activity of PGE_2[16], which in turn might facilitate the immigration of PMN due to the leukotactic stimulus of PGE_1[17,18] and E_2[19].

To test this assumption, the degree of vascular permeability and the resulting accumulation of plasma constituents was assayed using PVP (^{131}J)[20]. As may be seen in Figure 2, a more than 10-fold increase in vascular permeability was observed in the non-drugged animals 2 h after urate injection, whilst in the colchicine-treated animals the maximal increase in vascular permeability amounted to only 3 times the control values and was observed not earlier than 3 h after urate injection. Correspondingly the accumulation of plasma constituents in the joint fluid was smaller and appeared later in the drugged than in the non-drugged animals. Nevertheless, in both groups PMN invasion followed PG-release and increased vascular permeability, whilst the PG's disappear when vascular permeability reaches its peak.

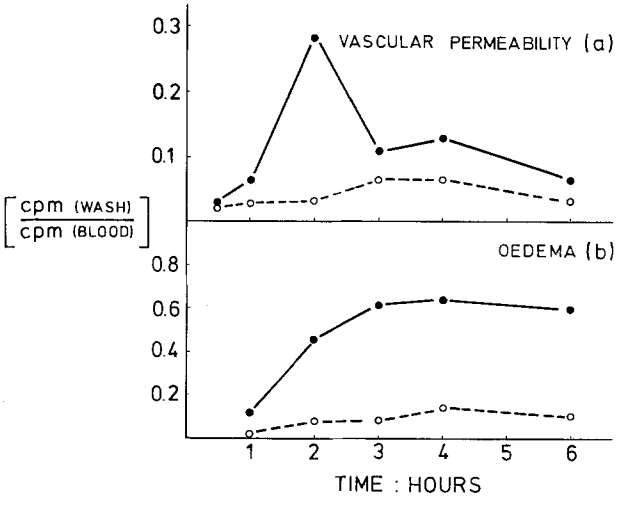

Fig. 2. Time course of changes in vascular permeability and oedema as measured with the PVP (^{131}J) method[20]. PVP (^{131}J) (rate constant of elimination, k_e in chicken = 0.1) was injected i.v. (10 µCi/kg) in chickens (2 kg weight) either 30 min before joint washes were performed or at zero time simultaneously with the intratarsal administration of urate crystals (details see Figure 1). At the times given, joint washes were performed with each animal and venous blood was obtained. The ratio cpm (per ml joint wash) to cpm (per ml blood) were taken as measure of the degree of vascular permeability during the preceding 30 min when PVP (^{131}J) was given 30 min before (a), or as a correlate of oedema when PVP (^{131}J) was given simultaneously with the urate crystals (b). The results are given for the urate injected joints of ● — ●, non-drugged and ○ --- ○, colchicine-treated animals (3 mg/kg, 3 h before urate injection). The ratios obtained from the saline-injected joints (not given in the figure) never exceeded 0.05. Each point in the figure represents a mean of 3 or more experiments.

In conclusion, it can be stated that PMN do not release significant amounts of PGE_2 or PGF_2 in the joint fluid in our model of acute inflammation. They could release these PG's while being trapped in the vessel wall before reaching the joint fluid. However, this appears unlikely because it implies that the PMN stop releasing PG's when they reach the joint fluid and start phagocytizing, a mechanism which is assumed to lead to cell breakage, enzyme release and PG's synthesis[5,7]. Instead we propose the thrombocytes as a more likely source of PG's in acute inflammation. These cells appear early enough in the joint fluid[9], they are well known to release PGE_2 and PGF_2[21] and in all experiments relating PG-synthesis in acute inflammation with PMN, thrombocytes (or platelets) were also present[4–7]. In addition it should be mentioned that the effect of colchicine on PMN invasion and on the vasculature response to inflammation is well compatible with the known impact fo this drug on microtubules[22,23].

Zusammenfassung. Bei einer akuten Entzündung wurde der zeitliche Verlauf der Prostaglandinfreisetzung, Granulozyteneinwanderung und Gefässpermeabilität verfolgt. Die höchsten Prostaglandinkonzentrationen wurden vor der Einwanderung von Granulozyten und vor einer massiven Steigerung der Gefässpermeabilität beobachtet. Dieser Befund spricht gegen die Behauptung, dass Granulozyten wesentlich an der Freisetzung von Prostaglandin beteiligt sind[24].

M. Glatt, B. Peskar and K. Brune[25]

Department of Pharmacology of the University, Biocenter, Klingelbergstrasse 70, CH-4056 Basel (Switzerland); and Department of Pharmacology of the University, Katharinenstrasse 29, D-78 Freiburg (Federal Republic of Germany), 29 August 1974.

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[25] Department of Pharmacology of the University, Katharinenstrasse 29, D-78 Freiburg (Fed. Rep. of Germany).

Ethylene as a Plant Hormone and Anaesthetic

Ethylene is now well established as a naturally occuring plant hormone[1]. Because the first effects of applied ethylene to be discovered were inhibitions of plant growth which disappeared on removal of the ethylene[2], it became generally known as a plant 'anaesthetic' and this led Luckhardt and Carter[3] to test and demonstrate its true anaesthetic effect on animals. The action of ethylene in plants occurs at much lower concentrations than in animals, for example, the stimulation of stem elongation in *Callitriche platycarpa* by ethylene is saturated at 1 nl ml^{-1} (ref. [4]) while the alveolar concentration of ethylene required for surgical levels in man is 67×10^4 nl ml^{-1} (ref. [5], though no measurement has been made of the concentration at its site of action). Also, ethylene is by

far the most active hydrocarbon in effecting plant growth, for example, it completely inhibits the elongation of etiolated pea stems at a concentration (1 nl ml^{-1}) at least 100 times lower than any related compounds[6].

However, we still know little about ethylene's mode of action in plants and how this is related to its effects on animal nervous tissue.

In animal systems, anaesthetics are thought to dissolve in the lipid phase of the cell membrane and cause it to expand[7,8]. This expansion can be antagonized by applying high pressure with the result that the physiological effects of the anaesthetic are reversed[9–12]. We have investigated whether hyperbaric pressures can reverse the stimulatory effect of ethylene on the growth of *Callitriche platycarpa*[4] and the inhibitory effect of ethylene on the elongation of etiolated pea plants[6] (*Pisum sativum* var. Meteor).

Table I. Effect of ethylene and pressure on *Callitriche* stem elongation

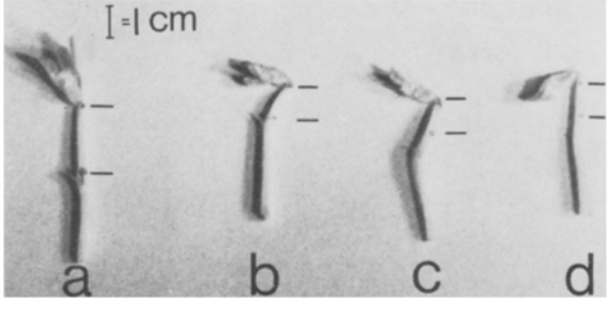
Treatment	Growth (% of control)
Control	100 ± 14
Ethylene (100 nl/ml^{-1})	190 ± 45
Pressure (120 atmospheres)	200 ± 40
Ethylene + pressure (as above)	180 ± 32

The data represent the mean of 3 separate experiments using 2–6 plants per treatment, ± 95% confidence limits. Control plants grew from 2 cm to approx. 3.5 cm in 24 h and the growth of treated plants is expressed as percent of control.

Table II. Effect of anaesthetics on *Callitriche* growth

Treatment	Growth (% of control)
Control	100 ± 34
Ethylene (100 nl/ml^{-1})	240 ± 27
Ethylene (12 PSI)	240 ± 49
Cyclopropane (15 PSI)	0
Cyclopropane (15 PSI) + pressure (80 atm.)	0
Nitrous oxide (80 PSI)	110 ± 20
Nitrous oxide (80 PSI) + pressure (90 atm.)	180 ± 45

Readings are from 1 experiment, 4–6 plants were used for each treatment. Data calculated as in Table I.



Effect of ethylene and pressure on elongation of etiolated pea stems. a) Control. b) Ethylene (100 nl ml^{-1}). c) Pressure (120 atmospheres). d) Ethylene + pressure (as above). Intact plants were treated for 24 h at 25 °C. Marked segments were 1 cm long at zero time.

Callitriche plants were cut to 2 cm stem length including the apical rosette and floated on half-strength Hoagland solution in 25 ml beakers. Seeds of *Pisum sativum* var. Meteor were planted in 25 ml beakers and grown in the dark for seven days at 25 °C. Beakers containing the plants were placed in 300 ml cylindrical stainless steel pressure vessels with perspex lids which were flushed with 100% O_2 for 1–2 min. The ethylene concentration was then raised to approx. 100 nl ml^{-1} and the pressure increased from ambient to various pressures up to 120 atmospheres with 99.9% helium (Air Products Ltd., Derby Road, Edmonton, U.K.). Control vessels contained beakers of 0.25 *M* mercuric perchlorate in 2.0 *M* perchloric acid to absorb ethylene and in the experiments with pea seedlings (carried out in the dark) KOH pellets were used to absorb CO_2. Experiments were run for approximately 24 h.

Table I gives the results of the application of 120 atmospheres pressure and 100 nl ml^{-1} of ethylene to *Callitriche*. The high pressure did not visibly damage the plants, and did not reverse the stimulatory effect of ethylene on stem elongation. Further experiments using 17, 33, and 67 atmospheres in combination with 100 nl ml^{-1} ethylene also showed no reversal of the ethylene effect. Similarly application of 120 atmospheres did not prevent the inhibition of etiolated pea stem growth by 100 nl ml^{-1} of ethylene (Figure).

It is evident from Table I and the Figure that 120 atmospheres of pressure alone had the same effect on *Callitriche* and peas as ethylene. It seems unlikely that pressure alone would cause opposite effects (stimulatory and inhibitory respectively) in the two systems, and we think this effect is due either to contamination of the helium with ethylene (about 0.01 nl ml^{-1} would be sufficient), or to a build-up of endogenous ethylene within the tissue caused by the reduced diffusion rate away from the plant surface in a compressed atmosphere[4]. Pressurizing plants may well stimulate endogenous ethylene production as even handling pea plants can induce temporary responses caused by 'wound' ethylene[13].

Since there was no pressure reversal using ethylene, we tried to 'anaesthetize' *Callitriche* plants with a variety of other anaesthetics (using concentrations that block peripheral nerve fibres) to see if their effects, if any, could be altered by pressures that reverse their action on nervous tissue (Table II). Of the anaesthetics that were not evidently toxic, cyclopropane (15 lb $inch^{-2}$) inhibited growth with no visible damage, but the inhibition was not

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reversed by pressure. Ethylene at 12 lb inch^{-2} (80×10^4 nl ml^{-1}) stimulated growth just as 100 nl ml^{-1}. Nitrous oxide at 80 lb inch^{-2} (ref. [14]) was without effect and the application of 90 atmospheres of pressure merely caused the usual growth stimulation.

In conclusion it seems that growth éffects caused in plants by ethylene are the result of a different mode of action from that involved in animal anaesthesia. If ethylene acts upon membranes in plants, it seems more likely that it interacts with protein receptors[15,16] rather than with the whole lipid phase of the membrane[16,17].

Résumé. Nous avons tenté d'inverser les effets de l'éthylène sur la croissance des plantes par une pression accrue, ainsi qu'il est possible de le faire lorsque l'éthylène agit comme anesthésique chez les animaux. Nous n'avons découvert aucun renversement, ce qui implique une différence fondamentale entre la mode d'action de l'éthylène chez les animaux et chez les plantes.

R. F. Irvine[18], S. H. Roth[19] and A. Musgrave[20]

Agricultural Research Council, Unit of Developmental Botany, 181A Huntingdon Road, Cambridge CB3 ODY (England); and Department of Pharmacology, South Parks Road, Oxford (England), 27 May 1974.

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[19] Present address: Division of Pharmacology and Therapeutics, Faculty of Medicine, The University of Calgary, Calgary, Alberta T2N 1N4, (Canada).
[20] Present address. Laboratorium voor Plantenfysiologie, Ijdijk 26, Amsterdam-Oost (The Netherlands).

Dopamine and Noradrenaline in the Salivary Glands and Brain of the Tick, *Boophilus microplus*: Effect of Reserpine

Salivary secretion has been recognized as the major route of water excretion in the female cattle tick, *Boophilus microplus*[1]. The mechanism by which salivary secretion is controlled is therefore of some interest to those concerned with the design of acaricides. Recent studies have revealed that the salivary glands of ixodid ticks are innervated by nerves containing dense core granules[2]. It is being increasingly realized that, although cholinergic agonists such as pilocarpine cause salivary secretion in ticks[1] and insects[3], these agents act indirectly[3–5]. It has, for example, been shown that while a number of organophosphorus insecticides cause salivary secretion in *B. microplus*, secretion can be blocked by pre-treatment with the catecholamine-depleting drug reserpine[4]. Adrenergic agonists are also known to stimulate tick salivary gland secretion both in vivo and in vitro[4,5]. There is now good evidence that dopamine is the transmitter at a number of insect salivary glands[3,6–9]. It was therefore of interest to ascertain whether a catecholamine was present in the salivary glands of the tick as well.

Fed ticks (*Boophilus microplus Canestrini*) were obtained from the Wellcome Research Laboratories, Beckhamstead, England. Reserpinized ticks received 10 µl of physiological solution containing 10 mg/ml reserpine (Sigma Chemical Co.). Controls received physiological solution only. After 14 h, salivary glands and brains were dissected out, weighed and frozen on dry ice. Tissue from at least 7 ticks was required for each assay. The frozen tissues were then assayed for dopamine and noradrenaline by an enzymatic-radiochemical technique[10] in which bovine liver catechol-*o*-methyltransferase is used to catalyse ^{3}H-methyl transfer from ^{3}H-S-adenosylmethionine to dopamine and noradrenaline. The 3-methoxytyramine and normetanephrine formed are separated from the reaction mixture by organic extraction and from each other by paper chromatography. ^{3}H-3-methoxytyramine and ^{3}H-normetanephrine are eluted from the chromatogram and the radioactivity determined by liquid scintillation counting. Dopamine and noradrenaline are quantitated by comparison with standards carried through the above procedure. The results for tick salivary glands and brain are summarized in the Table.

Salivary glands and brain contain both dopamine and noradrenaline. Dopamine is the major catecholamine in the salivary glands of the tick as in other invertebrates,

Dopamine and noradrenaline in the salivary glands and brain of the tick. Effect of reserpine

	Salivary glands		
	Control	Reserpinized	Decrease (%)
Dopamine	0.74 ± 0.04 (5)[a]	0.18 (4)[b]	76[c]
Noradrenaline	0.45 ± 0.11 (3)	0.07 ± 0.00 (3)	85[c]
	Brain		
	Control	Reserpinized	Decrease (%)
Dopamine	0.32 ± 0.05 (4)	0.21 ± 0.11 (3)	34 N.S.
Noradrenaline	0.36 ± 0.21 (4)	0.22 ± 0.04 (3)	39 N.S.

[a] Values shown are means (µg/g ± S.E.M., number of determinations in brackets) of determinations on salivary glands from at least 7 ticks. [b] 2 of the 4 determinations were below the detection level of the assay (0.11 µg/g). [c] $p < 0.05$; N.S., not significant.

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