



[ISSUE CoM] IGPM-Assistant Professor. Paola Magioncalda

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My academic training and research experience have provided me with a background in psychiatry and neuroscience. After graduating from medical school and specializing in Psychiatry (University of Genoa, Italy), I obtained a postgraduate research Master's degree in Affective Neuroscience (Maastricht University, Netherlands) and a Ph.D. degree in Neuroscience (University of Genoa, Italy). Firstly, as a research fellow, I conducted my research work at the Department of Neuroscience of the University of Genoa (Italy). I also spent several periods of training and research work at various international research centers as visiting researcher, including the Mind Brain Imaging and Neuroethics Center (University of Ottawa, Canada), Icahn School of Medicine at Mount Sinai (New York, U.S.), Research Center for Brain and Consciousness (Taipei Medical University, Taiwan), and Mental Health Centre and Psychiatric Laboratory (Sichuan University, Chengdu, China).

At the end of 2019, I started working as a medical researcher at Taipei Medical University – Shuang Ho Hospital. Finally, I joined Taipei Medical University as an assistant professor of the International Master/Ph.D. Program in Medicine (IGPM) of the College of Medicine and as a joint-appointment assistant professor of the Graduate Institute of Mind Brain and Consciousness (GIMBC).At Taipei Medical University, I had the opportunity to build my Lab, the Psychiatric Neuroscience Lab, which I am running with Dr. Matteo Martino (a TMU assistant professor of GIMBC), and continue developing my research work.

My long-term research interest is focused on understanding the relationship of brain functioning with phenomenal experience and behavior, as well as investigating the psychopathology and pathophysiology of major psychiatric disorders, especially bipolar disorder. These research areas are complementary in psychiatric neuroscience since a better understanding of brain physiology is fundamental for the study of brain alterations, while the investigation of the neurobiology of psychiatric disorders may provide valuable insights for a better comprehension of how the brain works. In this regard, bipolar disorder can be of particular relevance. Bipolar disorder is a major psychiatric disorder characterized by manic and depressive states that show opposite patterns of alterations across various psychopathological domains, including psychomotricity, affectivity, and thought. Thus, bipolar disorder can represent a unique model to investigate how changes in phenomenal and behavioral patterns are related to changes in brain functioning. On the other hand, a mechanistic understanding of the pathophysiology of psychiatric disorders, such as bipolar disorder, represents a crucial step for implementing specific diagnostic tools and effective therapies for these severe and debilitating illnesses.

In the last years, in strict collaboration with D

r. Matteo Martino, I conducted a series of research studies to investigate the potential neurobiological alterations underlying bipolar disorder, using various neuroimaging and laboratory techniques. Our research showed that mania and depression are related to distinct changes in the functional architecture of intrinsic brain activity. Such intrinsic brain activity represents the neuronal activity spontaneously p



duced by the brain and sets the baseline processing of inputs and outputs from and to the environment. Our findings suggested that a functional reconfiguration of intrinsic brain activity occurs in bipolar disorder with an opposite dysbalance between large-scale brain networks in mania and depression, which results in a relative over-tuning or de-tuning of intrinsic brain activity to the environment, finally manifesting in the opposite phenomenal-behavioral changes associated with manic and depressive symptomatology. Further results showed that such network dysbalance is associated with distinct changes in the subcortical-cortical coupling and neurotransmitter signaling. Finally, we detected abnormalities in the brain's white matter that correlated with immune-inflammatory alterations in patients with bipolar disorder.

Based on these data, we recently proposed a theoretical model of the pathophysiology of bipolar disorder aimed at linking changes in the immune-inflammatory status, damage of white matter, alterations in intrinsic brain activity, and psychopathology. Briefly, our model suggests that the core pathophysiological mechanism of bipolar disorder is traceable to immune/inflammatory-mediated damage of white matter involving the limbic tracts that destabilize the neurotransmitter signaling, and changes in such neurotransmitter signaling lead to phasic reconfigurations of intrinsic brain activity, resulting in a dysbalance between brain networks that clinically manifests in the manic-depressive symptomatology.

We are now conducting an ambitious project (funded by various TMU and NSTC grants) to test the specific hypotheses that emerged from our theoretical model of the pathophysiology of bipolar disorder. Moreover, based on this model, we are planning to implement studies using animal models to obtain a mechanistic understanding of the role of immune-mediated damage of white matter in bipolar disorder, as well as to test potential therapies to protect and repair the white matter. Data from these studies can make a relevant contribution to psychiatric neuroscience and potentially provide an innovative, effective, and neuroscientific-based treatment for bipolar disorder and other neuropsychiatric disorders.



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