Powdered neem seed kernel was extracted seven times with hexane to remove the oil content. Deoiled powder was further ground in a pestle and mortar and passed through 200 mesh sieve. Fine powder thus obtained was mixed with wheat flour (w/w) in the following manner. Weighed quantity 100 mg of the powder was dissolved in 20 ml acetone (only partly soluble) in a beaker. To this, 10 g of the wheat flour was added and stirred thoroughly. After evaporating the solvent the wheat flour was transferred into specimen tube (10 × 4 cm). Three replications were made for the treatment. Wheat flour treated with acetone alone served as control (two replicates). Ten first-stage larvae were released in each specimen tube and covered with muslin cloth and then transferred to a glass cupboard maintained at 35° ± 2°C and 65 ± 5% humidity respectively. Similar experiment using third instar larvae was also conducted to find out whether the larvae could be controlled at this stage of development. *Trogoderma* larvae were obtained from the culture maintained in the laboratory on wheat flour.

Weekly observations revealed that none of the 30 first instar larvae could develop beyond second instar and all died after the second week while 18 out of 20 larvae released in control emerged as adults. In the other experiment where third stage larvae were introduced all the 20 larvae in control emerged as normal adults by the end of third week while in the treated they were still in larval stage. Out of 30 larvae in the treatment, 5 pupated in the 4th week and one deformed adult emerged in the 5th week. Further development of the remaining larvae was almost completely arrested and all, except one which survived upto 15th week, died by 8th week. None of the pupae could emerge as adult.

Of the several biologically active compounds known to occur in the seed kernel[4] only azadirachtin has been reported to retard the growth and kill the insects by affecting hormonal balance of the insects[5,6]. Since deoiled kernel is reported to be rich in azadirachtin content, the growth retarding effect and the death of the larvae are attributed to the azadirachtin present in deoiled kernel and not due to kernel's antifeedant effect because there was some development in both first and third instar larvae. In the latter case it was more as 5 larvae could develop upto pupal stage and one of them emerged as deformed adult. This suggests that feeding did occur in larvae which affected them adversely. Further it has also been reported that insects fed on azadirachtin treated diet develop at much slower rate than control and many even die at the time of moulting[7].

The present finding offers a very cheap and an effective method for controlling this hard and serious pest of wheat in storage.

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EFFECT OF JUVENILE HORMONE ANALOGUE ON THE EGG NUMBERS AND EGG VIABILITY IN DIFFERENT EGG CYCLES OF THE BUG, *DYSDERCUS KOENIGII* FABR

K. P. SRIVASTAVA, R. L. KATIYAR and V. K. AWASTHI
Department of Zoology, Banaras Hindu University, Varanasi 221 005, India.

INVESTIGATIONS have shown that juvenile hormone analogue (JHA) adversely affects both fecundity and fertility of insects[1-3]. But in none of these studies, a correlation between the JHA effects on these parameters of reproduction could be established. The data presented in this note show such a correlation.

Dysdercus koenigii shows 6 egg cycles under laboratory conditions; the first one in 6 to 7 days after adult emergence and the rest at intervals of 3 to 4 days after the first. Ten to 12 hr old adult females were given a topical application of 10 and 100 μg of JHA R-394 (ethyl 9-cyclohexyl-3, 7-dimethyl-2,4 nonadienoate, kindly supplied by Dr Streinz of the Academy of Sciences, CSSR) dissolved in 1 μl of acetone, controls receiving 1 μl of acetone alone. The insects were provided with young adult males for mating and the

Table 1 *Effect of JHA on the egg number and viability in D. koenigii*

Dose (μg)		Egg cycles++					
		I	II	III	IV	V	VI
0	egg numbers (control)	128.60 ±2.21	110.33 ±2.50	91.00 ±2.01	70.25 ±2.78	49.66 ±1.50	44.30 ±2.22
	% variability	98.28	96.37	95.82	96.72	95.77	97.50
10	egg numbers	69.50 ±2.30	61.10 ±2.56	47.67 ±2.66	41.30 ±2.19	35.50 ±2.40	22.00 ±1.48
	% viability	0	14.40	43.90	46.17	66.19	78.19
100	egg numbers	62.40 ±3.50	49.60 ±2.01	35.46 ±1.75	25.70 ±1.32	15.00 ±1.60	+
	% viability	0	0	0	3.89	26.00	+

+ insects died; ++ each datum is an average of 10 insects

number of eggs laid and hatched in each egg cycle were recorded and the per cent hatching calculated.

The results are summarized in table 1, which shows that (i) the number of eggs declined towards the later egg cycles and egg viability remained more or less the same in the controls (ii) in the JHA-treated insects, the decline in the number of eggs is not only greatly magnified but also occurs in egg viability (iii) the reduction in the number of eggs increases from a lower to the higher egg cycles and in egg viability, the order is reversed. Some of the deleterious histological changes observed in the ovarioles after the JHA treatment are lateral orientation and resorption of oocytes (our unpublished observations).

Other factors (*viz* nutritional, environmental and mating) that generally affect fecundity in insects[4] remaining constant in these studies, the number of eggs will depend on the number of oogonia attaining maturity; and the egg hatches, on the fusion of gametes and successful completion of embryogenesis. Both the latter events occur after oviposition in the majority of the insects[5]. A reduction in the number of eggs will therefore, indicate damage to the sex cells (oogonia) prior to oviposition and damage to the eggs after oviposition in egg hatches. Decline in the number of eggs towards the later egg cycles in the controls of the present insect is a natural phenomenon of ageing[4]. But its greatly enhanced rate in the experimental insects cannot but be attributed to the JHA effect. This is one of the usual juvenile hormone effects reported in other insects[2,3,6] and is caused by cell death in the germarium, resorption of young oocyte in the vitellarium, reduced number of oocytes etc[7,8]. However, what merits our attention in these studies is the order—from lower to higher egg cycles in the case of the number of eggs, and from higher to lower egg cycles in case of the egg viability—in which the decline occurs. These sequences of decline can be explained as follows: the sex cells (oogonia or their precursors) that produce earlier egg cycles are expected to be relatively more differentiated than those that produce the later egg cycles. Since JHA adversely affects the sex cells at an early (proliferating) stage,[7,8] it will injure fewer of those cells that produce earlier egg cycles (they being more differentiated and so older) and more of those cells that produce later egg cycles (they being less differentiated and so younger) resulting in the observed order of decline in the number of eggs. Since in the case of egg viability, the deleterious effects of JHA are likely to occur in the eggs after oviposition, a time-lag of a fair duration between the JHA application and manifestation of its effects will precede oviposition during which some of the analogue may get metabolised[9,10] to elicit lesser effect in the later than in the earlier egg cycles resulting in more egg hatches in the former. Zero viability in some of the early egg cycles indicates a complete inhibition of embryogenesis by the JHA.

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NEWS

CANDIDA IS NO LONGER A HARMLESS YEAST

. . . "*Candida albicans* appears to live in most of us as part of the normal body flora. Billions of friendly bacteria help the immune system to keep the yeast under control . . . . Within the past few decades, however, levels of yeast have increased dramatically in many people. Part of the reason may be that modern drugs and diets stimulate *Candida* growth. And after more than 20 years of clinical observations involving nearly 3,000 patients, Orian Truss [a Birmingham, Ala., internist] is convinced that this yeast is implicated in a wide variety of human ills, from depression and hormonal disturbances to allergic reactions and autoimmune diseases . . . . Truss claims that according to mathematical calculations based on the number of antigens yeast produces, there may be trillions of strains of *Candida* in the world. 'And logic says we probably pick up new strains constantly—from a doorknob; from sleeping in a hotel bed. Regular disinfectants won't kill it,' he says. 'Certainly it is spread by kissing and by sexual intercourse. And I don't see any way that a newborn baby won't have its mother's strains.' But exposure to the yeast does not always mean it will produce a health problem. 'The illness comes about only when a *particular* immune system cannot deal with a *particular* strain of *Candida*.'"

[(Sherry Baker in *OMNI* 7(6):84–8, 120–8, Mar 85). (Reproduced with permission from Press Digest, *Current Contents*®, No. 21, May 27, 1985, p. 20. Published by the Institute for Scientific Information®, Philadelphia, PA, USA.)]

COMPUTER-CONTROLLED BUS TRAFFIC

A computer system to improve the operation of mass-transit facilities has been put to use in Kazan, capital of Tataria, a republic in the middle reaches of the Volga. Special sensors in city streets transmit to the computer information on the arrival of buses to a station. The computer fixes the time and monitors observance of the traffic timetable.

Part of the new system are TV cameras, which observe the busiest parts of the one million strong city. They help the traffic dispatchers, who send additional buses to the busiest lines (*Soviet Features*, Vol. XXIV, No. 104, July 1985)