insect prefers objects of a given angular size, motion parallax may be one mechanism to determine distance and absolute size of the object.

Although, in our experiments on the walking fly, a highly significant effect of pattern distance could be proved, that effect is not necessarily to be explained by binocular or monocular mechanisms of distance measuring. According to previous results, the spontaneous preference of *Drosophila* flies towards vertically inclined stripes is due to the fact that vertical stripes facilitate any mechanism of locomotor course control[12]. That explanation coincides with the experimental data that the preference for vertical stripes fades when the wavelength approaches the resolution threshold at $\lambda = 2\ \Delta\psi$ ($\Delta\psi$ = divergence angle of neighbouring visual units), and that it is even reversed in the range of $\Delta\psi < \lambda < 2\ \Delta\psi$ (preference for horizontal stripes). The recent results support the hypothesis that this mechanism of visual course control also depends on the distance of the vertical stripe pattern with regard to the fly's position (provided that λ is identical in all cases): the smaller that distance becomes, the more the capacity of maintaining a straight course is lost. One difference caused by near and far stripes which coincide in angular dimensions consists in the different angular velocities of the vertical images, when the fly approaches the stripes (motion parallax). The velocity of image motion increases with decreasing distance between walking fly and stripe pattern. According to some cinematographic measurements, the de-winged flies are walking by a speed of about 25 mm·sec^{-1} [15]. By this one can calculate the angular velocity of a contrast line moving laterally over the fly's eyes when the fly is approaching the center of a vertical stripe. When the fly is crossing the circle C1, these angular velocities are 0.5° sec^{-1} and 2.3° sec^{-1} in the large and small drum, respectively. The mechanism of locomotor course control, and thus the preference for vertical stripes, may well depend on those differences in the velocity of image motion and may have nothing at all to do with distance measuring[18].

Zusammenfassung. Fliegen bevorzugen sehwinkelkonstante Vertikal- gegenüber Horizontalstreifen bei grossen, nicht dagegen bei kleinen Musterentfernungen. Diese entfernungsabhängige Vertikalstreifenpräferenz wird als Folge eines Mechanismus zur optischen Kurskontrolle verstanden, der von Art und Geschwindigkeit der retinalen Bildverschiebung abhängt.

R. Wehner and E. Horn

Zoologisches Institut der Universität, Künstlergasse 16, CH-8006 Zürich (Switzerland), and Zoologisches Institut der Universität, Lehrstuhl II, Kaiserstrasse 12, D-75 Karlsruhe (German Federal Republic, BRD), 20 January 1975.

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Lysis of Zoospores of *Phytophthora palmivora* induced by Concanavalin A

Major changes occur on the surface of zoospores of *Phytophthora palmivora* undergoing encystment. Within 2 min, the wall-less zoospore secretes an amorphous cyst coat and elaborates de novo a microfibrillar cell wall made of β-glucans[1,2]. The unencysted zoospore binds concanavalin A (Con A) on its plasmalemma. This surface binding capacity increases greatly during encystment as new Con A receptor sites appear on the cell surface[3]. These new sites correspond to the amorphous coat material secreted by the so-called peripheral vesicles[4]. We believe that this Con A binding material is involved in cell adhesion and cyst wall formation.

In the course of studying the binding of Con A to zoospores, we noted a lethal action. Motile zoospores incubated with Con A at concentrations as low as, 25–50 µg/ml ceased swimming, agglutinated weakly and began lysing within a few minutes. By 30 min, essentially all zoospores had burst (Table I). The lytic action of Con A could be completely nullified by methyl-α-D-mannoside (0.166 *M*) if the mannoside was added within 3 min after exposing the motile zoospores to Con A (200 µg/ml). If the addition was made after 5 min, about half of the zoospores lysed while the rest encysted normally. After a 10 min exposure to Con A, the entire population lysed.

Annulment of the lytic effect of Con A by methyl-α-D-mannoside indicated that Con A was operating as a lectin[5]. It could be argued that methyl-α-D-mannoside prevented lysis by affording osmotic protection to the cells. However, this was ruled out because mannitol (0.166 *M*) added in lieu of methyl-α-D-mannoside did not prevent Con A-induced lysis. At 0.32 *M* mannitol, the zoospores also lysed by the action of Con A, but many of the cells did not burst; they became irregular in shape, showed a granulated cytoplasmic appearance, and failed to germinate. In the presence of 0.32 *M* mannitol alone, the cells germinated normally.

In sharp contrast to zoospores, cysts (prepared by vigorous agitation of a zoospore suspension[1]) were not susceptible to Con A. They germinated normally in the

Table I. Effect of concanavalin A on zoospores of *P. palmivora*

Concanavalin A (µg/ml)	Concanavalin A	Concanavalin A plus methyl-α-D-mannoside
200	++++	−
100	++++	−
50	++++	−
25	+	−
12.5	−	−

(++++) = total lysis, (−) = no lysis. The zoospore suspension was incubated at room temperature and scored for lysis after 45–60 min under a light microscope. The incubation mixture contained: 0.7 ml motile zoospore suspension (2×10^6 cells/ml), 0.2 ml Con A at the appropriate concn in 0.05 *M* NaCl and either 0.1 ml of 4 m*M* sodium phosphate buffer, pH 7.2, in 0.05 *M* NaCl, or 0.1 ml of 0.8 *M* methyl-α-D-mannoside in the same buffer/salt.

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Table II. Incorporation of UDP-^{14}C-glucose into alkali-insoluble glucan by cell free extracts of *P. palmivora* zoospores

Cell fractions	Con A	Con A + Methyl-α-D-mannoside	Methyl-α-D-mannoside	No addition
1000 × *g* Pellet	3.38	4.09	4.0	3.41
10,000 × *g* Pellet	5.11	6.69	7.57	7.49
100,000 × *g* Pellet	4.62	5.35	5.38	4.33

The values are percent of radioactivity incorporated into alkali-insoluble glucan. The reaction mixture (final volume 0.6 ml) consisted of 0.29 μmoles of cellobiose, 3.4 μmoles of UDP-^{14}C-glucose (250,000 dpm), 10.8 μmoles $MgCl_2$, 0.083 *M tris*-HCl buffer, pH 7.5, and 0.1 ml particulate enzyme. Con A (200 μg in 0.41 *M* NaCl) and/or 0.16 *M* methyl-α-D-mannoside was added to the appropriate samples. The cell fractions were prepared as described elsewhere[6].

presence of Con A concentrations that caused total lysis of unencysted cells. It seems unlikely that the cyst wall protected the cell simply by blocking the entry of Con A since it can penetrate through the wall and bind onto the cyst plasmalemma[3].

Since Con A binds intensely to the amorphous material secreted by encysting zoospores, the possibility was considered that Con A provoked zoospore lysis by interfering with the process of cyst wall formation. However, Con A did not markedly inhibit the activity of wall-glucan synthetases in a cell free system (Table II). In this cell free system, glucose residues are joined by β, 1–3 and β, 1–6 but not by β-1, 4 linkages[6,7], hence, the possibility that Con A might block specifically, cellulose synthesis could not be excluded. Alternately, the primary effect may be on the zoospore plasmalemma where Con A may disrupt its function. For instance, it may adversely affect the discharge of peripheral vesicles and thus selectively upset the process of cell wall neogenesis of the zoospore without affecting wall formation in subsequent developmental stages.

Con A can sometimes be toxic[8] but, to our knowledge, the drastic lethal action recorded herein has not been formerly described. The lysis of zoospores by Con A invites questions on the occurrence of this phenomenon particularly during host/parasite interactions, and raises the speculation, that it might be another way in which lectins contribute to defend higher plants against pathogens[9].

Resumen. La concanavalina A causa la lísis total de las zoosporas de *Phytophthora palmivora*. Una vez enquistadas, las células se vuelven resistentes a esta lectina. Aparentemente, la concanavalina A interfiere especificamente con el proceso de neogénesis de pared celular.

V. O. SING and S. BARTNICKI-GARCIA[10]

Department of Plant Pathology, University of California, Riverside (California 92502, USA), 3 February 1975.

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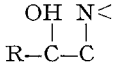
The Proposal of an Unified Model for the Interpretation of the Activity of Different Classes of β-Adrenergic Agents

The β-adrenergic agonists and antagonists can be divided[1] in two types, **A** and **B**. Although the structure-activity relationship of these drugs has been extensively studied, there is no satisfactory explanation for the way in which the Ar–O–CH_2 moiety of type **B** compounds can replace the single aromatic nucleus of type **A** agents in the drug-receptor interaction[2]. Therefore, we have initiated



A: R = Aryl; **B**: R = Aryl–O–CH_2

an investigation of the X-ray crystal structures of representative compounds of type **B**, and compared these three-dimensional structural data with similar kinds of data from type **A** compounds. The crystal structures of two type **B** β-blockers (propranolol [**2**] and alprenolol [**3**]) have been reported[3], and we have recently determined the structures of propranolol, propranolol hydrochloride and dichloroisoproterenol, a type **A** β-blocker.

The torsion angles about the C_1–C_2 bond are similar in both **2** and **2**·HCl, and correspond to a conformation in which nitrogen is approximately *anti*-periplanar to the

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