gamma globulin (RGG), is added to the tissue culture. This response could be elicited with various different macromolecules used for sensitization or as carriers.

The polymorphism of the phenomenon was explored by immunizing mice at various ages with DNP-KLH and then exposing spleen cells from these mice to DNP-RGG and KLH. Differences in age at which carrier-independent helper capacity is acquired were studied with 6 different inbred strains of mice, and are shown in figure 2. The age at which activity was observed varied markedly and occurred at the earliest age in SJL and at the latest in DBA/1. Age-dependent changes were also observed with another hapten, para-azobenzene sulfonic acid (PAS; fig. 3). In this case, the response depended on the availability of hapten-primed B cells, but even if they were available, the indirect plaque-forming hapten response was lower than that observed in the indirect plaque-forming response to DNP. The carrier-independent helper capacity of SJL was again detected relatively early. It will be noted that the development of helper capacity in A/J was greatly delayed by comparison to its appearance in C57BL/6 (fig. 3), whereas in the indirect PFC response to DNP (fig. 2) the carrier-independent helper capacity of C57BL/6 and A/J became demonstrable at comparable ages.

The difference between PAS and DNP need for hapten-primed B cells is presumably attributed to 'naturally' induced DNP sensitization, as a concomitant of aging.

In short, we have observed a helper capacity which is evoked by an antigen-specific event, but can provide help to memory B cells, irrespective of the carrier. This helper capacity increases with age at a rate that shows remarkable polymorphism.

* Present address: Sakura National Hospital, Sakura-shi, Chiba Prefecture, 285 (Japan).
1 To whom reprint requests should be addressed.

2 B. Cinader, T. Amagai, T. Matsuzawa and K. Nakano, in: Cellular and molecular mechanisms of immunologic tolerance, p. 187. Eds T. Hraba and M. Hašek. Marcel Dekker, New York 1981.
3 J.C. Roder, A.K. Duwe, D.A. Bell and S.K. Singhal, Immunology *35*, 837 (1978).
4 R.E. Callard, B.F. De St. Groth, A. Basten and I.F. McKenzie, J. Immun. *124*, 52 (1980).
5 M.R. Szewczuk, Fedn Proc. *39*, 462 (1980); abstr.
6 T. Amagai, K. Nakano and B. Cinader, Scand. J. Immun., in press (1982).
7 A.J. Crowle, J. Miller and N. Grove, Gerontology *27*, 241 (1981).
8 T. Tada, T. Takemori, K. Okumura, M. Nonaka and T. Tokuhisa, J. exp. Med. *147*, 446 (1978).
9 C.A. Janeway, Jr, D.L. Bert and F.-W. Shen, Eur. J. Immun. *10*, 231 (1980).

O,p'-DDT causes growth of an androgen-dependent gland in *Coturnix* quail[1]

E. Adkins-Regan and E.D. Hurvitz

Department of Psychology and Section of Neurobiology and Behavior, Uris Hall, Cornell University, Ithaca (New York 14853, USA), 12 March 1982

Summary. *O,p'*-DDT injected into adult castrated male quail *(Coturnix coturnix japonica)* stimulated growth of the proctodeal (foam) gland, a structure that is androgen but not estrogen responsive, indicating that this pesticide constituent, in addition to its known estrogenic actions, is also androgenic.

O,p'-DDT, which constitutes about 15–20% of commercial DDT[2], has been shown to have estrogenic actions at several target organs; it causes growth of immature mouse uteri, chick oviducts, and quail oviducts[3,4], and causes morphological feminization of avian embryos[2,5]. It binds to cytosol estrogen receptors[6], and may also bind to androgen receptors, because it has been shown to inhibit binding of 5α-dihydrotestosterone (DHT) in the rat prostate[7].

The proctodeal gland, unique to the genus *Coturnix*, is a foam-producing protuberance posterior to the cloaca that can be externally measured[8,9]. It regresses in castrates, and is restored to intact size by treatment with testosterone (T)[10]. In vitro, the proctodeal gland of mature males primarily metabolizes T to DHT[11]. In vivo, DHT or its propionate (DHTP) are as effective or more effective than T or TP at stimulating growth of the gland[11,12], while estradiol is totally ineffective[12,13], suggesting that growth is mediated by DHT receptors.

For the present study, adult male quail housed on a 16 h light: 8 h dark light:dark cycle were bilaterally castrated under Nembutal anesthesia; 1 week later (day 1) they were examined to ensure that all proctodeal glands had regressed, and were randomly assigned to 1 of 4 treatment groups. Each bird was injected in the pectoral muscle twice daily for 16 days; the 2 injections were 0.5–2 h apart. 1 group received injections of the sesame oil vehicle. A 2nd group received oil injections on days 1 and 2, and thereafter received oil followed by 0.5 mg 5α-DHTP (Steraloids). A 3rd group received 1.0 mg *o,p'*-DDT (Aldrich) followed by oil on days 1 and 2, and 1.0 mg *o,p'*-DDT followed by 0.5 mg 5α-DHTP thereafter. A 4th group received 1.0 mg *o,p'*-DDT followed by oil each day. These procedures were designed to facilitate detection of any antiandrogenic as well as androgenic actions of *o,p'*-DDT.

Proctodeal glands were measured according to standard procedures[9] on day 4 and every other day thereafter. At the end of the experiment each bird was weighed, killed, and examined to confirm the absence of testicular tissue. All procedures were then repeated with new males, and the data from both experiments were combined.

O,p'-DDT had no deleterious effect on overall condition. Weights are shown in the table; none of the means are significantly different from each other. As the figure indicates, DHTP markedly stimulated proctodeal gland growth,

Body weights of the males at the end of the experiment

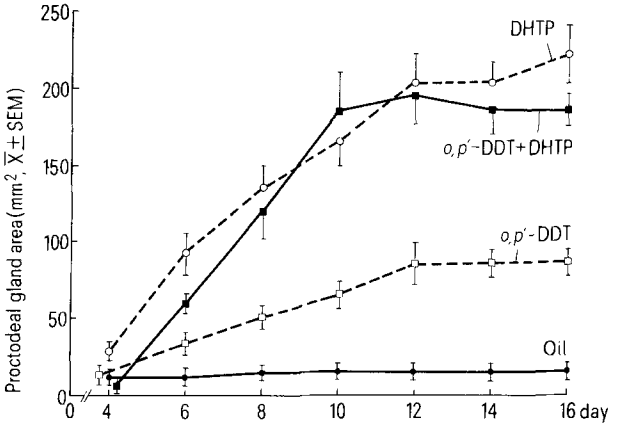
Treatment	N	Weight (g, $\bar{X} \pm$ SEM)
Oil	11	133±3
DHTP	11	154±4
o,p'-DDT+DHTP	11	150±4
o,p'-DDT	13	138±4

DHTP = 5α-dihydrotestosterone propionate.

and *o,p'*-DDT, when combined with DHTP, did not inhibit this response (i.e., had no antiandrogenic effect). But *o,p'*-DDT given alone significantly stimulated proctodeal gland growth; on each of days 8, 10, 12, 14, and 16 there was no overlap in the gland sizes of birds injected with *o,p'*-DDT vs oil.

Given that the proctodeal gland cannot be stimulated by estrogen, this result indicates that *o,p'*-DDT is acting as an androgen at this target organ. While *o,p'*-DDT-stimulated glands were smaller than DHTP-stimulated glands, or glands of intact males[12], they grew to about the same size as those of castrated males injected or implanted with the naturally-occurring androgens androsterone or androstenedione[13,14].

The significance of this result is 2-fold. First, some of the deleterious effects of DDT on the reproduction of wild animals[15] may be due to interactions of *o,p'*-DDT with androgen as well as estrogen receptors. Second, since other natural and artificial androgens (as well as estrogens) are known to aggravate certain cancers of the reproductive organs via receptor interactions[16,17], DDT could have a similar effect.



Proctodeal gland growth of castrated male quail injected with *o,p'*-DDT (1 mg/day), 5*α*-dihydrotestosterone propionate (DHTP, 0.5 mg/day), *o,p'*-DDT + DHTP, or oil. *O,p'*-DDT injections began on day 1; DHTP injections began on day 3. See the table for *N*'s.

1 This work was supported by NSF grant BNS76-24308 to E.A.R.
2 D.M. Fry and C.K. Toone, Science *213*, 922 (1981).
3 J. Bitman, H.C. Cecil, S.J. Harris and G.F. Fries, Science *162*, 371 (1968).
4 W.A. McBlain, V. Lewin and F.H. Wolfe, Can. J. Zool. *55*, 562 (1977).
5 H. Lutz and Y. Lutz-Ostertag, in: Intersexuality in the animal kingdom, p.382. Ed. R. Reinboth. Springer, New York 1975.
6 W.H. Bulger, R.M. Muccitelli and D. Kupfer, Steroids *32*, 165 (1978).
7 A.E. Wakeling and W.J. Visek, Science *181*, 659 (1973).
8 R.D. Klemm, C.E. Knight and S. Stein, J. Morph. *141*, 171 (1973).
9 B.D. Sachs, Science *157*, 201 (1967).
10 F.A. Beach and N.G. Inman, Proc. natl Acad. Sci. USA *54*, 1426 (1965).
11 R. Massa, D.T. Davies and L. Bottoni, J. Endocr. *84*, 223 (1980).
12 E.K. Adkins and E.E. Pniewski, J. comp. Physiol. Psychol. *92*, 1169 (1978).
13 E.K. Adkins, J.J. Boop, D.L. Koutnik, J.B. Morris and E.E. Pniewski, Physiol. Behav. *24*, 441 (1980).
14 E.K. Adkins, Horm. Behav. *8*, 201 (1977).
15 D.B. Peakall, Scient. Am. *222*, 73 (1970).
16 R.J.B. King, G.J. Cambray, R. Jagus-Smith, J.H. Robinson and J.A. Smith, in: Receptors and mechanisms of action of steroid hormones, part 1, p.275. Ed. J.R. Pasqualini. Dekker, New York 1976.
17 M.G. Mawhinney and J.A. Belis, in: Cellular mechanisms modulating gonadal action, p.331. Eds R.L. Singhal and J.A. Thomas. University Park, Baltimore 1976.

Influence of three synergists on the action of some insecticides against parental and resistant strains of the Egyptian cotton leafworm

E.I. El-Sayed, M.M. Abdel-Sattar and H.H. Mohanna

Laboratory of Plant Protection, National Research Centre, Dokki, Cairo (Egypt), and Central Agricultural Pesticide Laboratory, Ministry of Agriculture, Cairo (Egypt), 15 December 1981

Summary. Piperonyl butoxide, MGK 264 and DEF acted as synergists when used together with endrin and methomyl against the Egyptian cotton leafworm *Spodoptera littoralis*. The synergistic effect was higher with resistant strains than with the parental strain. The three substances tested as synergists actually antagonized the action of phospholan and cypermethrin on the parental strain, though they had some activity as synergists with these insecticides on resistant strains. The synergistic effect of DEF was higher than that of piperonyl butoxide or MGK 264, particularly on resistant strains.

The use of many insecticides has begun to suffer from a lowering of their potency due to insects acquiring an increased tolerance as successive generations are subjected to them. Synergists are of practical importance in increasing the efficiency of insecticides and increasing the spectrum of activity. In addition, they might restore the activity of an insecticide against resistant strains of insects. Also, it is often more economical to use synergists with an insecticide than to use the insecticide alone[1].

The purpose of this work is to study the action of 3 synergists, piperonyl butoxide, MGK 264 and DEF, on the efficiency of 4 insecticides used against the parental strain and against endrin-, phosfolan-, methomyl- and cypermethrin-resistant strains of *Spodoptera littoralis* BOISD (Lepidoptera, Noctudidae).

Materials and methods. The test insect was obtained from a parent strain, collected from the field and cultured in the laboratory for 2 generations, and from endrin-, phosfolan-, methomyl- and cypermethrin-resistant strains of *Spodoptera littoralis* maintained in the same laboratory. The insect was reared[2] under conditions of 25 ± 2 °C and 65 ± 5% relative humidity. The tests were done at levels of resistance of 16.98-, 8.11-, 9.13- and 13.89-fold, for endrin, phosfolan, methomyl and cypermethrin, respectively.

4th instar larvae, 40–45 mg each, were treated topically with 1 µl of acetone solution of the insecticide or an insecticide/synergist combination. The toxicants were applied to the dorsal region of the thorax. At least 5 replicates of 10 larvae each, were used at each concentration level. Mortality counts were taken 24 h after treatment. Data were corrected