Narrative therapy, visualization, and brain neurons in Parkinson's disease

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Narrative therapy (story telling) and expressive poetry (sensory words) can be used to engage the mirror neurons and motor neurons in the brain and decrease the symptoms in Parkinson's disease. The kind of stories we listen to and whether we identify with the narrator or not influence what parts of our brain "lights up," get more blood flow, more nutrients, and more stimulation causing it to better develop or heal. This means the kind of stories we tell in our families and communities, the kind of speakers we hear, the kind of music we listen to influences the ability of our cortex to function. Story telling provides another doorway to greater brain health. Mirror neurons cause us to feel the actions of others in our own body. Motor neurons can be engaged through seeing another person move or through guided visualizations. An engaging story about someone walking is a brisk and balanced way and can create an image in the mind of someone with Parkinson’s disease. That image is then translated into a subtle contracting of the muscles needed to walk in that particular way. This stimulates the brain and nerve pathways to the muscles that are needed in order to do these actions. Research has also shown that injured athletes who visualize themselves doing their sport come back to the game with better skills than an athlete who doesn’t do any visualization.

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Development of preclinical diagnostics of Parkinson’s disease - strategy and recent progress

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At Parkinson’s disease (PD) motor symptoms first appear many years after the onset of degeneration of nigrostriatal dopaminergic neurons that explains low efficiency of treatment. Therefore the development of preclinical diagnostics based on a search for biomarkers mainly as a change in the composition of plasma and expression of specific genes and phenotype of blood cells in drug-naïve patients at the early clinical stage is of a high priority. Still, there is no guarantee that biomarkers, found at clinical stage are also a characteristic of preclinical stage. Therefore, we searched for biomarkers in experimental models of the earlier clinical and preclinical stages of PD (MPTP-treated mice) in addition to drug-naïve patients shortly after the appearance of motor symptoms. According to our data, the concentration of some markers in plasma, e.g., L-DOPA, were modified in the same way in mice at the presymptomatic and symptomatic stages of Parkinsonism and patients. The concentration of others, e.g., DOPAC differed at the presymptomatic stage in mice from those in mice at the symptomatic stage and patients. Apparently, the former is more reliable than the latter. Moreover, we have developed at experimental models a novel complementary approach to the preclinical diagnosis of PD by using a pharmacological provocation test, which induces short-term increase of a failure of the nigrostriatal system and motor dysfunctions. Peripheral biomarkers and a positive provocation test, found at prophylactic examination of humans would allow including them in a risk group for the final diagnosis with positron emission tomography.

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