ing action potentials might reflect 2 classes of cells. The 1st of these explanations was not supported by the resting potentials, which in fact was slightly lower in the cells generating action potentials than in the remaining cells.

All-or-none action potentials were not elicited after superfusion with Na-free solution for several min. The main component of the inward action current in normal saline is thus carried by Na^+. However, even in Na-free solution a significant regenerative depolarisation with a maximum rate of rise less than 3 V/sec occurred at the termination of a hyperpolarising current pulse (figure 1, C). Small graded responses were superimposed on the electrotonic potential when a depolarising current pulse was applied (figure 1, D). It is reasonable to consider that an increase of the membrane permeability to Ca^{2+} is responsible for the regenerative responses in Na-free solution. When the Ca^{2+} concentration was increased 10-fold to 24 mM, the maximum rate of rise of the anodal break response increased from 1.7 ± 0.6 V/sec ($n = 9$) to 5.7 ± 2.1 V/sec ($n = 5$). Figure 2, A shows the fast rising off-response in Na-free saline with 24 mM Ca^{2+}. When the membrane potential was hyperpolarised to a more negative level than -70 mV by DC current, an all-or-none action potential could be evoked by an outward current pulse (figure 2, B). The action potential was resistant to 2×10^{-6} g/ml tetrodotoxin.

In the cells which generate Ca spikes, Sr^{2+} and Ba^{2+} can replace Ca^{2+} as inward current carrier[12]. This is also the case in the anterior pituitary cells. When 24 mM Ca^{2+} in Na-free solution was exchanged with isomolar Sr^{2+}, action potentials occurred in response to a depolarising current pulse as seen in figure 2, C. The maximum rate of rise attained 11.1 V/sec in this cell. In Na-free solution with 24 mM Ba^{2+}, the action potential was markedly prolonged, and it was often seen to overshoot (figure 2, D). This prolonged action potential is probably due to the suppressing effect of Ba^{2+} on the delayed rectification mechanism.

The recorded cells were not identified in the present study. However, the number of parenchymal glandular cells greatly exceeds any other cells within the gland. It is therefore likely that the reported electrical activity was recorded mainly from glandular cells. The capacity of pituitary tumor cells to generate electrically induced Na- and Ca-dependent action potentials thus seems to be a physiological property retained from normal pituitary glandular cells. Our data will be presented in more detail elsewhere[14].

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The effects of juvenile hormone analogues on the eggs of *Pieris brassicae* L.[1]

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Summary. Mortality was produced in the eggs of *P. brassicae* after treatment with 3 juvenile hormone analogues. The mortality was greatest in the early stages of embryonic development.

Embryonic development can be divided into a period of embryogenesis followed by a period of larval differentiation. These 2 periods are separated by the blastokinesis of the embryo. The effect of juvenile hormone analogues (JHAs) on post-oviposition egg development show that they are most effective in blocking embryonic development when they are applied during embryogenesis[2]. This disruption of embryonic development has been found in a wide variety of insects, e.g.: *Lygaeus kalmii* (Hemiptera)[3], *Lepismodes inquilunus* (Thysanura)[4], *Schistocerca gregaria* (Orthoptera)[5], *Hyalophora cecropia* (Lepidoptera)[6], and *Epilachna varivestis* and *Lasioderma serricorne* (Coleoptera)[7]. In the present experiment, the eggs of *P. brassicae* have been tested for mortality after applying JHAs to the eggs at different times between oviposition and hatching. The analogues used were Ro-84314[8], Ro-69550[9] and Law's Mimic[10].

Materials and methods. Cabbage plants with newly laid eggs on them were transferred from a greenhouse to a constant environment of 25 °C, 70% relative humidity and 16 h daylength. The eggs were treated topically with a JHA in acetone solution at the rate of 10 µg JHA/1 µl acetone/20 eggs. They were treated at the time of oviposition or at 1, 2 or 3 days later. Untreated eggs normally hatched at 4 days after oviposition. Control eggs were treated with acetone at the same time-intervals and with the same volume of acetone. The number of 1st instar larvae that hatched were counted and counts were continued daily until after ecdysis to the 4th instar.

Results. Although counts were made of the numbers of surviving larvae from hatching until the 4th instar, deaths were only found to occur at the time of hatching or at a larval ecdysis. The table shows the number of larvae surviving each of these critical periods. It can be seen that where the JHA produced a large mortality, most of this occurred before hatching to the 1st instar, but there was often another large mortality at the ecdysis to the 2nd instar. Most larvae that survived to the 2nd instar then survived to become adults. The most effective time for JHA treatment is immediately after oviposition, when there is nearly a 100% mortality for all analogues. 1 day after oviposition is also very effective in the analogues Ro-84314 and Law's Mimic but not Ro-69550. If the treatment is postponed until 2 or 3 days after oviposition, mortality is considerably reduced but this is still higher than the controls.

Discussion. The results show that the JHAs produce mortality in the eggs of *P. brassicae* throughout embryonic development, but mortality is greatest when the JHA is applied

Effect of juvenile hormone analogues on the eggs

Treatment	Time of JHA treatment in days after oviposition	No. of eggs treated	Larvae surviving to 1st instar No.	%	Larvae surviving to 2nd instar No.	%	Larvae surviving to 3rd instar No.	%	Larvae surviving to 4th instar No.	%
Ro-84314	0	169	19	11.2	0	0	0	0	0	0
	1	238	66	27.7	0	0	0	0	0	0
	2	197	170	86.3	118	59.9	115	58.4	105	53.3
	3	162	145	89.5	119	73.5	111	68.5	107	66.0
Ro-69550	0	196	0	0	0	0	0	0	0	0
	1	143	120	83.9	102	71.3	90	62.9	86	60.1
	2	220	199	90.5	142	64.6	122	55.4	118	53.6
	3	216	174	80.6	141	65.3	124	57.4	123	56.9
Law's Mimic	0	205	40	19.5	5	2.4	5	2.4	5	2.4
	1	312	88	28.2	60	19.2	47	15.1	43	13.8
	2	249	161	64.7	156	62.6	153	61.4	139	55.8
	3	180	145	80.6	139	72.2	139	77.2	133	73.9
Acetone	0	225	203	90.2	199	88.4	195	86.7	194	86.2
	1	206	199	96.6	192	93.2	183	88.8	179	86.9
	2	202	196	97.0	187	92.6	187	92.6	182	90.0
	3	152	146	96.1	133	87.5	130	85.5	126	82.9

at oviposition or 1 day later. This is consistent with previous work on other insects and suggests that the 1st day of embryonic development in *P. brassicae* is the highly susceptible period of embryogenesis. This is not consistent with previous work on *P. brassicae* in which no mortality effect on the eggs could be found[11]. The small mortality of the acetone controls may be due to an alteration in the structure of the chorion of the eggs which allows excessive water to be lost from them.

Although the majority of the mortality occurred in the eggs, there was also some mortality in the 1st and subsequent ecdyses. This is most likely explained as a delayed mortality effect due to an upset in the programming of the corpora allata, rather than a result of the persistence of hormone residues[3].

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Activity alterations of metabolic enzymes in the anterior pituitary of female rats during acute and chronic starvation, as well as after refeeding

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Summary. The activity of glycolysis and hexose monophosphate shunt decreases while the activity of some oxydative enzymes and acid phosphatase increases in the anterior pituitary of adult female rats during starvation. The alterations depend on the severity of starvation. The polypeptide hormone production also decreases. A close relationship exists between the metabolic activity of the gland and its endocrine function.

Restriction of food intake involves decreased pituitary secretion and weight reduction of the peripheral endocrine organs[3-5]. A substantial correlation between the decrease in prolactin, growth hormone, FSH, LH and TSH pituitary and blood concentration and/or content and the severity of starvation has been verified in our previous paper[5]. Similar results have been reported by other authors[4,6-11]. The aim of recent experiments was to clarify whether the various starvation conditions did affect anterior pituitary metabolic and lysosomal activity. An answer was sought to: 1. whether starvation-induced biochemical changes in the anterior pituitary were similar to those in other tissues, and 2. whether there was a correlation between pituitary metabolic activity and hormone secretion in starved animals.

Material and methods. The experiments have been carried out on adult CFY female rats. The animals were kept in individual cages. The daily intake of the granulated food was measured regularly. To prevent coprohagia a wide-meshed wire net was applied to the bottom of cages. The animals were grouped as follows: control group, fed ad libitum; acutely starved group, fed ad libitum to day 16, thereafter 6 days total food deprivation; chronically starved group, 6 days total food deprivation, thereafter ¼ of the original food intake; the so-called refed group, 6 days total food deprivation, then 10 days ¼ of the original food intake, thereafter ad libitum feeding. At the end of the experimental period, the animals were decapitated in light ether narcosis. The anterior pituitary has been homoge-