the membrane can be characterized by a single frequency, since multiple marker particles on a single RBC membrane moved with the same frequency during the motion (Fig. 3C). Figure 4 shows the tank tread frequency (f) measured as a function of $\dot{\gamma}$ and η_0. There was a linear increase of f with $\dot{\gamma}$ with the same slope for all values of η_0 tested. In contrast, the observed elongation for a particular shear rate strongly depends on η_0 (*16*, *17*).

The results reported here lead to a number of observations about red cell micromechanics:

1) The demonstration of the existence of shear flow within the cell proves the fluidity of the cytoplasm and rules out the assumption of elastic elements in the cytoplasm.

2) The constant phase relationship between multiple membrane markers during tank tread motion makes it unnecessary to postulate an extremely inviscid liquid membrane film (*4*) to explain the fluid droplike behavior of the entire red cell. In contrast, our observations show that despite the well-established shear elasticity of the membrane (*18*), continuous shear deformations are tolerated and allow the generation of stationary flow within the cytoplasm. Other results (*19*) show that tank tread motion can occur even after the normally low shear modulus of the membrane has been strongly increased on modification of membrane proteins by reagents with SH groups.

3) For better definition of individual RBC's, the tank tread motion in whole blood was observed at a small distance (5 μm) between cone and plate. This gives rise to the objection that the proximity of the cone and plate is responsible for the tank tread motion. However, elongation and orientation could be observed at a much greater distance (15 μm), where wall effects can be neglected. From this we conclude that the tank tread motion also occurs in the bulk flow of whole blood at sufficiently high shear rates. We postulate that the participation of the RBC cytoplasm in the shear flow of the whole suspension is an important factor in the low-bulk viscosity of whole blood and its shear thinning behavior at high shear rates.

4) Although an emulsion is a better model for blood than a suspension, there are decisive differences in the behavior of red cells and liquid droplets when the viscosities of continuous and dispersed phases are matched in both systems. In liquid droplets the frequency of motion of the boundary face depends on the viscosity ratio of the dispersed and the continuous phase, whereas in RBC's the tank tread frequency is independent of the suspending media investigated and thus of the viscosity ratio. This difference could be attributed either to a favorable ratio of the volume to the surface area of the RBC, allowing large deformation of the cell without an increase of the membrane area, or to the predominance of the mechanical properties of the membrane over the viscosity ratio of the cytoplasm and the suspending phase (*20*).

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Human Flicker Sensitivity: Two Stages of Retinal Diffusion

Abstract. *A well-known solution of the diffusion equation gives an exponential square-root function as the frequency response for a one-dimensional diffusion or transmission process. When two or more such processes are cascaded, the result is still an exponential square-root characteristic, but with a longer time constant. This seems to explain why flicker thresholds obey the Kelly-Veringa diffusion model at high frequencies, even though the psychophysically inferred diffusion process is much slower than the first stage of visual transduction measured by, for example, late receptor potentials. Two such stages in tandem are sufficient to account for the psychophysical data, because the psychophysical time constant is proportional to the square of the number of stages involved. In addition, the nonlinear behavior of flicker thresholds under intense light adaptation can be explained if the loss factor in the first stage is proportional to the amount of the photopigment bleached. Apparently the flicker thresholds are governed by first- and second-order retinal neurons.*

An old problem in psychophysics is the question of whether the thresholds for certain classes of simple stimuli are controlled primarily by retinal mechanisms or by higher visual centers. For photopic flicker thresholds, some insight into this question is provided by the photoreceptor diffusion model proposed by Veringa (*1*). As modified by Kelly (*2*), the diffusion model describes the flicker sensitivity as a function of frequency by the expression

$$G(\omega) \sim \exp(-\sqrt{\omega\tau}) \qquad (1)$$

where G is the amplitude sensitivity, ω is the flicker frequency multiplied by 2π, and τ is the time constant of the diffusion process. At relatively high flicker frequencies, this exponential square-root function fits the sinusoidal flicker data from many sources (*2*–*8*), both psycho-

Table 1. Comparison of receptor and psychophysical time constants from several sources.

Receptor time constants			Psychophysical time constants	
τ_1	Reference	$4\tau_1$	τ	Reference
0.19	(*5*)*	0.76	0.82	(*6*)‡
0.19	(*3*)†	0.76	0.74	(*8*)§
0.15	(*6*)*	0.60	0.50	(*2*, *7*)‖

*Late receptor potential. †Electrical phosphene. ‡10° field. §7° field. ‖Ganzfeld.

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physical and electrophysiological. It also accounts for the traditional data on critical flicker frequencies (CFF).

We will argue that this psychophysical flicker characteristic can be resolved into a small number of cascaded diffusion stages, very likely two. We confirm that the first stage represents "the diffusion of a transmitter molecule inside the retinal receptor cells," as Veringa hypothesized (*1*, p. 413). We suggest that the second stage may represent the passive, graded-potential response of bipolar cells. Near the flicker threshold, the rest of the visual system apparently performs no further frequency-dependent filtering on the output of this second retinal diffusion stage.

Two different types of experiments, involving electrical phosphenes (*3, 4*) and late receptor potentials (*5*), agree exactly on a value of 0.19 second for the time constant of the receptor stage. That agreement was first pointed out in a recent study by Kelly, Boynton, and Baron (*6*), who noted that this electrophysiological time constant is much smaller than the usual psychophysical one. They concluded that the psychophysical flicker characteristic is not controlled solely by transmitter diffusion within the receptor cells, but must involve more proximal stages of the visual process.

However, they also found that they could bring the psychophysical time constant into agreement with the electrophysiological one by superimposing an intense adapting background (25,000 trolands) on the psychophysical stimulus. Their explanation for this result was that the slower diffusion process that governed the psychophysical data must be somehow "desensitized" by the adapting background, allowing the faster, electrophysiological one to be detected. That is, they believed that the flicker characteristic revealed by intense adaptation was that of the receptors.

But a background intense enough to bleach half the available cone pigment would be more likely to suppress the frequency dependence of the photoreceptor response, leaving the remaining stages of the visual system in control of the shape of the psychophysical flicker characteristic. We hypothesize that an increase in the (normally negligible) losses of the receptor diffusion process causes just such a flattening of its contribution to the flicker characteristic. Indeed, by making the loss term of the receptor stage proportional to the (steady state) amount of cone pigment bleached, we can predict the nonlinear behavior of the flicker threshold under intense light adaptation.

Thus the agreement found by Kelly *et al.* (*6*) between the electrophysiological flicker characteristic and the intensely adapted psychophysical characteristic plays a different role in our interpretation: It suggests that the time constants of various neural diffusion processes in the retina may be nearly alike. With that assumption and a knowledge of the time constant of any one of these processes, we can calculate the number of diffusion stages controlling the psychophysical flicker data.

To do this, we first generalize Eq. 1 to represent the behavior of n different stages in cascade

$$\exp(-\sqrt{\omega\tau}) = [\exp(-\sqrt{\omega\tau_1})] \times [\exp(-\sqrt{\omega\tau_2})] \ldots [\exp(-\sqrt{\omega\tau_n})] \quad (2)$$

The more such stages we cascade, the slower the total process will appear to be; its time constant can be calculated as

$$\sqrt{\tau} = \sqrt{\tau_1} + \sqrt{\tau_2} \ldots + \sqrt{\tau_n} \quad (3)$$

If we now assume that all stages are alike (that $\tau_1 = \tau_2$, and so forth), then Eq. 2 reduces to

$$\exp(-\sqrt{\omega\tau}) = [\exp(-\sqrt{\omega\tau_1})]^n \quad (4)$$

and

$$n^2 = \tau/\tau_1 \quad (5)$$

That is, if the psychophysical characteristic is controlled by n identical diffusion processes in cascade, its time constant will be n^2 times the constant for a single such process.

All the available values that we could use for τ and τ_1 are given in Table 1; substituted in Eq. 5 they give values of n ranging from 1.6 to 2.3. Indeed, if we assume that $\tau_1 = 0.19$ (the value on which the phosphene data and receptor potentials agree), and $\tau = 0.76$ (a value between the 7° and 10° values of τ in Table 1), we obtain $n = 2$ exactly.

In deriving Eq. 5, we did not specify that τ_1 is a property of cone cells; only that it represents one stage in a chain of cascaded diffusion processes governing the psychophysical data. However, if the psychophysical flicker thresholds are controlled by two stages with equal time constants, there is no longer any reason to assume that the intense background of Kelly *et al.* (*6*) somehow flattened the frequency response of the second stage; first-stage flattening is much more likely. In fact, it can be predicted from photopigment bleaching, as we will show. Together with the receptor potentials and phosphene data, this result provides strong evidence that the first stage does in fact represent transmitter diffusion inside the receptor cells, as hypothesized by Veringa (*1*).

What about the second stage? We suggest that the retinal bipolar cells (second-order neurons that generate passive graded potentials) may be the site of a second diffusion process, with about the same time constant as the receptor diffusion process. Intracellular recordings in *Necturus* (*9*) show that response to a spot of light occurs at about 150 msec in the receptor cells and about 600 msec in the bipolar cells. That is the relation predicted by Eq. 5 with $n = 2$. It appears that no further mechanisms are needed to account for the high-frequency flicker thresholds (*10*).

Of course it might be fortuitous that the data appear to obey Eq. 5 so precisely. But in a well-designed signaling channel, the filtering characteristics of successive stages should be approximately matched. Moreover, the single-stage time constants required by the psychophysical characteristic decrease as the inverse square of the number of stages, so that values of n much greater than 2 become implausible. For example, maintaining $\tau_1 = 0.19$ second and apportioning the remainder of the psychophysical process equally among two stages makes $\tau_2 = 0.048$; with three stages, $\tau_2 = 0.021$ second, an order of magnitude smaller than τ_1.

On the other hand, τ_1 and τ_2 need not be exactly matched. If the former represents intrareceptor diffusion and the latter intrabipolar diffusion, they may well differ slightly. Our argument is not affected by small differences between these time constants, as shown by the following examples. Suppose we cascade $\tau_1 = 0.19$, the receptor time constant, with $\tau_2 = 0.15$, as measured by Kelly *et al.* (*6*), which we regard as the bipolar time constant. This gives $\tau = 0.68$ second, which is within the range of psychophysical values in Table 1. Or if we combine $\tau = 0.74$ with $\tau_2 = 0.15$, and solve for τ_1, we obtain 0.22 second for the receptor diffusion constant, which is probably within the accuracy of the phosphene and receptor-potential data.

Next, we show how intense light adaptation can eliminate the frequency dependence of receptor diffusion, thus revealing the second-stage characteristic psychophysically. In the presence of loss or decay of the diffusing substance, Eq. 1 becomes

$$G(\omega) \sim \exp\left(-\sqrt{k + \sqrt{k^2 + \omega^2\tau^2}}\right) \quad (6)$$

(7) where k is the rate constant for the decay (11). For adaptation levels at which pigment bleaching is negligible, the flicker data are adequately fitted with $k = 0$ (2, 6, 8). However, if $k >> \omega\tau$, k will dominate the frequency response of the first stage, which approaches $\exp(-\sqrt{2k})$; that is, the response of that stage becomes constant. Then the overall response of our two-stage model is

$$G(\omega) \sim \exp(-\sqrt{2k})\exp(-\sqrt{\omega\tau_2}) \quad (7)$$

Equation 7 predicts that extensive loss in the photoreceptor stage will (i) reduce the overall sensitivity of that stage and (ii) decrease the slope of the psychophysical flicker curve. These are exactly the two effects reported by Kelly *et al.* (6) under intense adaptation, where their data were fitted by the lower, dashed line in Fig. 1A.

If the adaptation level is not held constant, but varies with the stimulus amplitude (as in traditional flicker studies), a more complicated result is obtained (Fig. 1A). These data, all obtained with 100 percent modulated, sinusoidal waveforms, fit Eq. 1 to about 1000 trolands, but at higher levels, the sensitivity decreases so rapidly that the frequency response function becomes double-valued. Traditional flicker studies have extensively confirmed that the CFF increases to a maximum at about 10^4 trolands and decreases slightly at higher intensities (12). But previous attempts to account for the high-intensity decrease of flicker sensitivity in terms of pigment bleaching have not succeeded. As Kelly *et al.* (6) pointed out, the amplitude threshold at 20,000 trolands should be about twice as great as that predicted by Eq. 1 if flicker sensitivity is proportional to pigment concentration; but the actual threshold is about ten times greater than that prediction.

The reason for this failure, we suggest, is that the effect of pigment bleaching is not a simple sensitivity loss; it is frequency-dependent in the manner of Eq. 6. We hypothesize that the loss constant, k, of the first diffusion stage is proportional to the amount of photopigment bleached in the receptor cells. We have tested this hypothesis by calculating the values of k for the seven lowest points in Fig. 1A. The ratio (R) of these thresholds to the prediction of Eq. 1 varies from 1 to 122. The theoretical expression for these ratios

$$R = \exp\left(-\sqrt{\omega\tau_1} + \sqrt{k + \sqrt{k^2 + \omega^2\tau_1^2}}\right) \quad (8)$$

depends only on the parameters of receptor stage, τ_1 and k. Solving Eq. 8 for k, we obtained the seven values plotted against the adaptation level in Fig. 1B. These values of k are adequately fitted by the smooth curve, which represents the equation

$$k = \frac{95\,I}{I + 25{,}000 \text{ trolands}} \quad (9)$$

where I is the adaptation level. This expression has the correct form to represent the amount of photopigment bleached in the steady state, and its half-bleach value of 25,000 trolands is reasonably close to the value of 20,000 trolands measured by Rushton and Henry (13). We therefore conclude that k is directly proportional to the concentration of bleached photopigment, which implies that the first diffusion process must take place within the photoreceptors.

We can reverse the calculation (Fig. 1B to Fig. 1A) by using the overall expression for our two-stage flicker model,

$$G(\omega) = C \exp\left(-\sqrt{k + \sqrt{k^2 + \omega^2\tau_1^2}} - \sqrt{\omega\tau_2}\right) \quad (10)$$

where $C = 2222$ (troland^{-1}) fits the data of Fig. 1A. Since G is inversely proportional to I at 100 percent modulation, we can combine Eqs. 9 and 10 to obtain the theoretical prediction of ω versus I shown by the solid line in Fig. 1A, which fits the data well. We therefore conclude that the dynamics of intrareceptor diffusion explain the high-intensity maximum in the traditional CFF data, if pigment bleaching is taken into account.

Together with our conclusion that n is a small number, probably 2, this result provides strong support for the hypothesis that flicker thresholds are controlled by first- and second-order retinal units. It also tends to confirm the hypothesis that

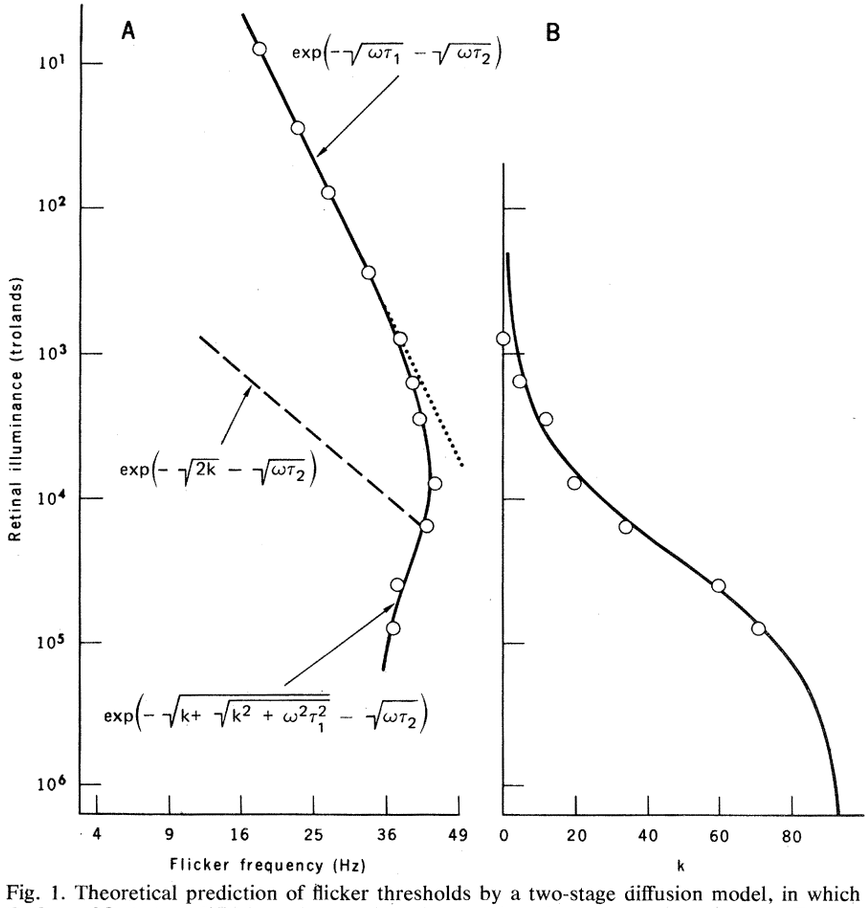

Fig. 1. Theoretical prediction of flicker thresholds by a two-stage diffusion model, in which the loss of first-stage diffusing substance is proportional to photopigment bleached. (A) Dashed line shows the variable-modulation threshold at a constant level of 25,000 trolands obtained by Kelley *et al.* (6). Points show their critical flicker frequencies for 100 percent modulated, sinusoidal waveforms up to 10^5 trolands. [The four most intense levels are beyond the range considered in (6), but all data points were collected in the same experiment.] The solid curve is theoretical. (B) Steady-state pigment bleaching function used to predict the theoretical CFF curve in (A), with a half-bleach level of 25,000 trolands. Points show values of the first-stage diffusion loss term, k, that were calculated from the threshold data in (A).

Eq. 10 does represent the temporal filtering effects of these visual cells on the signals they transmit.

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Cultural Transmission of Enemy Recognition: One Function of Mobbing

Abstract. *There are at least ten suggested hypotheses for the function of mobbing predators by fish, birds, and mammals. Experiments with captive European blackbirds support one of these—the "cultural transmission hypothesis." Perceiving a mobbing conspecific together with a novel, harmless bird induced blackbirds to mob the innocuous object. The mobbing response persisted during subsequent presentations of the novel bird alone, which was more effectively conditioned than an artificial control object. Enemy recognition could be culturally transmitted along a chain of at least six individuals.*

Ascribing a function to a particular trait of an organism implies that that trait enhances fitness. A given trait, however, may have more than one function (*1*), and it is likely that, of the selective forces molding that trait, only one or a few will select for its degree of adaptedness (*2*). Hence, when ascribing functions to a trait, fitness-enhancing properties of selection should be distinguished from those perfecting that trait.

The most serious drawback of the teleonomic approach, that is, the study of adaptations, is that one never knows when the list of functions suggested for a trait is exhausted (*1*). A self-checking procedure of detecting all functions of a given behavior by assessing its costs and benefits and relating these to fitness (*3*) may be a solution to this fundamental problem. The practical difficulties, however, are formidable.

Apart from notable exceptions [for example, (*4*)], the study of adaptation has remained largely guesswork, as evidenced, for example, by the numerous hypotheses regarding the function of mobbing predators. Although causal aspects of mobbing behavior have been frequently studied (*5–8*), little is known about its function or functions. There can be little doubt that mobbing has survival value, because it is potentially dangerous for the mobber (*9*) or its brood (*7*), is time-consuming, and is extremely widespread in vertebrates (*5, 8, 10, 11*). The type or degree of mobbing varies geographically as well as with the threat imposed by the respective predators (*7, 8*) and with the mobber's social organization (*12*). Yet the benefits of mobbing remain a matter of much speculation. The behavior has been suggested to confuse the predator (*13–15*), to discourage its presence through molestation (*5, 16*) or advertise the futility of further hunting (*12, 13*), to sensitize escape responses of other prey (*10, 17*), or to avoid the place where the encounter has previously occurred [(*10, 18*), but see also (*5*)]. The "cultural transmission hypothesis" suggests that perceiving other birds mob an object teaches an individual to fear that object and thus subsequently avoid it, mob it more strongly, or both (*14, 15, 17, 19*). Casual observations of jackdaws tend to support this suggestion (*20*). The benefits for the receiver are obvious, and while those for the transmitter are not so evident, the latter might benefit either immediately through confusing or molesting the predator, or at a future time from a larger number of knowledgeable warners it has engendered. However, these possible teacher benefits would be unnecessary if the receivers were relatives of the transmitter (*21*).

We examined the cultural transmission hypothesis directly by experimenting with captive blackbirds (*Turdus merula* L.) in the nonbreeding season. An "observer" bird was kept singly in an aviary (3 by 3 m) that was separated by a hallway 1 m wide from a second aviary (2 by 3 m) containing another blackbird that served as the "teacher" in all experiments, unless otherwise indicated. In the middle of the hallway a cardboard box with four radially and horizontally oriented chambers was rotated by a thread running to the experimenter behind a blind. By rotating the box 90°, an object in each of two opposite chambers was either exposed to the two birds on both sides of the hallway, or to only one of them. Two of the four chambers contained no object. A stuffed male noisy friarbird (*Philemon corniculatus*), an Australian honeyeater, was chosen as the conditioned object for the observer since it fulfilled the following necessary conditions: it is novel, resembles no genuine predator of blackbirds, yet is of a size similar to some actual predators. In order to effect the teacher's mobbing at the place of the honeyeater, a stuffed little owl (*Athene noctua*) was presented to it at the very moment rotation of the box exposed only the honeyeater to the observer. The stimulus objects occupied the two opposite chambers, 20 cm apart, that were hidden from view between trials. Juxtaposition of the two stimuli ensured, we think, that the observer associated the teacher's spatial orientation (if any) plus mobbing with the honeyeater alone. After presentation, the box was rotated back to its original position, revealing an empty chamber to each bird.

The first stimulus situation of an experiment, at 0930 hours, controlled for the stimulus effect of rotating an empty chamber back and forth. Rotation movements of a stimulus situation were spaced 5 minutes apart. The second situation, at 1000 hours, measured the stimulus effect of the novel honeyeater. The third situation, at 1200 hours, involved

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