

Bransfield, Robert

Robert C. Bransfield, MD, DLFAPA

Clinical Associate Professor

Rutgers Robert Wood Johnson Medical School

Private Practice in Psychiatry

Red Bank, NJ

www.MentalHealthandIllness.com



The Psychoimmunology of Lyme and Associated Diseases

Dr. Bransfield's primary activity is an office based private practice of psychiatry. In addition, Dr Bransfield is the Past President of the International Lyme and Associated Diseases Educational Foundation, Past President of the International Lyme and Associated Diseases Society, Past President of the New Jersey Psychiatric Association and has held a number of administrative positions with organizations involved with health, mental health and community related activities.

He is a Clinical Associate Professor of Psychiatry at Rutgers–Robert Wood Johnson Medical School and has previously held teaching appointments at Hahnemann Medical College, Drexel University School of Medicine and Eastern Virginia Medical School. He has taught in many settings to physicians, mental health professionals and the public. He has performed research, and has a particular interest in a unified theory of mental health and illness, the link between microbes and mental illness, the causes of autism, Lyme and other tick-borne disease, the link between microbes and violence, psychopharmacology, pharmaceutical benefit management, mental health parity, healthcare delivery issues and preserving the integrity of the physician patient relationship. Dr Bransfield he is the founder and administrator of the Microbes and Mental Illness Listserv. He has authored a number of articles on

topics related to tick-borne disease, psychiatry and healthcare policy issues. He has held a number of administrative positions for various organizations involved with a number of health, mental health and community related activities.

Conference Lecture Summary

Attention to psychoimmunology helps us understand the pathophysiological sequence that begins as a tick-borne or other infection and results in psychiatric symptoms. The nervous system and immune system communicate with each other and have many similarities—both have innate and adaptive capabilities, both involve complex communication between cells, both have similar pathophysiological processes. Many genes associated with mental illness involve immune functioning. Although there are multiple other contributors that provoke and weaken the immune system, acute and persistent infections are a major cause of pathological immune reactions resulting in disease progression. Adaptive functioning involves recognition of danger and early inflammation followed by adaptive immunity. *Borrelia burgdorferi* has the capacity to evade and suppress the immune system. In pathophysiological processes, this can result in persistent infection, persistent inflammation with cytokine effects without adaptive immunity, sometimes accompanied with autoimmune reactions. Persistent infection in the body can result in persistent immune effects that cross the blood brain barrier and result in neuropsychiatric symptoms. Sickness syndrome associated with interferon treatment and autoimmune limbic encephalopathies are models to understand inflammatory and molecular mimicry effects upon neuropsychiatric symptoms. Progressive inflammatory reactions have been proposed as a model to explain disease progression in depression, psychosis, dementia, epilepsy, autism, suicide, violence and other mental illnesses. Pathophysiological changes have been associated

with oxidative stress, excitotoxicity, changes in homocysteine metabolism and altered tryptophan catabolism. Lyme disease has been associated with the proinflammatory cytokines IL-6, IL-8, IL-12, IL-17, IL-18, IL-10 and interferon-gamma, the chemokines CXCL12 and CXCL13, CRP, CNS gliosis, Bb impacting neuronal and Schwann and glial cells, proinflammatory lipoproteins, increases in quinolinic acid, Bb surface glycolipids and flagella antibodies eliciting anti-neuronal antibodies and anti-neuronal antibodies and dissemination of immune reactions from the periphery to inflame the brain. Immune mediated effects of Lyme and other tick-borne diseases contribute to cognitive impairments, dementia, depression, anxiety, autism, violence and other psychiatric illnesses. Autism spectrum disorders associated with Lyme/tick-borne diseases may be mediated by a combination of inflammatory and molecular mimicry mechanisms. Reactivity to OspA and/or OspB appears significant in Lyme associated autism.

Greater interaction is needed between infectious disease specialists, immunologists and psychiatrists to benefit from this awareness and to further understand these mechanisms.