The cells exist in closed contact and not in a loose cluster. Therefore, the structure seems to be comparable with the globular stage of embryoid formation in *Nicotiana*[5]. The outer shape may have been formed, as in Fig. 1, by bursting out of the exine. Mitosis was not observed if the nutrients were replaced by those recommended by Bourgin and Nitsch[6], Murashige and Skoog[7] and Nagata and Takebe[8].

Trials with isolated microspores or pollen grains may lead to the discovery of the general conditions for the induction of androgenesis in angiosperms, independent from the obscure influence of the anther wall. Moreover, a higher yield of regenerating microspores in suspension cultures would make possible the application of biochemical methods for selection of auxotrophic mutants.

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Increased Vascular Resistance by Prostaglandins B_1 and B_2 in the Isolated Rat Pancreas

In view of the possibility that prostaglandins (PG) regulate local blood flow[1], we are investigating this activity in the pancreas. We have already found that PGE_2 reduces vascular resistance in the perfused rat pancreas whereas $PGF_{2\alpha}$ has the opposite effect[2]. These effects were seen at low doses (0.1 μg/ml.) and with good reproducibility.

PGB_1 has been reported[3] to have considerably less biological activity than prostaglandins of the E or A series, for example, PGB_1 has less than 5% of the activity of PGE_1 or PGA_1 on blood pressure or smooth muscle contraction[4]. A thorough examination of the biological activity of PGB_2 has not been reported. The PGB_1 and PGB_2 which we have used had virtually no effect on smooth muscle and less than 10% of the effect of PGA_2 on acute blood pressure lowering in the intact rat (J. Sanner and L. Rozek, personal communication), but we found that the PGB_1 and PGB_2 increased the vascular resistance to perfusion in the isolated pancreas.

The pancreas was surgically isolated from rats (200–250 g) previously fasted for 18 h. An arterial cannula was inserted into the coeliac axis with all adjoining arteries ligatured except for the pancreatico-duodenal artery, and an exit cannula was inserted into the portal vein[5]. The perfusion medium, similar to Krebs bicarbonate, was oxygenated, adjusted to *p*H 7.4 and perfused through the pancreas at a constant rate. A stopcock placed before the pump was used to switch from control medium to one containing the required drug. Changes in perfusion pressure indicated the vascular resistance of the organ.

Table 1 Influence of Phentolamine on the Pressor Response of Noradrenaline and PGB_2 in Isolated Rat Pancreas

Drug	Conc.	Obs.	Change from control pressure mm Hg ± s.e. Without phentolamine	With 1 μg/ml. phentolamine	% Inhibition
Noradrenaline	2 μg/ml.	4	+65±19	0	100
PGB_2	2 μg/ml.	4	+48±16	+27±4	56

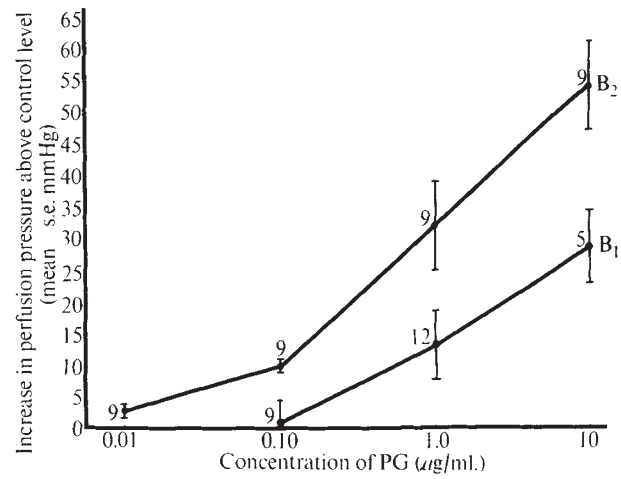

Fig. 1 Changes in pre-organ perfusion pressure expressed as the mean increase (mm Hg ± s.e.m.) produced by PGB_1 and PGB_2 in doses of 0.01 μg/ml. to 10 μg/ml. Control perfusion values were assigned a reference value of 0. The responses are the peak pressure change occurring during a two minute infusion of prostaglandins into an isolated rat pancreas.

PGB_1 and PGB_2 increased vascular resistance to perfusion (Fig. 1) in the isolated pancreas. The response was related to dose and linear between 0.1 μg/ml. and 10 μg/ml. The response to PGB_2 was of the same order of magnitude as that observed with adrenaline and noradrenaline which, however, had a much steeper dose-response curve. The weak activity of PGB_1 and PGB_2 on systemic blood pressure suggested that these agents might act indirectly by causing the release of a vasoconstrictor from the pancreas. The release of noradrenaline by PGB_2 was tested by comparing the perfusion pressure change resulting from PGB_2 and exogenous noradrenaline when phentolamine, an α-adrenergic blocking agent, was added to the perfusing medium. Phentolamine (1 μg/ml.) blocked the increase in pressure caused by 2 μg/ml. of noradrenaline but only halved the pressor effect of 2 μg/ml. of PGB_2 (Table 1). The differential effect of these two pressor compounds in the presence of an α-adrenergic blocking agent suggests that at least some of the pressor activity of PGB_2 does not depend on the release of stored noradrenaline.

In view of the low biological activity of PGB_1 and PGB_2 in other tissues, this effect in the pancreas is of great interest. The increased vascular resistance observed in the isolated pancreas suggests that a reduction in pancreatic blood flow could occur *in vivo* with these agents. The effect of exogenous PGB_1 and PGB_2 on the pancreas may be minimal in the intact animal because of the rapid catabolism of these compounds. It would be interesting, however, to determine whether either of these agents are synthesized within the pancreas and, if so, whether they are important local vasoconstrictor agents.

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