natural JH and methylenedioxyphenoxy analogue is more than 2-fold greater than that shown by larvae raised on wax-free diet in which such high response is obtained (at the most JH-sensitive stages) only on application of approximately a 5-fold higher dose of hormone. Even with ethyl trimethyl dodecadienoate the wax-fed larvae show a significantly higher morphogenetic response. Thus, the difference in the morphogenetic response between wax-fed and wax-deprived larvae appears to reflect differences in sensitivity to JH rather than being the result of individual variations in the experimental animals. Moreover, this difference in morphogenetic response was repeatable on each of the 3 occasions when it was tested. Preliminary studies using another JH analogue, chlorophenoxy 6-epoxy-3-ethyl nonene showed similar differences between wax-fed and wax-free diet-fed larvae. From the above results, we conclude that the presence of beeswax in diet of *Galleria mellonella* influences its morphogenetic response to JH in the last larval stadium.

Reasons for the observed differences in morphogenetic response correlated with the presence or absence of beeswax in the diet of *Galleria* larvae are not clear. Although the presence of beeswax in the diet is inessential for the normal growth of *Galleria*[10], its presence in the diet improved their rate of growth[11,12]. Assuming that observed differences were due to variations in the weights of the larvae raised on the 2 diets which resulted in de facto differences in the dose of applied hormone, we determined the weight of the larvae raised under the 2 diet conditions. These data show that under these conditions of humidity and temperature the weight of the larvae raised on both diets was more or less identical. Furthermore, to avoid any effect of weight we repeated these experiments with larvae of uniform weight and obtained identical results. Thus, the differences noted in morphogenetic response are not the result of variations in the rate of growth of the larvae on the 2 diets. The C^{17}-fatty acid and phospholipid content of *Galleria* maintained for short term on beeswax-containing-diet differs from those deprived of beeswax[11,12]. Whether these differences in fatty acid and phospholipid content or some other hypothetical growth factor in the beeswax or possible differences in lipolytic enzymes in the larvae can account for the differences in their morphogenetic response remains to be proven. If specific substances can be found in the wax-diet which enhance the morphogenetic responses of *Galleria* larvae to JH, they may prove useful in increasing the insecticidal property of juvenile hormones[13].

Zusammenfassung. Mit Juvenilhormon und JH-Derivaten injizierte Larven der Wachsmotte *Galleria mellonella* zeigten stärkere morphogenetische Effekte, wenn ihre Nahrung Bienenwachs enthielt, als wenn diese fehlt.

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A Survey on the Mutagenicity of Various Pesticides

The widespread use of an increasing number of chemicals in agriculture[1,2] and the resulting contamination of food with an increasing variety of pesticide residues imposes several problems. Apart from the dangers of acute and chronic poisoning, the possible mutagenic effects of such contaminations could threaten the genetic health of coming generations[3], and it is therefore of high importance that these effects be investigated. Thus, the present survey should help to establish a priority list of pesticides which would have to be tested thoroughly in mammalian systems to evaluate their mutagenic potential for mankind.

From the array of pesticides registered for use in Swiss agriculture[4] and their derivatives and contaminants, we chose those for testing which fulfilled one or more of the following requirements: a) Structural relationship to known mutagens; b) Structural relationship to natural nucleobases; c) Chemical reactivity towards nucleic acids; d) Pesticidal mode of action through interference with nucleic acid metabolism.

The testing procedure was as described elsewhere[5,6]. Two controls were run on each group, the standard control giving the rate of spontaneous mutations of the bacterial strain used, and a positive control with known mutagens demonstrating the mutagenic responsiveness of this particular strain. The results are summarized in the Table, of which some comments are made below.

It should be made clear that caution has to be exercized in extrapolating these results to higher organisms, a point which is instructively illustrated by the case of captan. Although this fungicide has been shown to act as a mutagenic substance through its alkylating potency[7,8], investigations failed to disclose a similar effect in Drosophila[9], as well as in the 'dominant lethal test' in mice[10]. Moreover it has recently been demonstrated that addition of blood serum destroys its mutagenic activity, apparently by binding the active principle – possibly thiophosgene[11] – to the thiol groups of serum proteins[12].

Another point of interest is the ambiguous response of part of the s-triazines. A mutagenic effect in bacteria could have been expected, since these compounds can be regarded as structurally related to the pyrimidine nucleobases. Supporting evidence for this view was provided by the findings of a small incorporation of s-triazine herbicides into nucleic acids of *Escherichia*

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Compound Chemical name	Common or Trade Name	Response to the *Salmonella* strains his G46	TA1530	TA1531	TA1532	TA1534
N-trichloromethylthio-tetrahydrophtalimide	Captan	++	++	−	−	−
N-tetrachloroethylthio-tetrahydrophthalimide	Captafol	++	++	−	−	−
N-trichloromethylthio-phtalimide	Folpet	++	++	−	−	−
cis-4-Tetrahydrophtalimide	−	−	−	−	−	−
2-chloro-4-ethylamino-6-isopropylamino-s-triazine	Atrazine	−	−	−	−	−
2-chloro-4,6-bis (ethylamino)-s-triazine	Simazine	−	−	−	−	−
2-hydroxy-4,6-bis (ethylamino)-s-triazine	Hydroxysimazine	±	±	−	−	−
2-hydroxy-4,6-diamino-s-triazine	Ammeline	−	−	−	−	−
2,4,6-triamino-s-triazine	Melamine	−	−	−	−	−
2-chloro-4,6-diamino-s-triazine	−	±	±	−	−	−
2-methoxy-4,6-bis (isopropylamino)-s-triazine	Prometone	−	−	−	−	−
2-methylthio-4,6-bis (isopropylamino)-s-triazine	Prometryne	−	−	−	−	−
diethyl-2-isopropyl-6-methyl-4-pyrimidinyl-phosphorothionate	Diazinon	−	−	−	−	−
2-diethylamino-6-methylpyrimidin-4-yl-dimethyl-phosphorothionate	Pirimiphos-methyl	++	++	±	±	+
2-amino-4-hydroxy-6-methylpyrimidine	−	−	−	−	−	−
1-(butylcarbamoyl)-benzimidazole-2-carbamic acid, methylester	Benomyl	++	++	−	−	−
benzimidazole-2-carbamic acid, methyl ester	−	++	++	−	−	−
2-amino benzimidazole	−	±	+	−	−	−
benzimidazole	−	±	+	−	−	−
2-(2′-furyl) benzimidazole	Furidazol	++	++	−	−	−
9-hydroxyfluorene-carbonic acid (9), n-butylester	Flurenol-ester	−	−	+	±	±
2,3,7,8-tetrachlorodibenzo-*p*-dioxine	—	−	−	±	+++	±
octachlorodibenzo-*p*-dioxine	—	−	−	−	±	±
manganese-ethylenebisdithiocarbamate	Maneb	−	−	−	±	±
zinc-dimethyl-dithiocarbamate	Ziram	−	±	−	−	+
pentachloronitrobenzene	Quintozen, PCNB	−	±	−	−	±
dimethylsulfoxide (spontaneous reversions)		−	−	−	−	−
diethylsulphate (positive control)		+++	+++	−	+	−
2-aminopurine (positive control)		+++	+++	−	−	−
2-aminofluorene (positive control)		−	−	+	++	++

Scoring of mutations and explanation of the signs: Mutagenic events were scored as the number of revertant colonies to histidine autotrophy on a selective medium. Relative mutagenicity is then expressed as the number of revertants from treated plates, per 10^8 bacteria, divided by the number of spontaneous reversions, per 10^8 bacteria. Mutagenic activity, corresponding to relative mutagenicity: −, no = 1; ±, doubtful = 1–2; +, weak = 2–5; ++, medium = 5–10; +++, strong = >10. Strains his G46 and TA1530, resp., are reverting through base substitutions, while strains TA1531, TA1532, and TA1534, resp., are reverting through frameshift mutations. The *Salmonella* strains used throughout this study have been received from Prof. B. N. Ames (University of California, Berkeley), whose gift is gratefully acknowledged.

coli[13–15] as well as a study on chromosome aberrations in plant cells[16]. However, another cytogenetic plant study[17], and studies with *Salmonella* strains[18] and *Drosophila*[19], came to negative conclusions regarding the mutagenicity of s-triazine herbicides.

The positive results with benzimidazoles, which have been published in detail elsewhere[6], are to be regarded as more serious, at least until a more thorough testing in mammalian systems has been done, because of their increasing use and because of their persistence and possible accumulation in soils[20].

The dioxines play a role as contaminants of chlorophenols and their derivatives. Our findings confirm earlier studies which showed the mutagenic activity of tetrachlorodioxin in *E. coli*[21] and *S. typhimurium*[22].

A last point should be mentioned here and thrown open for discussion. Is it wise to use pesticides, which produce mutations in bacteria, fungi and other lower organisms, even if no such effects in mammals can be detected, to enhance thus the probability of mutational resistance against therapeutic and pesticidal agents? Two recent cases may be cited to illustrate this question: The systemic fungicide benomyl has been shown to induce benomyl-resistance in *Penicillium*[23] and *Botrytis* species[24], as well as in mildew on cucumber cultures[25]. By the use of the strong mutagen ethylmethanesulfonate and appropriate selection, it has been possible to increase genetically the resistance of wheat and tomato against 2 herbicides[26]. Although in this latter case these mutations were selected as desired results, analogous mutations of weeds would not be very much appreciated. It might therefore well be worthwhile to follow and study these problems and questions further[27].

Zusammenfassung. Die Resultate einer Prüfung von 16 ausgewählten Pestiziden auf ihre Mutagenität im *Salmonella*-test werden diskutiert.

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