P.S. 1 - 1

EARLY-LIFE CRYPTOSPORIDIAL GASTROENTERITIS IS ASSOCIATED WITH EMBRYONAL FEATURES IN THE ADULT RAT HEART

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There is considerable evidence that hypertension and coronary heart diseases in adults originate from impaired growth and inflammation during neonatal development. Based on medical statistics, indicating that gastroenteritis is the most common cause of growth retardation and inflammation in children, we investigated the postponed effects of neonatal cryptospopridiosis on embryonic traits in the adult rat heart. We choose to investigate these traits because the returning to embryonic program is a well known adaptive mechanism of the failing heart to pathophysiologic conditions. Our data indicated that cryptosporidiosis is associated with embryonic traits in heart anatomy, cell morphology and molecular markers. The data of electrocardiography and doplerography revealed pulse rate retardation and interatrial septal defect known as patent foramen ovale.The results of cardiomyocyte image analysis and cytophotometry discovered cadriomyocyte remodeling seen from cell elongation and increased DNA copy number. Real-time PCR and immunocytochemistry identified overexpression of well-known fetal markers including myosin heavy chain beta (MYH7), hypoxia inducible factor 1 (HIF1a) and cyclin 1A (CCNA1). Overall, these results suggest that neonatal cryptosporidiosis triggers long-term and coordinated stress response including the return to embryonic gene program associated with progressive heart function decline.

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