What do twin studies look like for MDs?

What env factor may contribute to MD pre-natal, post-natal (baby, child, teen, young adult)

How could IPSCs help allocate individual treatment?

How could stem cells possibly help relieving drug attrition? (coming up with new drugs with are pre-clinically sound enough to pass clinical trials)

Does it make sense that EPCs are found in reduced numbers or with impaired functions in people with cardio-vascular (CV) issues?

Are the results of the 5 studies presented consistent: can EPCs be used as biomarkers for MD?

Would EPCs be just biomarkers or more than that? What are scientists suggesting?

What about VSELs?

Limitations of animals models to study MD/BD and using post-mortem human brains? How can one model try to model these diseases more appropriately?

If you want to use IPSCs to model MD/BD: criteria for the donors and how many do you think?

Explain epigenetics and how environmental cues may be lost in the reprogramming by losing epigenetics marks.

IPSCs-derived in BD- neurons seem to be functional and showcase how they can be used to predict response to Lithium treatment. (personalised medicine)

What do you think they mean by fibroblasts directly converted into neurons? Is there more than one way to do this? is this a problem?

These neurons also seem to respond to drugs as well as showing good phenotypes- model validation.

Is it going to be easy to model diseases in a dish and use them for drug screening? What about drug repurposing?

What do you think they mean by cell-type heterogeneity as an obstacle?

What is effect of increasing neurogenesis with MSC therapy in depressive rats versus normal rats?

Reduce depressive behaviour in depressed rats – does not affect behaviour of normal rats.

Try to summarise paper in a mind map