Exploration of the direction of cause-effect relationship between psychopathology and neuroplasticity

- Shrirang Bakhle, India. **World Congress of Psychiatry, March 2021**

Introduction

Neuroimaging reveals neuroplastic changes in various Mental Disorders.

Do these neuroplastic changes arise spontaneously and then cause the psychopathology? Or, are they the long-term effects of specific thinking of these patients?

Do these neuroplastic changes prove that the disorder is 'biological' in origin and not 'psychological' in origin?

Does repeated use of muscles (as in exercise) come *first* and then *later* give rise to muscle hypertrophy*? Or,* does muscle hypertrophy arise spontaneously – and that leads to repeated exercise?

Activity-dependent LTP, synaptic plasticity and neuroplasticity

These are commonly known mechanisms of memory formation. Specific repeated mental activity – such as trying to memorize – leads to long lasting changes in synapses and neurons.

Studies that show activity-dependent neuroplasticity

Maguire found significantly enlarged posterior hippocampi in London taxi drivers as compared to controls. (1) Did these people develop larger posterior hippocampi *first* - and *then* became taxi drivers? Or, the repeated mental activities required for driving were done *first* – and that gave rise to the neuroplastic changes *over time*?

Psychotherapy has been shown to induce neuroplastic changes in prospective studies. (2) This is another example of activity-dependent neuroplasticity.

Neuroplastic changes are found in patients of Gambling Disorder and other Behavioural Addictions. Do these changes occur spontaneously and then give rise to addicting thinking? Or, these changes develop as a result of the patients indulging in addiction related mental activities repeatedly?

Borders et al have shown how rumination gives rise to PTSD and depression. Rumination is nothing but doing specific mental activities repeatedly. (3)

Conclusion

Neuroimaging reveals neuroplastic changes. But it is important *not to* jump to the conclusion that these 'biological' changes are the *cause* of the specific 'psychological' patterns of thinking. They may be the *effect* of the thinking.

The only way to be sure of the direction of the cause-effect relationship is doing prospective studies.

References: (1) Maguire et al, (2000). Navigation-related structural change in the hippocampi of taxi drivers. Proceedings of the National Academy of Sciences of the United States of America, 97(8), 4398–4403. (2) Månsson et al, Neuroplasticity in response to cognitive behavior therapy for social anxiety disorder. Transl Psychiatry. 2016 Feb; 6(2): e727. (3) Borders et al, (2015). Sleep problems may mediate associations between rumination and PTSD and depressive symptoms among OIF/OEF veterans. Psychological Trauma: Theory, Research, Practice, and Policy, 7(1), 76-84. a0036937