and one at room temperature. Results, shown in Table 1, indicate that cold is required for GA-triggering of germination over the range studied. The time course of germination was in all cases similar to that observed in previous experiments, involving 1000 parts per million of GA in continuous contact with seeds. The percentages of germination were approximately proportional to concentration of GA over the range studied.

The above-described experiment shows that the lag in germination after GA treatment is not due to difficulty in penetration of the seed testa by growth promoter. Seeds which received only a 12- or 24-hour application of GA, followed by thorough washing, started and stopped germination after the same amount of cold storage as those in continuous contact with the promoter. Thus it seems that in *Melampyrum lineare* all germination is associated with a final cold process, whether germination is initiated (i) with GA, or (ii) in its absence (for a percentage of the seeds) by preconditioning on the plant or forest floor, or at room temperature in the laboratory. Chilling before initiation cannot replace this post-activation cold process. A search of the literature indicates that such a germination pattern has not been noted previously.

In the field up to about 40 percent of the seeds present in the forest floor germinate by late spring. The remainder lie viable but dormant and are apparently subject to heavy depradation by the biota of the forest floor. Nevertheless, it seems likely that the field germination counts in late spring include some seeds from more than the preceding year's crop. In the laboratory, dormancy in the ungerminated seeds can be broken by GA, although a natural agency has not been found. Preliminary experiments suggest that a light process is not involved. Possible natural activators include short periods of seed-exposure to desiccation or to elevated temperatures, or a relatively long exposure to a sequence of temperatures corresponding to the seasonal changes of temperature in the field. Also, germination promoters might be present in host-root exudates or their natural decomposition products. These possibilities are being investigated (*9*).

E. J. C. CURTIS
J. E. CANTLON
Department of Botany and Plant Pathology, Michigan State University, East Lansing

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9. Supported by grant G-13991 from the National Science Foundation.

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Temperature Dependence of Wing Abnormality in Tribolium confusum

Abstract. *When* Tribolium confusum *pupae were exposed continuously to temperatures from 10°C to 40°C, the adults which finally emerged exhibited wing abnormalities typical of those obtained with x-irradiation. The data indicate the implication of two processes. Two mathematical models are suggested to account for this phenomenon.*

Pupae of *Tribolium confusum* Duval were harvested within 30 hours of formation and incubated in the dark at temperatures from 10°C to 40°C. The particular wing abnormalities were indistinguishable from kinds seen when pupae were irradiated with a variety of accelerated atomic nuclei (*1*) or x-rays (*2*). These abnormalities are uniform but exhibit varying degrees of intensity; the membranous wing is displaced with irregular warping of the elytra. Continuous incubation at 10°C prevented eclosion for periods up to at least one month, while the higher temperature entirely inactivated the system and resulted in the death of all of the organisms within 7 days. Minimal effects were observed between 30°C and 35°C.

Table 1. Abnormalities (percentage) as a function of temperature.

(deg. C)	X	
	Exptl.	Calc.
20	27.1	26.9
25	5.2	5.88
30	1.6	1.46
35	1.6	1.60
38	5.3	4.41
39	5.8	6.47

At intermediate temperatures adults emerged with varying wing abnormalities. When the percentages of abnormalities were plotted against temperature, the resulting curve (Fig. 1) had two branches. It appears that two processes are implicated. The experimental data appear to be best fit by the function

$$X = 1.19 \cdot 10^{4}\, e^{-0.305t} + 1.32 \cdot 10^{-6}\, e^{+0.394t}$$

as shown in Table 1, where t is the temperature in degrees Celsius. Thus we believe that the wing abnormalities are caused by two additive independent processes with each separate process causing a change in the relative effect proportional to the change in temperature. In differential form, the equation given may be written

$$X = X_1 + X_2$$

$$\frac{dX_1}{dt} = -0.305X_1$$

and

$$\frac{dX_2}{dt} = +0.394X_2$$

where $X_1 = 100$ for $t = 15.16$ and $X_2 = 100$ for $t = 46.1$

In a second, more elaborate, model one may suppose that X is a function of two independent quantities X_1 and X_2, where X_1 is the percentage of abnormalities caused by the first process and X_2 is the percentage of abnormalities caused by the second process. The percentage of abnormalities caused then by the second, but not by the first process, is given by $X_2 \cdot (100 - X_1)/100$. Therefore

$$X = X_1 + X_2 \cdot [(100 - X_1)/100]$$

and

$$X = X_1 + X_2 - (X_1X_2/100)$$

The processes responsible for production of abnormality in the second model may also be considered to occur as follows, where two sets of precursors may be in dynamic equilibrium with two distinct end products:

$$nA + B \underset{}{\overset{k_1}{\leftrightarrows}} A_nB$$

$$k_1 = (A_nB)/(A)^n(B)$$

$$(A_nB) = k_1(A)^n(B)$$

and

$$(A_nB) = (A)^n(B) \cdot ae^{(-b/T)}$$

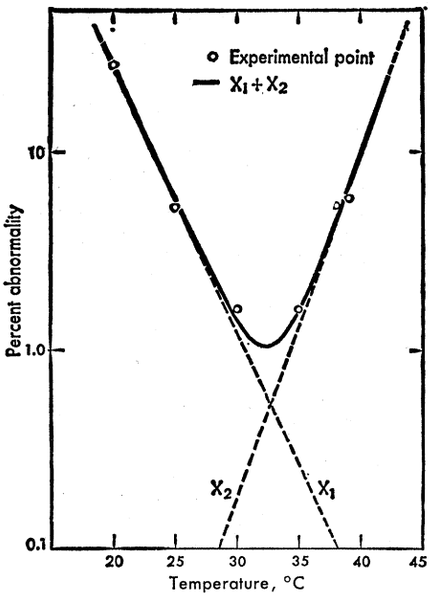

Fig. 1. Abnormalities as a function of temperature in degrees Celsius.

where T is the absolute temperature and α and b are constants.

Also,

$$mC + D \overset{k_2}{\rightleftarrows} C_mD$$

$$k_2 = (C_mD)/(C)^m(D)$$

and

$$(D) = [(C_mD)/(C)^m] \cdot \gamma e^{(+d/T)}$$

where γ and d are constants. Therefore

$$(A_nB) = ae^{(-b/T)} \qquad (1)$$

and

$$(D) = ce^{(+d/T)} \qquad (2)$$

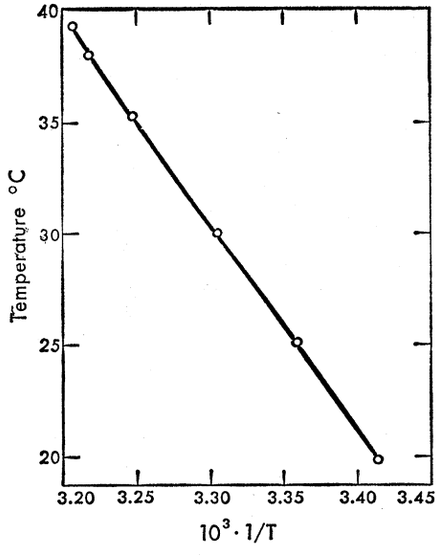

Fig. 2. Correspondence between t (in degrees Celsius) and $1/T$.

where a and c can be considered to be constant if $(A_nB) \ll (B)$ and (A), and if $(D) \ll (C_mD)$ and (C) over the temperature range considered (where k_1 is very small and k_2 is very large). Thus, X_1, the first process, is proportional to Eq. 1, and the second process, X_2, is proportional to Eq. 2. From this hypothesis it can be seen that abnormal development may be caused by the presence of either an unusual quantity of some product or by an imbalance in the presence of one of the precursors of a separate set of reactions. From Fig. 2 it is evident that $1/T$ is approximately a linear function of t. Furthermore, from Fig. 1 it can be seen that $X_1X_2/100$ is much smaller than X. Therefore our experimental data fit both models equally well. The exponential constants of the second model were calculated to be $b = 2.5 \times 10^4$ and $d = 3.2 \times 10^4$. From Fig. 1 it is evident that the values of

$$Q_{10} = (K_{T+10})/K_T$$

for the two separate reactions are approximately 20 and 50, thus being 10 to 20 times greater than one usually expects for simple biochemical reactions (*3*). This consideration suggests that we discard the hypothesis previously indicated where $(A_nB) \ll (A)$ and (B), and $(D) \ll (C_mD)$ and (C). Now by putting

$$n(A_nB) + (A) = (A)_t$$

and

$$(A_nB) + (B) = (B)_t$$

where $(A)_t$ and $(B)_t$ are constant, we arrive at

$$(A_nB) = k_1 [(A)_t - n(A_nB)]^n \cdot [(B)_t - (A_nB)]$$

for the first reaction, and, with similar notation,

$$(D) = \frac{1}{k_2} \frac{(D)_t - (D)}{[(C)_t - m(D)_t + m(D)]^m}$$

for the second reaction. Certain values of the constants $(A)_t$, $(B)_t$, $(C)_t$, $(D)_t$, m, and n may explain the high slopes of the two branches of our curve, even with values of k_1, k_2, dk_1/dT, and dk_2/dT that are common in biochemical reactions. The degrees of freedom of this model, however, are too many to calculate any parameter (*4*).

JOHN V. SLATER, A. RESCIGNO
NABIL M. AMER, C. A. TOBIAS
Space Sciences Laboratory and
Donner Laboratory of Medical Physics, University of California, Berkeley 4

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Cytochrome in Thiobacillus thiooxidans

Abstract. *Cytochrome is present in the autotrophic sulfur bacterium,* Thiobacillus thiooxidans. *As mediator in the final electron transfer to oxygen, the cytochrome participates in the oxidation of sulfur compounds by extracts of the organism.*

During an extensive study of the Thiobacillus group, Szczepkowski and Skarzynski (*1*) failed to observe cytochrome in *Thiobacillus thiooxidans.* The absence of this important constituent usually present in aerobic cells was explained (*2*) by suggesting that the bulk of energy from sulfur oxidation was derived through substrate phosphorylation by way of an adenosine phosphosulfate complex, whereby oxidative phosphorylation would be made superfluous. Nevertheless, cytochromes are readily detectable in other members of the genus (*3*), which would imply that the metabolic activities of *T. thiooxidans* differ fundamentally from those of other thiobacilli. Because this seemed unlikely, a reexamination of the situation was undertaken.

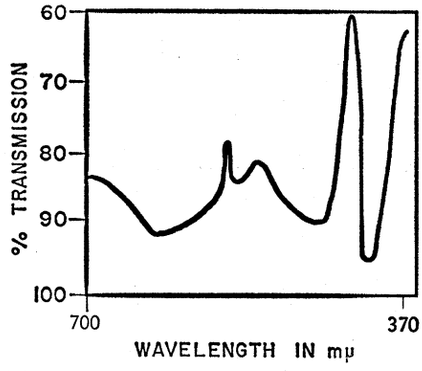

Fig. 1. The reduction of cytochrome from *T. thiooxidans* by sodium dithionite. Cuvette contents: 2 ml of extract (13 mg protein): 1 ml of 0.1*M* phosphate buffer, *p*H 6.9; 0.2 ml of $Na_2S_2O_4$ (20 μM); distilled water to 4 ml.