On the Mechanism of Xylem Vessel Length Regulation

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The mechanism by which the plant regulates the length of xylem vessels has not yet been elucidated. The length of a xylem vessel depends on the number of fused vessel elements and their individual lengths. In this paper, a straightforward mechanism is postulated to explain how the length of xylem vessels in plants is regulated. The mechanism is based on a steerable, similar chance for all tracheary elements to be a terminal element. As a result, an exponential vessel length distribution is created with mostly short and fewer long vessels, as is commonly found in plants.

Angiosperm plants have xylem vessels, which are responsible for long-distance transport of water and nutrients. During vascular development, single cells fuse into linear strands. After fusion and formation of a secondary cell wall, these tracheary elements lose their nucleus and cell contents, leaving a hollow, dead, finite capillary (the vessel). Xylem vessels are laterally interconnected by bordered pits and thus form the transport pathway for upward water transport. Long vessels transport water more efficiently than short vessels, because intervessel transport, i.e. transport through bordered pit pairs, is considered to be of influence on total vessel length (Tyree and Zimmermann, 2002).

Direct measurement of the length of xylem vessels is virtually impossible because of the enormous length compared with the microscopic diameter. All current vessel length measurements are indirect and based on counting the number of continuous vessels at varying lengths of a stem segment. Often, an exponential relationship is found for the number of continuous vessels as function of the length of the stem segment (Fig. 1; see also for examples Zimmermann and Potter, 1982; Darlington and Dixon, 1991; Nijsse et al., 2001; Tyree and Zimmermann, 2002). It is intriguing to consider that this exponential relationship is analogous to radioactive decay of atoms in time: All atoms have per unit of time a similar chance to decay, and an observer cannot predict the moment of decay for a single atom. In the same way, xylem vessels have for every additional length unit a similar chance to terminate. The plant anatomist cannot predict the length of a single vessel; he can only describe the length distribution of a population of vessels.

Analogous to the lifetime distribution of radioactive atoms the length description of a population of vessels is described by:

\[ (N) = e^{-kN} \]  

(Fig. 2)

where \( N \) is the number of fused vessel elements and \( P(N) \) is the fraction of the vessels that consist of \( N \) or more elements. \( k \) is the exponential factor, from which, analogous to the radioactive halftime value, the half-length value \( \tau \) can be calculated:

\[ \tau = \frac{\ln 2}{k} \]

The length distribution \( D \), which is the fraction of vessels with a length of \( N \) as a function of \( N \) is:

\[ D(N) = \frac{-\partial P(N)}{\partial N} = ke^{-kN} \]  

(Fig. 3)

Vessel length is defined here as the number of fused elements. If vessel elements have a stable average length, the vessel length distribution function can also be written in length units. Vessel element length can vary (Baas et al., 1983) but is not thought to be of influence on total vessel length (Tyree and Zimmermann, 2002).

In general, vessels are randomly located in the longitudinal direction (Skene and Balodis, 1968; Tyree and Zimmermann, 2002). It is important to consider that the vessel length distribution in a cross section is biased to the longer vessels. This is due to the fact that longer vessels exist over a proportionally longer length of the stem segment than the shorter vessels. The length distribution of vessels in a cross-sectional plane \( D_{cross} \) is therefore:

\[ D_{cross}(N) = cND(N) = k^2Ne^{-kN} \]  

(Fig. 4)

The constant \( c \) is a normalization factor, which appears to be equal to \( k \). The same formula has recently been derived by Cohen et al. (2003) in an-
other way to describe a unimodal vessel length distribution.

Long vessels contribute more to the xylem sap transport than short vessels: During transport a unit of sap stays longer in long vessels. If the length distribution is corrected for the impact on water transport, a "hydraulic vessel length distribution" is obtained:

\[ D_{\text{hydr}}(N) = cN D(N) = k^2 N e^{-kN} = D_{\text{cross}}(N) \]

Hence, the hydraulic vessel length distribution is equivalent to the cross-sectional vessel length distribution.

With the current vessel length measurements, a cross-sectional cut is made and the continuity of the cut open vessels is traced starting from this cut plane. Of these vessels, the part below the cut plane is not measured. Normally, the fraction of the vessels that is below the cut surface is randomly distributed. However long vessels can have a proportionally longer part cut off than short vessels. Therefore the length distribution of the measured parts of the cut open vessels is:

\[ D_{\text{cut}}(N) = \frac{c}{N} D_{\text{cross}}(N) = k e^{-kN} = D(N) \]

which is the same distribution as the overall vessel length distribution. This means that the overall vessel length distribution \( D(N) \) without further calculations can be deduced from the exponential relationship that is found in vessel length measurements (Fig. 1B, inset). It must be noted that maximum vessel length cannot be calculated, but that the length of the longest 1% of vessels is given by \( \ln(100)/k \).

The vessel length regulation mechanism postulated here only requires the existence of a similar chance for each added vessel element to be the terminal element. Note the analogy with the lifetime of radioactive atoms. \( \tau \) is the half-length value.
(hydraulic) vessel length distribution consists of a range of vessel lengths, depending on the half-length value. In terms of regulatory mechanisms in the plant, the chance mechanism is surely the simplest alternative to obtain such a predictable range of vessel lengths. It is known that vessel length distribution varies in different branching levels and under different environmental conditions. More distal branches mostly have shorter vessels, and many trees produce longer vessels in spring than in summer (Zimmermann and Potter, 1982). The chance mechanism proposed here allows plants to regulate vessel lengths (either spatially or temporally) by just changing the termination chance. In plants, special regions can exist where many vessels terminate, e.g. in branch junctions or in abscission layers. These regions might represent sites of an increased vessel termination chance.

Strikingly, the question of how a plant regulates vessel length is not commonly found in literature and is probably even lacking. The present contribution postulates the most straightforward mechanism to explain commonly found vessel length distributions. Trying to understand the mechanism of xylem vessel length regulation opens a way to better understand xylogenesis at the tissue level, including some implicit restrictions. Regulation of vessel length by means of a steerable termination chance implies that homogeneous wood always has an exponential length distribution, which can be tested experimentally. Additionally, ringporous wood will also contain exponential vessel length distributions in its homogeneous subdomains.

After predicting the existence of the regulation mechanism, it is important to search for the cytological and molecular basis of this mechanism. Vessel length is correlated with vessel diameter: Wide vessels are in general long, and narrow vessels are in general short (Tyree and Zimmermann, 2002). Auxins and cytokinins play roles in vessel element differentiation and vascular patterning (Ye, 2002). The vessel termination chance might be the result of a hormone flux or gradient. However, taking into account the stochastic nature of the proposed vessel length regulation mechanism, it is well possible that vessel termination is not directly regulated, but emerging from other developmental traits, such as vessel diameter or cell alignment. In this view, vessel termination could be regarded as an accidental yet very functional interruption of the default mechanism of tracheary element fusion. The regulation mechanism proposed here for the length of xylem vessels fits within the paradigm of the non-determined fractal-like nature of the architecture of plants (West et al., 1999). It provides a useful insight into plant structure, both to explain development of simply ruled complex structures and to further facilitate the modeling of plant vascular architecture.

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