actograms (level of significance 1%). That means: the events determining the transition from activity to rest and from rest to activity have no influence on one another. The frequency histograms of A and R could be fitted in 66% and 90% of all actograms to exponential functions. Therefore the conclusion is permitted that in these cases the transition probabilities from activity to rest and vice versa are independent of the duration of activity respectively rest. The transition probabilities $p_{A\to R}$ and $p_{R\to A}$ are negatively correlated with each other ($r=-0.75$, $a<0.1\%$). - The transition probabilities are not temperature-compensated. An increase of temperature reduces the probability for the transition from activity to rest and enlarges the probability for the transition from rest to activity (2 experiments, each with 10 animals; 1st experiment: 11 days 23.5 °C → 11 days 28 °C; 2nd experiment: 7 days 28.2 °C → 7 days 33 °C → 7 days 28.5 °C). Whereas the endogenous circadian periodicity of intact cockroaches is hardly influenced by temperature cycles, in operated animals 12/12 h as well as 15/15 or 24/24 h cycles induce corresponding locomotion rhythms (12/12 h cycles: 23/29 °C, 19/29 °C, 25/30 °C; 15/15 h cycles: 25/30 °C; 24/24 h cycles: 25/30 °C; each experiment with at least 7 bilobectomized animals, figure 3) (see the partly contrary results in Gryllus[9]). In constant conditions, the induced rhythms seem to cease immediately (figure 3).

Discussion. The activity patterns of bilobectomized cockroaches can largely be described by the assumption of simple stochastic regularities. Possibly other higher animals also are endowed with at least 2 nervous mechanisms by which spontaneous activity and rest can be controlled. 1 mechanism is the circadian pacemaker, the other probably consists of 2 random generators which produce endogenous signals starting and closing activity bursts and rest pauses. Normally the random mechanism seems to be superimposed by the circadian clock. The importance of the random mechanism in intact animals is unknown. Perhaps it controls the subtle distribution of activity in rhythmically structured locomotion patterns. In animals with degenerated circadian clock (for instance *Uca* species[6,7] or cavernicolous animals[10,11]) the activity sequence is predominantly or exclusively structured by an endogenous random mechanism.

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Effects of an insect growth regulator, ZR-2646 on egg fertility in the fleshfly, *Sarcophaga bullata*[1]

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Summary. Topical application of an insect growth regulator, ZR-2646 on adult females of *Sarcophaga bullata* affects the viability of the eggs. The compound is most effective if applied at the time of ovulation.

Though the presence of optimal titers of juvenile hormone (JH) is essential for the normal reproductive performance of insects, the same hormone or compound with JH activity can act as an ovicide if applied at a certain time during the ovarian cycle[3-5]. Usually this is the period of ovulation when the endogenous JH titer is at its low. On these grounds several insect growth regulators (IGR) with JH activity have been synthesized and their potential use against insect pests tested[6]. I report here the ovicidal effects of one such IGR, ZR-2646 on the fleshfly, *Sarcophaga bullata.*

Material and methods. The fleshfly, *Sarcophaga bullata* was reared in the laboratory under constant conditions of photoperiod (16 h light/8 h dark) and temperature (25 °C)[7]. Under these conditions yolk deposition starts on the 4th day of eclosion and mature eggs are ovulated on the 7th day after eclosion. After 4 days of embryonic development 1st instar larvae are laid on the 11th day after eclosion.

The IGR ZR-2646[8] was dissolved in 99.9% pure acetone and 5 µl of the solution containing known amounts of IGR was applied topically onto mid dorsal abdominal cuticle of each female fly. Since this compound was reported to act as a juvenile hormone mimic as well as a juvenile hormone antagonist in *Manduca sexta* with high and low doses respectively[9] the effect of 20 µg or 5 µg per fly was tested. Control flies received 5 µl of acetone only.

Results and discussion. The table shows the effect of ZR-2646 on egg fecundity. Neither of the doses used caused any reduction in the total number of matured eggs produced per fly. However, fertility was affected by 20 µg of IGR. The effect was dependent on the day of treatment.

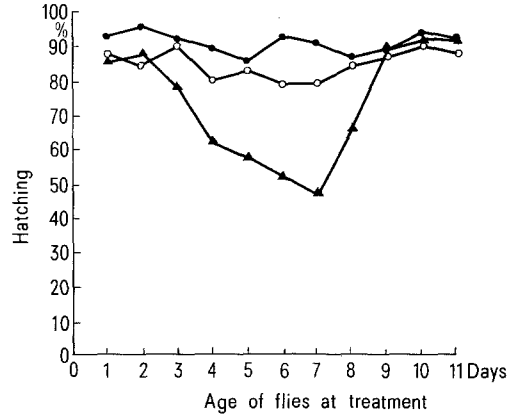

Fig. 1. Effect of ZR-2646 on the percentage of eggs that hatch into 1st instar larvae. ●, Control; ○, 5 µg of ZR-2646; ▲, 20 µg of ZR-2646.

Maximum reduction in the number of fertile eggs was observed when the flies were treated on the 7th day after eclosion (figure 1).

The identity of the stages at which mortality occurred during the peak period of sensitivity (6th and 7th days after eclosion) is shown in figure 2. While considerable numbers of embryos failed to complete embryogenesis in 6-day-old flies, this effect was not noticed when the flies were treated on the 7th day after eclosion. At this stage a large proportion of the mortality was due to inhibition of hatching of fully developed embryos. On both days of treatment a significant number of 1st instar larvae failed to molt to 3rd instar. There was no further mortality associated with larval-pupal and pupal-adult molts.

The peak sensitivity period of flies reported in this study coincides with the time of ovulation. In this respect these results parallel those reported for *Pyrrhocoris apterus*[3], *Hayalophora cecropia*[4], and *Drosophila melanogaster*[10]. Arrest of embryogenesis was significant only if the eggs were exposed to IGR just before ovulation; if the eggs were already ovulated at the time of IGR application (7-day-old flies) embryonic development was not affected, but hatching of fully developed embryos was blocked. Many of these embryos that hatched as 1st instar larvae also died before molting to 3rd instar (figure 2). Similar results of differential sensitivity of different stages of embryos was also observed in *Drosophila melanogaster*[10].

The insensitivity of the flies to IGR during the period of oocyte growth explains the absence of any effect on fecundity. Reduction in the total number of eggs produced per female was reported only for those insects which were sensitive to IGR during the period of oogenesis[5,11]. In *Sarcophaga bullata* maximal sensitivity was observed only during the period of ovulation, that is after the completion of egg maturation and therefore there was no reduction in the fecundity of the flies.

It is clear from these results that ZR-2646 acts as a juvenile hormone mimic for *Sarcaphoga bullata* with 20 µg per fly causing significant ovicidal effects.

Total number of mature eggs produced per female after topical application of ZR-2646*

Age of flies at treatment (days after eclosion)	Acetone 5 µl	ZR-2646 5 µg	ZR-2646 20 µg
1	102	100	98
2	110	92	95
3	98	96	105
4	108	101	112
5	100	104	96
6	96	94	104
7	98	108	106

* Average from 50 flies for each treatment.

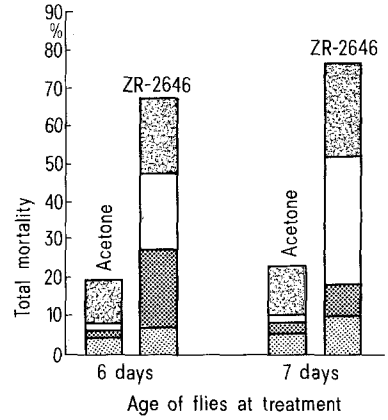

Fig. 2. Mortality of embryonic and postembryonic stages from treatment of female flies with 20 µg of ZR-2646. The unhatched fully developed embryos and hatched 1st instar larvae were cultured on beef liver. The undeveloped and poorly developed eggs were dechorionated and examined under phase contrast microscope. 1st and 2nd instar larvae; fully developed embryos; poorly developed embryos; undeveloped eggs.

1 The work was supported by the National Research Council of Canada.
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Satellite cells in denervated muscles

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Summary. It is known that, in a denervated striated muscle, the satellite cells multiply by mitotic division. A liaison between these satellite cells and the Schwann cell in front of the post-synaptic membrane in denervated frog muscle has been observed. It is probable that such cell connections help in the subsistence of the Schwann cell in a denervated muscle.

During the course of experiments on denervated muscles (rectus internus major and sartorius) of *Rana esculenta*, we observed by electron microscopy the multiplication of mononucleated cells designated as 'satellite cells' by Katz[2] and Mauro[3]. It is known that these satellite cells are capable of mitotic division, and that multiplication results from denervation[4–6]. During the 2nd month after denervation, the activation of satellite cells is manifested by an increase in their size and organelles. There is an expansion of endoplasmic reticulum and Golgi complex; ribosomes and pinocytotic vesicles are in abundance. At this stage of denervation, these cells are seen multiplying, and often 2–3 of them are aligned one after the other between the basal lamina and sarcoplasmic membrane (figure 1). It seems that, after proliferation, these satellite cells are capable of protracting themselves from the original site of multiplica-