In another experiment where only the dilute dye was used, no photodynamic action could be demonstrated when the mixture was rubbed on leaves prior to illumination.

Oster and McLaren (*6*) have shown that the fluorescent dye, acriflavine, mediates a visible light inactivation of intact tobacco mosaic virus. However, to my knowledge, similar photodynamic action has not previously been demonstrated for infectious nucleic acid (*7*).

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Temperature and Charge Transfer in a Receptor Membrane

Abstract. The rate of rise and the amplitude of a mechanically elicited generator potential in a receptor membrane (Pacinian corpuscle) increases markedly with temperature. By contrast, the amplitude of the action potential of the Ranvier node adjacent to the receptor membrane remains practically unchanged over a wide range of temperature. The activation energy of the rate-limiting process in excitation of the receptor membrane is high; it indicates the existence of a high potential energy barrier for charge transfer.

Mechanical stimulation of the nerve ending of Pacinian corpuscles produces transfer of charges through its receptor membrane. The energy requirements for the transfer are markedly influenced by temperature. For example, the strength of a mechanical stimulus necessary to produce a given generator potential at 25°C may be reduced to one-third at 35°C. A temperature change alone, however, elicits no detectable transfer.

The experiments reported here were done on single intact Pacinian corpuscles of the cat and on single nerve endings isolated by dissection from the corpuscles (*1*). The receptor ending was stimulated with mechanical pulses of known strength from a piezoelectric crystal, and the resulting electrical activity was led off from the axon or directly from the nerve ending (*2*). Experiments with intact corpuscles and with denuded nerve endings yielded essentially the same results.

The main effect of a temperature change is to vary the rate of rise and the amplitude of the mechanically elicited generator potential (Figs. 1 and 2). The rate of rise and the amplitude of the generator potential in response to a mechanical stimulus of a given (submaximal) strength increase approximately linearly with temperature with a mean Q_{10} of 2.5 and 2.0, respectively (15° to 35°C). The decay time of the generator potential is not appreciably affected by temperature.

The membrane of the first node of Ranvier adjacent to the receptor membrane, the site at which the nerve impulse arises, behaves quite differently. The amplitude of the action potential of the node, like that of other membranes with regenerative excitability (*3*), remains rather constant over a wide range of temperature (20° to 40°C), and its duration increases with temperature with a Q_{10} of 3 or higher. The electrical threshold for firing of impulses at the node varies inversely with temperature; the initiation of impulses at the node fails completely below 12°C, although the receptor membrane still produces generator potentials.

It may be thought that the observed results reflect mechanical effects due to changes in viscosity or rigidity of the preparation rather than effects on the excitation process of the receptor membrane. This possibility seems, however, unlikely. The visco-elastic properties of our preparation—namely, the denuded nerve ending—are not expected to differ from those of aqueous protein jellies whose temperature coefficients of viscosity and rigidity are as low as those of water. For instance, the Q_{10}'s of viscosity of blood plasma (*4*), egg albumin (*5*), and water are all approximately 1.2 between 20° and 40°C.

The energy of activation of the rate-limiting process in receptor excitation, as calculated from the temperature dependence of the rate of rise of the generator potential, is equivalent to 16,300 cal/mole. This reveals that at least at one stage in the excitation process there is a high potential energy barrier for charge transfer. It has been shown that charge transfer increases as a function of the electrical gradients across the receptor membrane (*6*). Thus a simple and, heretofore, quite plausible model for receptor excitation is that of ions diffusing simply along their gradients through mechanically stretched "pores" of the receptor membrane. This model must now be modified. The high activation energy found in our experiments forces at least one additional element, a high energy barrier, upon this or any other model one may prefer to choose. To surmount the barrier, energy may be supplied directly through heat transfer, or indirectly through a mechanically coupled chemical reaction. In any event, the immediate source that supplies the energy to surmount this barrier cannot be identical with the one that serves to trigger the excitation process: excitation, in our experiments, could only be brought about by mechanical stimulation; heat transfer alone, however steep its gradients, was found to be ineffective in exciting charge transfer.

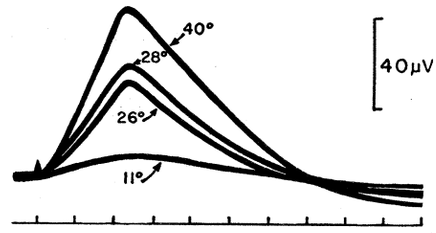

Fig. 1. Effect of temperature on charge transfer through a receptor membrane. The nerve ending is stimulated with equal mechanical pulses from a piezoelectric crystal, and the resulting generator potentials at various temperatures are superimposed. Time calibration, 1 msec.

Receptor excitation is thus thought to operate according to one of the following schemes. (i) The mechanical stimulus causes a directly coupled increase in permeability of the receptor membrane, and ions flow across the membrane along their electrochemical gradients, after overcoming an energy

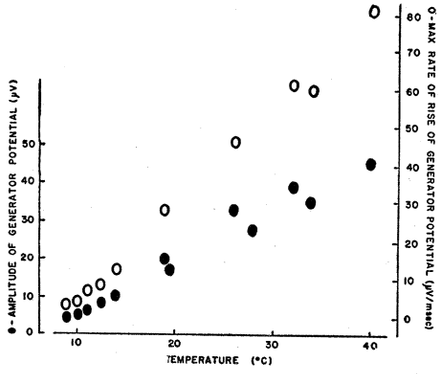

Fig. 2. Amplitude and rate of rise of generator potential as a function of temperature. The nerve ending is stimulated with equal mechanical pulses, and the mean amplitude and rate of rise of the resulting generator potential are determined at various temperatures.

barrier. A nonmechanical source provides the immediate energy to overcome the barrier. (ii) The mechanical stimulus activates a chemical reaction of about 16,300 cal of activation energy per mole, which in turn causes the permeability of the membrane to increase and ions to flow along their gradients (*7*).

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Frequency of Mutations Induced by Radiations in Hexaploid Species of Triticum

Abstract. The frequency of visible mutations induced by x-rays, phosphorus-32, and sulfur-35 was calculated in six hexaploid *Triticum* species. The species with spelted ears and winter habit showed a much lower mutation rate than the free-threshing, spring wheats.

Results from mutation experiments carried out during recent years in several countries in varieties of bread wheat (*Triticum aestivum* L.; $2n = 42$) have borne out Gustafsson's (*1*) statement that in this species "with suitable x-ray doses a mass mutating sets in." MacKey (*2*) has shown that over 30 percent of the mutations isolated in the progenies of irradiated plants of bread wheat result from either the loss or the duplication of the speltoid suppressor gene *Q* situated in the distal end of the long arm of chromosome IX [chromosome 5A according to the new system of nomenclature proposed by Sears (*3*)]. the hexaploid wheats, *T. spelta* L., *T. macha* Dek. et Men., and *T. vavilovi* Jakub., lack the *Q* factor; hence an experiment was undertaken for finding out the frequency and types of mutations induced by radiations in *T. aestivum* L., *T. compactum* Host., *T. sphaerococcum* Perc., *T. spelta* L., *T. macha* Dek. et Men., and *T. vavilovi* Jakub., the six commonly recognized hexaploid species. One stable and homogenous strain was chosen in each species; the *T. aestivum* and *T. macha* varieties used were awned and the rest were awnless. The *T. aestivum*, *T. compactum*, and *T. sphaerococcum* varieties were spring types, while the others had a winter habit.

Table 1. Frequency of mutations observed in the M_2 generation in *Triticum* species.

Species	Total M_2 families (No.)	M_2 families segregating for mutants (No.)	M_2 plants studied (No.)	Mutations in M_2 generation (No.)	Mutation rate (%)	
					Segregating families	Mutations per M_2 family
T. aestivum	338	88	6103	206	26.04	60.95
T. compactum	337	92	6335	253	27.30	75.07
T. sphaerococcum	333	82	6106	219	24.62	65.77
T. spelta	346	27	3390	36	7.80	10.40
T. macha	190	4	1987	13	2.11	6.84
T. vavilovi	147	3	1556	5	2.04	3.40

Dry seeds (5 to 6 percent moisture content) were treated with x-rays (11,000 and 16,000 r), phosphorus-32 (5 μc per seed), and sulfur-35 (5 μc per seed). One hundred seeds were used in each treatment, and the treated seeds were planted in the field along with the respective controls. The main tiller and one or two more tillers of each plant were selfed, and the second generation progenies were raised the following year by sowing the seeds from each plant in individual rows. While no visible mutations occurred in the control material, many such mutations were found in the progenies of treated plants. The population was scored for all phenotypically detectable mutations, and the mutation frequencies observed in the different species were calculated both in terms of the percentage of M_2 families segregating for mutations and the percentage of mutants per M_2 family (Table 1). Since the trend in the frequency and spectrum of mutations induced by the different treatments was similar in all the species, the pooled data are given in Table 1.

Statistical analysis showed that *T. spelta*, *T. macha*, and *T. vavilovi* had a significantly lower mutation frequency than *T. aestivum*, *T. compactum*, and *T. sphaerococcum*. The differences between species within each of these two groups were not significant. It is now known that *T. spelta*, *T. sphaerococcum*, and *T. compactum* are each separated from *T. aestivum* by a single gene: *Q* located on chromosome IX (5A), *S* on XVI (3D), and *C* on XX (2D), respectively (*4*). The number and location of the genes differentiating *T. macha* and *T. vavilovi* from *T. aestivum* have not yet been precisely determined, though there is evidence to suggest that only one or two genes may be involved in these cases also (*5*). A study of the relative frequencies of different types of mutations found during the present study in each species revealed that 31.07, 39.92, and 63.93 percent, respectively, of the mutations isolated in *T. aestivum*, *T. compactum*, and *T. sphaerococcum* could be attributed to the loss or duplication of the appropriate species differentiating locus (that is, *Q*, *C*, or *S*). The mutations found in *T. spelta* related mostly to awning or ear density, while a change to the *T. aestivum* type of ear and internode structure was the only type of mutation recorded in *T. macha* and *T. vavilovi*. Visible mutations can occur in a polyploid only at loci in which phenotypic buffering induced by duplications does not exist (*6*). These results suggest that the number of such loci, while generally few in hexaploid *Triticum* species, is relatively more in the free-threshing spring wheats (*7*).

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