During normal brain development in our fetal model, neurons (brain cells) rapidly grow processes called dendrites that allow them to communicate with other neurons. In panel A, a neuron equivalent to that from a 28-week-old human fetus displays very few dendritic processes and appears very simple. Panel B shows that four weeks later, neurons appear to be much more complex with many dendritic processes and branches present.

Figure 2
When fetal brains are exposed to decreased oxygen and blood flow (hypoxia and ischemia), the neurons do not appear to mature normally. On the left is a tracing of
a brain cell (neuron) from a control (Con) animal showing normal development of its complex branching pattern. On the right is a tracing of a neuron of the same age as the control, which was exposed to a brief period of hypoxia/ischemia (HI). This cell displays fewer processes and a simpler branch pattern. Thus, despite being the same age as the control animal, brain cells in the HI animal are more immature.

![Figure 3](image)

In the companion paper by Vinall et al., MRI anisotropy measurements of water diffusion demonstrated that human cortical development is abnormal in survivors of preterm birth. Here we address the cellular basis for the abnormal cortical MRI.

The figure on the left (A) is the cortical neuron tracing that was used to generate the 3D structure on the right (B). From the 3D structure in B, a calculation of theoretical water diffusion along the cell's processes was generated. With this approach, it was determined that water diffusion (anisotropy) was abnormal in the immature cells from the ischemic animals when compared with controls.

The anisotropy disturbances predicted from the cell-based calculations were in close agreement with the anisotropy disturbances detected by MRI of the cerebral cortex.
Thus, the cortical MRI abnormalities observed in the preterm survivors were in close agreement with the predictions of abnormal water diffusion calculated from the structure of the individual cortical cells.