

# DEVELOPMENT OF THE NERVOUS SYSTEM

## WINTER 2014

1. What are the characteristics of schizophrenia? What could be reasons for the initial name of the disease “Dementia praecox”?
2. Why can the occurrence of autism spectrum disorder not completely be explained by genetics?
3. Why is neurotrophin a coincidence detector? Why could mutations in this gene cause intellectual disability?
4. Draw a topographic and a discrete map. Where do they occur? How do they differ?
5. What are neurotrophins? Give two examples of neurotrophins.
6. How can social behaviour be studied in *C. elegans*? How is this regulated?
7. What are the two types of radial migration in the cortex? How does cortical development proceed?
8. Which cells generate most of the cortical neurons?
9. What are two positive regulators of neurogenesis?
10. What is the labelled pathway hypothesis?
11. Give examples of how axons change their behaviour at choice points.

changes occur in where they grow and changes to the surface may occur:

example of floor plate crossing: axonin-1 on precommissural axon needed and NrCAM on outer cells of floor plate needed for successful entry. These axons will continue their journey contralaterally (and rostrally) while other axons lacking axonin-1 will project ipsilaterally. Also, the crossing axons will express a new receptor on their surface so that Shh becomes repulsive (it was initially attractive) - receptor: Hhip1. Therefore Shh will be more repulsive when bound to Robo1 than it is attractive when bound to the precommissural attractive receptors (Boc, Ptc, Smo)

2nd example: retinal axons can either project contralaterally (cross) or ipsilaterally (didn't cross). It depends on the present pioneer axons, which way they will take.