The History of Tissue Tension

W. S. PETERS* and A. D. TOMOS

School of Biological Sciences, University of Wales, Bangor, Gwynedd LL57 2UW, Wales, UK

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In recent years the phenomenon of tissue tension and its functional connection to elongation growth has regained much interest. In the present study we reconstruct older models of mechanical inhomogeneities in growing plant organs, in order to establish an accurate historical background for the current discussion. We focus on the iatromechanic model developed in Stephen Hales’ Vegetable Staticks, Wilhelm Hofmeister’s mechanical model of negative geotropism, Julius Sachs’ explanation of the development of tissue tension, and the differential-auxin-response-hypothesis by Kenneth Thimann and Charles Schneider. Each of these models is considered in the context of its respective historic and theoretical environment. In particular, the dependency of the biomechanical hypotheses on the cell theory and the hormone concept is discussed. We arrive at the conclusion that the historical development until the middle of our century is adequately described as a development towards more detailed explanations of how differential tensions are established during elongation growth in plant organs. Then we compare with the older models the structure of more recent criticism of hormonal theories of tropic curvature, and particularly the epidermal-growth-control hypothesis of Ulrich Kutschera. In contrast to the more elaborate of the older hypotheses, the recent models do not attempt an explanation of differential tensions, but instead focus on mechanical processes in organs, in which tissue tension already exists. Some conceptual implications of this discrepancy, which apparently were overlooked in the recent discussion, are briefly evaluated. © 1996 Annals of Botany Company

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The historian, and especially the historian-scientist, can, I believe, become too easily beguiled by the power of present scientific theory, and consequently imagine that its ancestor theory carried the same logical implications, which are then presumed to have stood clear to the earlier practitioners.

R. J. Richards (1992)

INTRODUCTION

Plant organs do not consist of mechanically homogenous material, but usually are found to exist in states of mutual tensions between their different parts. Such tissue tensions become apparent upon organ dissection, when dramatic bendings or coilings occur. Their nature was vividly debated in the second half of the last century (e.g. Sachs, 1874). Remarkably, original research on tissue tension seems to have ceased at about 1940. Tissue tension has regained interest in recent years (Firn and Digby, 1977; Heijnowicz and Sievers, 1995), leading to the formulation of the hypothesis that plant organ growth is controlled by the epidermal tissue (Kutschera, 1987, 1992). Surprisingly, the origin of this notion has been ascribed to scientists as distant in time and theoretical background as Kraus (1867; Kutschera, 1992) or Thimann and Schneider (1938; Edelmann, Bergfeld and Schopfer, 1989). We feel that here, and in some other cases, assessments of the historical development of the topic are inaccurate. As a result important aspects of the control of tissue tension have been overlooked.

In the present paper we attempt to reconstruct some of the more influential hypotheses on organ growth mechanics. In particular we will concentrate on the theoretical framework, in which the models were formulated, and on the experimental strategies deduced from them. While analysing the models, we purposefully will refrain from re-evaluating the significance of experimental results per se. By doing so we hope to avoid interpreting the historic theories on the basis of present knowledge. Interpretations of the latter type are likely to over-emphasize factors, which presumptively correspond to modern views, and neglect aspects, which have gone out of fashion, even if they were highly important at their time. For due discussion of these different historiographical approaches see Agassi (1963), Kuhn (1969, 1977) and Bayertz (1980).

Our aim in this paper, however, is more than a mere correction of inaccurate views of a historic process. Experimental strategies do not simply emerge from bodies of established knowledge; rather they are constructed on the basis of hypotheses. One may doubt whether it is always the most recent hypotheses which automatically provides the soundest foundation for future research (Kuhn, 1969). In returning some older concepts to the current discussion, we hope to broaden the spectrum of justifiable research strategies.
Vegetable Staticks

In the 17th century, the transfer to the medical sciences of mathematical and mechanical principles, which had proven so successful in physics, resulted in the 'iatromechanics' of Sanctorius, Giovanni Borelli, William Harvey and others. The iatromechanical concept, namely to search causal explanations for life activities in their exact mechanical descriptions, was applied to botany on a large scale by Stephen Hales. In his *Vegetable Staticks* (1961; originally published 1727) he described growth tests, which resemble modern measurements of relative elemental growth rates, and discussed the mechanical role of various parts of shoots in elongation growth (see his Experiment CXXIII). He based his considerations on Borelli's assumption (1927; first published 1680), that elongation was driven by the attraction of water by the 'spongy pith'. The 'dilating spongy substance' exerted its force on 'plinth, or abutments', which he identified with the partitions of the shoot at the nodes (Fig. 1). Thereby the outer parts of the shoot ('vessels') were 'distended like soft wax'. This distention was understood to be passive. Hales compared the lengthening of the 'vessels' with the 'effect in melted glass tubes, which retain a hollowness, tho' drawn out to the finest thread'.

Iatromechanic models typically interpreted organisms as machines performing mechanical work. Iatromechanic studies centred on the immediate causes (such as specific structure), which determined the types of action a particular machine could possibly perform. The question of how the machines came into existence was beyond the scope of these models (Reif, 1985), and therefore no developmental causes were considered. Consequently, Hales did not comment on the origin of the organismic machine he described. He stressed that nature was 'always carefully providing for the succeeding year's growth by preserving a tender ductile part in the bud replete with succulent pith'. But this preservation of en miniature machines as remnants of last year's ones could not explain the origin of the mechanical apparatus itself from the seed unless ideas of preformation were employed.

The theoretical background in the 19th century

One character of the development of biological sciences in Europe during the first half of the 19th Century was the controversy between the idealistic concepts of the *Naturphilosophie* on one side, and the reductionism of the inductivistic programme on the other. A major breakthrough for the latter is marked by the formulation of the cell theory (Schleiden, 1838; Schwann, 1839; for review see Jahn, 1987). It stated that multicellular organisms are built up from individual elementary organisms, namely the cells, which as such were thought independent of each other, and of the multicellular entity. The practical implication of this concept was that 'the quest for the fundamental power of organisms is thereby reduced to the quest for the fundamental power of single cells' [Schwann, 1839, p. 229 (All translations of quotations from the German by WSP.)]. Relevant physiological research thus should be performed on the cellular level, since the organism represents but an arrangement of cells (Schleiden, 1842).

Wilhelm Hofmeister challenged the cell theoreticists' doctrine (Hagemann, 1992; Kaplan, 1992), and maintained that 'the growth of single cells within a meristem is regulated and determined by...the mass increase of the meristem as a whole. This mass increase cannot be construed as the sum of the generating powers within the individual
cells' (Hofmeister, 1867, p. 129). Experimental strategies derived from this organismal concept, within which ‘the degree to which so-called cell enlargement occurs is simply a marker of organ expansion’ (Kaplan, 1992, p. 331), will obviously differ from the above. It appears that despite the intervening 130 years the implications of this dichotomy remain to be resolved (Kaplan and Hagemann, 1991; Sitte, 1992). We believe that the subsequent development of theories on growth mechanics can only be properly understood against this theoretical background.

**Hofmeister’s model of shoot tropisms**

Hofmeister (1859, 1863) gave precise descriptions of tissue tension phenomena in growing shoots and discussed functional implications, thereby surpassing the contributions of his predecessors, which he briefly reviewed (Hofmeister, 1863). Although he spoke of ‘differences of tension of tissues’ (Differenzen der Spannung der Gewebe) instead of using the term ‘tissue tension’ (Gewebe-spannung), he actually introduced the concept into modern botany.

For our purpose, it is enlightening to understand how Hofmeister established a role of differential tensions in his theories. He started with an investigation of the bending of shoots as induced by external mechanical stimuli (Hofmeister, 1859). He found growing internodes bent in response to vigorous shaking of the plant, or repeated gentle blows from one side (for a recent review of related effects see Jaffé, 1985). He then looked into the mechanical architecture of the stems to find that ‘the pith possesses a tendency to expand considerably, which is kept within bounds by the resistance offered by the cortex and wood’ (p. 254). He explained the mechanically-induced bending by assuming that the mechanical stimuli loosen the expansion-resisting organ parts in an inhomogeneous manner. Convex bending occurred on the side in which greatest wall loosening had been induced. This hypothesis seemed corroborated by the fact that only growing internodes bend. Only those are in a state of tissue tension, which was a precondition for the bending response.

In 1860 Hofmeister (Hofmeister, 1863) used this very argument to explain the mechanism of geotropic shoot curvature. Negative geotropic curvature occurred, because ‘in the lower half of an organ the extensibility of those walls, which restrict expansion..., increases’ (p. 88) due to the gravitational force. Here gravitation is functionally analogous to the external mechanical stimuli discussed above. It can only cause an effect in shoot portions which are in a state of tissue tension. Hofmeister consequently stressed that negative geotropism occurs exclusively in those. To him the change in differential tensions was the mechanical cause of the curvatures. In contrast, in his opinion positive geotropic curvatures in roots were brought about by entirely different mechanisms, which did not depend on differential tensions in the organ.

Hofmeister conceived the immediate cause for differential tensions in different states of imbibition of different cell walls, and denied a crucial rôle of ‘endosmosis’ and turgor pressure. Similar ideas found supporters until the end of the century (Sachs, 1865; Kraus, 1867; Müller, 1877; Boehm, 1886; Küster, 1899), before cellular mechanics in the modern sense were unanimously accepted (see, e.g. Pfeffer, 1904). Hofmeister did not go into detail regarding the developmental causes of tissue tension, which therefore remained an unexplained presupposition in his model. There is but one short remark on this issue in his description of geotropism in rhizomes: ‘During and after elongation, differences of tension occur between the central tissues and the epidermis of the elongated internode: the latter is stretched due to the former’s tendency to expand.’ (Hofmeister, 1863, p. 108). In his logic, elongation growth somehow establishes the necessary condition (tissue tension) for the *explanandum* (geotropic curvature) to occur. We suggest that herein lies the reason for the lack of an explicit explanation of the developmental causes of differential tensions. He considered tissue tension an effect of elongation growth, the mechanism of which he had not set out to explain. This lack of causal explanation of the ontogenetical development of shoot mechanical architecture is a character shared by Hofmeister’s theory with the much older iatromechanical models.

**The causes of tissue tension pursued**

Julius Sachs’ review-like essay of 1865 (chapter XIII) appears to mark the starting point of the developmental analysis of differential tensions. Kraus (1867, p. 106) judged about this work: ‘What confers lucid clearness on the whole treatise, is that the fundamental cause of tissue tension is recognized within the differential growth of the different connected tissues. Thereby the whole of tissue tension phenomena became... a mere function of the best known character of all tissues, namely growth’. In Sachs’ hands Hofmeister’s incidental remark mutated into a central causal statement. To Sachs, tissue tension was a side-effect of elongation growth. This is clear not only from his texts (e.g. 1875, 1887), but also from the structure of his arguments. For example, he inferred from the fact that isolated root tissues elongate at different rates, that tissue tensions exist in the intact organs, although they are not readily demonstrated directly (Sachs, 1875, p. 720). In this context the term ‘growth’ on the cellular level meant the ‘constant overstepping of the limit of elasticity of the growing cell-wall constantly being neutralized by intussusception’ (Sachs, 1875, p. 712; compare Pfeffer, 1904, §§ 9, 18).

The differential rates of cell growth established a differentiated mechanical architecture, with consequences for growth on the organ level: ‘We have seen how elongation growth in internodes is produced by two factors; how the inner tissues, the pith in particular, form the actually expanding element or driving force of growth, and how an end is set to the steady elongation of those elements by the pressure which the very elastic peripheral tissues exert on them; how the peripheral tissues, so to speak, only determine the extent of growth of the internode’ (Kraus, 1867, p. 141. Note that ‘elastic’ means ‘possessing a high elastic modulus’ in Kraus’ text). The importance of inner tissues for the growth of the whole organ was underlined by the terminological use. Inner tissues were referred to as being in a
'state of active tension', in contrast to the 'passive tension' of the outer tissues (Vines, 1886, pp. 343, our italics). [For the proper understanding of the historic texts it is important to note that ‘tension’ (Germ.: Spannung) was used as a generic term, referring to both tensile and compressive stresses (Germ.: Zugspannung and Druckspannung). 'Tissue tension' (Germ.: Gewebespannung) as introduced by Sachs therefore correctly describes the whole of mutual tissue stresses in an organ.] In connection with the finding that isolated pith keeps on growing for days, whereas isolated outer tissues do not elongate at all (Sachs, 1875), this notion led to the idea that some tissues (e.g. the epidermis) are only capable of 'passive growth', i.e. elongation driven by forces from outside the growing cells (Sachs, 1875; for a particularly straight-forward application of the concept see Küster, 1899). The causal analysis of differential tensions in shoots thus seems to have supported a similar interpretation of the rôle of the outer tissues in organ growth as that of Hales' iatromechanical approach (see above).

The insight that ‘tissue tension is indeed a result of differential tissue growth’ (Kraus, 1867, p. 108) had consequences for Hofmeister's model of negative geotropism. Changes in the intensity of differential tensions now were interpreted as results of growth processes, just like differential tensions themselves resulted from growth. It followed that 'the change in tissue tension in the convex and concave halves of bending plant organs is not the cause of the movement, but results with mathematical necessity from the curved form of the whole' (Frank, 1868, p. 74; cited after the review by Schober, 1899).

Further research focused on the nature of cell growth in general, and the regulation of differential growth in particular. At the end of the century the cell theory was practically unanimously accepted (Hansen, 1897). In this theoretical environment, differential tensions had only limited value, serving as indicators of prior differential cell growth. Ludwig Jost (1908) put it unmistakably: 'In former times tissue tension was studied with great care, since insights into various physiological phenomena were expected from such investigations. However, these expectations were not fulfilled. Therefore we will no longer discuss those matters, but consider again the differentiation of cells in a meristem' (p. 350). The mechanism of differential cell growth was obscure still. Differential cell turgor was suggested to play the key rôle (de Vries, 1919; Went, 1933).

But it was not before the application to the problem of the hormone concept, that a generally accepted theory was formulated.

The advent of hormones

One difficulty implicit to the cell theory lies in the necessity to explain the neat coordination of the huge number of individual 'elementary organisms', which form the multicellular entity. The endocrinological hormone concept provides a mechanism by which such correlations could be explained. After a series of successes in zoological fields in the 1920s (for review see Karlson, 1982), the concept was readily acquired by plant physiologists (e.g. Boysen-Jensen, 1936; Went and Thimann, 1937), although numerous inconsistencies appeared (for discussion see Zimmermann and Wilcoxson, 1935; Trewavas, 1981, 1986; Firn and Myers, 1987; Hathway, 1990). Not surprisingly ‘the leading idea that correlations in plants are due to the influence of special substances' (Went and Thimann, 1937, p. 2) was soon utilized to formulate a model for the development of tissue tension in growing shoots.

Kenneth Thimann had proposed in 1937 that ‘the curve of response against auxin concentration is... of the same general shape for each organ, though shifted horizontally or vertically’ (quoted from Thimann and Schneider, 1938, p. 635), namely bell-shaped. This differential-auxin-response hypothesis is commonly found in modern textbooks still. The explanation of how tissue tensions could arise was constructed by transferring its logic from organs to cells: ‘...the same relations hold for the individual layers of cells within a given organ. In the pea stem, the innermost layers—i.e. the pith—reach their maximum elongation at relatively low auxin concentrations, the outermost, on the other hand, at relatively high' (Thimann and Schneider, 1938, p. 635). Highest sensitivity in terms of lowest effective auxin concentrations thus occurs in the pith. On the contrary, highest sensitivity in terms of maximum inducible effect is found in the epidermis (Fig. 2; compare Firn, 1986, for a discussion of the ambiguity of the term ‘sensitivity’). In the intact plant, however, auxin concentrations had to be postulated to be in a range in which elongation was promoted stronger in the inner tissues than in the epidermis, in order to account for the observed direction of the tension gradient (Fig. 2, arrow A). Following the logic of the hypothesis, the Went-reaction (the auxin-dependent reversal of tissue tension in the split-pea test; Went, 1934) had to be
interpreted as an effect induced by unphysiologically high hormone concentrations (Fig. 2, arrow B). Thimann and Schneider saw their theory supported by several lines of evidence. First there was the finding that ‘peeled sections of both Pisum and Avena respond very well to auxin’ (Thimann and Schneider, 1938, p. 629), or ‘in other words, the sensitivity of peeled material to auxin is certainly no less than that of normal material in the same experiment’ (p. 630). Second, auxin dose-response curves of isolated tissues appeared to be in accord with the theory (Thimann and Schneider, 1938). Third, in auxin-depleted material very low auxin concentrations seemed to increase the naturally occurring concave bending, an effect readily explained by the theory (Thimann and Schneider, 1938, 1939. Compare Fig. 2, arrow C). Thimann and Schneider (1938, p. 641) believed they had solved a classical problem: ‘The differences in the auxin response of different tissues are the principal cause of the tissue tension studied by the older botanists’.

The differential-auxin-response hypothesis was a typical cell-theoretical model insofar as the cells in an organ were envisaged as qualitatively identical entities, the behaviour of which explained the behaviour of the organ as a whole. The model bore two obvious implications. First, tissue tension was a solved problem within the cell-theoretical paradigm, i.e. the elements of the compound phenomenon were understood, and no more investigations into its nature were necessary. In fact, Thimann and Schneider’s (1938) graphs were reproduced in numerous text-books and research reports (e.g. Rietsema, 1950; Bunning, 1953). We suggest that this situation explains the almost complete absence of research on differential tensions and their relation to growth in the following four decades. Second, for future research on the cellular mechanism of auxin action the cell type studied would be irrelevant. Against this theoretical background any doubts about the qualitative identity of cells concerning auxin action would, of course, necessitate reconsideration of the developmental causes of tissue tension.

Criticism of the model, which was raised immediately (Went, 1939), did not question the basic similarity of all cells with respect to auxin action. Instead, cell-theoretical notions were applied in an even stricter sense. Suggesting quantitatively identical auxin sensitivities in all cells, Went (1939, p. 406) claimed that ‘tissue tension is due to differential auxin content of the inner and outer tissue’.

The epidermis in control

Studies on the nature of tissue tension, or the implications of organ mechanical architecture for cell physiology, were rare in the decades following Thimann and Schneider’s work, indicating a wide acceptance of their model. The few exceptions (e.g. Masuda and Yamamoto, 1972; Yamamoto et al., 1974), though undoubtedly valuable contributions, did not trigger the elaboration of an alternative biomechanical hypothesis. Models of similar structure, i.e. explanations of complex developmental processes by gradients of either growth hormone or hormone sensitivity, had been established for many problems, particularly for tropic curvatures (Chodony, 1928; Went, 1928).

In a review on this topic Firn and Digby (1980) suggested that ‘it is now time to go back and look in detail what happens during a tropic curvature and to build theories on firmer foundations’ (p. 145). Thus the hormone concept within its cell-theoretical framework apparently was unsuccessful in supplying a unanimously accepted theory of tropisms. Firn and Digby (1977) had indeed formulated a model of shoot geotropism, which did not include hormones at all. Their starting point was virtually identical to Hofmeister’s, inasmuch as a characterization of differential tensions in an organ formed the basis of the modelling of the mechanics of the organ’s geotropic curvature. The outer tissues, found to constrain organ elongation, were suggested to be the sole geoperceptive and georesponsive elements within the organ. Gravitation-induced elongation growth would only occur in the peripheral layers of the lower side, thus evoking the curvature. Again, a developmental explanation for differential tensions is beyond the scope of the model. Tissue tension rather is included as a condition under which the proposed mechanism can occur.

More recently, Kutschera (1987) proposed a similar mechanism of auxin-mediated elongation growth. The hypothesis is based on a mechanical model of growing plant organs (Fig. 3), in which the peripheral wall(s) act analogously to the wall of the Nitella cell, whereas the inner tissue as a whole can be regarded as a giant, pressurized protoplast (Kutschera and Köhler, 1992, p. 1380). Since such a system is characterized by the distribution of tensile and compressive stresses, Kutschera (1989) suggested to speak of ‘tissue stresses’ rather than of ‘tissue tension’ (but see the above remark on the original meaning of the term Gewebespamnung).

The model departs from classical cell theory by interpreting the whole organ as one supercell, to which concepts of cellular biomechanics can be applied. Just as the cell wall restricts the expansion of the protoplast in a Nitella cell, the outer tissues are thought to restrict growth in an organ. This seems to imply that ‘the control over the rate of organ growth can only be brought about by changes in the mechanical properties of the rigid, extension-limiting peripheral walls’ (Kutschera, 1992, p. 251). The model of auxin action in shoots is constructed accordingly. Auxin is postulated to exert its growth promoting cellular effects (leading to wall loosening) exclusively in the epidermis. On the other hand, inner tissues elongate by means of an unknown IAA-independent growth process’ (Kutschera, 1987, p. 223). Consequently, cellular auxin effects should be classifiable into growth-relevant and -irrelevant ones by the criterion of their exclusive occurrence in the epidermal tissue (Kutschera, 1987; Dietz, Kutschera and Ray, 1990).

Tissue tension forms a conditio sine qua non for the suggested mechanism of auxin-mediated elongation. Wall-loosening in the epidermis exclusively would remain without effect if this issue would not have gained a special function by the prior establishment of differential tensions. The explanation for this developmental process, however, is beyond the scope of the model. Kutschera (1992) lists a number of structural features which appear to establish a special mechanical rôle of the epidermal walls. However, no developmental causation for differential tensions is offered.
Fig. 3. Model of a growing shoot, as formulated as the basis of the epidermal-growth-control hypothesis (combined after Kutschera 1987, 1989, 1992). The thick and rigid walls of the outer tissues (OT), especially the outer one of the epidermis (E), bear the turgor-induced tensile stress of the whole organ. The walls of the inner tissues (IT) do not contribute to this stress-bearing; they are kept in a state of compression by the outer tissues, which resist their tendency to expand. The mutually induced stresses in the different tissues are schematically indicated in the stress profile at the bottom. Multicellular shoots are thus mechanically analogous to the giant internode cells of Nitella: the outer tissues resemble the cell wall, whereas the inner tissues are equivalent to a single protoplast.

In the logic of the hypothesis, auxin occupies a position identical to the one external mechanical stimuli did in Hofmeister's model. Compared to the model of Thimann and Schneider, the result is turned into a premise: auxin acts on tissue tension—it does not cause it any more. It is a telling detail that the mechanical analogy between single cells (such as Nitella internodes) and multicellular organs formerly had been employed to underline the significance of differential tensions to the stiffness of non-lignified organs (e.g. Sachs, 1875; Strasburger et al., 1898; Bennecke and Jost, 1923; Bower, 1923). To judge from the contemporary literature, these researchers were fully aware of the functional implications of their concept: 'Since the pith is in a state of compression, any increased length of the cortical tissue must result in an increase in the length of the whole shoot' (Darwin and Acton, 1907). But since the cell-analogous organ was understood to be a result of the growth process, the conclusions concerning the control of organ growth, which appear plain and inevitable if the existence of differential tensions is presupposed (Kutschera, 1987, 1992), had in fact never been drawn.

**COMMENTS ON THE CURRENT DISCUSSION**

In the present study we discuss the historical development in the field of the biomechanics of elongating plant stems in the context of major conceptual changes ("paradigm-changes" in the sense of Kuhn, 1974), such as the establishment of the cell theory, or the break-through of the hormone concept. Our historical reconstruction suggests that a major line of development led from predominantly descriptive models (e.g. Hales' iatromechanic model) to functional hypotheses, which considered differential tensions and their immediate causes as a precondition for certain responses (e.g. Hofmeister's theory of negative geotropism), and further to causal hypotheses, which aimed to explain tissue tension itself by providing developmental causes for its existence. The most advanced theory of the latter type was the differential-growth-response hypothesis by Thimann and Schneider. We interpreted the almost complete absence of original research on tissue tension in the four decades after 1940 as an indicator of the wide acceptance of this hypothesis.

Functional and causal hypotheses as defined above differ in scope. Therefore, a number of functional hypotheses might be compatible with a single causal one. For example, the ideas on shoot geotropism put forward by Firn and Digby (1977) are as such compatible with the differential-growth-response hypothesis (Thimann and Schneider, 1938). The case is different with the currently much-discussed epidermal-growth-control hypothesis (Kutschera, 1987, 1992), which undoubtedly classifies as a functional hypothesis. It is simply impossible that both this model, which explains auxin action on the background of pre-existing differential tensions, and the differential-growth-response hypothesis, which explains differential tensions as an effect of auxin action, are equally valid. Similarly, the claim that elongation growth is controlled by the epidermal layers in general (Kutschera, 1992), calls into question all interpretations of tissue tension since the 1860s, which explained the elongation-restricting rôle of the epidermis as a result of the growth process. Thus, if one accepts the epidermal-growth-control hypothesis as it stands, one abandons all causal hypotheses on differential tensions so far formulated. As an alternative, it has been suggested that structural characters of organs, such as different wall diameters in different tissues, might be sufficient to explain the occurrence of differential tensions (Kutschera, 1992). However, such explanations are merely descriptive, and in this respect do not differ from iatromechanic accounts.
Moreover, some older studies suggest that the evaluation of the structural causes of tissue tensions might not be simple at all. Tissue tensions are not found only in highly developed plants, but also in organs lacking histological differentiation such as mushroom stalks (Sachs, 1874; Pfeffer, 1904). Pronounced tissue tensions occur in macroscopic marine algae as well. Here the outer tissues’ small, thick-walled cells are kept under compression by the bigger, thin-walled ones of the central tissues (Küster, 1899; we find his results readily reproducible). The correlation between wall-thickness distribution and differential tensions in marine algae therefore is the reverse of that in stems of most seed-plants. However, the situation in macroalgae may be comparable to the formation of the central cavity in angiosperm stems, where the thin-walled pith does not follow the growth of the thick-walled outer tissues and disintegrates (Küster, 1899). Another argument questioning the validity of structural characters as causal explanations for the occurrence of tissue tension can be drawn from the function of pulvini, the motor organs for leaf movement. In his historic sketch, Wetherell (1990, p. 73) concluded with reference to Sachs (1887): ‘Sachs understood pulvinar movements, as we do today, in terms of reversible ‘...tissue-tensions of extraordinary magnitude’...’ (our italics). Apparently, regular reversions of differential tension can occur without modification of organ structure. Thus, tissue tensions can certainly not be satisfactorily explained by structural characters.

We have to infer that differential wall-thickness or histological differentiation do not suffice to explain the immediate, let alone developmental causes of differential tensions. We conclude that to date we lack any sound alternative to Sachs’ assumption, that a mechanical architecture allowing for tissue tension to occur is established in an organ by differential growth (i.e. differential rates of wall-loosening; compare Tomos, Malone and Pritchard, 1989; Brown, Sommer and Pienaar, 1995). Recent data from rice (Oryza sativa L.) internodes, in which gibberellin is involved in the response to submergence by promoting elongation growth (Raskin and Kende, 1984), should be considered in this context. Whereas slowly growing internode sections in water do not possess significant tissue tensions, sections growing rapidly in gibberellin solutions do (Hoffmann-Benning and Kende, 1994). The induction of tissue tension by gibberellin, concurrent with growth promotion, suggests that this ‘hormone’ induces higher rates of wall-loosening in the inner tissues than in the outer ones, at least during the initial phase of the response. Gibberellin action in rice internodes thus could provide a model for the establishment of differential tensions during organ elongation.

The above problems passed unnoticed in the current debate. This may be because recent historical accounts did not attempt to reconstruct historic hypotheses as they were understood by contemporary practitioners. As a result, gross misinterpretations occurred. For example, summarizing Sachs’ contribution, Kutschera (1987, p. 216) claimed that ‘Sachs suggested that a plant organ can only grow as long as tissue tension is established’. This interpretation is not only untenable, since it reverses the cause-effect relationship of tissue tension and growth as elaborated by Sachs; it actually misconstrues Sachs’ causal hypothesis as a functional one, thereby obscuring the fundamental difference between the epidermal-growth-control hypothesis and its predecessor.

Finally, we feel that some remarks on the ‘organismal concept of multicellularity’ (Kaplan, 1992) should be made. In short, the organismal concept claims that the evolution of multicellularity in plants took place by compartmentation of unicellular organisms, and not by accumulation of the latter (Kaplan and Hagemann, 1991). Noteworthily this is a phylogenetic statement. As such the concept is compatible with functional changes of different organ parts during further evolutionary developments. The idea that plant organs behave according to the concept, if their outermost tissue functions analogously to the wall of a single cell (Kutschera, 1995), interprets the phylogenetic concept as a functional one. However, a functional interpretation of plant organs as ‘supercells’ is not implied by the organismal concept. In fact, on the background of the organismal concept the suggestion has been put forward that the adaptive significance of multicellularity lies within the inner cell walls’ contribution to the mechanical properties of the whole organ (Niklas and Kaplan, 1991).

With respect to organ growth, the organismal concept claims that an understanding of whole organ mechanics forms the basis of the understanding of the mechanical rôle single cells may play within the organ (Hofmeister, 1867; Kaplan and Hagemann, 1991; Kaplan, 1992). Considering differential tensions, one may doubt whether either type of understanding has been achieved yet.

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