pool. But when the consequences of the foregoing postulates are worked out, we obtain the family of curves shown in Fig. 5, which correspond well enough with Donner's observations.

In the Appendix to this paper are derived the expectations which follow mathematically from the postulates. We now proceed to compare those expectations with the experimental results.

Silent substitution

The mesopic state. Figure 3*A* of our first paper (Donner & Rushton, 1959*a*) shows the spectral relation for silent substitution at an adaptation level of 10–50 times the absolute threshold. The curve nearly coincides with the rhodopsin absorption spectrum. Figure 3*B* shows results from the same ganglion when the adapting light had been increased 400 times. There was no blue hump and so we assume that we are still below threshold for the green rods. But we are well above threshold for the rhodopsin rods and also for the cones. So the condition is one where the excitation E of the pool lies on the straight part of the slope (Fig. 2) and hence is proportional to $\log J/\theta$, and the relation given by equation (4) of the Appendix (p. 343) applies.

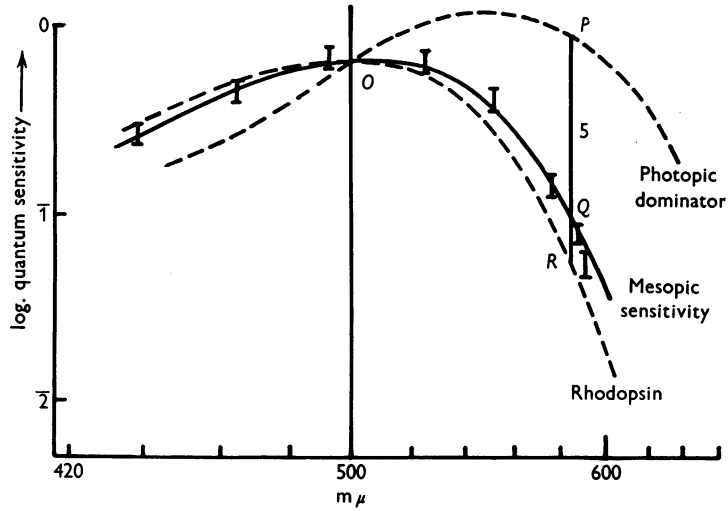

Fig. 3. Experimental points replotted from fig. 3*B* of Donner & Rushton, 1959*a*. Dotted curves are the rhodopsin and the photopic dominator on a logarithmic plot, adjusted to cross at *O*. Full curve is the weighted mean of the two dotted curves, *RQ* being 1/5 of *QP*.

In Fig. 3 of this paper the experimental points of curve *B* are replotted, together with the rhodopsin curve and the photopic dominator (both dotted). The continuous curve is obtained by dividing the ordinate difference between rod and cone sensitivity in the proportions of 1:5, which is the theoretical curve of equation (4) of the Appendix, if we assume that the connexion coefficient for rods is 5 times that of cones. This curve adequately fits the experimental points, including the region in the blue where they fall slightly below the rhodopsin curve.

From equation (5) of the Appendix the Fechner fraction should be constant throughout the curve at a value 5/6 that of curve *A* (Donner & Rushton, 1959*a*, fig. 3). This also is satisfied within experimental limits. Thus the hypothesis

of this paper explains quantitatively the fact of silent substitution, the form of the spectral sensitivity curve, the constancy of the Fechner fraction and the amount it is reduced by the entry of cone excitation, simply by assuming that the connexion coefficient for cones is 1/5 of that for rods. In the frog's retina there are about half as many cones as rods.

Before passing to the more difficult situation when green rods are also involved we may note how the geometry of Fig. 3 illustrates the contribution to E made by the various receptors. The log. light stimulus represented by the point Q lies a distance QP below the cone sensitivity curve and hence generates in the cones an excitation QP more than does light O which lies on the curve. But Q lies a distance QR above the rod sensitivity curve. So in changing from O to Q the cones are excited QP more, the rods QR less. QP is 5 times QR but the connexion coefficient of rods is 5 times as great, hence the change of E at the pool is exactly neutralized. And so it will be for every point Q which lies 1/6 of the way from the scotopic to the photopic curve.

Now this argument must be modified when receptors are involved which are exposed to lights not far above threshold or even below it, as evidently is the case for the green rods, which are sometimes present and sometimes absent at mesopic levels. In Fig. 4 are re-drawn the experimental points of fig. 5*B* from Donner & Rushton, 1959*a*, the mesopic sensitivity curve of Fig. 3 (obtained by combining the rhodopsin curve with the photopic dominator in the proportions 5:1), and a curve maximal at 430 mμ to represent the sensitivity of the green rods. This latter curve is given the same shape as the rhodopsin curve but is shifted along the axis of wave frequencies so that the maximum lies at 430, to correspond with the pigment which Dartnall has found by partial bleaching in extracts of frogs' retinas (Dartnall, 1957, p. 191, fig. 3.7), though a maximum at 450 mμ or longer would suit our results better.

The horizontal shift of the blue curve is thus defined, but the vertical shift is left arbitrary. Let us suppose that in Fig. 4 the vertical position shown corresponds to the *threshold* level of the green rods (but cones and rhodopsin rods of course have their thresholds at much weaker intensities). Then since all points on the blue curve represent intensities which are just threshold, points below the curve excite, points above do not (note: increasing sensitivities are represented upwards, increasing intensities downwards).

It is now clear how we may combine the contributions to the pool both of green rods and the other receptors represented by the mesopic curve (Fig. 4). The part of the mesopic curve to the right of the intersection corresponds to intensities below threshold for the green rods. In this region, then, the mesopic curve alone represents the spectral sensitivity (as it did in Fig. 3 when *all* the curve was below the threshold of blue receptors).

The curve on the left of the intersection involves the green rods which add

to the mesopic curve by the rule of the weighted mean (Appendix equation 4). The connexion coefficient is here taken as 0·1 of the sum of the coefficients of all the receptors. As constructed, the green rod contribution meets the mesopic curve at a kink. This is because the relation between E and log J (Fig. 2) was taken as the dotted line running straight to the axis. Since the ψ relation is a curve, the transition in Fig. 4 should also be smoothed and not kinked. The final sensitivity curve of Fig. 4 is seen adequately to fit the experimental

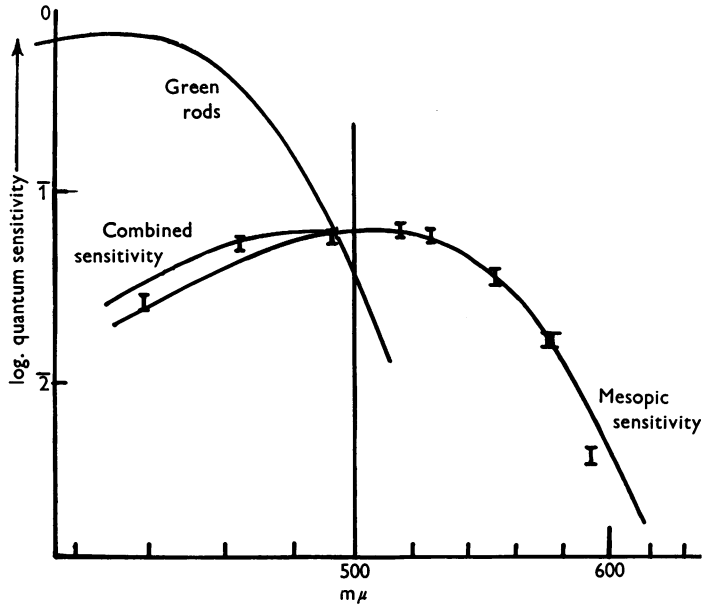

Fig. 4. Experimental points replotted from fig. 5B (Donner & Rushton, 1959a). The mesopic curve is replotted from Fig. 3, the green rod curve is taken from Dartnall (1957), and to the left of its intersection is combined with the mesopic curve by the rule of weighted mean, pulling 1/10 of the total weight.

points of fig. 5B of Donner & Rushton, 1959a, which are here represented as bars. And the observed fact that sensitivity at longer wave-lengths is the same whether the blue hump is present or absent is satisfactorily explained if the threshold level of the green rods cuts the mesopic curve at wave-lengths near 500 mμ.

But this type of explanation is hard to reconcile with the results of fig. 5 of Donner & Rushton, 1959a. The striking feature there was that the sensitivity curve appeared to be exactly the same shape, though curve B was at an adaptation level 25 times as great as in A and in both curves identical results were obtained with adaptation wave-lengths of 576 or 464 mμ. Stability of the sensitivity curve despite change in adaptation would be expected if all the receptors involved were well above their thresholds. But this cannot

easily be said of the blue sensitivity curve which never seems to depress the green hump of the mesopic curve as it would if it was there involved according to the rule of the weighted mean. The expedient of keeping the green rods out of the long-wave half of the mesopic sensitivity curve, by supposing that they reach threshold at about the 500 mμ adaptation level, is well enough for one curve of fig. 5 (Donner & Rushton, 1959*a*) but it can hardly apply to all four of them. So we are left with an awkward discrepancy between the 'excitation pool' theory and observation, and a need to know the actual spectral sensitivity of the green rods.

The photopic state. Wald, Brown & Smith (1955) have pointed out that the spectral absorption of iodopsin fits the photopic dominator curve about as accurately as rhodopsin fits the scotopic curve. If iodopsin is a homogeneous pigment this suggests that frogs and many other animals have only one class of cone contributing to the dominator curve. On the other hand, the photopic dominator is similar to the luminosity curve in man, which is chiefly due to the combined effect of two pigments which cannot be iodopsin (Rushton, 1958). Granit has long ago suggested that the photopic dominator might be a combination of narrower curves (modulators) and Donner (1953) has produced good evidence that in the pigeon this is the case.

Stimulation by silent substitution, though undertaken in the hope that it might afford decisive evidence, is seen to advance the matter hardly at all. To be sure, all the conditions of silent substitution in the photopic state (fig. 4, Donner & Rushton, 1959*a*) are precisely what would be expected if every cone contained iodopsin, but the analysis of mesopic conditions has required the concept of an excitation pool which predicts identical relations from mixed receptors.

Histologists (e.g. Rochon-Duvigneaud (1943), fig. 212, p. 351; Saxén, 1954) describe two distinct cone types, and Donner (1959) has some preliminary physiological observations which support this. But since in nearly every result of our three papers the cones appear all to act together in a fixed proportion, in the present analysis it will be sufficient to assume that cones may be represented by a single type of contribution to the pool, namely that with the sensitivity of the photopic dominator.

The break-down of silent substitution. There are two conditions where a ganglion which normally exhibits a silent change loses this property. One is at the high mesopic adaptation level, in transition to the pure photopic dominator curve (Donner & Rushton, 1959*a*, fig. 6), and the other is at any level when the spatial distribution of light upon the retina changes. For instance, instead of changing from one colour to another, each a uniform field and precisely coinciding, we may change from one small spot to another small spot very near to it. In this case it is usually impossible to get a silent change even if the two lights have the same colour. Indeed, it was pointed out that

the wedge W in fig. 1 (Donner & Rushton, 1959*a*) required a small compensator to make the field uniform, otherwise it was impossible to get silent substitution in a sensitive preparation, since not all parts of the field could be matched simultaneously when changing from the graded field of the wedge to the uniform field of the substituted light. Barlow (1953) observed the same kind of response except with pure 'off' ganglions. The diagram in Fig. 1*A* of this paper does not take account of such spatial effects. For if the ganglion was connected to only one pool which showed a unique level of excitation, a silent change should be obtainable whatever the spatial distribution of the adaptation light or of its substitute.

But since the above observations show that pooling occurs only over areas which are much smaller than the receptive field of the ganglion, we must conclude that there are many pools connected to each ganglion. And the simplest modification is that of Fig. 1*B*, where bipolars or horizontal cells may be regarded as the pools. On the input side they receive and mix the streams of excitation from various receptors (with perhaps one receptor supplying more than one pool); on the output side they transmit a nerve signal when the level $\pm E$ changes sufficiently. The pools must be supposed normally not to differ much as to the proportions in which they receive contributions from the different types of receptors, so with a uniform field a substitution which is silent for one will be silent for all, and hence for the ganglion connected. But it is probable that, during the transition from mesopia to photopia, the pools show marked individual differences. There is a very big change in spectral sensitivity between the mesopic and photopic states, due largely to the loss of contribution from the rhodopsin rods, and, if the various bipolar or horizontal cells were not all at precisely the same stage in their rod loss, no stimulation by light substitution could match all of them at once. Thus at the first stage of the transition it is only with an extreme colour change that the breakdown is detected, but, as the heterogeneity of rod connexion develops, a smaller and smaller change will be silent (as seen in fig. 6, Donner & Rushton, 1959*a*), until at length only a change to a nearly identical colour can remain without discharge. But when the process is complete, and all the rod contributions abolished, the pools will be restored anew to homogeneity, and thus the silent substitution of the photopic dominator will be obtained.

Increment threshold

The concept of retinal excitation to which we have been led by the experiments on silent substitution is one where rods and cones of all kinds pool their excitations and become indistinguishable from a single type of receptor, whose log. sensitivity is the weighted mean from all those receptors. This is a very different concept from that derived from Stiles's (1939, 1949, 1959) two-colour increment thresholds. The striking thing about that great body of

experimental work is the way that the rod mechanism and the various cone mechanisms behave as *independent* units. Whatever the mechanism excited by the flash, its increment threshold depends upon its own spectral sensitivity, and that only. Test flashes of various colours are effective to stimulate just in proportion to the sensitivity of the mechanism to that colour: adaptation fields of various colours are effective in depressing excitability to the flash in proportion to that same sensitivity relation. And when changing conditions of flash or adaptation favour the excitation of a new mechanism the transition is abrupt, showing a pronounced kink in the log. increment threshold curve.

Such independence of component mechanisms is not what would be expected of ganglion cells which exhibit silent substitution, for with them it appears that individual mechanisms pour their contributions towards excitation into a common pool. Yet at first sight the careful experiments which Donner has undertaken to study this matter confirm Stiles in all the main particulars. In Donner's family of curves (Donner, 1959) each member shows a lower branch corresponding to the (rhodopsin) rods, and an upper branch corresponding to the (photopic dominator) cones, and the results are consistent with a fairly sharp transition between the two branches.

Now the rather precise relations between receptors which have been put forward to explain silent substitution allow us also to calculate the family of curves to be expected for the conditions of Donner's fig. 3. These curves are shown here in Fig. 5 together with a reproduction of Donner's 77 points. The curves were calculated from the excitation pool hypothesis, making the following specific assumptions.

(i) With the deep-blue adaptation field which was used, the green rods will not be involved in the increment flashes of wave-lengths 527–646 mμ. So the receptors present will be rods with rhodopsin sensitivity and cones with sensitivity corresponding to the photopic dominator.

(ii) The relation between E the excitation level in the pool and log I is given by the ψ curve in Fig. 2. Its derivative ψ' is shown in the Appendix (Fig. 6).

(iii) The increment threshold is small enough for this increment to be obtainable from the first derivative ψ' only.

(iv) Suitable numbers are given to four constants so as to introduce the actual values of the thresholds and Fechner fractions of rods and of cones in Donner's experiment (Donner, 1959, fig. 3). The assumptions, in short, are that rods and cones have their known spectral sensitivity, and that E is generated according to the initial postulate of Fig. 2, which will give the increment threshold curve for rods alone. Then the choice of the four ringed points out of the 77 in Fig. 5 allows the positions and shapes of all the curves to be defined absolutely. The way that this calculation is done is indicated in the Appendix.

The curves of Fig. 5 do not fit the points so well as the curves which Donner has drawn in his fig. 3. But this is not surprising, since his were illustrative only and not intended to be exact according to Stiles's concepts. Neither are all the rod portions of his curves formed exactly by one curve slid vertically,

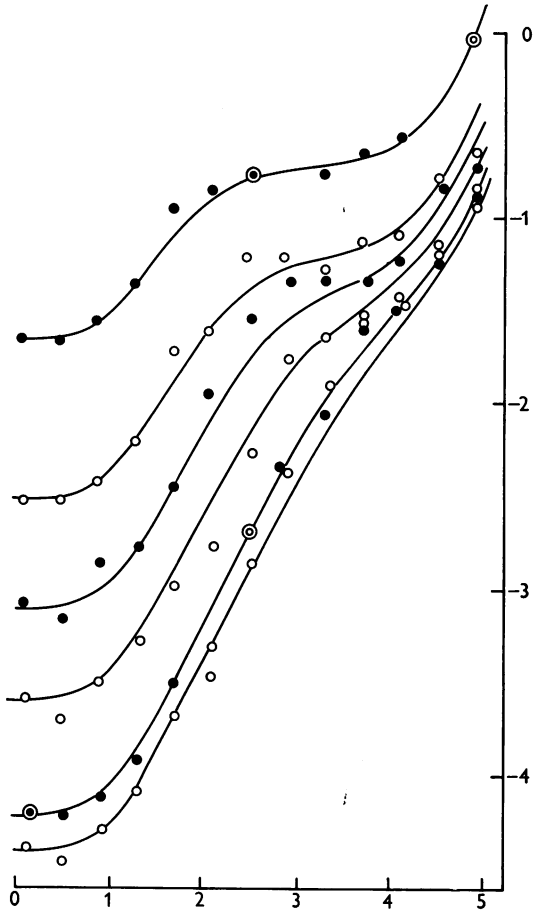

Fig. 5. The points are from fig. 3 (Donner, 1959). The curves are calculated from the excitation pool postulates using the four ringed points to give the thresholds and Fechner fractions of rods and of cones.

nor are all the cone portions one curve slid vertically, nor is the cone curve at all the same as the rod curve slid horizontally, nor is the cone sensitivity fitted to the photopic dominator curve. The curves were drawn to fit the points and fit them well and so illustrate clearly the general relations displayed. Donner in fact is careful to point out that the upper branch does not keep its shape like a single Stiles's mechanism and that many elements show only a gradual transition from one branch to the other, which makes analysis

in terms of Stiles's mechanisms unreliable. The family of calculated curves in Fig. 5 on the other hand involves very little that is arbitrary (as was pointed out above) and the over-all correspondence with experiment is very fair. The principle conclusion to be drawn is that the concept of excitation pools which was put forward to account for silent substitution is consistent with the increment threshold results of Donner (1959). For though at first these appeared rather incompatible they turn out to be a predictable consequence.

We must be careful, however, not to conclude that Stiles's results in man should be interpreted in the same way. In addition to the fact that transitions from one mechanism to another are much sharper in Stiles's results than in the curves of Fig. 5 there is a more fundamental difference. The excitation pools we have studied have only one parameter, E; the increment threshold is simply an intensity threshold for the pool, whatever λ and μ. But in Stiles's experiments the subject may be able to say that the flash appearing upon the blue field is red and not green, now bright, now just visible. If in this way it is a two-parameter system we should be cautious about identifying it with the colourless excitation pool in the frog.

Spectral sensitivity

It is worth noting that the idea of excitation pools predicts quite different spectral sensitivity curves in (*a*) silent substitution and in (*b*) increment threshold. In both cases the experimenter may use as index the smallest intensity of new light which will elicit a response (*a*) when the adaptation light is removed and replaced by light of a different wave-length, (*b*) when the adapting light is left unchanged, but added to it is an increment of new wave-length.

Equation (4) (p. 343) shows that for (*a*) the log. sensitivity curve should be the weighted mean of the log. sensitivities of all the contributing receptors. Equation (6) on the other hand shows that for (*b*) a different relation obtains, which may be re-written

$$\frac{I\Delta E}{\Delta I} = \frac{D_1(\lambda)}{D_1(\mu)} b_1 + \frac{D_2(\lambda)}{D_2(\mu)} b_2 + \frac{D_3(\lambda)}{D_3(\mu)} b_3 + \ldots \qquad (6A)$$

for conditions where the light is bright enough for ψ to be on the straight 45° slope (Fig. 2) for all the receptors 1, 2, 3 ... involved. $D_n(\mu)$, $D_n(\lambda)$ are the relative pigment densities or quantum sensitivities of receptor n to the adapting wave-length μ and the increment wave-length λ respectively. It is seen that for fixed adaptation to intensity I of wave-length μ, the increment threshold ΔI depends upon the weighted means of the sensitivities (not log. sensitivities) of the contributory receptors. This result is formally identical with the condition where the pigments are mixed in suitable proportions in a single class of receptor. There are however important differences. If I is weak enough to leave the pigments substantially unbleached (as is generally the case) then a change

in the wave-length μ will not affect the proportions in which the pigments are mixed in the receptors, but it may change very much the proportions in which the receptors enter into relation ($6A$) by altering the values of the various denominators. This in fact is the basis of Donner's (1959) preliminary evidence that the photopic dominator involves more than one cone.

If on the other hand it is ΔI and λ which are fixed and I and μ are adjusted so that the increment flash is just threshold, then ($6A$) shows that I depends upon the weighted mean of the *reciprocals* of the receptor sensitivity. Thus the relation between I and μ should not be the same as that between ΔI and λ, except when the pool is fed by a single type of receptor. In Stiles's experiments the two relations were the same, which is consistent with the view that colour mechanisms enter *singly* into each branch of his curves.

Intraretinal recording

When the concept of Excitation Pools was first inferred from the results of silent substitution, it seemed presumptuous to propose this specific mechanism for which there was no evidence from intracellular recording. But in the last year or two records, mainly from the eyes of fish, have exhibited properties very similar to those here postulated.

The records which Svaetichin (1953, 1956) first obtained were attributed to single cones (where they could hardly represent pools derived from many kinds of receptors). But as the result of careful location of the recording site, Svaetichin & MacNicol (1958) and MacNicol & Svaetichin (1958) have now shown that the cells responsible lie in the region of the horizontal or bipolar layers, where they may well be affected by receptors of different types. The characteristic of these records when they are 'luminosity responses' is, that so long as a steady light falls upon the eye there is a steady hyperpolarization of the region recorded. The potential rises and falls rapidly with the light like the output of a photocell, except that the change is linear not with the intensity of the light but with its logarithm. In fact the relation of amplitude with $\log I$ shown in fig. $2B$ of Svaetichin & Jonasson (1956, p. 8) is almost identical with the ψ function in Fig. 2 of the present paper. The spectral sensitivity of this 'luminosity response' is very similar to the photopic dominator curve, and may well receive contributions from receptors covering the whole spectral range.

Similar records have been obtained by Motokawa, Oikawa & Tasaki (1957), Tomita (1957), Tomita, Tosaka, Watanabe & Sato (1958), Grüsser (1957) and Brown & Wiesel (1958), the latter two from the eye of the cat.

Though it is too early yet to form a precise opinion upon the histology and electrophysiology of the structures involved, these most important new types of retinal record encourage us to hope that this paper may receive substantial support in its general concepts, and in its details valuable corrections.

SUMMARY

1. In the mesopic state rods and cones are simultaneously contributing excitation to the ganglion cell. Yet it is possible to substitute light of one colour for that of another without discharge.

2. This leads to the idea of an excitation pool whose level depends upon the streams of excitation from many kinds of receptors, but which only excites the ganglion through a change in the level of the pool.

3. A set of restricted rigid properties are postulated, determining the way that light influences the pool's level, and this the ganglion discharge.

4. In the mesopic state, where rhodopsin rods and cones are well above threshold, the 'excitation pools' hypothesis accords well with observation. But the 'grass-green' rods, which are near threshold, do not fit well.

5. The non-silent transition from the mesopic to the photopic state requires that a ganglion be connected to more than one pool.

6. The two-colour increment-threshold results of Donner are also in reasonable accord with the excitation pools hypothesis.

7. The intraretinal potentials which have been recorded by Svaetichin and others, especially from fish, have properties very similar to those postulated for the excitation pools.

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APPENDIX

A quantitative formulation

Let I_λ = intensity of light of wave-length λ falling upon the retina in units of quanta $\sec^{-1}$ cm^{-2},

J_1 = rate at which quanta are being absorbed by the visual pigment of receptor (1),

$D_1(\lambda)$ = density of this pigment at wave-length λ measured in units such that $J_1 = I.D_1(\lambda)$,

E = level of total excitation in the Pool,

b_1 = connexion coefficient which determines what proportion of excitation from the group of receptors (1) reaches the pool,

θ_1 = 'threshold' for excitation of receptor (1) defined by the relation of Fig. 2,

$\pm\Delta E$ = change in the level of the pool necessary for a ganglion response. Since 'on' and 'off' thresholds are usually quite different $+\Delta E$ and $-\Delta E$ must be taken as quite different in magnitude. It is likely that ΔE may change in various conditions, e.g. 'fatigue' by quickly repeated flashes, but in the present treatment it is assumed to remain constant.

$\psi(\log J/\theta)$ is the function relating E with J/θ shown in Fig. 2.

$$\text{For } J/\theta \gg 1, \quad \psi(\log J/\theta) = \log J/\theta, \tag{1}$$

$$\text{For } J/\theta \ll 1, \quad \psi(\log J/\theta) = 0.$$

1. *Silent substitution.* In general

$$E = \sum^{n} b_n \psi\left(\log \frac{J_n}{\theta_n}\right), \tag{2}$$

where the summation $\sum^{n}$ includes all the receptor types. Consider an adaptation level where receptors 1, ..., m are well above their thresholds, and all the rest are well below. Then from (1)

$$E = \sum_{n=1}^{m} b_n \log \frac{J_n}{\theta_n} = \sum_{1}^{m} b_n \log \frac{I_\lambda . D_n(\lambda)}{\theta_n},$$

therefore

$$\frac{E + \sum_{1}^{m} b_n \log \theta_n}{\sum_{1}^{m} b_n} = \log I_\lambda + \frac{\sum_{1}^{m} b_n \log D_n(\lambda)}{\sum_{1}^{m} b_n}. \tag{3}$$

Now the condition for silent substitution is that I_λ should change in wavelength and intensity in such a way that E does not alter. In that case the left-hand side of equation (3) will not alter, and may be written as $\log K$. So equation (3) becomes

$$\log \frac{K}{I_\lambda} = \frac{\sum_{1}^{m} b_n \log D_n(\lambda)}{\sum_{1}^{m} b_n}, \tag{4}$$

where the left side is the log. spectral sensitivity for silent substitution, and the right side is the weighted mean of the log. absorption curves of all the visual pigments 1, ..., m.

The Fechner fraction in silent substitution may be found by differentiating equation (3) where E and I are the only variables.

$$\frac{2{\cdot}3\ \Delta E}{\sum_{1}^{m} b_n} = (\Delta I/I)_\lambda. \tag{5}$$

The right side is the Fechner fraction. It is therefore independent of λ, being directly proportional to ΔE which we are taking to be always constant, and inversely to the sum of all the connexion coefficients involved.

2. *Increment threshold.* We treat the case where only the rhodopsin rods and the cones are involved, with spectral sensitivities $D_1(\lambda)$, $D_2(\lambda)$ respectively. Then from equation (2)

$$E = b_1 \psi\left(\log \frac{J_1}{\theta_1}\right) + b_2 \psi\left(\log \frac{J_2}{\theta_2}\right).$$

Therefore $$2{\cdot}3\,\Delta E = b_1 \frac{\Delta J_1}{J_1}\psi_1' + b_2 \frac{\Delta J_2}{J_2}\psi_2',$$

writing ψ_1', ψ_2' for the derivatives

$$\frac{d\psi(\log J_1/\theta_1)}{d\log(J_1/\theta_1)}, \quad \frac{d\psi(\log J_2/\theta_2)}{d\log(J_2/\theta_2)} \text{ respectively.}$$

Now the adapting light I is at wave-length μ,

therefore $$J = I.D(\mu).$$

But the flash ΔI is at wave-length λ:

therefore $$\Delta J = \Delta I.D(\lambda);$$

therefore $$2{\cdot}3\,\Delta E = \frac{\Delta I}{I}\left(\frac{D_1(\lambda)}{D_1(\mu)} b_1 \psi_1' + \frac{D_2(\lambda)}{D_2(\mu)} b_2 \psi_2'\right).$$

or $$\log(2{\cdot}3\,\Delta E) + \log I - \log \Delta I = \log\left(\frac{D_1(\lambda)}{D_1(\mu)} b_1 \psi_1' + \frac{D_2(\lambda)}{D_2(\mu)} b_2 \psi_2'\right). \qquad (6$$

To calculate the curves of Fig. 5 (in all of which $\mu = 464$) we need to evaluate the four constants of equation (6), by introducing the actual values of four points in Fig. 5. Consider the curve where $\lambda = 552$ and introduce the condition that at $\log I = 3{\cdot}0$, $\log \Delta I = -2{\cdot}2$, at $\log I = 0$, $\log \Delta I = -4{\cdot}2$. These appear to lie below the cone threshold so we put $\psi_2' = 0$ and justify it by the fit of the curve so computed. Substituting the first pair of values in equation (6) gives

$$\log(2{\cdot}3\,\Delta E) + 3{\cdot}0 + 2{\cdot}2 = \log b_1 \frac{D_1(552)}{D_1(464)} + \log \psi_1'(3). \qquad (7)$$

Subtracting from this the similar equation with the second pair of values gives

$$1{\cdot}0 = \log \psi_1'(3) - \log \psi_1'(0). \qquad (8)$$

Now the shape of ψ_1' is shown in Fig. 6, but its position along the horizontal axis is not yet determined. $\psi_1'(3)$ is clearly 1, therefore $\log \psi_1'(3) = 0$, therefore from equation (8) $\psi_1'(0) = 0{\cdot}1$. So the curve ψ_1' in Fig. 6 is correctly placed.

Now in Fig. 5 the results of $\lambda = 646$ show that when $\log I = 4{\cdot}8$, $\log \Delta I = -0{\cdot}1$, and $\psi_1' = 1 = \psi_2'$.

Substituting these values in equation (6), and subtracting equation (7) with $\log \psi_1'(3)$ put zero, gives

$$-0{\cdot}3 = \log\left[\frac{D_1(646)}{D_1(552)} + C\right],$$

where $$C = \frac{b_2}{b_1} \cdot \frac{D_1(464)}{D_1(552)} \cdot \frac{D_2(646)}{D_2(464)}. \qquad (9)$$

Since $D_1(646)/D_1(552)$ is very small compared with C

$$C = \text{antilog}\,(-0{\cdot}3) = 0{\cdot}5.$$

The final constant required is the lateral shift of ψ_1' in Fig. 6 which will bring it into the correct position for ψ_2'. This has been found by trial computation and is shown in Fig. 6.

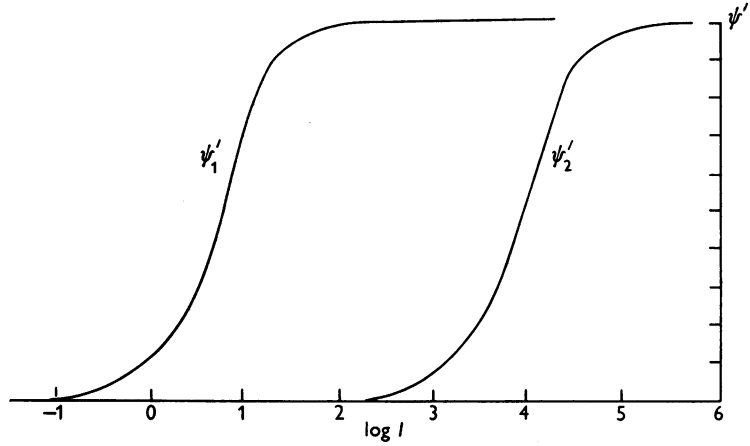

Fig. 6. Abscissae give log. intensity, I. Ordinates give the derivative of the function ψ shown in Fig. 2. The positions of ψ_1' and ψ_2' correspond to the thresholds for rods and cones in Fig. 5.

The formula for the family of curves in Fig. 5 is therefore found by subtracting equation (7) from equation (6), which gives

$$\log I - \log \Delta I = 5{\cdot}2 + \log\left[\frac{D_1(\lambda)}{D_1(552)}\,\psi_1' + 0{\cdot}5\,\frac{D_2(\lambda)}{D_2(646)}\,\psi_2'\right]. \qquad (10)$$

For any given λ we measure $D_1(\lambda)/D_1(552)$ upon the rhodopsin sensitivity curve, and $D_2(\lambda)/D_2(646)$ upon the photopic dominator curve. For any given $\log I$ we measure ψ_1' and ψ_2' upon the curves of Fig. 6. The values of $\log \Delta I$ so computed are plotted in Fig. 5.