a gene dosage effect. However, the phenotypic distinction between the two genotypes is not always clear.

During the winter of 1954 another sample of the same F2 family had been grown, giving 31 variegated and 25 non-variegated plants, while in another backcross family there were 10 variegated and 20 non-variegated plants. Assuming that the character was always expressed by homozygotes but only by a proportion p of heterozygotes, the method of maximum likelihood gives a value of p of 0·63 with 95% fiducial limits of 0·38 and 0·88. If 37% of heterozygotes were non-variegated, then approximately 10 of the 25 non-variegated F2 plants should have been heterozygotes. 5 of these plants were tested by selfing and 8 by backcrossing to proven homozygous non-variegated plants. The resulting families were grown in 1955 and 4 of the 13 plants tested were shown to have been heterozygotes because they segregated the expected proportions of variegated progeny. The only obvious seasonal difference between 1954 and 1955 to which the difference could be attributed was the colder temperature in May, the mean daily maxima and minima being 2°C lower in 1955.

Another sample of this F2 family was sown in November 1956. During the ensuing summer only 5 out of 33 plants showed the variegation and these only to a limited extent. The ratio variegated: non-variegated is approximately 1:3. It seems probable that the variegated plants were homozygotes and that the character was not expressed in the heterozygotes. During summer variegation was apparently recessive. There was a reversal of dominance.

These experiments show that variegation in 'Ornamental kale' is not only temperature-sensitive but, in addition, the frequency with which it is expressed differs in homozygotes and heterozygotes. The interaction of these two features may give rise to the phenomenon of reversal of dominance at different temperatures.

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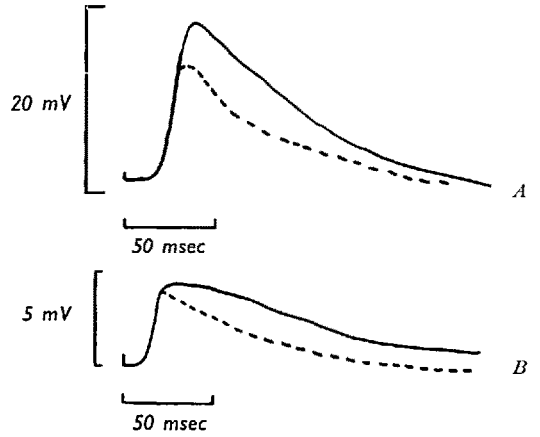
Résumé

La transmission de l'albinisme panaché de *Brassica oleracea* L. ne dépend que d'un seul facteur. Chez les hétérozygotes ce caractère apparaît à basse température (7°C), mais pas à haute température (21°C). Chez les homozygotes le caractère se manifeste probablement toujours, tout en restant peu prononcé à des températures plus élevées. Ainsi il y a une inversion du caractère dominant avec changement de température.

The Effects of γ-Aminobutyric Acid and Picrotoxin on the Junctional Potential and the Contraction of Crayfish Muscle[1]

Stimulation of the inhibitory neuron of the opener muscle of the crayfish claw normally reduces the contraction. But the inhibition resulting from this stimulation is greatly reduced in the presence of picrotoxin[2]. It is also known that γ-aminobutyric acid (GAB) inhibits the contraction of crayfish muscle[3]. It therefore seems possible that GAB mimics the action of the inhibitory transmitter on the muscle. This idea would be supported if the action of GAB, like that of the inhibitory transmitter, were blocked by picrotoxin (the blocking of GAB's action by picrotoxin has been demonstrated on the Crustacean heart[4]).



A and *B* Junctional potentials recorded with an intracellular electrode before (——) and after (– – –) the perfusion of the muscle with $2{\cdot}4 \times 10^{-4}$ *M* GAB

Our results [5] show that when GAB was introduced into the perfusion fluid, the response by the muscle to stimulation of the motor neuron at 60/s was slightly reduced by concentrations of GAB as low as $3{\cdot}7 \times 10^{-6}$ *M* (0·38 μg/ml); invariably, contractions were completely eliminated by 10^{-3} *M* GAB. The GAB effect was readily reversed by washing the claw free of the amino acid or by perfusing with a solution containing 10^{-4} *M* picrotoxin along with the GAB. From this result it is clear that picrotoxin does block the inhibitory action of GAB on crayfish muscle.

This evidence by itself does not prove that GAB is the inhibitory transmitter, because contractions were also blocked completely by other amino acids, 10^{-2} *M* β-alanine for example. And the action of β-alanine was also blocked by picrotoxin[6].

We have studied the effects of GAB and of picrotoxin on the membrane of the closer muscle by the use of intracellular electrodes. After $2{\cdot}4 \times 10^{-4}$ *M* GAB was perfused around the muscle, there was little change in the resting potential of the muscle fiber. But when the 'fast' motor neuron was stimulated while GAB was present there was usually a distinct decrease in the height of the junctional potential (Figure *A*). And GAB also produced a pronounced increase in the rate of decay of the junctional potential, which suggests that GAB reduces the resistance

[1] The study was supported by Grants B-1089 and B-31 from the National Institute of Neurological Diseases and Blindness, United States Public Health Service. Some of this work was done at the Department of Zoology, Cornell University. We are grateful to Prof. S. C. WANG, Columbia University, for generous assistance.

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[3] J. A. BROCKMAN, JR. and S. L. BURSON, JR., Proc. Soc. exp. Biol. N. Y. *94*, 450 (1957).

[4] E. FLOREY, Naturwissenschaften *44*, 424 (1957).

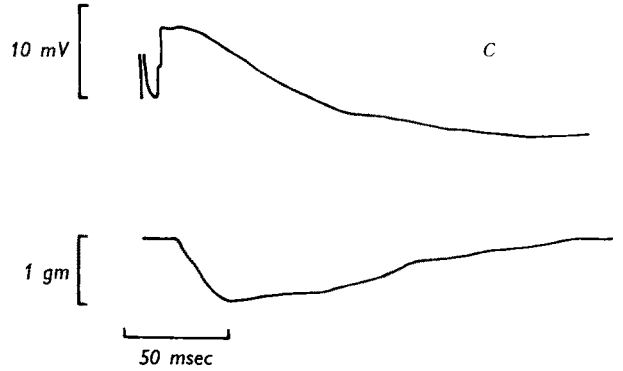
[5] The crayfish studied were *Cambarus clarkii*, *C. virilus*, and *Orconectes immunis*.

[6] A. BAZEMORE, K. A. C. ELLIOTT, and E. FLOREY, Nature *178*, 1052 (1956), made the important discovery that GAB and β-alanine block impulse generation in the Crustacean stretch receptor. Apparently GAB mimics the action of the inhibitory transmitter on the receptor, C. EDWARDS and S. W. KUFFLER, Fed. Proc. *16*, 34 (1957).

of the muscle fiber. This observation is important, for it will be recalled that a similar decrease in the resistance of the muscle membrane has been shown in the crab to result from the stimulation of the inhibitory neuron[7]. All of the effects of GAB on the junctional potential were eliminated by washing the muscle free of the amino acid or by perfusing with a solution containing 10^{-3} *M* picrotoxin and $2{\cdot}4 \times 10^{-4}$ *M* GAB.

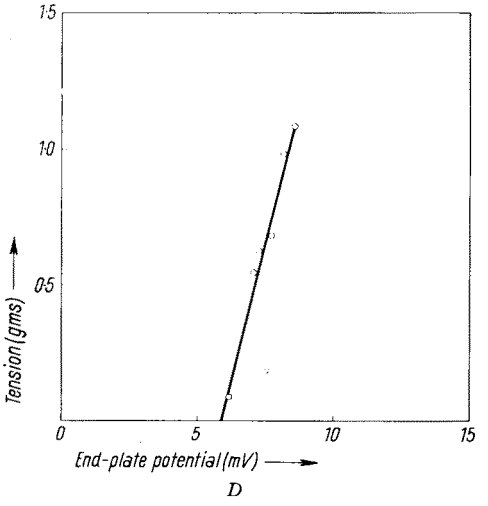
In some instances, however, there was merely a slight change in the height of the junctional potential after GAB was added (Figure *B*), even though the contraction was considerably reduced. This result recalled the idea that the inhibitory transmitter may act at some point between the junctional potential and the contractile machinery, and that changes in the membrane may be merely incidental side effects[8]. The problem then is whether a small change in the height of the junctional potential could possibly lead to a large change in the tension produced by the muscle.

This question was explored by stimulating the motor neuron with pairs of closely spaced stimuli while measuring the junctional potential generated in one fiber and the tension exerted by the entire muscle. Pairs of stimuli separated by more than 2·5 ms produced a double junctional potential (Figure *C*). As the interval between the first and the second stimuli was increased, the peak depolarization of the muscle membrane was also increased, until a maximum was reached when the interval between stimuli was about 5·3 ms. So by this technique the height of the junctional potential could be varied, and the relation between the height of the junctional potential and the tension produced by the muscle could be determined (the assumption underlying this method is that the behavior of the single junction at which the electrode is lodged is typical of junctions throughout the muscle; recordings with extracellular electrodes appear to support the assumption).



C The double junctional or end plate potential produced by a pair of closely spaced stimuli applied to the 'fast' motor neuron (upper trace) and the tension produced by the closer muscle (lower trace; an increased tension gives a downward deflection)

The results of a typical experiment are plotted as Figure *D*. Apparently a small change in the peak depolarization of the junctional potential produced a large change in the tension exerted by the muscle. So it seems that even the small decrease in the junctional potential sometimes found after exposure to GAB may be sufficient to account for a pronounced change in the contraction of the muscle. From our results, it appears probable that GAB inhibits the contraction of the muscle by producing a decrease in the height of the junctional potential.



D

D The relation between the peak depolarization in the junctional potential and the maximum tension produced by the closer muscle

We conclude that GAB mimics the effects of the inhibitory transmitter on crayfish muscle because this amino acid inhibits the contraction of the muscle and acts on the junctional potential like the inhibitory transmitter, and also because its action is blocked by picrotoxin.

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Résumé

L'acide γ-amino-butyrique et la β-alanine reproduisent l'action du transmetteur inhibiteur du système neuromusculaire de l'écrevisse. Ces deux acides aminés diminuent la contraction musculaire. L'acide γ-amino-butyrique réduit la différence de potentiel et augmente sa décomposition. L'action de ces acides aminés est bloquée par la picrotoxine comme l'est celle du transmetteur inhibiteur.

Hypothalamic Nerve Fibres in the pars tuberalis and Pia-arachnoid Tissue of the Cat and their Degeneration Pattern after a Lesion in the Hypothalamus

Much experimental data of the last ten years have shown that the function of the anterior pituitary gland is influenced by the hypothalamus. However, the structures and the precise mechanisms of the «connecting link» between the hypothalamus and the adenohypophysis remains still unsettled. It is widely accepted that the secretion of the anterior pituitary is regulated by humoral substances from nerve endings in the median eminence, which are transmitted by hypophyseal portal vessels to the cells of the pars distalis, but unequivocal data which support or establish this thesis as the sole or major mechanism of regulation are absent (SAYERS, REDGATE and ROYCE[1]). Therefore, also other structures and mecha-

[7] P. FATT and B. KATZ, J. Physiol. *121*, 374 (1953).

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