1. We know there are multiple plasticity mechanisms active all at once. Hebbian plasticity underlies learning, but de-stabilises networks. Homeostatic plasticity ensures that even when learning occurs, the network remains in a stable state
2. Generally, though, when we speak of these, we refer to synaptic plasticity only. But, lots of evidence now confirms that, in fact, structural plasticity is active in the adult brain. So not only are synaptic weights changing, the connectivity of networks is changing too!
3. So, it isn't hard to imaging how if changes in synaptic strengths makes plasticity-stability an issue, how the removal and formation of whole synapses would have a much larger effect to plasticity-stability.
4. For the purpose of our analysis, we do not consider the modulation of synaptic strengths as structural plasticity (even though it is).

1. In the adult brain, now that we have the tech to look in at the microscopic structures that form synapses, we see that these are highly dynamic. They sprout and retract, forming and removing synapses. However, this must happen in a way that the brain remains functional: so, are their Hebbian and Homeostatic components of structural plasticity too?

1. The simplest way of studying homeostatic mechanism is to nudge the network out of its stable state. Lesion studies have, for a long time, observed structural changes after peripheral lesioning. A peripheral lesion is where you dont destroy the network itself, but you disrupt the input to it—we'll come to this in detail later.

1. With more tech, neuroscientists have been able to mark, track, and analyse micro-structures that are involved in synapses: boutons, spines, dendritic trees. So, there's recently quite a bit of data from these.

1. The protocol is pretty standard. Here, for a study in the visual cortex, the retinal field of a rat or a mouse is mapped.

1. Then, a part of the retina is lesioned. This cuts off inputs to a part of the visual cortex, as shown in the first figure. This forms the Lesion Projection Zone (LPZ). By repeated imaging of the region over months, the reorganisation of the network is tracked.
2. Other lesion studies use similar methods: digit removal, whisker trimming, and so on—anything that cuts off projecting activity on to a set of neurons.

1. So, if this a simple schematic, of the regions around the LPZ, this is what we know from these studies.
2. To permit a window for structural re-organisation.
3. To make up for lost excitation.
4. To make up for lost inhibition.

1. In the paper, we simply cite this bit and discuss it briefly, but here I'll explain it in more detail here.
2. Computational modelling stems from evidence from decades years ago. It was established that outgrowth depends on the change in the Calcium concentration in neurons.
3. So, in this figure, we see that a neurotransmitter causes a change in the Calcium of the neuron, and that causes some change in its outgrowth.
4. What this suggests, is that there's an optimal level of Calcium for neurons to have "normal" growth.
5. Based on this, Butz and van Ooyen came up with a framework for modelling structural plasticity. In this, they modelled the rate of change of synaptic elements as a Gaussian function of the neuron's Calcium concentration.

1. A stable fixed point where nothing occurs. If activity is less, sprouting, if more, retraction, but if very less, retraction
2. Note that they applied the same for excitatory and inhibitory post-synaptic elements
3. And, the same for all neurons: excitatory and inhibitory.

1. They replicated the peripheral lesion study, as you see.
2. You have repair from the outside in, and you have ingrowth of excitatory axons.

1. They suggested that post-synaptic elements should form at a lower activity level than pre-synaptic elements
2. They used an arbitrary homeostatic point, for all neurons
3. No discussion of inhibitory circuit here.
4. No discussion of biological realism of the model either.

1. We decided to start with a biologically realistic model: a cortical model proposed by Vogels et al.
2. This includes realistic conductances, for example.
3. This model is balanced by homeostatic inhibitory synaptic plasticity, and exhibits AI characteristics similar to cortical networks
4. This model does not have any spatial information incorporated it, but to model the LPZ and spatial analysis we do need it.

1. A few of these were made while incorporating spatial information in the model, keeping in mind that we want' to keep the model as realistica as we can manage.

1. Explain the simulation protocol.