method[7]. The gastric mucosa was homogenized in 0.1 *M* phosphate buffer, pH 8.0. Final concentration: 20 mg (wet weight) per ml. After centrifugation at 10,000 × *g* for 10 min at 0 °C, aliquots (5–20 µl) were added to 0.5 ml of 0.1 *M* acetate buffer, pH 3.5, and mixed with ^{57}Co-labelled cyanocobalamin (approx. 20,000 cpm). 0.2 ml of the mixture was passed through a Sephadex G 25 column (length 200 mm, inner diameter 8 mm). The column was washed with 0.1 *M* phosphate buffer, pH 7.0. Protein-bound vitamin B_{12} was excluded from the gel and appeared in the first 3 ml fraction after the void volume. This fraction was collected and quantitated by γ-spectrometry. Blank values were obtained by running a parallel separation of identical amounts of free vitamin B_{12} and collecting the same fraction for quantitation.

Results and comments. In the rat, reserpine was found to mobilize gastric vitamin B_{12}-binding proteins. In the mouse, hamster, guinea-pig and rabbit, however, reserpine was without effect (Table I).

Total truncal vagotomy of the rat did not significantly affect the concentration of gastric histamine or vitamin B_{12}-binding proteins. Vagal denervation did, however, cause a total inhibition of the capacity of reserpine to mobilize both gastric histamine and vitamin B_{12}-binding proteins (Table II). The mechanism behind the effect of vagotomy on the action of reserpine is unknown. Interestingly, gastrin, which is believed to originate from a similar type of gastric endocrine cell in the pyloric gland area[17–19], is mobilized by reserpine; this gastrin-mobilizing effect of reserpine is also abolished by vagal denervation[20].

While reserpine fails to deplete the gastric stores of vitamin B_{12}-binding proteins in the mouse, hamster, guinea-pig and rabbit, the B_{12}-binders of the rat reside in a reserpine-sensitive storage pool. This seems to agree with the existence of amine-storing gastric endocrine cells, which are markedly different as regards their sensitivity to the amine-releasing action of reserpine[9–11]. In the rat, reserpine mobilizes both histamine and vitamin B_{12}-binding proteins from the gastric mucosa. After vagal denervation, reserpine fails to affect the gastric content of histamine as well as of B_{12}-binding proteins. Together these observations support the concept that locates IF to some cell type within the system of gastric endocrine cells[21].

Zusammenfassung. Reserpin mobilisiert Histamin und den «Intrinsic factor» aus dem Rattenmagen. Eine trunkuläre Vagotomie hebt diese Wirkung von Reserpin auf. Reserpin hat keinen Effekt auf den «Intrinsic factor» des Magens von Maus, Hamster, Meerschweinchen und Kaninchen.

R. HÅKANSON, G. LIEDBERG and K. LINDSTRAND

Departments of Pharmacology, Surgery and Hematology, University of Lund, Lund (Sweden), 30 December 1970.

[17] J. E. MCGUIGAN, Gastroenterology *55*, 315 (1968).
[18] W. G. FORSSMANN and L. ORCI, Z. Zellforsch. *101*, 419 (1969).
[19] G. BUSSOLATI and A. G. E. PEARSE, Histochemie *27*, 1 (1970).
[20] S. EMÅS and B. FYRÖ, Acta physiol. scand. *63*, 358 (1965).
[21] Supported by grants from the Swedish Medical Research Council (No. B71-14X-1007-05C and No. B71-19X-766-05C), the Medical Faculty of Lund, and Albert Påhlson's Foundation.

The Herbicide Eptam® 6-E: A Selective Female Chemosterilant for the Egyptian Cotton Leafworm, *Spodoptera littoralis*

Several adult Lepidoptera have been successfully sterilized in the laboratory through the use of the most common insect chemosterilants[1]. But for practical interest, the lack of specific food and generally the ignorance of effective attractants require their pre-emergence chemosterilization. As many other lepidopterous pests[2,3], the larval treatment of the Egyptian cotton leafworm *Spodoptera littoralis* Boisduval (Noctuidae); the most voracious pest in UAR, with aziridines[4,5] or antimetabolites[5] that mostly have been proved effective when administered to imagoes resulted in a partial (if any) sterility in the resulting adults even at toxic doses[4,5].

Therefore, compounds with apparently new mode of action were sought for and investigations were initiated in our laboratory to find effective agents that can be used as larval chemosterilants in an efficient and practically integrated program of eradication for this insect pest.

This paper reports the effects of the larval feeding with the herbicidal thiolcarbamate Eptam® 6-E (S-ethyl-dipropylthiolcarbamate (75.5% active ingredient)) on the growth rate, adult emergence, number of eggs laid and hatch. The effectiveness of other compounds against this pest will be reported elsewhere.

The rearing and the feeding techniques, as well as the assessment of the sterilant activity, were previously described[6]. The exploratory dosage-mortality tests of this study, which are not presented, showed that the optimum rate tolerated at 24 h was 250 µg active ingredient/larva where no kill observed. Each test contained at least 75 Eptam-fed last instar larvae along with about an equal number for the check group. Experiments, including untreated checks, were replicated 4 times. The Table summarizes the data obtained.

When fed to larvae, the herbicide Eptam apparently inhibited egg laying in the ensuing females that were mated to untreated males. Although in some replicates (No. 2 and 4) very few eggs were deposited in a single patch, no sign of hatch was detected. Untreated females mated to males that developed from treated larvae laid fewer eggs which were less viable than untreated checks. The average reduction in the biotic potential of these females was found to be 23.3%. The herbicide had also high selectivity for inducing a deteriorating development in females. At immature stages, the late mortality and teratogenesis were so conspicuous among females that

[1] A. B. BOŘKOVEC, Adv. Pest Contr. Res. *7*, 1 (1966).
[2] L. BONNEMAISON, Phytiat. Phytopharm. *15*, 79 (1966).
[3] D. O. HATHAWAY, L. V. LYDIN and B. A. BUTT, J. econ. Ent. *59*, 851 (1966).
[4] A. TOPPOZADA, S. ABDALLAH and M. E. ELDEFRAWI, J. econ. Ent. *59*, 1125 (1966).
[5] M. T. EL-IBRASHY and N. F. SHEHATA, unpublished observations.
[6] M. T. EL-IBRASHY, in preparation.

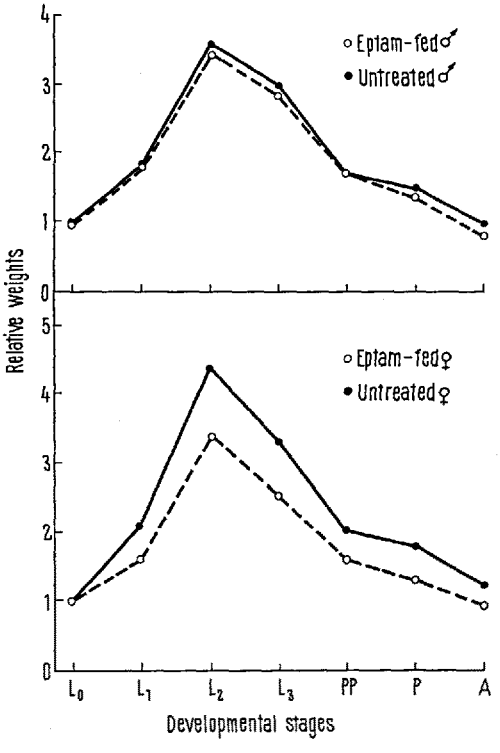
Effect of the herbicide Eptam® 6-E fed to the newly-ecdysed last (= sixth) instar larvae of *S. littoralis* upon number and biotic potential of emerging moths[a]

	Replicate No.															
	1				2				3				4			
	Treated		Check		Treated		Check		Treated		Check		Treated		Check	
	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀
No. larvae tested[b]	75		80		100		100		100		90		85		85	
No. moths produced[c]	12	3	15	16	16	4	20	20	14	4	15	17	13	3	16	15
Mean eggs laid (♀)	972	–	1172		988	30	1204		881	–	1101		1050	21	1280	
Mean hatched eggs (♀)	836	–	1078		865	0.0	1143		758	–	992		883	0.0	1144	
Decrease in egg viability (%)	22.4	–	–		24.3	100.0	–		23.6	–	–		22.8	100.0	–	

[a] The dose used was 250 µg/larva. [b] External sex discrimination is impossible in the larval stage of the insect. [c] The calculated sex ratio was almost 1:4 and 1:1 (females:males) in treated and untreated insects respectively. See Text for explanation.

the chemical reduced the emergence of adult females by about 80%. The female to male ratio among the resulting moths was about 1:4, compared with a sex-ratio of almost 1:1 in untreated checks. The chemical, at the applied dose, had no apparent effect on adult longevity of either sex. Evidently, the surviving larvae that produced adults were not injured. However, the growth rate of developing females was selectively reduced in contrast to males which were practically not affected (Figure).

The nature of the mechanism by which Eptam brings about a selective feminine inhibitory action on growth and biotic potential of the insect remains to be elucidated. However, for a chemical to modify growth in a broad sense, it must arrive at the physiological site through which its actions are manifested. Inasmuch as ovarian maturation is a part of the overall growth, it is probable



The growth rate of *S. littoralis* fed the herbicide Eptam at the outset of the last (= sixth) larval instar compared with the corresponding untreated sexes. The rate of each indicated group is presented as insect weights relative to the initial weight of larvae at the time of outset. L_0–L_3, age in days of the last larval instar; PP, prepupal stage; P, pupal stage; A, adult stage.

that the herbicide Eptam would intervene mainly with lipid metabolism, abolishing thereby the normal tissue response to the corpus allatum hormone. Allatectomy in the last larval instar of *S. littoralis* led to the emergence of non-ovipositing females[7], due mainly to the cessation of the probable lipogenetic activity of the ovaries[8]. The growth rate of allatectomized insects was also so reduced that the emerging moths were considerably smaller than sham controls[7,9]. Sexual dimorphism in lipid content and use has been demonstrated in several Lepidoptera and correlated with storage of reserves for egg production[10]. The possibility still exists that the herbicide may change the normal sensitivity of the tissues to the hormone rather than interference with the hormone activity (El-Ibrashy[11]). This could be a result of one or the combination of the known pharmacological activities of other carbamate derivatives; viz. being mitotic poisons[12] and enzyme inhibitors[13].

In fact, the findings discussed here have raised a number of questions that warrant further investigations before any definite conclusion could be drawn. Nevertheless, the evidence available may arise interest in thiolcarbamate as apparently highly effective and innocuous chemosterilants for the economically important noctuids; but first there is a real need to identify the sites through which these chemicals express their actions at the cellular and molecular levels. This problem is presently being tested in our laboratory[14].

Zusammenfassung. Das herbizide Thiolcarbamat Eptam® hemmt die Entwicklung der Weibchen der ägyptischen Baumwollraupe *Spodoptera littoralis*. Die schlüpfenden Weibchen sind relativ klein und legen keine oder nur sterile Eier.

M. T. El-Ibrashy

Laboratory of Plant Protection,
National Research Centre, Cairo (Egypt),
15 December 1970.

[7] M. T. El-Ibrashy, J. Insect Physiol., in press.
[8] M. T. El-Ibrashy and I. Z. Boctor, Z. vergl. Physiol. *68*, 111 (1970).
[9] M. T. El-Ibrashy and N. F. Shehata, J. Endocr., in press.
[10] L. I. Gilbert and H. A. Schneiderman, Gen. comp. Endocr. *1*, 453 (1961).
[11] M. T. El-Ibrashy, Z. angew. Ent. *66*, 113 (1970).
[12] G. W. Ivens and G. E. Blackman, Symp. Soc. exp. Biol. *3*, 226 (1949).
[13] B. Bacetti, Redia *47*, 199 (1962).
[14] Acknowledgment. The author expresses appreciation to Dr. E. N. Abou-Zeid, Laboratory of Plant Physiology, N.R.C., Cairo, for furnishing the herbicide Eptam; a product of Stauffer Chemical Co., 380 Madison Ave., New York 17 (N.Y., USA).