which grow under the influence of its hormone is known not to affect receptivity[4]. Accordingly, the effect on injecting mature corpora allata into pupae just before eclosion has been tested.

Corpora allata, together with the associated complex of fused corpus cardiacum and hypocerebral ganglion, were dissected from 6–12 day old virgin females and briefly stored in *Drosophila* Ringer's solution[5]. Single glands, together with a little Ringer's solution, were injected into the abdomens of pupae, 17–19 h before eclosion, through a glass needle using the technique of Ephrussi and Beadle[5]. Control pupae received a small piece of the aorta dissected from the same donor flies. Mortality was about 50 per cent, but with two exceptions all flies which survived day 0 went on to mate and lay fertile eggs.

Injected flies were tested for receptivity on the morning of day 1. Each was placed with a single male in a 'Perspex' observation cell[6] 1 in. in diameter. Males court females of all ages with equal persistence and will continue courting unreceptive virgin females for several hours. In practice, 30 min without acceptance is a suitable criterion because more than 90 per cent of receptive females accept in less than 10 min. Forced matings are not uncommon and the female can be seen struggling to push the male off, but unless she succeeded in doing so within a few minutes she was recorded as receptive.

Table 1. RECEPTIVITY OF *D. melanogaster* FEMALES MEASURED ON DAY 1

	Receptive on day 1	Unreceptive on day 1	Total
Corpus allatum injected	20	0*	20
Control, aorta injected	7	16†	23
Control, not injected	7	18	25

The difference between the two injected groups is highly significant (χ^2 with Yates's correction = 19·23, $P < 0·001$).

* Two flies injected with allata were unreceptive on day 1 but died on day 2.

† These flies were tested for the normal appearance of receptivity on days 2 or 3, and all were receptive.

The results are given in Table 1 and strongly suggest that the presence of a mature corpus allatum in young flies induces the precocious onset of receptivity. This could result from the direct action of its hormone on some receptivity mechanism in the central nervous system. Alternatively, as Highnam[7] points out, the hormone may facilitate the release of stored neurosecretory products and these may affect behaviour. Preliminary experiments indicate that, given at this stage, the hormone requires some time to take effect and that females with implanted glands are not receptive on day 0.

The assistance of Miss M. C. Hill is gratefully acknowledged. This work was supported by a grant from the Science Research Council.

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Impairment of Oxidative Phosphorylation by D-threo- and L-threo-Chloramphenicol

WE reported earlier that high concentrations of chloramphenicol impair certain energy-linked functions of mitochondria[1,2]. The experiments were carried out with the D-threo isomer (the antibiotic) which effectively blocks protein synthesis in bacteria[3]. Of principal interest is the suppression of respiration and oxidative phosphorylation. If these effects are produced *in vivo*, it cannot be assumed that responses produced in tissue by D-threo-chloramphenicol are necessarily related to inhibition of protein synthesis. This is not to deny that protein synthesis can be blocked; but it becomes essential that the investigator establishes that impairment of phosphorylation is not also, or even primarily, involved whenever concentrations of the order of 0·5 mg/ml. or greater are used.

One way of avoiding this difficulty is to use L-threo-chloramphenicol as a control treatment. The L-threo isomer is relatively ineffective in inhibiting protein synthesis[3]. Ellis[4] has reported that both the D-threo and L-threo isomers are effective in inhibition of ion accumulation, and he suggests that there is not necessarily any connexion between the uptake of ions and the synthesis of protein. Recently, Billet *et al.*[5] have used the L-threo isomer as a control to establish that the effects of the D-threo isomer on the development of the chick embryo result from inhibition of protein synthesis. The D-isomer was effective in arresting development while the L-isomer was not. There is thus support for the postulate that the antibiotic impairs development through inhibition of protein synthesis.

Table 1. IMPAIRMENT OF OXIDATION AND PHOSPHORYLATION BY D-THREO AND L-THREO-CHLORAMPHENICOL

Chloramphenicol	$Qo_4(N)$	P/O
none (0·1 ml. ethanol/vessel)	2443	1·70
D-threo (1 mg/ml.)	1102	0·98
L-threo (1 mg/ml.)	717	0·46

R. J. Ellis and W. Landauer have questioned (personal communications) whether L-threo-chloramphenicol will also suppress oxidative phosphorylation. In order to answer this point we carried out experiments with mitochondria from 3 day etiolated corn shoots as previously described except that 40 μmoles each of pyruvate and malate were used as substrate instead of pyruvate and succinate. The compounds were dissolved in absolute ethanol, with equivalent ethanol added to the control. The ethanol tends to have an inhibitory effect on oxidative phosphorylation. A typical result is given in Table 1. This shows that the L-threo isomer effectively suppresses oxidation and phosphorylation; indeed, it is consistently more effective than the D-threo isomer. This result can be correlated with the slightly greater inhibitory effect of root growth with the L-threo isomer[6].

Thus when only the D-threo isomer is effective on tissue (for example, Billet *et al.*[5]) there is reason to believe that protein synthesis is the process being inhibited. Where both the D and L compounds are effective (for example, Ellis[4] and Ronnicke[6]), the impairment of phosphorylation is probably involved. The mechanism responsible for the impairment of phosphorylation is still unknown. As reported previously[2], the antibiotic does not act like a true uncoupler; however, it does exhibit certain similarities to the respiration-suppressing phase of uncoupler action. More recent work has shown that chloramphenicol increases the rate and extent of mitochondrial swelling[7], which suggests that the compound acts by damaging the mitochondrial membrane rather than by specifically interfering with a phosphorylated intermediate.

We thank Dr. R. J. Ellis and Parke Davis and Co. for supplies of D-threo- and L-threo-chloramphenicol. This work was supported by the U.S. Atomic Energy Commission.

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