Two faces: Galectin-3 in the gut-brain axis, neuroinflammation and behavior

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Gut-immune-nervous interactions became a fundamental research area for the comprising of mental diseases. Bidirectional pathways seem to regulate intestinal permeability and consequently the delivery of harmful molecules to the brain. Several studies appoint that increased intestinal permeability are directly related with symptoms as anxiety, depression, and addiction. Galectin-3, a betagaloctosides binding proteins, seems to be a potential link between intestinal permeability and brain disorders. At enterocytes, galectin-3 maintain cell-cell adhesions, limiting translocation of luminal factors to the bloodstream. Galectin-3 also promotes anti-inflammatory polarization of macrophages outside nervous system and regulates lymphocytes-plasma cells differentiation. Previous works of our group suggest that galectin-3 protects mice for gastric inflammation, however, other groups demonstrated galectin-3 in the central nervous system as a harmful molecule. In fact, a dichotomy in galectin-3 and behavior seems to be present. Several data suggest that global galectin-3 depletion or precisely Gut-Liver depletion affects mice behavior, while central nervous system administration of recombinant Galectin-3 promotes neuroinflammation. In this lecture, my aim is to demonstrate the dichotomy of galectin-3 in periphery and central nervous system as a potential mechanism of therapeutic approach for gut-brain linked disorders.