Commentary

'Mechanisms of auxin-induced plant cell elongation': a reply to the commentary by H. Göring

Benno Brummer and Roger W. Parish

Cytology, Plant Biology Institute, University of Zürich, Zollikerstrasse 107, CH-8008 Zürich, Switzerland

Received 3 June 1985

In May 1983, we submitted a hypothesis on the mechanisms of auxin-induced plant cell elongation to FEBS Letters. This appeared in September 1983 [1] and in a commentary on this paper (November 1984, [2]) H. Göring wrote: "It can be concluded that the ideas published in the paper of Brummer and Parish and presented as a new hypothesis were published in earlier papers" (by Göring himself) "some of them in easily available journals". Göring presented a list of six references to substantiate his claim [3–8].

These are strong words and we feel obliged to answer them.

Firstly, Göring implies that we wilfully or through negligence failed to cite his work. In fact only two of the references were available to us [3,4], although we had not read them. This underlines a weakness of the system to which we are all prone. We formulated our hypothesis in 1981–82, using a set of core references relevant to our ideas. This core assisted us in selecting earlier papers. It was never our aim to review the field. [3] was only cited once up to the end of 1984 (in 1983) and [4] was cited 7-times, 5-times in 1979–82, two of these citations in journals unknown to us. (Information from the Science Citation Index.) The literature dealing with auxins is enormous and, when citation is so low, omissions are almost impossible to rectify. It is doubtful that we would have quoted [3] for reasons mentioned below. [4] would have been mentioned because it surprisingly reports "in most cases" a continuing membrane depolarization of coleoptile cells following indole-3-acetic acid (IAA) treatment. This would have contradicted part of our hypothesis (see below). It is grossly unfair to have expected us to have read the book article [5], since it contains proceedings of a meeting and was published in the year we were writing our paper. It was not available to us and, obviously, neither were the 3 remaining references (even if the summaries of the Russian papers *were* in English, as Göring points out) [6–8].

Secondly, how true is Göring's claim that he had already published the ideas we presented?

We questioned the wall acidification theory of auxin-induced elongation growth [9], discussing the inconsistencies of this hypothesis. We postulated that a central effect of auxin is cytosolic acidification, possibly via an increase in levels of cytosolic Ca^{2+} or release of vacuolar protons. Activation of the electrogenic proton pump would lead to changes in membrane potential (hyperpolarization) and transmembrane ion gradients, the latter serving as a second messenger. We argued that these changes rather than wall acidification are involved in elongation growth.

We shall attempt to summarize the ideas published by Göring. He suggested that within minutes IAA "inhibits consumption in glycolysis", thereby decreasing ATP levels and causing membrane *depolarization* [3,6]. Subsequently, "only after change-over glycolysis via PEP-carboxylase reaction... energy metabolism is promoted again", because the ATP level now rises. "H^+ secretion is stimulated and, in

Published by Elsevier Science Publishers B.V. (Biomedical Division)
00145793/85/$3.30

dependence on other conditions, a partial or complete repolarization of membrane potential and even hyperpolarization will occur" [3]. However, Göring writes in a later paper (in German) that IAA-induced growth was independent of the degree of repolarization in this second phase [6]. (He had previously reported a continuing depolarization "in most cases" [4].)

We fail to see similarities to our hypothesis. On the contrary, the activation of the proton pump resulting from cytosolic acidification would cause membrane *hyperpolarization*. The *initial* depolarization measured in some laboratories following auxin application is not specific for IAA. It is also caused by low concentrations of benzoic and butyric acids [10]. However, the latter does not induce subsequent hyperpolarization or growth and hyperpolarization appears to be a "specific auxin effect" [10].

Göring further states that "at higher IAA concentrations th changes in metabolism would be greater. The result would be an acidification of the cytoplasm and an induction of ethylene production" [3]. The impression is made that cytosolic acidification is related to ethylene production but not to elongation growth. The summary of [3] states: "...the hypothesis is deduced that both high IAA concentrations and water stress reduce cytoplasmic pH... this acidification of cytoplasm is claimed to be responsible for the stimulation of ethylene production". This is also stated in [6].

Only in the book article [5] does Göring at last write: "It could be...supposed that auxin directly or indirectly affects metabolism and produces a trend towards acidification of the cytoplasm. Then auxin-induced H^+ extrusion should be considered only a secondary result and a component of pHc regulation". How this fits in with the original hypothesis is not discussed.

Hence, Göring first suggested a relationship between cytosolic acidification and growth in 1982, but we could hardly have been expected to know this. (The article has been cited once, in 1984.)

We should point out that, if auxin is stimulating the H^+ pump, its two most likely modes of action are to increase the efficiency of the pump (e.g. by configuration changes) or to provide more substrate (cytosolic acidification) [11]. Certainly, neither Göring nor ourselves were the first to think of this. (We also postulated a role for cytosolic Ca^{2+} in cytosolic acidification. Hopefully not all those who have suggested hormones raise cytosolic Ca^{2+} levels will attack us for stealing their ideas.)

Our hypothesis postulated a sequence of events of which cytosolic acidification is only one. In particular, we wished to implicate changes in ψ_m and transmembrane ion gradients in growth, almost a third of the paper discussing evidence from various systems. These ideas were presented as an alternative to the wall-acidification theory, a theory which Göring has not questioned in his work.

Changes of cytosolic pH are of immediate interest because they can now be measured. We have recently shown that the degree of growth stimulation by weak acids is positively correlated with the extent of their cytosolic acidification and stimulation of the proton pump [12]. We suggested that acids induce growth by acidifying the cytosol and stimulating the proton pump rather than via direct acidification of the wall. This has now also been shown by others [13]. Under conditions where the carboxylic ionophore monensin transports protons from the cytoplasm to the wall, no growth was induced, presumably because no pump stimulation (or cytosolic acidification) occurred [14]. Finally, the fungal toxin fusicoccin, which induces elongation in a variety of tissues, rapidly lowers the cytosolic pH of root cells [15] and coleoptiles [13]. Preliminary results indicate IAA also lowers the cytosolic pH of coleoptile cells [15]. Evidence against the wall acidification theory of auxin-induced growth has also recently been presented by Kutschera and Schopfer [16].

REFERENCES

[1] Brummer, B. and Parish, R.W. (1983) FEBS Lett. 161, 9–13.
[2] Göring, H. (1984) FEBS Lett. 177, 1.
[3] Göring, H. and Zoglauer, K. (1979) Biochem. Physiol. Pflanzen 174, 568–578.
[4] Göring, H., Polevoy, V.V., Stahlberg, R. and Stumpe, G. (1979) Plant Cell Physiol. 20, 649–656.
[5] Göring, H. and Bleiss, W. (1982) in: Plasmalemma and Tonoplast: Their Function in the Plant Cell (Marmé, D. et al. eds) pp.263–269, Elsevier, Amsterdam, New York.
[6] Göring, H. (1980) Wiss. Z. Ernst-Moritz-Arndt-Universität Greifswald, Math.-Naturwiss. Reihe 24, 87–89.

[7] Göring, H. and Bleiss, W. (1981) in: Rost Rastenij i Differenzirovka (Kefeli, V.I. ed.) pp.40–56, Izdatelstvo Nauka, Moscow.
[8] Göring, H. (1981) in: Selfregulation of Plant Metabolism (Georgiev, G. et al. eds) c/o Jusantov, Sofia.
[9] Hager, A., Menzel, H. and Krauss, A. (1971) Planta 100, 47–75.
[10] Bates, G.W. and Goldsmith, M.H.M. (1983) Planta 159, 231–237.
[11] Smith, F.A. and Raven, J.A. (1979) Annu. Rev. Plant Physiol. 30, 289–311.
[12] Brummer, B., Felle, H. and Parish, R.W. (1984) FEBS Lett. 174, 223–227.
[13] Hager, A. and Moser, I. (1985) Planta 163, 391–400.
[14] Brummer, B., Potrykus, I. and Parish, R.W. (1984) Planta 164, 345–352.
[15] Brummer, B., Bertl, A., Potrykus, I., Felle, H. and Parish, R.W. (1985) FEBS Lett., in press.
[16] Kutschera, V. and Schopfer, P. (1985) Planta 163, 483–493.