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## 6 A simple model can unify a broad range of phenomena in retinotectal map development.

Simpson HD, Goodhill GJ.  
Biol Cybern. 2011 Feb; 104(1-2):9-29

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**Michael Crair** and **Moran Furman**, Yale University, CT, USA. **F1000 Neuroscience** **6**  
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Simpson and Goodhill employ a computational model to explore the roles of chemoaffinity cues, axonal competition, and axon-axon interactions during retinotectal map development. They extend previous computational models of retinotectal/retinocollicular map development to simulate and analyze a wide range of surgical and genetic manipulations.

Each retinal ganglion cell (RGC) axon in the model is represented by a set of axonal branches that advance across the tectum, initiating at its rostral border. The central equation of the model instructs the movement of a branch in response to three 'forces': 1) chemoaffinity, which drives each branch towards its target termination zone, defined based on molecular labels; 2) competition for physical space, acting to move branches away from areas where branch density is high; and 3) axon-axon interactions that are based on relative receptor expression levels and that repel nearby axons if their levels of receptor expression (such as ephrin-A [EphA]) are above a certain threshold.

In their simulations, Simpson and Goodhill first show that the model successfully recapitulates a set of classical experiments in which parts of the retina and/or tectum are ablated, rotated or re-positioned. These surgical manipulations demonstrate key roles for chemoaffinity and axonal competition during retinotectal map development. Simpson and Goodhill then proceed to simulate more refined genetic manipulations, in which EphA3 is selectively knocked-in to half of the RGCs at random, resulting in duplicated retinotopic maps {1,2}. Notably, these duplicated maps merge or 'collapse' at the rostral superior colliculus (SC) in heterozygous knock-in (KI) mice. In the spirit of Reber et al. {2}, Simpson and Goodhill's model suggests that accounting for the EphA3 KI mapping phenotypes requires a form of axon-axon interaction that is based on relative receptor levels.

What are the main differences between Simpson and Goodhill's model and other existing models of retinotectal map development? First, the proposed model distinguishes between repulsive axon-axon interactions that are based on relative receptor levels and axon competition for physical space. The authors argue that this distinction is particularly important when modeling the EphA3 KI phenotypes. Other models have postulated, in contrast, a role for correlated retinal activity for the map collapse in the EphA3 KI mice {3}. Reflecting this difference in interpretation, one of the predictions of the Simpson and Goodhill's model is that map collapse in the EphA3 KI phenotypes will remain stable under altered or absent, correlated retinal activity. Another difference between the present model and some of the existing models is that it explicitly simulates the growth of retinal axon branches into the tectum, whereas other models often assume that branches are present in the tectum to start with, and undergo a re-positioning process during development.

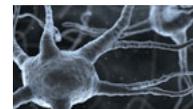
The study by Simpson and Goodhill does a nice job in accounting for a wide range of manipulations under a unified and relatively simple modeling framework. It would be interesting to expand the model to include activity-dependent mechanisms and examine the mapping phenotypes in mice where retinal activity patterns are altered. Also, the present version of the model assumes a fixed number of axonal branches that navigate across the SC. It would be interesting to extend the model to explicitly represent the process of retinal axon branch elaboration {4}, and to simulate the roles of activity and molecular cues in promoting/inhibiting axon branching during map development.

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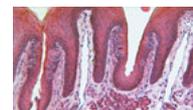
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#### Competing interests

None declared

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