are supposed to be attached and, as the above results show, all essential thiol groups connected with the K-ATPase of myosin are protected from alkylation. The intermediate inactivation rate, resulting from alkylation in the presence of high Mg-ADP concentrations, implies some extent of ADP binding to myosin. It is unlikely that this effect is due to the existence of different myosin species (e.g. some with both heads protected by rigor attachment and others inactivated because of dissociation of both heads from actin) since it is not influenced by a 50-fold change in the ADP cencentration. Hence it may be concluded that all myosin molecules are in the same state, binding at least one ADP and interacting at the same time with actin. The additional evidence that when myosin-ADP complexes combine with actin, ADP becomes liberated[26], suggests that this specific interaction occurring in myofibrils comprises one ADP-free rigor-like interacting, and one ADP-loaded non-interacting head. Furthermore this particular type of cooperative interaction is regulated by the troponin-tropomyosin complex. In the context of active muscle, this view is in line with the suggestion of VINIEGRA-GONZALEZ and MORALES[27] that the myosin heads of one cross-bridge bind alternately on, and exert force at, two different monomers of the actin filament.

Zusammenfassung. Alkylierung von 2 Thiolgruppen pro Myosin mit NEM bei 0 °C in Gegenwart einer Diphosphatkette inaktiviert die K-ATPase vollständig. In Myofibrillen werden diese Thiolgruppen durch Rigor-«interaction» beider Myosinköpfchen mit Actin vor Alkylierung geschützt. In Gegenwart von Mg-ADP tritt eine spezifische vom Rigor verschiedene «interaction» zwischen Myosin und Actin auf. Man muss annehemen, dass dabei nur 1 Myosinköpfchen ans Actin bindet und dass das andere Köpfchen ein ADP gebunden hat.

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[27] G. VINIEGRA-GONZALEZ and M. F. MORALES, Bioenergetics *3*, 55 (1972).

Storage of Insecticides in the Fat Body of *Spodoptera littoralis* (Boisd.) as a Possible Mechanism of Resistance

Storage of insecticides in the insect fat body has been shown by many workers to be a contributing factor in developing resistance. Lipids may act as competitive sites for toxic agents, especially if the latter are lipid soluble compounds (MUNSON et al.[1]). The fat body is also considered to be one of the most active tissues in the metabolism of some insecticides (FEWICK[2]; KUHR[3]).

Resistance of *Spodoptera littoralis* against insecticides is now a common phenomenon in Egypt. Among the factors that might contribute to the total resistance of this pest is the enhanced rate of storage of insecticides in the fat body. This possibility was investigated.

Materials and methods. Three R-strains and 1 S-strain of *S. littoralis* were used. The R-strains were reared under continuous pressure of the corresponding insecticides, DDT, methyl parathion and/or carbaryl. By injection, all the R-strains had an almost identical resistance level, i.e., 3-fold. By topical application, however, the DDT R-strain was 25-fold, the carbaryl R-strain was 13-fold, and the methyl parathion R-strain was 3-fold. Total fat content was determined by extracting the dried larvae according to BENNET and THOMAS[4].

Analytical grade samples of DDT, methyl parathion and carbaryl were used. All strains were injected with the LD_{50} of the S-strain. Batches of 50 full grown larvae were dissected to remove the fat body at various intervals after injection, and 0.2–0.3 g. samples were extracted by homogenizing in the appropriate solvent.

The extraction and clean up procedures of KLEIN et al.[5], MOLLHOFF[6] and JOHANSON et al.[7] and those of SCHECHTER et al.[8], COFFIN and MCKINELY[9], and MISKUS et al.[10] for the colourimetric determination, were adopted for the samples containing DDT, methyl parathion and carbaryl, respectively.

Storage of insecticides in the fat body of susceptible and resistant strains of the 6th instar larvae of *S. littoralis*

Insecticides applied	Time after treatment (h)	Amount of insecticide stored	
		S-strain (ppm)	R-strain (ppm)
DDT	0	Negligible	Negligible
(Amount injected	1.5	36	47
200 µg/g)	3	280	533
	6	167	208
	12	144	197
	18	150	131
	24	150	125
Methyl parathion	0	Negligible	Negligible
(Amount injected	1.5	17	43
30 µg/g)	3	28	48
	6	9	34
	12	7	15
	18	5	3
	24	0	0
Carbaryl	0	Negligible	Negligible
(Amount injected	1.5	5	8
400 µg/g)	3	16	15
	6	10	11
	12	9	8
	24	7	8

[1] S. C. MUNSON, G. M. PADILLA and M. L. WEISMANN, J. econ. Ent. *47*, 578 (1954).
[2] M. L. FEWICK, Biochem. J. *70*, 373 (1958).
[3] J. R. KUHR, J. agric. Food Chem. *18*, 1023 (1970).
[4] S. E. BENNET and C. A. THOMAS, J. econ. Ent. *56*, 239 (1963).
[5] A. K. KLEIN, J. O. WATTS and J. N. DAMICO, J. Ass. off. agric. Chem. *46*, 165 (1963).
[6] E. MOLLHOFF, Bayer PflSchutz-Nachr. *20*, 557 (1967).
[7] D. B. JOHANSON, F. E. CRITCHFIELD and B. W. ARTHUR, J. agric. Food Chem. *11*, 77 (1963).
[8] M. S. SCHECHTER, S. B. SOLOWAY, R. A. HAYES and H. L. HALLER, Indian eng. Chem. *17*, 704 (1945).
[9] D. E. COFFIN and W. B. MCKINELY, J. Ass. off. agric. Chem. *46*, 223 (1963).
[10] R. MISKUS, H. T. GORDON and D. A. GEORGE, J. agric. Food Chem. *7*, 631 (1959).

Results and discussion. The total fat content in the S-strain and the 3 R-strains did not vary greatly, i.e. 3.2, 2.9, 3.9 and 3.1% for the S-, DDT R-, methyl parathion R-, and carbaryl R-strains, respectively. This is in contrast to several publications showing a positive correlation between insect resistance to different insecticides and the amount of insect fat (MUNSON and GOTTLEIB[11]; MUNSON et al.[1]; BENNET and THOMAS[4]; KHAN and BROWN[12]. The results of BRIDGES and COX[13], and ASCHER and NERI[14] are generally in agreement with ours.

Storage and rates of disappearance of DDT, methyl parathion and carbaryl are shown in the Table. The DDT R-strain (3-fold resistance by injection) stored about double the amount of DDT that was stored by the S-strain in the first 3 h after application. This suggests that storage of DDT is an important defence mechanism in *S. littoralis*. The enhanced rate of disappearance of the stored DDT in the R-strain is probably due to an increased rate of detoxication, as was found in the housefly by MIYAKE et al.[15]. 6 h after treatment, similar amounts of DDT were found in both strains and remained so for the rest of the experimental period.

Methyl parathion was also stored in considerable amounts in the R-strain. Compared with DDT, its rate of disappearance was much lower. On the other hand, storage of carbaryl and its rate of disappearance were almost equal in both S- and R-strains. This might be accounted for by a low lipid solubility and/or a high detoxication rate in the fat bodies of both S- and R-strains.

These results suggest that the levels of storage of DDT and methyl parathion in the R-strains is almost due to differences in the molecular species of fat, as compared to the S-strain.

Zusammenfassung. Signifikante Unterschiede des gesamten Fettgehaltes zwischen S- und R-Arten der *Spodoptera littoralis* wurden nicht festgestellt. DDT und Methylparathion wurden im Gegensatz zu Carbaryl in den R-Arten gespeichert und die Reduktionsrate von Methylparathion erwies sich als wesentlich niedriger als bei DDT.

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[11] S. C. MUNSON and M. E. GOTTLEIB, J. econ. Ent. *46*, 798 (1953).
[12] N. A. Q. KHAN and A. W. A. BROWN, J. econ. Ent. *59*, 1512 (1966).
[13] R. C. BRIDGES and J. T. COX, Nature, Lond. *184*, 1740 (1959).
[14] K. R. S. ASCHER and I. NERI, Entomologia exp. appl. *4*, 7 (1961).
[15] S. S. MIYAKE, C. W. KEARNS and H. LIPKE, J. econ. Ent. *50*, 359 (1957).
[16] Present address: Canada Department of Agriculture, Research Branch, University Campus, Saskatoon (Saskatchewan, Canada).

Effect of Prostaglandin $F_{2\alpha}$ on the Secretion of Pancreatic Juice Induced by Secretin and by Dopamine

Prostaglandins (PGs) are biosynthesized in most tissues from essential fatty acid. Thus neither specific tissue for production nor specific organs for their action have been identified yet. Many PGs, however, have potent peripheral vascular action[1–3] and a variety of actions on the gastrointestinal tract[4,5]. Recently, it was reported that prostaglandin E_1 (PGE_1) inhibited the volume of pancreatic juice induced by secretin and pancreozymine in dogs[6]. Since a very potent effect of prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$) on the dog's submaxillary gland has been reported[7], the response of the pancreas to $PGF_{2\alpha}$ attracted our interest. Previously we have developed the blood-perfused isolated canine pancreas preparation and have found specific secretogogue activity of dopamine[8]. In the present study, the effect of $PGF_{2\alpha}$ on the secretin- and dopamine-induced pancreatic secretion has been investigated using this preparation.

Experiments were performed on 7 adults mongrel dogs weighing 11–14 kg. The animals were anesthetized with 30 mg/kg of sodium pentobarbital injected i.v. The supranavel abdomen was opened by a midline incision. A polyethylene tube was inserted into the main pancreatic duct and the rate of secretion measured by a drop counter. The accessory pancreatic duct was ligated. Polyethylene cannulae were inserted into the gastroduodenal and the splenic arteries through which the pancreas was perfused with the animal's own blood from the left femoral artery by means of a Harvard peristaltic pump (Model 500–1200). All experiments were performed under constant arterial pressure at 100 mm Hg by means of a pneumatic resistance which was placed in parallel with the perfusion system. The details of the preparation were described in a previous paper[8]. A dose of 300 units/kg of sodium heparin was given at the beginning of the perfusion and a supplementary dose of 200 units was given i.v. at 1 h intervals. $PGF_{2\alpha}$ was injected into a rubber tube connected to the arterial cannula on a volume of 0.1 ml for 4 sec. Secretin and dopamine were infused intra-arterially at a constant rate of 0.1 ml/min by a Harvard infusion pump (Model 600–900). Drugs used in this study were secretin (kindly given from Professor J. E. JORPES, Karolinska Institute, Stockholm, Sweden), dopamine hydrochloride (ICN) and prostaglandin $F_{2\alpha}$ tromethamine (kindly given from Nippon Upjohn Ltd.).

A typical result is shown in the Figure a) and b). When secretin was infused at a rate of 0.05 units/min intra-arterially, the pancreatic secretion started immediately and reached a constant level. Then, $PGF_{2\alpha}$ in a dose of 100 µg was intra-arterially injected. The rate of secretion diminished gradually and reached the maximum inhibition at about 5 min later, but it returned gradually to the initial level. Perfusion blood flow transiently increased about 5 ml/min and systemic blood pressure decreased about 10 mm Hg by the injection of $PGF_{2\alpha}$ even though

[1] S. BERGSTROM, Science *157*, 382 (1967).
[2] J. NAKANO and J. R. MCCURDY, J. Pharmac. exp. Ther. *156*, 538 (1967).
[3] D. W. DUCHARME, J. R. WEEKS and R. G. MONTGOMERY, J. Pharmac. exp. Ther. *160*, 1 (1968).
[4] A. ROBERT, J. E. NEZAMIS and J. P. PHILLIPS, Am. J. dig. Dis. *12*, 1073 (1967).
[5] L. WAY and R. P. DURBIN, Nature, Lond. *221*, 874 (1969).
[6] J. RUDICK, M. GONDA, D. A. DREILING and H. D. JANOWITZ, Gastroenterology *60*, 272 (1971).
[7] R. A. HAHN and P. N. PATIL, Br. J. Pharmac. *44*, 427 (1972).
[8] K. HASHIMOTO, S. SATOH and O. TAKEUCHI, Br. J. Pharmac. *43*, 739 (1971).