istic IR, MS and ^{1}H-NMR spectra as methyl 8-acetylaminononanoate and methyl 9-acetylaminononanoate.
In conclusion, all these results point to structure **2** for the diacetylderivative and thus the natural compound is necessarily the corresponding diaminoderivative **1**.
Comparison of the spectral properties of **2** with those of the diacetylated derivative of the polar Dragendorff positive compound, previously reported in the basic extracts of *Adonia variegata*, *Harmonia 4-punctata* and *Semiadalia 11-notata*[4] shows that **1** is also present in these species. Moreover, **1** could also be isolated besides hippodamine and n-octylamine from a basic extract of *Hippodamia convergens*, surprisingly devoid of convergine[12].
The synthesis of **1** in order to evaluate its biological activities is under way, as well as the determination of its absolute configuration.

1 Maître de Recherches du Fonds National de la Recherche Scientifique.
2 We gratefully acknowledge the 'Fonds National de la Recherche Scientifique' for financial support. We express our sincere thanks to Professor K.S. Hagen for a generous gift of *H. leis conformis*.
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0014-4754/85/040519-02$1.50 + 0.20/0

Selectivity of action between pyrethroids and combined DDT-pyrethroid insecticides on Na^+ influx into mammalian neuroblastoma

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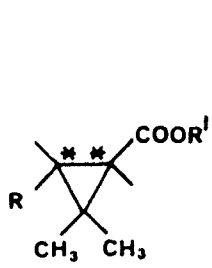
Summary. Several of the most active synthetic pyrethroid insecticides in the presence of sea anemone toxin II, induced a dose related influx of sodium ion into the C9 mouse neuroblastoma. The influx of sodium ion into this mammalian cell did not take place with a DDT analogue, EDO and several new combined DDT-pyrethroid insecticides, although these have been reported to cause excess sodium influx into arthropod axons, related to their insecticidal activity. This difference between species in the action of the new insecticides at the nerve sodium channel explains their low mammalian toxicity.
Key words. C9 cell; NIE 115 neuroblastoma; $^{22}Na^+$ influx; pyrethroid insecticides; combined DDT-pyrethroid insecticides.

Jacques and co-workers[2] have examined in detail the influx of $^{22}Na^+$ into rat C9 cell and NIE 115 adrenergic neuroblastoma. They have demonstrated that micromolar amounts of synthetic pyrethroid insecticides stimulated the entry of $^{22}Na^+$ into these cells via the Na^+ channel. To induce this influx of the sodium ion they have shown the requirement for synergy of the added pyrethroids with toxins specific to the gating system of the C9 cell and the NIE 115 neuroblastoma Na^+ channels. Without these toxins e.g. veratridine, batrachotoxin, or sea anemone toxin II, the pyrethroids did not stimulate the $^{22}Na^+$ ion influx. The C9 cell has a special property in that its Na^+ channels which are electrophysiologically silent, can be chemically opened by these toxins. The NIE 115 neuroblastoma is a electrophysiologically fully active nerve cell.
Two new series of insecticidally active compounds have been reported[3,4], whose design was based on the combination of DDT and pyrethroid structures. The new insecticides have exceptionally low acute mammalian toxicity when compared with the synthetic pyrethroids. In this note we report the results of $^{22}Na^+$ influx experiment for the new structures and one DDT analogue, which also gives a low mammalian toxicity.

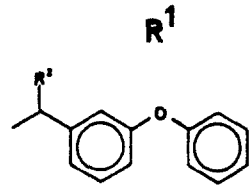
Results and discussion. EDO (GH149, table), an insecticide isosteric with DDT was shown by electrophysiological measurements to have an effect on Na^+ flux an arthropod (lobster) axon[5] similar to DDT itself. In this preparation it inactivated the closure of the Na^+ channel and caused a delay in the falling phase of the Na^+ mediated potential. This effect was blocked by tetrodotoxin. In the present experiments using the method reported previously[2] no stimulation of the $^{22}Na^+$ influx took place in the C9 cell and the NIE 115 neuroblastoma preparations, even on application of high concentrations (2.0 mmol) of EDO in the presence of sea anemone toxin II.
Four of the new DDT-pyrethroid compounds (table) were then tested in these two preparations. Listed values in the table show, that GH401, GH414 and GH601 have insecticidal activities intermediate between those of Kadethrin and Deltamethrin. However, similarly to EDO, these combined DDT-pyrethroid structures, added to a concentration of 0.1 mmol, did not stimulate the influx of $^{22}Na^+$ into the mammalian nerve cell preparations, in the presence of sea anemone toxin II.
In the experiments on the synthetic pyrethroids all had the 3,3-dimethylcyclopropane structure disubstituted at the 1,2-position of the ring. We could not correlate (table) the reported[2] rate of influx of ^{22}Na induced by the addition of the pyrethroids, with insecticidal activity measured in the Australian sheep blowfly (*Lucilia cuprina* W.), an insect we used previously[4] for the ranking of insecticidal activity.
In C9 cell preparations in which the Na+ channels are not capable of activation by electric stimulation, it was observed[2] that the pyrethroids activated the channel in a manner similar to that of veratridine which is known to slow down the inactivation process of a functional Na^+-channel[2]. In these preparation the increase of the rate of Na^+ influx could be stopped by tetrodotoxin, which was shown however, to exert its action at a different receptor to the pyrethroids. In that study it was also demonstrated that the Na^+ influx experiments in presence of sea anemone toxin II, closely simulated electrophysiological measurements of the delay in the falling phase of Na^+ mediated potentials in sodium channels induced by pyrethroids, in

Table 1. Differences of $^{22}Na^+$ influx into C9 cell and NIE 115 neuroblastoma for active pyrethroid and combined DDT-pyrethroid insecticides

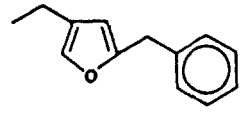
PYRETHROIDS

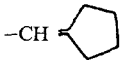
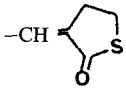


A



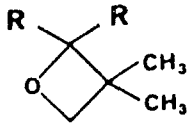
B



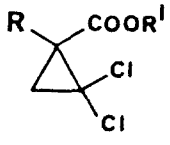
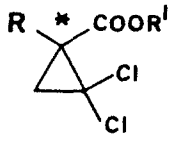
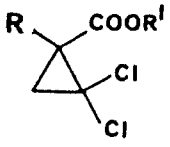
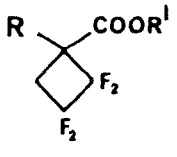
Name	R	R^1	R^2	Configuration	$K_{0.5}$, $^{22}Na^+$* influx, (μM)	Insecticidal** Activity (LD_{50}, μg/insect)
Deltamethrin	$-CH=C(Br)_2$	A	C≡N	1R,3S	0.5	0.0026
Cispermethrin	$-CH=C(Cl)_2$	A	H	1R,3S	0.4	–
Bioresmethrin	$-CH=C(CH_3)_2$	B	–	1R,3R	2.8	0.0036
Cismethrin	$-CH=(CH_3)_2$	B	–	1R,3S	0.6	–
'Ethanochrysanthemate'		B	–	1R,3R	0.5	0.027
Kadethrin		B	–	1R,3S (E)	0.1	0.026
RU 25475	$-CHBrCBrCl_2$	A	C≡N	1R,3S (R+S)	0.2	–

DDT-ANALOGUE

R=4-ethoxyphenyl R^1, R^2= as above

Name	Structure	R^1	R^2			
GH149 (EDO)		–	–	–	no $^{22}Na^+$ influx	0.52

DDT-PYRETHROID TYPE

GH380		A	H	racemic	no $^{22}Na^+$ influx	0.19
GH401		A	H	S(−)	no $^{22}Na^+$ influx	0.022
GH414 Cycloprothrin		A	C≡N	racemic	no $^{22}Na^+$ influx	0.017
GH601		A	−C≡CH	racemic	no $^{22}Na^+$ influx	0.014

*$K_{0.5}$ are half-maximal values of concentrations of each compound, required to stimulate the influx of $^{22}Na^+$ into the C9 cell and NIE 115 neuroblastoma in the presence of 10 μmol of sea-anemone toxin II. All experiments where no influx was observed were tested to a concentration of 0.1 mM. All compounds were tested in duplicate. Where active, the experimental error was within 5% for each cell line. **Insecticidal activities are 48 h, mortality (LD_{50}) values in Australian sheep blowfly (*Lucilia cuprina* W.), obtained by topical application of each compound in acetone.

electrophysiologically active mammalian NIE 115 neuroblastoma cells.

While a separate toxicological classification was claimed[6] for the α-cyano-pyrethroids and those not containing this group, the pyrethroids in the table which induce the sodium ion influx belong to both classes of structures. In contrast, the combined DDT-pyrethroid – GH414 : which does not induce the influx contains the α-cyano group.

This selectivity of effects in the mammalian neuroblastoma also contrasts with the finding of Lund and Narahashi[7] who reported, that both the α-cyano pyrethroids and those not containing this group, including the compounds EDO and the combined DDT-pyrethroid insecticide GH401 (table), exert the same qualitative effect in the induction of the influx of Na^+ ion in the voltage clamped arthropod (crayfish) axon. All these active insecticides differed only at the quantitative level in their response, on the prolongation of the sodium tail current on step depolarization of this arthropod axon.

We conclude from the above evidence that there is a qualitative difference between the receptors in arthropods and mammals for the DDT-isostere EDO and the DDT-pyrethroid compounds, when they act in the Na^+ channel gating system. Such selectivity has been previously observed for a family of sea anemone toxins which are polypeptides and comprise toxins particularly specific against Na^+ channels in crustaceans, while other members of the family are more specific against Na^+ channels in mammalian tissue[8].

We have to assume that the combined DDT-pyrethroid insecticides, are either selective for the Na^+ channels of arthropod nerve membranes, or that their action is completely different from that of the pyrethroid class of insecticides. Considering their structural similarities to the pyrethroid class of compounds, we assume for the DDT-pyrethroid esters a lack of attachment to the gating system structure of the Na^+ channel in the mammalian nerve cell.

This finding also indicates that the observed[2] graded Na^+ influx induced by differing concentrations of the pyrethroid insecticides in the mammalian preparation does not reflect their insecticidal activity, but possibly indicates neurophysiological events which alone, or in combination with other more recently reported CNS effects[9], lead to the observed acute mammalian toxicity for some of the pyrethroids[10].

This contrasts with a lack of mammalian toxicity for EDO and the combined DDT-pyrethroid compounds listed in the table. EDO was tested without toxic effects to a dose of 2000 mg/kg. Compound GH380 and GH601 gave not toxic effects to a dose level of 16,000 mg/kg (i.p. in olive oil, albino mouse, 5 animals/dose, 7-day observation period)[11]. Moreover, the α-cyano-group containing insecticide GH414 to the tested limit dose of 5000 mg/kg, i.p. in the mouse (or same dose orally, rat), also gave no symptoms of acute toxicity.

These findings diminish the requirement to invoke a differential biochemical degradation to explain the decreased mammalian toxicity of the combined DDT-pyrethroid insecticides. The study also demonstrates that great care has to be exercised in the extrapolation of neurophysiological data from one species to another, in the search for detailed explanations of the mode of action of insecticides.

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0014-4754/85/040520-03$1.50 + 0.20/0

The siderophore mediated release of iron and magnesium from Mt St. Helens' ash and silicate rock standards[1]

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Summary. Substantial amounts of iron and magnesium were observed to be released from ash from Mt St. Helens' and US Geological Survey Silicate Rock Standards by the siderophore rhodotorulic acid.

Key words. Rhodotorula pilimanae; Mt St. Helens' ash; siderophore-mediated release; silicate rock standards, rhodotorulic acid.

The involvement of microbes in rock weathering has been of interest for some time. The classical concept of lichens as the pioneering organism has generated some literature support[3]. The most convincing evidence was the demonstration that compounds isolated from lichens could extract metal ions from rock samples[4]. Unfortunately, the biological function of these latter compounds is unknown. With the 1980 eruption of Mt St. Helens' there has been new interests in the events in the biological colonization of 'new' geological areas[5].

Because of the limited solubility of ferric salts in aerobic environments, one of the problems that a colonizer would encounter is an iron source. To circumvent this situation microbes excrete ferric specific chelators (siderophores) into their environment and then reabsorb the ferric-siderophore complex to obtain iron[6]. Siderophores have been observed in soils[7], are thought to be involved in plant nutrition and in the suppression of plant diseases[8–10], but no data exist on their involvement in the weathering of rocks.

In order to determine the role of siderophores in the release of ions from rocks, an aqueous solution of the siderophore rhodotorulic acid (RA) was shaken with various rock samples and the resulting supernatant was examined for metal ions.

Materials and methods. United States Geological Survey 200 mesh Silicate Rock standards (granite, G-2; granodiorite, GSP-1; andesite, AGV-1; peridotite, PCC-1 and basalt, BCR-1) were from Dr F.J. Flanagan[11]. Ash from Mt St. Helens was from Moscow, Idaho (18 May 1980, eruption) and Hillsboro, Oregon (12 June 1980, eruption). A sand sample from Camp