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A discussion on some aspects of the Turing model of morphogenesis applied to plants

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ABSTRACT

In 1952, British mathematician Alan Turing (1912–1954) published the reaction-diffusion model, in which two interacting species of molecules can generate a complex pattern in the course of plant or animal development if the substances (termed morphogens) differ in their diffusion rate. Turing's theory got concrete biochemical and molecular support during recent 15 years. Several pairs of interacting and diffusing chemicals have been suggested for various morphogenetic processes in multicellular animals. Therefore, the principal points of Turing's theory have been confirmed, though there is no universal pair of interacting morphogens in animal development. These recent data are briefly considered in the present essay and an attempt is made to consider current applications of the Turing's model to the development of vascular plants. In the latter case, however, the situation seems today to be less clear than in the case of the metazoan morphogenesis.

Keywords : Activator-inhibitor systems, auxin, pattern formation, plant morphogenesis, Turing's theory.

The reaction-diffusion model of Turing (1952) postulates a pair of activator and inhibitor, one of which diffuses through a tissue much faster than the other. According to his seminal theoretical work, these two interacting chemical substances (morphogens) can generate stable inhomogenous complex patterns (for example, an isolated ring of cells). In this connection Turing (1952, p. 68) mentioned the tentacle pattern of *Hydra* (invertebrate freshwater animal) and the whorls of leaves of certain plants such as woodruff, *Galium odoratum* (*Asperula odorata*).

Afterwards, Turing's ideas were developed further, first of all by the group of German theoreticians (Gierer and Meinhardt 1972, Koch and Meinhardt 1994, Meinhardt 1996, 2012). Their concept of local short-range activation and long-range inhibition (also termed a lateral inhibition) seems to be a general patterning principle. These authors dealt predominantly with the developing animal models (such as head and foot formation in *Hydra*, organogenesis in *Drosophila* or formation of vertebrate limbs), but Meinhardt (1996) emphasized on the occurrence of a common mechanism in plant and animal ontogeny.

Finally, Turing's theory received concrete biochemical and molecular support during recent 15 years. Several pairs of interacting and diffusing chemical substances have been suggested for various morphogenetic processes in invertebrate and vertebrate animals (see Table 1 for details). It should be remarked that the activator-inhibitor pair cAMP/ATP was suggested as a result of a theoretical analysis (Schiffmann 2005, 2017). By contrast, the other activator-inhibitor pairs (WNT/DKK, Nodal/Lefty, SHH or FGF-4/BMP-2 or BMP-4) were identified in the course of experimental studies and these morphogens are represented by the diffusing proteinaceous molecules. Therefore, it is reasonable to say that the principal points of Turing's reaction-diffusion model have been confirmed for the case of metazoan organisms (Kondo and Miura 2010, Painter *et al.* 2012, 2018), though there is no universal pair of interacting morphogens in multicellular animal development. Different suits of the diffusing activator and inhibitor are used in different morphogenetic processes. This idea seems to be appropriate within a framework of the original reaction-diffusion model.

The model was also used in the analysis of morphogenesis in the primitive green algae, such as *Acetabularia* or *Micrasterias* (e.g., Harrison *et al.* 1984, Holloway 2010), but this is not within the scope of present paper. I will now consider whether the Turing's model may be currently applied to the development of the vascular plants. It is appropriate to remind in this connection that Turing's paper (1952) was a subject of benevolent and constructive discussion in the botanical literature very soon after its publication (Wardlaw 1953, 1955). Besides, the eminent plant morphogenesis researcher Edmund Sinnott regarded Turing's research as "a promising hypothesis" (Sinnott 1960, p. 456).

First of all, it should be noted that molecular and cellular processes underlying pattern formation in plants are not identical with those in animals (Hernández-Hernández *et al.* 2012). Owing to the presence of cell walls, for instance, there are no migrations of individual cells or cell sheets during higher plant development (Ivanov 2011, Torii 2012, Nick 2014). Many authors (Smith 2008, Nick 2009, 2014, Korasick *et al.* 2013, van Berkel *et al.* 2013, Draelants *et al.* 2015, Laskowski and ten Tusscher 2017 and others) believe that the phytohormone auxin plays a crucial role in the processes of plant growth, morphogenesis and differentiation. Therefore,

Table 1 — Various authors about the activator-inhibitor systems in animal ontogeny

Activator/Inhibitor	Organisms, structures, morphogenetic processes	References
cAMP/ATP	Multicellular animals: first of all, echinoderms and amphibians; gastrulation, embryonic induction, organogenesis.	Schiffmann 2005
WNT/DKK	Hair follicle patterning in developing murine skin.	Maini *et al.* 2006, Sick *et al.* 2006
Nodal/Lefty	Sea urchin (*Strongylocentrotus purpuratus*) early embryogenesis.	Ertl *et al.* 2011
	Zebrafish (*Danio rerio*) ealy embryogenesis.	Müller *et al.* 2012
SHH or FGF-4/BMP-2 or BMP-4	Chick feather bud patterning.	Painter *et al.* 2012

Abbreviations for the putative morphogens: cAMP – cyclic adenosine monophosphate, ATP – adenosine triphosphate, WNT – wnt signaling protein, DKK – dickkopf secreted protein, SHH – sonic hedgehog secreted protein, FGF-4 – proteinaceous fibroblast growth factor, BMP-2 and BMP-4 – bone morphogenetic proteins. Nodal and Lefty are proteinaceous growth factors.

this hormone may be regarded as a principal plant morphogen, but the situation concerning plants seems to be different from that with the pairs of interacting morphogens in the developing multicellular animals. Nonetheless, there is an interesting suggestion (Nick 2009, 2014) that instead of actual inhibitory molecules (as in the Turing–Gierer–Meinhardt model) the developing plant organisms achieve the lateral inhibition by mutual competition for an activator (auxin). It is appropriate to remark, however, that "auxin is different from the morphogens considered by Turing. Instead of moving primarily via diffusion, it is actively pumped from cell to cell by the action of import and export proteins" (Smith 2008, p. 2631). These PIN-formed (PIN) proteins are polarly localized membrane proteins that fulfill the transport of auxin in higher plants (van Berkel *et al.* 2013). Several groups of researchers, mainly working with the auxin (e.g., Prusinkiewicz and Rolland-Lagan 2006, van Berkel *et al.* 2013, Draelants *et al.* 2015, Laskowski and ten Tusscher 2017), are skeptical about the occurrence of the Turing-type patterning during higher plant development.

On the other hand, recent study of patterning in *Arabidopsis thaliana* leaf has revealed a few interacting activator-inhibitor pairs (Torii 2012). In the process of stomatal development, for instance, the transcription factor SPCH (SPEECHLESS) represents an activator, while the secreted peptide EPF (epidermal patterning factor) represents an inhibitor. So the SPCH/EPF relation in the *Arabidopsis* leaf strongly resembles certain aspects of the original reaction-diffusion model.

In concluding, it is reasonable to say that recent results and ideas of different authors on the developmental control in higher plants cannot be reconciled completely and arranged within the framework of the reaction-diffusion Turing– Gierer –Meinhardt model (because of the specific features of plant morphogenetic processes and their multi-faceted nature). Further perspective for the broad application of this model to the vascular plants is today not clear (unlike the case of the metazoan morphogenesis).

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