BIOGRAPHICAL SKETCH

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NAME	POSITION TITLE
Kelley, Keith W.	**Professor of Immunophysiology**
eRA COMMONS USER NAME (credential, e.g., agency login) kwkelley	

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	MM/YY	FIELD OF STUDY
Illinois State University, Normal, IL	B.S. (Honors)	1969	Agriculture
University of Illinois, Urbana-Champaign, IL	M.S.	1973	Anim. Sci., Physiology
University of Illinois, Urbana-Champaign, IL	Ph.D.	1976	Anim. Sci., Physiology

A. Personal Statement

Dr. Kelley is an American immunophysiologist, researcher and academic who served as Editor-In-Chief of the scientific journal *Brain, Behavior, and Immunity* from 2003-2017 (Impact Factor > 6.0). Prior to this he served as President of the international Psychoneuroimmunology Research Society (PNIRS). The broad goal of Dr. Kelley's research program has focused on discovering interactions between the nervous and immune systems and the relationship between behavior and health. These career research contributions have significantly impacted the major fields in psychoneuroimmunology, including some of the first studies on the effects of stress on immunity that continue today with Chinese colleagues, the restoration of immune functions in aged subjects by pituitary-derived hormones, the role of hormones on susceptibility to infectious disease and hematopoiesis, the molecular mechanisms that mediate the inhibitory effects of the proinflammatory cytokine TNFα on muscle, cancer and neuronal cells and the underpinnings of sickness and depressive-like behaviors. Throughout his 40 year career, Dr. Kelley has published over 350 scientific articles and book chapters and trained 58 graduate and post-doctorate students. Since 1978 he has been the principal or co-investigator of consecutive NIH grants totaling almost 40 million USD and served on six dozen NIH study sections. Professor Kelley has presented over 350 invited lectures in countries that include Australia, China, Taiwan, South Korea, Vietnam, Western and Eastern Europe, Russia, and South Africa. In 2011, he was elected as a Fellow of the American Association for the Advancement of Science in Medical Sciences for "…exceptional and scholarly contributions in brain, behavior, and immunity by recognizing and advancing the physiology of immunology and its role in communicating with the brain." Since 2012, he has served as the Founder and Chairperson of the PNIRS**Asia-Pacific** affiliate and recently launched the PNIRS**Asia-Pacific** Global Research Connections website (https://pnirs.ansc.illinois.edu/#).

B. Positions and Honors

1976-1984	Assistant Professor, Associate Professor, Department of Animal Science, Washington State University
1982-1983	Chargé de Recherches d'Immunologie, Institut National de la Recherche Agronomique (INRA), Station de Recherches de Virologie et d'Immunologie, Paris, France (Sabbatical leave)
1983	Research Fellow, Immunology, Institut National de la Santé et de la Recherche Medicale (INSERM), Laboratoire de Neurobiologie du Comportement, Bordeaux, France (leave of absence, 3 mos.)
1984-2011	Professor of Immunophysiology, Department of Animal Sciences in the College of ACES and in the Department of Pathology in the College of Medicine, Graduate School Faculty, Integrative Immunology and Behavior Faculty, Neuroscience Program Faculty, Nutritional Sciences Program Faculty, University of Illinois at Urbana-Champaign
1987	Professor of Immunology, INSERM, Laboratoire de Neurobiologie du Comportement, Bordeaux, France (4 mos.)
1987-1988	USDA Grants Program Manager, Competitive Biotechnology Research Grant Program (25%)
2011-2021	Professor, Department of Pathology in the College of Medicine, University of Illinois at Urbana-Champaign
2011-Present	Professor Emeritus of Immunophysiology, Department of Animal Sciences in the College of ACES, Neuroscience Program Faculty, Nutritional Sciences Program Faculty, University of Illinois at Urbana-Champaign
2021-Present	Visiting Scholar, Shenzhen University, School of Psychological Sciences (https://psy.szu.edu.cn/en/)

Outstanding Achievements and Service

1969	Honors Graduate, Illinois State University
1982	Visiting Professor of Immunology, INRA, Paris, France
1983	Visiting Professor of Immunology, INSERM, Bordeaux, France
1987	Visiting Professor of Immunology, Ecole Practique des Hautes Etudes and INRA, Bordeaux, France
1987	National Animal Management Award, ASAS, Sponsored by Merck Research Laboratories

1988 Gordon Conference Invited Speaker, "Biology of Aging"
1991 Distinguished Lecturer, UCLA Task Force in Psychoneuroimmunology
1991 USSR Academy of Medical Sciences, Invited Scientist to USSR
1992 Scientific Council, Psychoneuroimmunology Research Society, Elected by Membership
1992 College Paul A. Funk Recognition Award for Outstanding Research, ACES, University of Illinois
1992 Samuel Brody Memorial Lecture, University of Missouri
1994 Gordon Conference Invited Speaker, "NeuroEndocrinImmunology"
1994 Secretary-Treasurer, Psychoneuroimmunology Research Society, Elected by Membership
1994 University-Wide Senior University Scholar Award for Faculty Excellence, University of Illinois
1995 Wellcome Visiting Professorship in Basic Medical Sciences, Kansas State University
1996 Gordon Conference Invited Speaker, "Prolactin and Growth Hormone in the Immune System"
1996 Scientific Council, Society of Leukocyte Biology
1997 Gordon Conference Invited Speaker, "NeuroEndocrinImmunology"
1997 Department H.H. Mitchell Award for Excellence in Graduate Teaching & Research, University of Illinois
1997 College Senior Faculty Award for Excellence in Research, ACES, University of Illinois
1997 National Animal Physiology and Endocrinology Award, ASAS, Sponsored by Protiva, a Unit of Monsanto Company
1999 President, Psychoneuroimmunology Research Society, Elected by Membership
2000 Scientific Council, International Society of Neuroimmunomodulation, Elected by Membership
2003 Editor-in-Chief of "*Brain, Behavior, and Immunity*," Selected by the PsychoNeuroImmunology Research Society
2003 National Norman Cousins Memorial Lecture and Award, Psychoneuroimmunology Research Society
2005 The Jim Flood Memorial Lecture Distinguished Lectureship, Saint Louis University Summer Geriatric Institute
2009 International Advisory Board of Brainimmune.com
2011 Elected as a Fellow, Medical Sciences, in the American Association for the Advancement of Science (AAAS)
2014 Scientific Organizing Committee, International Society of Neuroimmunomodulation, Liege, Belgium
2015 Plenary International Lecture, "Interaction of nervous and immune systems," St. Petersburg, Russia
2016 Keynote Speaker, Triangle Chapter of the Society for Neuroscience, 2015 Spring Neuroscience Day, Chapel Hill, N.C.
2016 Organizer and Chair, PNIRS**China** symposium International Conference of Physiological Sciences, Beijing, China
2016 Co-Chair of joint meeting symposium for PNIRS**China**, International Stress and Behavior Society, Zhanjiang, China
2017 Keynote Speaker, "Nourishing the Mind & Body: From Immune to Neuroscience," China Medical University, Taiwan
2017 Keynote Speaker, III Congress of the Mexican Society of Neuroimmunoendocrinology, Mazatlán, Sinaloa, Mexico
2017 Organizer and Chair, PNIRS**Asia-Pacific** symposium, Australasian Neuroscience Society, Sydney, Australia
2018 Co-organizer and Chair, PNIRS**Asia-Pacific** symposium China Medical University, Taichung, Taiwan
2018 Organizer and Chair, PNIRS**Asia-Pacific** symposium, Conference of Physiological Sciences, Nanchang, China
2019 Co-organizer and Chair, PNIRS**Asia-Pacific** symposium, Japanese Neuroscience Society, Niigata Japan
2019 Organizer and Chair, PNIRS**Asia-Pacific** symposium, International Brain Research Organization World Congress of Neuroscience Daegu, Korea
2019 Organizer and Chair, PNIRS**Asia-Pacific** symposium China Medical University, Taichung, Taiwan
2020 Organizer and Chair, PNIRS**Asia-Pacific** symposium China Medical University, Taichung, Taiwan
2021 Organizer and Chair, PNIRS**Asia-Pacific** symposium China Medical University, Taichung, Taiwan

Service: Full Member, Neuroendocrinology, Neuroimmunology, Rhythms and Sleep (NNRS) Study Section (14-18); NIMH SEP K99/R00 Awards (15); NIGMS Review Panel, SEP for COBRE (14); Transformative TR01 NIH Review Panel (13); DoD CDMRP Clinical/Innovative Review Panel (13); Transformative TR01 NIH Review Panel (12); Neurotoxicology and Alcohol (NAL) NIH Study Section (11); NIH Distinguished Review Panel, "Neuroscience, Brain Diseases and Aging" (10); Biological Rhythms and Sleep SEP (10); Inflammation & Obesity, NIDDKD PO1 (09); DoD CDMRP Clinical/Innovative Review Panel (09); Stress, the HPA and Aging, NIA PO1 (07,09); RC1 Challenge Grants (09); Innate Immunity & Inflammation SEP (08); NIMH Board of Scientific Counselors, Office of NIMH Director (08); IFCN-H SEP (08); NIMH Pathway to Independence K99 (07); SBIR/STTR SEP Cell Biology (07); Innate Immunity and Inflammation (05, 06); NIMH SEP on Exploratory Grant Applications (06); Full Member IFCN-2 (98-03); NIMH SEP on HIV and Psychiatric Comorbidity (05); Cell. Molec. Immunology A (04, 05); Cell Molec. Immunology B (04, 05); Allergy and Immunology (03); Expt. Immunology (88, 03, 04); Biobehavioral Regulation, Learning and Ethology (04); Neurobiology of Motivated Behavior (03); NINDS SEP on NeuroAIDS (02); Hematology-2 (01); IFCN-4 SEP (01); Neuroimmunology, Virology and AIDS (97-98; *ad hoc* in 96); IFCN-1 (99, 00); Respiratory and Applied Physiology (00); NIH Site Visit Team for NCI (Survival Signals in Mammalian Cells, 97; 98) and NIA (Immunobiology of Aging, 90; 92; 96); Brain Disorders and Clinical Neuroscience-4 (98); Biobehavioral and Behavioral Processes-2 (99); Allergy and Immunology (91; 95); Geriatrics and Rehabilitative Medicine (97); Psychobiological, Biological and Neurosciences (96; 98); Psychobiology, Behavior and Neurosciences (96); Psychopathology and Clinical Biology (88); Immunology (95); Neurology (87); Canadian Breast Cancer Research Initiative (00); USDA Competitive Biotechnology Grants Study Section (86-87; Program Manager in 88); Mentor for 5 Research Assistant Professors; Major Professor for 47 Post-Doctoral/Doctoral/M.D./Ph.D./M.S. students; Editorial Boards of Progress in NeuroEndocrinImmunology (88-92); Brain, Behavior, and Immunity (90-02); Animal Biotechnology (89-94); Neuroimmunomodulation (93-); Endocrinology (94-98); Neuroendocrinology (95-99), Current Pharmaceutical Design (03-),

International Journal of Medical Sciences (06-); International Journal of Tryptophan Research (10-19); Advisory Board of Brainimmune.com (09-)

C. Contributions to Science (https://en.wikipedia.org/wiki/Keith_W._Kelley; and www.orcid.org/0000-0002-6837-8793**)**

Selected Publications: (h-index = 65 by Web of Science; 80 by Scopus; 96 by Google Scholar; 284 peer-reviewed papers, 74 chapters, speaker at >350 international and national meetings)

1. Stress and immunity. Not long after synthetic glucocorticoids were shown to inhibit lymphocyte proliferation, but before IL-1 and IL-2 were characterized, the possibility that acute and chronic stressors might regulate cell-mediated immunity was unknown. We tested this hypothesis in mice, domestic animals and birds and found that a variety of stressors regulate delayed-type hypersensitivity and contact sensitivity responses *in vivo*. We were the first to discover that stressors could actually increase cell-mediated immune reactions in the skin, which contrasted with the popular view at that time that stress would only reduce immune responses. Since physiological responses to stressors require the brain to induce adaptive responses, data in these papers were some of the first to firmly establish a connection between the immune and central nervous systems. These data have been replicated many times in many laboratories.

a. Kelley, K.W. 1980. Stress and immune function: A bibliographic review. Ann. Rech. Vet. 11:445-478.
b. Blecha, F., R.A. Barry and K.W. Kelley. 1982. Stress induced alterations in delayed type hypersensitivity to SRBC and contact sensitivity to DNFB in mice. Proc. Soc. Exp. Biol. Med. 169:239-246.
c. Dantzer, R. and K.W. Kelley. 1989. Stress and immunity: An integrated view of relationships between the brain and the immune system. Life Sciences 44:1995-2008.
d. Wen, H., H. Ma, Q Cai, S. Lin, X. Lei, B. He, S. Wu, Z. Wang, Y. Gao, W. Liu, W. Liu, Q. Tao, Z. Long, M. Yan, D. Li, K.W. Kelley, Y. Yang, H. Huang and Q. Liu. 2018. Recurrent ECSIT V140A mutation triggers hyperinflammation and promotes hemophagocytic syndrome in extranodal NK/T-cell lymphoma. Nature Medicine 24:154-164
e. Cui, B., Y. Luo, P. Tian, F. Peng, J. Lu, Y. Yang, Q. Su, B. Liu, J. Yu, X. Luo, L. Yin, W. Cheng, F. An, B. He, D. Liang, S. Wu, P. Chu, L. Song, X. Liu, H. Luo, J. Xu, Y. Pan, Y. Wang, D. Li, P. Huang, Q. Yang, L. Zhang, B.P. Zhou, S. Liu, G. Xu, E.W.F. Lam, K.W. Kelley and Q. Liu. 2019. Stress-induced epinephrine enhances lactate dehydrogenase A and promotes cancer stem-like cells. J. Clin. Invest. 129:1030-1046.
f. Cui, B., F Peng, J. Lu, B. He, Q. Su, H. Luo, Z. Deng, T. Jiang, K. Su, Y. Huang, Z.U. Din, E.W.F. Lam, K.W. Kelley and Q. Liu. 2020. Cancer and stress: NextGen strategies. Brain, Behavior, and Immunity 93:368-383. https://doi.org/10.1016/j.bbi.2020.11.005

2. ***Pituitary Gland is Required for Resistance to Bacterial Infection***. The idea that protein hormones from the adenohypophysis could regulate immune events was relatively unexplored before 1980. Growth hormone declines with aging. We postulated and subsequently established that replacement therapy with GH reverses thymic atrophy that occurs during the aging process of all animals, a process that was once considered to be irreversible. We went on to demonstrate the expression and functional relevance of specific GH receptors on lymphoid and myeloid cells. Treatment of both macrophages and neutrophils with either GH or its downstream mediator, IGF-I, increased lysosomal enzyme content and superoxide anion production. These experiments culminated with data showing that animals lacking a pituitary gland die as a result of infection with *Salmonella typhimurium*, an effect that was reversed by administration of GH.

a. Kelley, K.W., S. Brief, H.J. Westly, J. Novakofski, P.J. Bechtel, J. Simon and E.B. Walker. 1986. GH_3 pituitary adenoma implants can reverse thymic aging. Proc. Natl. Acad. Sci. USA 83:5663-5667.
b. Edwards, C.K. III, S.M. Ghiasuddin, J.M. Schepper, L.M. Yunger and K.W. Kelley. 1988. A newly defined property of somatotropin: Priming of macrophages for production of superoxide anion. Science 239:769-771
c. Edwards, C.K., III., L.M. Yunger, R.M. Lorence, R. Dantzer and K.W. Kelley. 1991. The pituitary gland is required for protection against lethal effects of *Salmonella typhimurium.* Proc. Natl. Acad. Sci. USA 88:2274-2277.
d. Fu, Y.K., S. Arkins, G. Fuh, B.C. Cunningham, J.A. Wells, S. Fong, M.J. Cronin, R. Dantzer and K.W. Kelley. 1992. Growth hormone augments superoxide anion secretion of human neutrophils by binding to the prolactin receptor. J. Clin. Invest. 89:451-457.

3. Leukocytes Synthesize Hormones. Cells of the immune system were once believed to be the source of all cytokines. At that time, hormones were considered to be synthesized only by cells of the endocrine system. We were the first to use the relatively new technique of Northern blotting to clearing establish the presence of steady-state transcripts for the pro-opiomelanocortin precursor of ACTH in cells of the immune system. We went on to show the leukocytes could also synthesize prolactin and IGF-I and to establish that these neuroendocrine hormones influence a variety of immune events. These data paved the way for development of new concepts that now recognize the existence of bidirectional communication pathways between the nervous and immune systems. It is now known that several neurotransmitters are also synthesized by leukocytes, including acetylcholine, which was the first neurotransmitter ever discovered. We went on to show that communication is possible because both the nervous and immune systems share a common biochemical language involving shared ligands such as neurotransmitters, neuropeptides, neuroendocrine hormones and cytokines along with their respective receptors.

a. Westly, H.J., A.J. Kleiss, K.W. Kelley, P.K.Y. Wong and P.H. Yuen. 1986. Newcastle disease virus infected splenocytes express the pro opiomelanocortin gene. J. Exp. Med. 163:1589-1594.
b. Arkins, S., N. Rebeiz, D.L. Brunke-Reese, A. Biragyn and K.W. Kelley. 1995. Interferon-γ inhibits macrophage insulin-like growth factor-I synthesis at the transcriptional level. Molecular Endocrinology 9:350-360.
c. Sabharwal, P., R. Glaser, W. Lafuse, S. Varma, Q. Liu, S. Arkins, R. Kooijman, L. Kutz, K.W. Kelley and W.B. Malarkey. 1992. Prolactin synthesized and secreted by human peripheral blood mononuclear cells: An autocrine growth factor for lymphoproliferation. Proc. Natl. Acad. Sci. USA 89:7713-7716.
d. Liu, Q., R.W. VanHoy, J.H. Zhou, R. Dantzer, G.G. Freund and K.W. Kelley. 1999. Elevated cyclin E levels, inactive retinoblastoma protein and suppression of the $p27^{KIP1}$ inhibitor characterize early development of promyeloid cells into macrophages. Molecular and Cellular Biology 19:6229-6239.

4. ***Pro-inflammatory Cytokines Cause Hormone Resistance***. The flip side of pituitary hormones enhancing immunity is cytokines from the innate immune system affecting the biological properties of hormones. Many cytokines and hormones share intracellular biochemical substrates and transcription factors, which provide a molecular basis for intracellular crosstalk between the endocrine and immune systems. In response to inflammatory processes such as tissue injury, low grade chronic infections or stress, pro-inflammatory cytokines not only reduce food consumption but they also antagonize cellular responses to the major anabolic growth-promoting hormones, insulin, GH and IGF-I. The latter event leads to a state of cytokine-induced hormone resistance. Our discoveries contributed to acceptance of this concept by proving that low picogram concentrations of proinflammatory cytokines directly impair IGF-I receptor signaling, thereby inhibiting the actions of IGF-I, leading to the induction of apoptosis, impairment in cell proliferation and a reduction in protein synthesis. These findings established that IGF-I acts like an anti-inflammatory cytokine to counterbalance the inflammatory properties of the major pro-inflammatory cytokines, IL-1 and TNF. These discoveries provided a conceptual advance in understanding how the immune and endocrine systems communicate with one another.

a. Venters, H.D., R. Dantzer and K.W. Kelley. 2000. A new concept in neurodegeneration: TNFα is a silencer of survival signals. Trends in Neurosciences 23:175-180.
b. Shen, W.H., Y. Yin, S.R. Broussard, R.H. McCusker, G.G. Freund, R. Dantzer and K.W. Kelley. 2004. Tumor necrosis factor α inhibits cyclin A expression and retinoblastoma hyperphosphorylation triggered by insulin-like growth factor-I induction of new E2F-1 synthesis. J. Biological Chemistry 279:7438-7446.
c. Broussard, S.R., R.H. McCusker, J.E. Novakofski, K. Strle, W.H. Shen, R.W. Johnson, R. Dantzer and K.W. Kelley. 2004. IL-1β impairs insulin-like growth factor I-induced differentiation and downstream activation signals of the insulin-like I growth factor receptor in myoblasts. J. Immunology 172:7713-7720.
d. Strle, K., S.R. Broussard, R.H. McCusker, W.H. Shen, J.M. LeCleir, R.W. Johnson, G.G. Freund, R. Dantzer and K.W. Kelley. 2006. C-jun N-terminal kinase mediates TNFα suppression of differentiation in myoblasts. Endocrinology 147:4363-4373.

5. ***Systemic Inflammation Causes Sickness and Depression***. A former post-doc, Steven Kent, showed that injection of IL-1, either systemically in the form of intraperitoneal administration or centrally via an intracerebroventricular route, caused clinical signs of sickness, as determined by motivational deficits in both eating and social behaviors. The amount of IL-1 required to induce similar amounts of sickness was approximately 100-200 times less when injected into the brain than when injected systemically. The most significant finding was that pretreatment of rats with the IL-1 receptor antagonist in the lateral ventricles of the brain significantly impaired the ability of systemic IL-1 to cause behavioral deficits. These early data showed clearly that inflammatory events in the periphery somehow communicate that message to the brain and lead to a change in social and eating behavior. We termed this and other behavioral events associated with acute inflammation as sickness behavior, which is orchestrated by brain cytokines. This concept of sickness behavior has now been extended to depression, If the peripheral immune response is too intense or remains unabated, symptoms of depression can emerge. The transition from sickness to depression is mediated by activation of the tryptophan degrading enzyme, indoleamine 2,3 dioxygenase (IDO) and the production of kynurenine metabolites. Pharmacologic inhibition or genetic deletion of IDO prevents development of depression-like behaviors following peripheral immune activation. IGF-I was also shown to impair development of inflammation-induced sickness and depression behaviors. These discoveries created a paradigm shift that offered a new framework to understand the basis of a number of normal and abnormal behaviors as well as regulation of immunity by T_{regs}.

a. Kent, S., R.M. Bluthé, R. Dantzer, A.J. Hardwick, K.W. Kelley, N.J. Rothwell and J.L. Vannice. 1992. Different receptor mechanisms mediate the pyrogenic and behavioral effects of interleukin 1. Proc. Natl. Acad. Sci. USA 89:9117-9120.
b. O'Connor, J.C., M.A. Lawson, C. André, E.M. Briley, S.S. Szegedi, J. Lestage, N. Castanon, M. Herkenham, R. Dantzer and K.W. Kelley. 2009. Induction of IDO by Bacille Calmette-Guérin is responsible for development of murine depressive-like behavior. J. Immunology 182:3202-3212.
c. Dantzer, R., J.C. O'Connor, G.G. Freund, R.W. Johnson and K.W. Kelley. 2008. From inflammation to sickness and depression: When the immune system subjugates the brain. Nature Reviews Neuroscience 9:46-57.
d. Raison, C.L., M. Lawson, R. Dantzer, K.W. Kelley, B.J. Woolwine, G. Vogt, J. Spivey, K. Saito and A.H. Miller. 2010. CSF concentrations of brain tryptophan and kynurenines during immune stimulation with IFNα: Relationship to CNS immune responses and depression. Molecular Psychiatry 15:393-403.
e. Kelley, K.W. 2017. To boldly go where no one has gone before. Brain, Behavior, and Immunity 66:1-8.

f. He, B, R. R. Gao, D. Lv, Y. Wen, L. Song, X. Wang, S. Lin, Q. Huang, Z. Deng, Z. Wang, M. Yan, F. Zheng, E.W.F. Lam, K.W. Kelley, Z. Li and Q. Liu. 2019. The prognostic landscape of interactive biological processes presents treatment responses in cancer. EBio Medicine pii: S2352-3964(19)30070-2. doi: 10.1016/j.ebiom.2019.01.064.

g. Kelley, K.W. and A. Shimada. 2020. Neuroinflammation and the blood-brain-interface: new findings in brain pathology. Clinical and Experimental Neuroimmunology. doi: 10.1111/cen3.12558.

h.Kelley, K.W. and S. Kent. 2020. The legacy of sickness behaviors. Frontiers in Psychiatry. 11:607269. https://doi.org/10.3389/fpsyt.2020.607269

i. Kelley, K.W., Y.P. Peng, Q. Liu, H.C. Chang, S.J. Spencer, M. Hutchinson and A. Shimada. 2020. PsychoNeuroImmunology goes east: Development of the PNIRS**China** affiliate and its expansion into PNIRS **Asia-Pacific**. Brain, Behavior, and Immunity. 88:75-87. doi.org/10.1016/j.bbi.2020.04.026

j. Kelley, K.W. 2021. From psychoneuroimmunology to immunopsychiatry: An historical perspective. In G. Khandaker, N. Harrison, R. Dantzer and E. Bullmore (eds.) Immunopsychiatry: An Introduction. Cambridge University Press, Cambridge, United Kingdom. pages 25-50. www.cambridge.org/9781108424042