(4) In fish No. 4 the main branch of the ramus lateralis vagi was cut anteriorly on both sides; the ventral branch of this nerve, however, was cut only at the left side. So, of all trunk lateralis sense organs, only the small pit organs at the ventral part of the right side were still innervated. A number of clear and oriented reactions were observed on stimulation of the tail region at the right side, though only with relatively strong currents. In a few cases a 'reversed' response occurred on stimulation of the left side; again (see above) intact electroreceptors of the right side were evidently involved.

The following conclusions can be drawn: (a) From 1 and 2: the external taste buds are not at all involved in electroreception. (b) From 2: electroreception resides in lateral sense organs. (c) From 3 and 4: the small pit organs are electroreceptors.

The experiments are to be continued.

Zusammenfassung. Zwergwelse (*Amiurus nebulosus*) sind elektrisch sehr empfindlich. Geblendete Tiere sprechen, je nach der Stromstärke, auf schwache Ströme ohne Dressur mit biologisch bedeutsamen Reaktionen an: Ausweichen oder Annähern und gegebenenfalls Schnappen. Auf Grund einseitiger Denervierungsversuche konnte festgestellt werden, dass die sehr zahlreichen äusseren Geschmacksknospen mit dem Empfang schwachelektrischer Reize nichts zu tun haben. Als Elektrorezeptoren erweisen sich vielmehr die anatomisch zur Gruppe der ampullären Seitensinnesorgane gehörigen, ebenfalls in weiter Verbreitung am Körper vorkommenden «small pit organs».

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Protection against Hyperbaric Oxygen Toxicity after Feeding *N,N*-diphenyl-*p*-phenylene diamine

JAMIESON and VAN DEN BRENK[1] have shown that intraperitoneal injections of *N,N*-diphenyl-*p*-phenylene diamine (DPPD) protects rats and Walker and Eliza strain mice against toxic effects of later exposure to 100% oxygen at 5 atmospheres. These effects included convulsions, lung damage and eventual death. We have obtained similar results in C_3H mice after feeding DPPD. The degree of protection is linked to the number of feedings and presumably to tissue accumulation of DPPD.

Methods. A 2% solution of DPPD[2] in sesame oil was fed by stomach tube to groups of adult C_3H mice (some obtained from Jackson Memorial Laboratory, Bar Harbor, Maine and some from Cumberland View Farms, Cumberland, Virginia). Dose was adjusted to body weight: in the first 2 experiments a total of 0.56 g of DPPD/kg mouse was divided evenly among 7 daily feedings. Previous experience showed this to be near the lethal limit; 8 feedings (0.64 g/kg) usually kills a large percentage.

In subsequent experiments 6, 4, and 3 daily feedings of the same solution were used. Control animals were fed sesame oil in the same proportion to weight as animals receiving DPPD in sesame oil.

In each experiment 5 DPPD and 5 sesame oil fed mice were used. They were placed in a high-pressure chamber[3] on opposite sides of a dividing partition that permitted free passage of gas but not mingling of mice. The chamber was vented with 100% oxygen at 5 l/min until a pure oxygen atmosphere was attained to the end of each experiment. The oxygen concentration was determined by sampling and testing vented gas with a PO_2 electrode[4]. Pure oxygen was compressed at 1 atmosphere/min to 60 p.s.i.g. (5 atm. abs.) and maintained to the end of the experiment.

The time in oxygen at 5 atmospheres at which the mice died was recorded: the sign used was complete cessation of respiratory movements.

Dead animals were dissected, the lungs grossly and microscopically examined.

Results. Within 2–5 min after arriving at 5 atmospheres, distress, followed by full tetanic seizures, was observed in all animals regardless of treatment. Such seizures lasted approximately 15 sec and the animals appeared to fully recover. Within 5–10 min seizures reappeared. They recurred thereafter with increasing frequency alternating with ever-shortening recovery periods. Finally, after 45 min to 1 h, the mice lay on their sides, breathing with gasping movements, having occasional seizures but little organized voluntary movement until death. The Table gives the survival data.

An analysis of the variance in survival time between DPPD and sesame oil fed animals shows highly significant differences ($P < 0.001$ in all cases except 3 feedings where $P < 0.01$ but > 0.001).

The difference between the 2 experiments with 7 feedings was not significant.

At autopsy, the lungs of treated animals had fewer hemorrhagic and collapsed areas than those of untreated animals. Histological sections bore out this observation.

No. of feedings of DPPD	No. of animals	Mean survival time (min)	Range of survival time (min)	Protection factor
7	5	661.4	319–950	4.17
0[a]	5	158.6	148–170	
7	5	723.4	574–962	4.50
0[a]	5	161.6	149–169	
6	5	561.8	470–641	3.17
0[a]	5	177.5	160–192	
4	5	424.6	368–478	2.26
0[a]	5	187.4	168–203	
3	5	376.6	266–587	2.06
0[a]	5	167.8	147–180	

[a] These received sesame oil in the same number of feedings as DPPD above.

[1] D. JAMIESON and H. A. S. VAN DEN BRENK, Biochem. Pharmac. *13*, 159 (1964).

[2] Obtained through the courtesy of the U.S. Rubber Company.

[3] Bethlehem Corporation, Table Top Hyperbaric Chamber Model No. 614, Bethlehem, Pennsylvania.

[4] Instrumentation Laboratory Inc. II-113-51 Ultra Micro PO_2 electrode.

Conclusions. DPPD protects when given by mouth against oxygen toxicity. The degree of protection varies directly with the number of applications, indicating that DPPD is accumulated in tissue.

Since the same central nervous system response to hyperbaric oxygen was seen in all mice it does not seem to be involved in the mechanism of DPPD protection. The mechanism is not known, but contrary to the results of JAMIESON and VAN DEN BRENK, the lungs were less damaged in treated animals.

These data, save for the exception noted, substantially confirm those of JAMIESON and VAN DEN BRENK. There is, however, a great strain difference in the tolerance of mice to hyperbaric oxygen. Untreated C_3H mice survived the same pressure much longer than the strain they used.

Some preliminary experiments in which untreated and DPPD fed mice were returned to air at atmospheric pressure after varying intervals in hyperbaric oxygen show that DPPD greatly extends the time they can spend in hyperbaric oxygen without obvious damage[5].

Résumé. Des souris adultes traités au préalable par du *N,N*-diphenyl-*p*-phénylène diamine sont protégés contre la toxicité du oxygène hyperbarié. La protection est dépendente du dosage. Le méchanisme est inconnu.

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[5] Supported by a grant from the U.S. Rubber Company.

Cytokinin and Gibberellin-Like Activity in the Spring Sap of Trees

The hormonal control of the physiological processes by which deciduous trees begin active growth in spring has been the subject of much speculation and investigation[1–3]. Of direct interest to the present work is the demonstration by NANDA and PUROHIT[4] that gibberellic acid will initiate the release of sugars from starch stored in twigs. As measured by the increase in bud burst (dormancy release), applied gibberellic acid will also overcome the effect of a substance causing dormancy in birch[5]. EAGLES and WAREING[5] also observed in *Acer pseudoplatanus* 'significant changes in the levels of endogenous gibberellins during December–April'. Recently SKENE[6] has demonstrated gibberellin activity in the bleeding sap collected from *Vitis vinifera* prior to bud burst.

Kinin-like compounds, together with gibberellins, may also be implicated in bud burst since benzyladenine will break dormancy in *V. vinifera*[7] and in unchilled seeds of a number of species of deciduous trees[8]. Application of kinetin to lateral buds of peas enables them to overcome apical dominance[9], although auxin must be added to the bud if its growth rate is to be of the same magnitude as that of an uninhibited lateral bud[10]. The bleeding sap of *Acer saccharum* was shown by NITSCH and NITSCH[11] to contain substances with cytokinin-like activity shortly before bud burst. Thus in early spring supplies of gibberellins and cytokins may become available to buds either by release or by synthesis within the buds themselves, or as imports, together with nutrients, in the ascending sap. We have already shown that the xylem sap of a number of herbaceous plants contains both kinin-like[12] and gibberellin-like[13] substances (see also[14]).

This communication deals with an examination of the kinin and gibberellin activities of the spring sap of 2 tree species.

Xylem sap was collected from sycamore (*A. pseudoplatanus*) and birch (*Betula pubescens*) in March 1966, immediately prior to bud burst. Sap used for gibberellin determination was obtained by sawing down a 6 m high sycamore tree and collecting the sap bleeding from the remaining stump (30 cm high) and root system. Sap for cytokinin determination was collected by boring a hole (30 cm above ground level) through the bark into the xylem of birch and sycamore trees, and fitting a piece of polythene tube into the hole. In both cases the sap was collected for 12 h, immediately frozen and kept at − 20 °C until required.

Gibberellin determination. After thawing, 100 ml of sap was processed as described elsewhere[15]. The final eluants, from the thin layer chromatograms of the 2 fractions (acidic, and basic + neutral) were assayed for gibberellin content using the barley endosperm assay[16,17], and the Meteor pea bioassay[15]. The results obtained from the fraction extracted at pH 7.5 are shown in Figure 1. Only this fraction showed any significant ($P < 0.05$) amounts of gibberellin-like substances. While the barley endosperm assay showed one zone of activity, Rf 0.3–0.6, 2 peaks (Rf 0.2–0.3 and 0.5–0.6) were obtained using the Meteor

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