Brain Inflammatory Responses Compromise NG2-Glial Homeostasis during Depression

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Major Depressive Disorder is a severe, chronic disorder affecting over 350 million people per year, and one out of every six people will be affected in their lifetime. Not only is this debilitating disease the leading cause of disability worldwide, but it also costs the United States alone over $50 billion per year. Current antidepressant treatments are ineffective in over one third of patients suffering from this disease, and the underlying mechanisms of depression are still unknown. There are several prominent hypotheses on what drives depression, including the inflammatory and glutamatergic hypotheses, which are heavily correlated with the specific glial cell populations microglia and NG2 glia, respectively. In this study, stress-induced depression was investigated in a murine model to compare the effects of depression on both microglia and NG2 glia. Fixed mouse brain tissues were obtained from control mice and mice that exhibited depressive-like symptoms. Tissues were sectioned and stained, and cells were traced to identify morphological changes. Statistically significant changes in microglial morphology of depressed mice suggest evidence of activation and inflammatory responses. Additionally, NG2 glial morphology, which is instrumental in their function, was affected with statistically significant decreases found in all dendritic aspects. Based on these results, stress signals appear to play a part in the alterations of NG2 glia and microglial activation, and these findings may lay the groundwork for further investigation into the relationship between depression, inflammation, and glutamatergic systems.

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