УНИВЕРЗИТЕТ У КРАГУЈЕВЦУ
ФАКУЛТЕТ МЕДИЦИНСКИХ НАУКА
КАТЕДРА ЗА ПСИХИЈАТРИЈУ

ДЕКАНУ ФАКУЛТЕТА МЕДИЦИНСКИХ НАУКА
УНИВЕРЗИТЕТА У КРАГУЈЕВЦУ
ПРОФ. ДР ВЛАДИМИРУ ЈАКОВЉЕВИЋУ

Предмет: **Записник са седнице Катедре за психијатрију**

Састанак Катедре за психијатрију је заказан 25. 1. 2024. године, са почетком у 12 сати, са следећим дневним редом:

1. Утврђивање предлога, који је поднела проф. Милица Боровчанин, за избор проф. Моноџит Дебната у звање гостујућег професора;
2. Организација наставе у наступајућем семестру;
3. Израда уџбеника.

Одсуство су оправдали доц. др Данијела Ђоковић и асс. др Немања Мурић., остали чланови катедре су били присутни.

Након дискусије су донете следеће одлуке:

- Већином гласова утврђен је предлог за избор проф. Моноџит Дебната у звање визитинг професора. Проф. Мирјана Јовановић је била против избора са објшњењем да проф. Моноџит Дебнат нема задовољавајуће референце за избор, да његова област базичног образовања није медицина ни психијатрија, те да су његово поље истраживања базичне науке, а не област психијатрије
- Једногласно је утврђен предлог за именовање доц. др Бранимира Радмановића за руководиоца предмета Психијатрија на Интегрисаним академским студијама медицине на енглеском језику;
- Усвојене су теме, обим текста и начин припреме уџбеника.

проф. др Горан Михајловић,
Шеф Катедре за психијатрију

У Крагујевцу, 25. 1. 2024. године

Curriculum vitae

Name	: DR. MONOJIT DEBNATH
Present Position	: Professor and Head
Address	: Department of Human Genetics National Institute of Mental Health & Neurosciences Hosur Road, Bangalore-560029, INDIA
Qualifications	: M.Sc., Ph.D.
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Nationality	: Indian

ACADEMIC PROFILE

2006	**PhD (Thrust Area: Immunogenetics),** University of North Bengal, W.B., INDIA **Title of the Thesis**: Analysis of etiology of the delusional disorder, a model monosymptomatic psychotic disorder on the basis of immunogenetic and cytological investigations.
1998	**Master of Science (MS)** in Zoology (specialization in Immunology and Cell Biology), University of North Bengal, W.B., INDIA.
1996	**Bachelor of Science (BS)** in Zoology, St. Joseph's College, Darjeeling, W.B., INDIA.

RESEARCH AND PROFESSIONAL EXPERIENCE

Duration	**Post held**	**Name of the Institute**	**Thrust Area**
1999-2005	Doctoral student	North Bengal University, INDIA	Immunogenetics
2006-2007	Postdoctoral Researcher	Kunming Institute of Zoology, CHINA	Population Genetics
2007-2008	Junior Scientist	National Institute of Biologicals, INDIA	Quality control studies of blood products
2009	Visiting Researcher	Kunming Institute of Zoology, CHINA	Population Genetics
2009 – 2010	Postdoctoral Researcher	St. Louis Hospital, Paris and Mondor Institute of Biomedical Research, Creteail, France	Immunogenetics
Jan, 2012-June, 2015	Assistant Professor	NIMAHNS, Bangalore, India	Immunogenetics of CNS disorders
June 2015-June, 2018, 2018	Associate Professor	NIMAHNS, Bangalore, India	Immunogenetics of CNS disorders
July, 2018- June, 2022	Additional Professor	NIMAHNS, Bangalore, India	Immunogenetics of CNS disorders
April 2018-March 2022	Adjunct faculty	Department of Clinical Neuroscience, NIMHAANS, India	Immunogenetics of CNS disorders
July 2022- till date	Professor	NIMHANS, Bangalore, India	Immunogenetics of CNS disorders
April, 2023-till date	Professor and Head of the Department	NIMHANS, Bangalore, India	Immunogenetics of CNS disorders

AWARDS/MEDAL/ACADEMIC DISTINCTIONS:

- **University Gold Medal** for securing first position in M.Sc.(MS) Examination, University of North Bengal, Siliguri, W.B., INDIA.

- **Travel Support Award** from 13th International Histocompatibility Workshop and Congress group, May 12-22, 2002, Seattle, USA.

- **Young Scientist Award** by the Department of Zoology, University of North Bengal and Zoological Society of Calcutta in the National Symposium on "Assessment and Management of Bioresources", 2003.

- **Sudev Bhusan Ghosh Young Scientist Award** for the year 2003, Zoological Society, Kolkata, INDIA.
- **First Grade Research Award (2006),** China Postdoctoral Science Foundation, China (No. 20060400308).
- **Best Poster Award (1st Prize),** in the International update on Neuromuscular Disorders (NERVECON-III), August 24-26, 2018, Hyderabad.
- **International Society of Gene and Cell Therapy (ISGCT) Faculty Award**, 2019, Gene Research Foundation, Bangalore, India.

FELLOWSHIPS

- **Post-Doctoral Fellowship (2009-2010),** The Neuropole of research of Ile-de-**France** area (**NeRF**), France.
- **Post-Doctoral Fellowship (2009),** Kunming Institute of Zoology, Yunnan, P.R. China.
- **Post-Doctoral Fellowship 92006-2007),** Kunming Institute of Zoology, Yunnan, P.R. China.
- **Senior Research Fellowship (2004-2006),** Department of Science & Technology ,Govt. of India, New Delhi, India.
- **Senior Research Fellowship (2003-2003),** Labonya Prova Bose Trust, Kolkata, India.
- **Senior Research Fellowship (2000-2002),** Lady Tata Memorial Trust, Mumbai, India.

PUBLICATIONS

2001

1) **Debnath M,** Das SK, Ghosh P, Mandal BB and Chaudhuri TK(2000). Role of HLA Class-I antigens in Delusional Disorder. *Indian Journal of Psychiatry,* 42(3):275-279.

2003

2) **Debnath M,** Das SK, Bera NK, Nayak CR and Chaudhuri TK (2003). Association of HLA class-I antigens with delusional disorder. *International Journal of Human Genetics,* 3(3): 187-189.

2005

3) **Debnath M**, Das SK, Bera NK, Nayak CR and Chaudhuri TK (2005). A study of HLA-linked genes in a monosymptomatic psychotic disorder in an Indian Bengali Population. *Canadian Journal of Psychiatry*, 50(5):269-274.
4) **Debnath M**, Das SK, Bera N, Nayak C and Chaudhuri TK (2005). Study of HLA –linked genes in paranoid schizophrenia in an Indian Bengalee population. *International Journal of Human Genetics* 5(4):273-277.

2006

5) **Debnath M** and Chaudhuri TK (2006). Study of Genetic relationships of Indian Gurkha population on the basis of polymorphic HLA loci. *International Journal of Human Genetics* 6(2): 159-162.
6) **Debnath M**. and Chaudhuri, T.K. (2006). Distribution of HLA-A and B loci allele in Toto: a Sub Himalayan vanishing Indian tribe. *Tissue Antigens*, 67*:* 64-65.
7) **Debnath M**, Das SK, Bera NK, Nayak CR, Chaudhuri TK (2006). Genetic associations between Delusional Disorder and Paranoid Schizophrenia: a novel etiologic approach. *Canadian Journal of Psychiatry,* 51(6):342-349.
8) **Debnath M**. and Chaudhuri TK (2006). The role of HLA-G in cytokine homeostasis during early pregnancy complicated with maternal infections: a novel etiopathological approach to the neurodevelopmental understanding of schizophrenia. *Medical Hypotheses*, 66(2):286-293.

2011

9) **Debnath M**, Doyle KM, Langan C, McDonald C, Leonard B, Cannon DM. Recent advances in Psychoneuroimmunology: Inflammation in Psychiatric Disorder. *Translational Neuroscience* 2011;2:121-137.
10) **Debnath M,** Palanichamy MG, Mitra B, Jin QJ, Chaudhuri TK, Zhang YP. Y-chromosome haplogroup diversity in the sub-Himalayan Terai and Duars populations of East India. *Journal of Human Genetics*,2011 56:765-771.

2013

11) **Debnath M**, Cannon DM, Venkatasubramanian G. Variation in the major histocompatibility complex [MHC] gene family in schizophrenia: Associations and functional implications. *Prog Neuropsychopharmacol Biol Psychiatry*, 2013;42: 49-62.
12) Anderson G, Berk M, Dodd S, Bechter K, Altamura AC, Dell'Osso B, Kanba S, Monji A, Fatemi SH, Buckley P, **Debnath M**, Das UN, Meyer U, Muller N, Kanchanatawan B, Maes M. Immuno-inflammatory, oxidative and nitrosative stress, and neuroprogressive pathways in the etiology, course and treatment of schizophrenia, *Progress in Neuropsychopharmacology & Biological Psychiatry* 2013;42:1-4.
13) **Debnath** M, Mitra B, Bera NK, Chaudhuri TK, Zhang YP. Lack of association of IL-6 (-174 G>C) and TNF-α (-238 G>A) variants with Paranoid Schizophrenia in an Indian Bengalee Population. *Cytokine* 2013;61:455-458.
14) **Debnath M,** Busson M, Jamain S, Etain B, Hamdani N, Oliveira J, Boukouaci W, Amokrane K, Moins-Teisserenc H, Lajnef M, Bengoufa D, Malafosse A, Bellivier F, Hnery C, Kahn JP, Krishnamoorthy R, Charron D, Leboyer M, Tamouza R. The HLA-G low expressor genotype is associated with protection against bipolar disorder. *Human Immunology*, 2013:74:593-597.

15) Venkatasubramanian G, **Debnath M.** The TRIPS (Toll-like Receptors in Immune-inflammatory PathogenesiS) Hypothesis: A Novel Postulate to Understand Schizophrenia. *Progress in Neuropsychopharmacology & Biological Psychiatry,* 2013;44C:301-311.

16) **Debnath M**, Venkatasubramanian G. Recent advances in psychoneuroimmunology relevant to schizophrenia therapeutics. Current Opinion in Psychiatry 2013;26:433-439.

2014

17) Venkatasubramanian G, **Debnath M.** Neuroimmunological aberrations and cerebral asymmetry abnormalities in schizophrenia: select perspectives on pathogenesis. *Clinical Psychopharmacology and Neuroscience* 2014;12:8-18.

18) **Debnath M**, Berk M. Th17 pathway-mediated immunopathogenesis of schizophrenia: mechanisms and implications. *Schizophrenia Bulletin* 2014. 40(6):1412-21.

19) Palanichamy MG, Mitra B, **Debnath M**, Agrawal S, Chaudhuri TK, Zhang YP. Tamil merchant in ancient Mesopotamia. *PLoS ONE*. 2014 Oct 9;9(10):e109331.

2015

20) Rajasekaran A, Venkatasubramanian G, Berk M, **Debnath M**. Mitochondrial dysfunction in schizophrenia: Pathways, mechanisms and implications. *Neuroscience and Biobehavioral Reviews*, 2015 ;48C:10-21.

21) **Debnath M,** Venkatasubramanian G, Berk M. Fetal programming of schizophrenia: select mechanisms. *Neuroscience and Biobehavioral Reviews,* 2015; 49: 90-104.

22) Palanichamy MG, Mitra B, Zhang CL, **Debnath M**, Li GM, Wang HW, Agrawal S, Chaudhuri TK, Zhang YP. West Eurasian mtDNA lineages in India: an insight into the spread of Dravidian language and the caste origin. Human Genetics, 2015 ;134(6):637-47.

23) Callaly E, Walder K, Morris G, Maes M, **Debnath M**, Berk M. Mitochondrial Dysfunction in the Pathophysiology of Bipolar Disorder: Effects of Pharmacotherapy. Mini Rev Med Chem. 2015;15(5):355-65.

24) Oliveira J, **Debnath M**, Etain B, Bennabi M, Hamdani N, Lajnef M, Bengoufa D, Fortier C, Boukouaci W, Bellivier F, Kahn JP, Henry C, Charron D,Krishnamoorthy R, Leboyer M, Tamouza R. Violent suicidal behavior in bipolar disorder is associated with nitric oxide synthase 3 gene polymorphism. Acta Psychiatr Scand. 2015 Sep;132(3):218-25.

25) Rajasekaran A, Shivakumar V, Kalmady SV, Narayanaswamy JC, Venugopal D, Amaresha AC, Venkatasubramanian G, **Debnath M.** Soluble Human Leukocyte Antigen (sHLA)-G levels may predict early onset of schizophrenia in male patients. Tissue Antigens, 2015;86: 36-37.

26) Shivakumar V, Chhabra H, Subbanna M, Agarwal SM, Bose A, Kalmady SV, Narayanaswamy JC, **Debnath M**, Venkatasubramanian G. Effect of tDCS on Auditory Hallucinations in Schizophrenia: Influence of Catechol-O-Methyltransferase (COMT) Val158Met Polymorphism. Asian Journal of Psychiatry, 2015 Aug;16:75-7.

27) **Debnath M.** Adaptive Immunity in Schizophrenia: functional implications of T cells in the etiology, course and treatment. Journal of Neuroimmune Pharmacology 2015 Dec;10(4):610-9.

2016

28) Chhabra H, Shivakumar V, Agarwal SM, Bose A, Venugopal D, Rajasekaran A, Subbanna M, Kalmady SV, Narayanaswamy JC, **Debnath M**, Venkatasubramanian G. Transcranial

Direct Current Stimulation & Neuroplasticity Genes: Implications for Psychiatric Disorders. Acta Neuropsychiatrica, 2016 Feb;28(1):1-10.

29) Davis J, Eyre H, Jacka FN, Dodd S, Dean O, McEwen S, Debnath M, McGrath J, Maes M, Amminger P, McGorry PD, Pantelis C, Berk M. A review of vulnerability and risks for schizophrenia; beyond the two hit hypothesis. Neuroscience and Biobehavioral Reviews,2016;65:185-94.
30) Rajasekaran A, Shivakumar V, Kalmady SV, Narayanaswamy JC, Subbana M, Venugopal D, Amaresha AC, Venkatasubdramanian G, **Debnath M**. The impact of IL-10 polymorphisms and sHLA-G levels on the risk of schizophrenia. Asian Journal of Psychiatry, 2016;23: 39-43.
31) Rajasekaran A, Shivakumar V, Kalmady SV, Narayanaswamy JC, Subbana M, Venugopal D, Amaresha AC, Venkatasubramanian G, Berk M, **Debnath M**. The impact of HLA-G 3′ UTR variants and sHLA-G on risk and clinical correlates of schizophrenia. Human Immunology, 2016; 77(12):1166-1171.

2017

32) **Debnath M,** Berk M. Functional implications of the IL-23/IL-17 immune axis in Schizophrenia. Molecular Neurobiology, 2017;54(10):8170-8178.

33) Nagappa M, **Debnath M**, Taly AB. Enigmas in immunobiology of Guillain-Barré syndrome: Ganglioside antibodies and beyond! Neurol India. 2017 Sep-Oct;65(5):973-974.

2018

34) Shivakumar V, **Debnath M**, Venugopal D; Rajasekaran A, Kalmady SV, Subbanna M, Narayanaswamy JC, Amaresha AC; Venkatasubramanian G. Influence of correlation between HLA-G polymorphism and Interleukin-6 (IL6) gene expression on the risk of schizophrenia. Cytokine, 2018 Jul;107:59-64.
35) **Debnath M**, Nagappa M, Murari G, Taly AB. IL-23/IL-17 immune axis in Guillain Barré Syndrome: Exploring newer vistas for understanding pathobiology and therapeutic implications. Cytokine. 2018 Jan 10;103:77-82.
36) Kalmady SV, Agrawal R, Venugopal D, Shivakumar V, Amaresha AC, Agarwal SM, Subbanna M, Rajasekaran A, Narayanaswamy JC, **Debnath M,** Venkatasubramanian G. CHRFAM7A gene expression in schizophrenia: clinical correlates and the effect of antipsychotic treatment. J Neural Transm (Vienna). 2018 Apr;125(4):741-748.
37) Chhabra H, Shivakumar V, Subbanna M, Kalmady SV, Bose A, Agarwal SM, Sreeraj VS, Dinakaran D, Narayanaswamy JC, **Debath M,** Venkatasubramanian G.Gene Polymorphisms and response to Transcranial Direct Current Stimulation for Auditory Verbal Hallucinations in Schizophrenia. Acta Neuropsychiatrica 2018 Aug;30(4):218-225.
38) **Debnath M,** Berk M, Leboyer M, Tamouza R. The MHC/HLA complex in Major Psychiatric Disorders: Emerging roles and implications. Current Behavioral Neuroscience Reports, 2018;5: 179-188.

39) **Debnath M,** Nagappa M, Talukdar PM, Subbanna M, Sundaravadivel P, Shivakumar V, Dutta D, Wahatule R, Sinha S, Bindu PS, Periyavan S, Umamaheswara Rao GS, Taly

AB. Comprehensive cytokine profiling provides evidence for a multi-lineage Th responses in Guillain Barré Syndrome. Cytokine. 2018 Apr 25;110:58-62.

40) **Debnath M,** Nagappa M, Subbanna M, Sundaravadivel P, Talukdar PM, Shivakumar V, Wahatule R, Dutta D, Sinha S, Bindu PS, Periyavan S, Umamaheswara Rao GS, Taly AB. Th17 pathway signatures in a large Indian cohort of Guillain Barré syndrome. Journal of Neuroimmunology 2018; 323:125-130.

41) Subbanna M, Shivakumar V, Talukdar PM, Narayanaswamy JC, Venugopal D, Berk M, Varambally S, Venkatasubramanian G, **Debnath M.** Role of IL-6/RORC/IL-22 axis in driving Th17 pathway mediated immunopathogenesis of schizophrenia. Cytokine 2018;111:112-118.

42) Venugopal D, Shivakumar V, Subbanna M, Kalmady SV, Amaresha AC, Agarwal SM, Narayanaswamy JC, Banerjee M, **Debnath M**, Venkatasubramanian G. Impact of antipsychotic treatment on methylation status of Interleukin-6 [IL-6] gene in Schizophrenia. J Psychiatr Res. 2018 Sep;104:88-95.

43) Balachander GM, Talukdar PM, **Debnath M**, Rangarajan A, Chatterjee K.Inflammatory Role of Cancer Associated Fibroblasts in Invasive Breast Tumors Revealed Using a Fibrous Polymer Scaffold. ACS Appl Mater Interfaces. 2018 Oct 10;10(40):33814-33826.

2019

44) Polavarapu K, Preethish-Kumar V, Sekar D, Vengalil S, Nashi S, Mahajan NP, Thomas PT, Sadasivan A, Warrier M, Gupta A, Arunachal G, **Debnath M,** Keerthipriya MS, Pradeep-Chandra-Reddy C, Puttegowda A, John AP, Tavvala A, Gunasekaran S, Sathyaprabha TN, Chandra SR, Kramer B, Delhaas T, Nalini A. Mutation pattern in 606 Duchenne muscular dystrophy children with a comparison between familial and non-familial forms: a study in an Indian large single-center cohort. J Neurol. 2019 Sep;266(9):2177-2185.

45) Varun CN, Venkataswamy MM, Ravikumar R, Nagaraju R, **Debnath M**, Varambally S, Venkatasubramanian G, Ravi V. Th17 and MAIT cell mediated inflammation in antipsychotic free schizophrenia patients. Schizophr Res. 2019 Oct;212:47-53.

46) Varambally S, Venkatasubramanian G, Govindaraj R, Shivakumar V, Mullapudi T, Christopher R, **Debnath M.** Philip M, Bharath RD, Gangadhar BN. Yoga and schizophrenia—a comprehensive assessment of neuroplasticity: Protocol for a single blind randomized controlled study of yoga in schizophrenia. Medicine, 2019. Oct;98(43):e17399.

2020

47) Subbanna M, Shivakumar V, Venugopal D, Narayanaswamy JC, Berk M, Varambally S, Venkatasubramanian G, **Debnath M.** The impact of antipsychotic medication on IL-6/STAT3 signalling axis in peripheral blood mononuclear cells of drug-naïve schizophrenia patients. Psychiatry Clin Neurosci. 2020 Jan;74(1):64-69.

48) Shivakumar V, Sreeraj VS, Subbanna M, Kalmady SV, Amaresha AC, Narayanaswamy JC, **Debnath M**, Venkatasubramanian. Differential impact of IL6 promoter gene polymorphism on hippocampal volume in antipsychotic-naïve Schizophrenia patients. Indian Journal of Psychiatry, 2020 Jan-Feb;62(1):36-42.

49) Balaji R, Subbanna M, Shivakumar V, Abdul F, Venkatasubramanian G., **Debnath M**. Pattern of expression of Toll like receptor (TLR)-3 and -4 genes in drug-naïve and antipsychotic treated patients diagnosed with schizophrenia. Psychiatry Research, 2020 Mar;285:112727.

50) **Debnath M**, Nagappa M, Dutta D, Talukdar PM, Subbanna M, Shivakumar V, Wahatule R, Sinha S, Bindu PS, Periyavan S, Umamaheswara Rao GS, Kumar MA, Taly AB. Evidence of altered Th17 pathway signatures in the cerebrospinal fluid of patients with Guillain Barré Syndrome. J Clin Neurosci. 2020 May;75:176-180.

51) Nagappa M, Sharma S, Govindaraj P, Chickabasaviah YT, Siram R, Shroti A, **Debnath M**, Sinha S, Bindu PS, Taly AB PMP22 Gene-Associated Neuropathies: Phenotypic Spectrum in a Cohort from India. J Mol Neurosci. 2020 May;70(5):778-789.

52) **Debnath M,** Banerjee M, Berk M. Genetic gateways to COVID-19 infection: implications for risk, severity and outcomes. FASEB J, 2020 Jul;34(7):8787-8795.

53) Shivakumar V, Rajasekaran A, Subbanna M, Kalmady SV, Venugopal D, Agrawal R, Amaresha AC, Agarwal SM, Joseph B, Narayanaswamy JC, **Debnath M,** Venkatasubramanian G, Gangadhar BN. Leukocyte mitochondrial DNA copy number in schizophrenia. Asian Journal of Psychiatry, 2020;53:102193.

54) **Debnath M,** Berk M, Maes M. Changing dynamics of psychoneuroimmunology during COVID-19 pandemic. Brain, Behavior & Immunity-Health, 5 (2020) 100096.

55) Rajan R, Divya KP, Kandadai RM, Yadav R, Satagopam VP, Madhusoodanan UK, Agarwal P, Kumar N, Ferreira T, Kumar H, Sreeram Prasad AV, Shetty K, Mehta S, Desai S, Kumar S, Prashant LK, Bhatt M, Wadia P, Ramalingam S, Wali GM, Pandey S, Bartusch F, Hannussek M, Krüger J, Kumar-Sreelatha A, Grover S, Lichtner P, Sturm M, Roeper J, Busskamp V, Chandak GR, Schwamborn J, Seth P, Gasser T, Riess O, Goyal V, Pal PK, Borgohain R, Krüger R, Kishore A, Sharma M and the Lux-GIANT Consortium. Genetic Architecture of Parkinson's Disease in the Indian Population: Harnessing Genetic Diversity to Address Critical Gaps in Parkinson's Disease Research. Front. Neurol. 2020, 11:524. doi: 10.3389/fneur.2020.00524.

56) Talukdar PM, Abdul F, Maes M, Venkatasubramanian G, Kutty BM, **Debnath M.** Maternal immune activation causes schizophrenia-like behaviors in the offspring through activation of immune-inflammatory, oxidative and apoptotic pathways, and lowered antioxidant defences and neuroprotection. Molecular Neurobiology, 2020 Oct;57(10):4345-4361

57) Wahatule W, Dutta D, **Debnath M**, Nagappa M, Mahadevan A, Sinha S, Bindu PS, Sundaravadivel P, Rao GSU, Periyavan S, Rao S, Taly AB. Ganglioside complex antibodies in an Indian cohort of GBS. Muscle and Nerve, 2020 Sep-Oct;68(5):1084-1091.

58) Bhargav PH, Reddy PV, Govindaraj R, Gulati K, Ravindran A, Gayathri D, Karmani SJ, Udupa K, Venkatasubramanian G, Philip M, **Debnath M**, Bharath RD, Sathyaprabha TN, Gangadhar BN, Muralidharan K. Impact of a Course of Add-on Supervised Yoga on Cortical Inhibition in Major Depressive Disorder: A Randomized Controlled Trial. Can J Psychiatry. 2021 Feb;66(2):179-181.

59) Rajasekaran A, Shivakumar V, Kalmady SV, Parlikar R, Chhabra H, Prabhu A, Subbanna M, Venugopal D, Amaresha AC, Agarwal SM, Bose A, Narayanaswamy JC, **Debnath M,** Venkatasubramanian G. Impact of NRG1 HapICE gene variants on digit ratio and dermatoglyphic measures in schizophrenia. Asian Journal of Psychiatry, 2020: 54:102363.

60) Nagappa M, Sinha S, Mahadevan A, Bindu PS, Krishnan A, Ray S, **Debnath M,** Bharath RD, Rangan K, Jena SS, Mathuranath PS, Taly AB. Genetically Established Familial Amyloidotic Polyneuropathy from India: Narrating the Diagnostic 'Odyssey' and a Mini Review'. Neurology India, 2020. DOI: 10.4103/0028-3886.294550.

2021

61) Subbanna M, Shivakumar V, Jose D, Venkataswamy M, **Debnath M,** Ravi Vasanthapuram, Reddy J, Venkatasubramanian G, Narayanaswamy JC. Reduced T cell immunity in unmedicated, comorbidity free Obsessive-Compulsive Disorder: An Immunophrnotyping Study. Journal of Psychiatry Research, 2021 May;137:521-524.

62) **Debnath M,** Berk M, Maes M. Translational evidence for the Inflammatory Response System (IRS)/Compensatory Immune Response System (CIRS) and neuroprogression theory of major depression. Prog Neuropsychopharmacol Biol Psychiatry. 2021, 111:110343.

63) Abdul F, Sreenivas N, Kommu JVS, Banerjee M, Berk M, Maes M, Leboyer M, **Debnath M** Disruption of circadian rhythm and risk of autism spectrum disorder: role of immune-inflammatory, oxidative stress, metabolic and neurotransmitter pathways. Rev Neurosci. 2021 May 28;33(1):93-109.

64) Talukdar PM, Abdul F, Maes M, Berk M, Venkatasubramanian G, Kutty BM, **Debnath M**. A proof-of-concept study of maternal immune activation mediated induction of Toll-like receptor (TLR) and inflammasome pathways leading to neuroprogressive changes and schizophrenia-like behaviours in offspring. European Neuropsychopharmacology, 2021;52: 48-61.

65) Dutta D, **Debnath M**, Nagappa M, Das SK, Wahatule R, Sinha S, Taly AB, Ravi V. Antecedent infections in Guillain-Barré syndrome patients from south India. Journal of the Peripheral Nervous System, 2021; 26(3):298-306.

66) Subbanna M, Reddy PV, Bhargav PH, Talukdar PM, Abdul F, Karmani S, Arasappa R, Venkatasubramanian G, Muralidharan K, Gangadhar BN, **Debnath M.** Long-term add-on Yoga therapy offers clinical benefits in Major Depressive Disorder by modulating the complement pathway-a randomized controlled trial. Asian Journal of Psychiatry 2021;66:102876.

67) Gulati K, Bhargav PH, Reddy PV, Govindaraj R, Ravindran A, Gayathri D, Karmani SJ, Udupa K, Philip M, **Debnath M**, Bharath RD, Sathyaprabha TN, Venkatasubramanian G, Muralidharan K. Adjunct yoga therapy: Influence on heart rate variability in major depressive disorder - A randomized controlled trial. Asian Journal of Psychiatry, 2021 Sep 10;65:102832.

2022

68) Subbanna M, Shivakumar V, Bhalerao G, Varambally S, Venkatasubramanian G, **Debnath M**. Variants of Th17 pathway-related genes influence brain morphometric changes and the risk of schizophrenia through epistatic interactions. Psychiatric Genetics, 2022;32(4):146-155.

69) Sharma PP, Seshagiri DV, Nagappa M, Mullapudi T, Sreenivas N, Dey S, Shivaram S, Wahatule R, Kumawat V, Nair NVS, Kamath S, Sinha S, Taly AB, **Debnath M.** Role of altered IL-33/ST2 immune axis in the immunobiology of Guillain-Barré syndrome. European Journal of Neurology, 2022;29(7):2074-2083.

70) **Debnath M,** Dey S, Sreenivas, Pal PK, Yadav R. Genetic and epigenetic constructs of progressive supranuclear palsy. Annals of Neurosciences, 2022 Apr;29(2-3):177-188.

71) Maes M, Rachayon M, Jirakran K, Sodsai P, Klinchanhom S, **Debnath M**, Basta-Kaim A, Kubera M, Almulla AF, Sughondhabirom A. Adverse Childhood Experiences Predict the Phenome of Affective Disorders and These Effects Are Mediated by Staging, Neuroimmunotoxic and Growth Factor Profiles. Cells. 2022 May 7;11(9):1564.

72) Ramaswamy P, Christopher R, Kumar Pal P, **Debnath M**, Yadav R. Plasma microRNAs as a Potential Biomarker for Identification of Progressive Supranuclear Palsy. Diagnostics (Basel). 2022 May 11;12(5):1204.

73) Minic Janicijevic S, Jovanovic IP, Gajovic NM, Jurisevic MM, **Debnath M**, Arsenijevic NN, Borovcanin MM. Galectin-3 mediated risk of inflammation in stable schizophrenia, with only possible secondary consequences for cognition. World J Psychiatry. 2022 Sep 19;12(9):1183-1193.

74) Selvaraj S, Shivakumar V, Kavya PV, Mullapudi T, Bhalerao G, Sreeraj VS, Suhas S, Dinakaran D, Parlikar R, Chhabra H, Narayanaswamy JC, **Debnath M**, Rao NP, Muralidharan K, Venkatasubramanian G. Neurohemodynamic correlates of BDNF gene

expression in schizophrenia patients with working memory deficits: A functional MRI study. Asian J Psychiatr. 2022 Sep 17;77:103261.

75) Sharma S, Govindaraj P, Chickabasaviah YT, Siram R, Shroti A, Seshagiri DV, **Debnath M**, Bindu PS, Taly AB, Nagappa M. Genetic Spectrum of Inherited Neuropathies in India. Ann Indian Acad Neurol. 2022 May-Jun;25(3):407-416.

76) Dutta D, **Debnath M**, Seshagiri DV, Nair BVS, Das SK, Wahatule R, Sinha S, Ravi V, Taly AB, Nagappa M. Impact of Antecedent Infections on the Antibodies against Gangliosides and Ganglioside Complexes in Guillain-Barré Syndrome: A Correlative Study. Ann Indian Acad Neurol. 2022 May-Jun;25(3):401-406.

77) Gokulakrishnan K, Nikhil J, Vs S, Holla B, Thirumoorthy C, Sandhya N, Nichenametla S, Pathak H, Shivakumar V, **Debnath M**, Venkatasubramanian G, Varambally S. Altered Intestinal Permeability Biomarkers in Schizophrenia: A Possible Link with Subclinical Inflammation. Ann Neurosci. 2022 Apr;29(2-3):151-158.

78) Al-Hakeim HK, Al-Musawi AF, Al-Mulla A, Al-Dujaili AH, **Debnath M**, Maes M. The interleukin-6/interleukin-23/T helper 17-axis as a driver of neuro-immune toxicity in the major neurocognitive psychosis or deficit schizophrenia: A precision nomothetic psychiatry analysis. PLoS One. 2022 Oct 18;17(10):e0275839.

79) Sharma PP, Seshagiri DV, Nagappa M, Mullapudi T, Sreenivas N, Dey S, Shivaram S, Wahatule R, Kumawat V, Sreekumaran Nair BV, Kamath S, Sinha S, Taly AB, **Debnath M.** Modulatory effects of vitamin D on IL -33/ ST2 immune axis in Guillain-Barré syndrome…quo vadis? Eur J Neurol. 2022 Jul;29(7):e22-e23.

80) Dutta D, Nagappa M, Sreekumaran Nair BV, Das SK, Wahatule R, Sinha S, Vasanthapuram R, Taly AB, **Debnath M.** Variations within Toll-like receptor (TLR) and TLR signaling pathway-related genes and their synergistic effects on the risk of Guillain-Barré syndrome. J Peripher Nerv Syst. 2022 Jun;27(2):131-143.

2023

81) Gokulakrishnan K, Nikhil J, Viswanath B, Thirumoorthy C, Narasimhan S, Devarajan B, Joseph E, David AKD, Sharma S, Vasudevan K, Sreeraj VS, Holla B, Shivakumar V, **Debnath M**, Venkatasubramanian G, Varambally S. Comparison of gut microbiome profile in patients with schizophrenia and healthy controls - A plausible non-invasive biomarker? J Psychiatr Res. 2023 Jun;162:140-149.

82) Mullapudi T, **Debnath M**, Govindaraj R, Raj P, Banerjee M, Varambally S Effects of a six-month yoga intervention on the immune-inflammatory pathway in antipsychotic-stabilized schizophrenia patients: A randomized controlled trial. Asian J Psychiatr. 2023 May 24;86:103636.

83) Reddy PV, Talukdar PM, Subbanna M, Bhargav PH, Arasappa R, Venkatasubramanian G, Muralidharan K, **Debnath M.** Multiple complement pathway-related proteins might regulate immunopathogenesis of Major Depressive Disorder. Clinical Psychopharmacology and Neuroscience, 2023; 21(2):313-319.

84) Khanra S, Reddy P, Giménez-Palomo A, Park CHJ, Panizzutti B, McCallum M, Arumugham SS, Umesh S, **Debnath M**, Das B, Venkatasubramanian G, Ashton M, Turner A, Dean OM, Walder K, Vieta E, Yatham LN, Pacchiarotti I, Reddy YCJ, Goyal N, Kesavan M, Colomer L, Berk M, Kim JH. Metabolic regulation to treat bipolar depression: mechanisms and targeting by trimetazidine. Mol Psychiatry. 2023 Jun 29. doi: 10.1038/s41380-023-02134-8.

85) Bhaskar L, Kharya C, **Debnath M,** Mullapudi T, Subbanna M, Chhabra D, Kumar N, Sharma PK, Bhagat OL, Kochupillai V. Effects of Sudarshan KriyaYoga and Advanced Meditation Program on Genetic Expression of Pro-inflammatory and Antioxidants Genes. Cureus, 2023; 15(7): e41377. doi:10.7759/cureus.41377

86) **Debnath M**, Berk M. Is paternal immune activation just as important as maternal immune activation? Time to rethink the bi-parental immune priming of neurodevelopmental model of schizophrenia. Medical Hypotheses, 2023;174: 111059

87) John DV, Sreenivas N, Deora H, Purushottam M, **Debnath M**, Mahadevan A, Patil SA. Cerebrospinal fluid inflammatory cytokine profiles of patients with neurotropic parasitic infections. Tropical Biomedicine 2023, 40(4): 406-415.

88) Nagappa M, Sharma S, Govindaraj P, Chickabasaviah YT, Siram R, Shroti A, Seshagiri DV, **Debnath M**, Sinha S, Bindu PS, Taly AB. Characteristics of patients with SH3TC2 associated neuropathy in an Indian cohort. Neurology India, 2023 Sep-Oct;71(5):940-945.

2024

89) Sarkar A, Nagappa M, Dey S, Mondal S, Babu GS, Pal Choudhury S, Akhil P, **Debnath M.** Synergistic effects of Immune checkpoints and Checkpoint Inhibitors in inflammatory neuropathies: Implications and Mechanisms. Journal of Peripheral Nervous System, 2024 (in press).

90) Talukdar PM, Reddy PV, Bhargav PH, Subbanna M, Karmani S, Arasappa R, Venkatasubramanian G, Muralidharan K, **Debnath M.** Long-term add-on Yoga therapy

modulates oxidative stress pathway and offers clinical benefits in Major Depressive Disorder-a randomized controlled trial. International Journal of Yoga, 2024 (in press).

91) Dey S., Yelamanchi R, Mullapudi T, Holla VV, Kamble N, Satyaprabha TN, Pal PK, **Debnath M**, Yadav R. Association of Insulin-like Growth Factor-1 and Neurofilament Light Chain in Patients with Progressive Supranuclear Palsy. Annals of Indian Academy of Neurology, 2024 (in press).

92) Sreenivas N, Maes M, Padmanabha H, Dharmendra A, Chakkera P, Paul Choudhury S, Abdul F, Mullapudi T, Gowda VK, Berk M, Vijay Sagar Kommu J, **Debnath M.** Comprehensive immunoprofiling of neurodevelopmental disorders suggests three distinct classes based on increased neurogenesis, Th-1 polarization or IL-1 signaling. Brain Behavior Immunity, 2024 Nov 14;115:505-516.

Total citations: 2464 **h-index:** 23 **i10- index:** 51

Book Chapter:

1. **Debnath M,** Dada R, Berk M. Insights into Mode of action of Yoga in Mental Disorders: A summary of Biological Evidence. In "The Science and Art of Yoga in Mental and Neurological Healthcare (Editors: Varambally S, George S and Srinivasan TM), 1st Edition, Jaypee Brothers, New Delhi. Pages: 69-75.

2. **Debnath M**, Raison CL, Maes M, Berk M. Role of T cell network in Psychiatric Disorders. In "Immuno-Psychiatry: Facts and Prospects (Eds. Berk, Leboyer and Sommer), Springer, Germany, 2021.

3. **Debnath M**, Venkatasubramanian G. Funding Opportunities (Resources) in Biomedical Sciences: Indian Perspective. In "The Quintessence of Basic and Clinical Research and Scientific Publishing (Eds. Jagadeesh, Sanatore and Balakumar), Springer, Singapore, 2023. PP. 837-852.

Resource Person/ Administrative/Academic Assignments:

Member:

- External Member, Board of Studies, School of Life Sciences, Sikkim University, Sikkim.
- Member of the Executive Council of the Indian Society of Human Genetics (ISHG).
- Member, Institute Animal Ethics Committee, NIMHANS, Bangalore.
- Co-ordiantor, Ph.D. Coursework Programme, NIMHANS, Bangalore.
- Member, Library Advisory Committee, NIMHANS, Bangalore.

International Grant Reviewer:

- The Netherlands Organisation for Health Research and Development (ZonMw).
- Israel Science Foundation (ISF), Israel.
- Medical Research Council, UK.
- National Science Centre, Poland
- Czech Health Research Council, Ministry of Health of the Czech Republic

National Grant Reviewer:

- Science and Engineering Research Board (SERB), Government of India.
- Department of Biotechnology (DBT), Government of India.
- Sri Devraj Urs Academy of Higher Education and Research, Kolar, Karnataka, India.

Ph.D. thesis examiner:

- Sri Devraj Urs Academy of Higher Education and Research, Kolar, Karnataka, India.
- All India Institute of Medical Science (AIIMS), New Delhi, India.
- Bharathidasan University, Tamil Nadu, India.
- Kerala University, Thiruvananthapuram, Kerala, India.
- Pondicherry University, Pondicherry, India.
- Calcutta University, Kolkata, India.

Doctoral Committee member:

- Sri Devraj Urs Academy of Higher Education and Research, Kolar, Karnataka, India.
- School of Regenerative Medicine, Manipal Academy of Higher Education, Bangalore, India.
- Jawaharlal Institute of Postgraduate Medical Education & Research (JIPMER), Pondicherry, India.

Members of Ethics/ Biorepository Committee:

- Member of Institutional Committee for Stem Cell Research (IC-SCR) of School of Regenerative Medicine, Manipal Academy of Higher Education, Bangalore.
- Member, Institutional Biorepository Committee, NIMHANS, Bangalore.

Chairperson in Conferences:

- In the International Symposium on Translational Neuroscience & XXXII Annual Conference of the Indian Academy of Neurosciences" November 1-3, 2014, held in NIMAHNS, Bangalore, India.
- In 3rd TS Srinivasan -NIMHANS Knowledge Conclave, 24th February,2017; NIMHANS, Bangalore, India.

- In NIMHANS Annual Reviews in Practical Neurology (ARPrN)-2019, August 16-18, 2019, NIMHANS, Bangalore, India.

- In 3rd International Society of Gene and Cell Therapy Conference, November 10-12, 2019, Bangalore Medical College and Research Institute, Bangalore, India.

- Serve as a 'Judge' in the Young Scientist Session in in the 4th Regional Science & Technology Congress, December 18-19, 2019, Alipurduar College, WB.

- Served as a Judge for Best Poster Award in 45th Annual Meeting of Indian Society of Human Genetics, February 13-15, 2020 at Sri Ramachandra Institute of Higher Education & Research, Chennai, Tamil Nadu.

- Served as a Judge in the Oral Presentation Competition in the "Yoga and Neurosciences: Traditions and Research Approaches (YANTRA)" conference, during 9th-10th and 16th - 17th October, 2020, NIMHANS, Bangalore.

- Served as a Judge in the Oral Presentation Competition in the "Yoga and Neurosciences: Traditions and Research Approaches (YANTRA)" conference, during 23-25 November 2023, NIMHANS, Bangalore.

Invited talk:

- Delivered an invited talk on "Pregnancy-Immunity-Psychitry Nexus: Fetal programming of mental disorders", April 22, 2014 at Department of Zoology, University of North Bengal, Siliguri, West Bengal, India.

- Delivered an invited talk on "Neurodevelopmental connection of schizophrenia: Evidences from immunogenetics studies" in the International Conference on Environment, Genes, Health and Diseases (EGHD-2017), August 22-24, 2017, Bharathiar University, Coimbatore, Tamil Nadu, India.

- Delivered an invited talk on "Fetal programming of schizophrenia: immunogenetic insights", XXXV Annual Meeting of Indian Academy of Neurosciences, October 29-31, 2017, Ravenshaw University, Cuttack, Odisha, India.

- Delivered an invited talk on " Nexus between Immunity and Psychiatry: Genetic-inflammatory synthesis of Schizophrenia", in the National Symposium on Trends in Biochemistry in Post-Genomic Era, February 28, 2018. Pondicherry University, Pondicherry, India.

- Delivered a talk on "Altered Th17 pathway in schizophrenia: evidences from genetic, gene expression and biochemical studies" in the 26th European Congress of Psychiatry, March 3-6, 2018, Nice, France.

- Delivered an invited talk on "Introduction to basic and applied genetics and steps towards building genetic basis of a disorder" in the Five days workshop on "Primer on genetics for mental and neurological health care professionals", April03, 2018, NIMHANS, Bangalore, India.

- Delivered an invited talk on "Research on Guillain Barre Syndrome at NIMHANS: small steps towards understanding the Immunobiology" in the NIMHANS -Liverpool University Annual Symposium, July 7, 2018, NIMHANS, Bangalore, India.

- Delivered an invited talk on "Immuno-inflammatory synthesis of schizophrenia" in the NIMHANS-Deakin University Mini-Symposium on "Translational Immunobiology of Schizophrenia and Bipolar Disorder" August 27, 2018, NIMHANS, Bangalore, India.

- Delivered an invited talk on "Immunobiology of Guillain Barre Syndrome: Role of Th17 pathway" in the 3rd International Society of Gene and Cell Therapy Conference, November 10-12, 2019, Bangalore Medical College and Research Institute, Bangalore, India.

- Delivered an invited talk on "Fetal programming of mental disorders: role of the immune system and environmental determinants", in the 4th Regional Science & Technology Congress, December 18-19, 2019, Alipurduar College, WB.

- Delivered invited talk on "Impaired immune-genetic synthesis of schizophrenia; a trans-species study", in the 45th Annual Meeting of Indian Society of Human Genetics, February 13-15, 2020 at Sri Ramachandra Institute of Higher Education & Research, Chennai, Tamil Nadu.

- Delivered an invited talk on "The storm and the aftermath of COVID-19: The bumpy road ahead for neurobiology and mental health" in the Two-day National Webinar on "COVID-19: Challenges for management of mental health and risk factors" June 19-20, 2021, Organized by the PG Department of Zoology, ABN Seal College and MJN Medical College and Hospital, Cooch Behar, West Bengal.

- Delivered a plenary talk on "Psychoneuroimmunology: The rise of the seventh sense in neuroscience and Yin and Yang of this sense", in the IBRO-APRC Nepal School on Understanding of Neuroscience and spectrum of neurogenetic disorders, August 22, 2021, Kathmandu, Nepal.

- Delivered an advanced lecture on "Genetic/epigenetic basis of neuropsychiatric disorders", in the IBRO-APRC Nepal School on Understanding of Neuroscience and spectrum of neurogenetic disorders, August 21, 2021, Kathmandu, Nepal.

- Delivered a talk on "Human intelligence: A genetic affair" in the Vigyan Anwesha: An initiative to connect students with Scientist programme of North Bengal Science Centre, Siliguri, A unit of National Council of Science Museums, Ministry of Culture, GOI, August 16, 2021.

Organizing workshop/conferences:

- **Organizing Secretary** of National Science Day, 28th February, 2017, NIMHANS, Bangalore, India.
- **Served as core member of the** Organizing Committee of Indian Congress of Parasitology, April 25-27, 2017, NIMHANS, Bangalore, India.
- **Organizing Secretary** of One Day Symposium on "Yoga and Indian Psychology", 25th June, 2017, NIMHANS, Bangalore, India.
- **Organizing Secretary,** five days workshop on 'Primer of genetics for mental and neurological health care professionals" April 03-08, 2-018, NIMHANS, Bangalore, India.
- **Organizing Secretary,** of the 3rd International Society of Gene and Cell Therapy Conference, November 10-12, 2019, Bangalore Medical College and Research Institute, Bangalore, India.
- **Co-ordinator** for the six months certificate course on "Genetic diagnosis and counselling", June-November, 2019, Department of Human Genetics, NIMHANS, Bangalore, India.
- **Organizing Secretary,** five days workshop on "Genetics and epigenetics for mental and neurological healthcare professionals", July 03-07, 2023, NIMAHNS, Bangalore, India.

MEMBER OF EDITORIAL BOARD:

1. Associate Editor, Frontiers in Psychiatry

2. Journal of Genetics & Genome Research, Published by ClinMed International Library, Newark, DE 19711, USA.

INVITED REVIEWER:

- Molecular Psychiatry
- Schizophrenia Bulletin
- Brain Behavior and Immunity
- Psychoneuroendocrinology
- Human Immunology
- Schizophrenia Research
- PLoS ONE
- Gene

- Journal of Clinical Psychiatry
- Molecular Biology Reports
- Translational Neuroscience
- Journal of Genetics
- Medical Hypotheses
- CNS Neurol Disord Drug Targets
- Asian Journal of Psychiatry
- Psychopharmacology
- Frontiers in Human Neuroscience
- Physiology and Behavior
- Indian Journal of Medical Research
- Immunological Investigations
- Psychiatry and Clinical Neurosciences
- BMC Neurology
- Current Pharmaceutical Design
- Psychiatry Research
- Scientific Reports
- Clinical and Experimental Dermatology
- Journal of Psychosomatic Research
- Translational Psychiatry
- Progress in Neuropsychopharmacology & Biological Psychiatry
- Australian and New Zealand Journal of Psychiatry
- National Science Review
- Current Topics in Medicinal Chemistry
- Journal of Neuroimmunology
- Ageing
- Neuroscience Bulletin
- Neurology India
- Frontiers in Psychiatry
- BMC Psychiatry

RESEARCH GRANT:

No.	Title of Project	Funding Agency	Amount	Duration
1	Genetic and expression studies of HLA-G and cytokines to evaluate the immune-mediated risk of schizophrenia **(As PI)**	NIMHANS Intra-mural Grant, India	Rs. 5.0 lakhs	2013- 2015
2.	The Immuno-Psychiatry in South India Study (IPS)-Immunogenetic and Immuno-phenotype characterization of major psychoses **(As Co-I). International grant (Indo-French)**	Indo-French Centre for the Promotion of Advanced Research	Rs. 78,85,160/-	2014-2018
3.	Understanding the Th17 cytokine-mediated pathogenesis of Guillain Barré Syndrome: An integrative biochemical, genetic and gene expression study **(As PI)**	Science & Engineering research Board, India.	Rs. 35,20,000/-	2014-2017
4.	Understanding the prenatal infection induced immune mediated risk of schizophrenia in a rat model (AS Mentor)	Department of Science & Technology, (WOS-A), India	Rs. 16,00,000/-	2015-2018
5.	Yoga and Schizophrenia - a Comprehensive Assessment of Neuroplasticity (Y - SCAN) **(As Co-I)**	Wellcome Trust-DBT India Alliance	Rs.2.97 crore	2016-2021
6.	Effect of Shakti kriya versus sudarshan kriya and pranayama (SK&P) on electroencephalo-gram (EEG), gene expression, heart rate variability (HRV), galvin skin resistance (GSR) and quality of life **(As Co-I)**	Department of Science & Technology, India.	Rs. 11,00,000/-	2017-2018
7.	Therapeutic effects of yoga in depression: A neurobiological investigation. **(As Co-I)**	Department of Science & Technology, India.	Rs.46,51,600/-	2016-2019
8.	Therapeutic effects of Yoga on immuno-inflammatory profile in Major Depressive Disorder **(As PI)**	NIMHANS Integrated Centre for Yoga	Rs. 10,00,000/-	2017-2020
9.	Understanding the Role of Mitochondrial Dysfunction in Inherited Peripheral Neuropathies: A Phenotype-Genotype Correlative Study Using Next Generation Sequencing **(As Co-I)**	Indian Council of Medical Research, India.	Rs. 24,00,000/-	2017-2019
10.	Generation of induced pluripotent stem cells and midbrain floor plate cells from Indian ethnicity Parkinson's disease patients **(As Co-I)**	DBT (Under PACE scheme of BIRAC)	Rs. 45,45,000/-	2018-2020
11.	Susceptibility of brain white matter to inflammatory mediators in Parkinson's disease: An exploratory DTI study **(As Co-I)**	Science & Engineering Research Board,	Rs. 36,38,000/-	2018-2021

		India.		
12.	Neurobiological Correlates of the Effect of Add-On HD-tDCS for Working Memory Deficit in Schizophrenia. **Co-supervisor** (Under Research Training Fellowship for Clinicians).	Wellcome Trust/DBT India Alliance	Rs. 33,98,450/-	2017-2019
13.	Empowering scheduled castes (SCs) through guided meditation **(As Co-I)**	Department of Science & Technology, India, SEED Division, India.	Rs. 57,70,000/-	2019-2022
14.	Genetic architecture of Parkinson's disease in India **(As Co-I).** **International grant (USA).**	The Michael J. Fox Foundation, USA.	Rs.78,00,000/-	2019-2024
15.	Spectrum of genetic mutations and their association with clinical phenotypes of progressive supranuclear palsy **(As Co-I)**	Indian Council of Medical Research, India	Rs. 46,00,000/-	2019-2022
16.	Identification of blood-based biomarkers in children with neurodevelopmental disorders. **(As PI: Centre for Excellence grant)**	VGST, Govt. of Karnataka.	Rs. 60,00,000/-	2019-2022
17.	Cognitive Dysfunction and Sleep Dysregulation as Endophenotypes of Psychosis: A Study of Neurophysiological, Neuroimaging and Neuroimmune Correlates. **(As Collaborator Under DBT-Wellcome trust India Alliance Early Career Fellowship).**	Wellcome Trust/DBT India Alliance	Rs. 1.86 crore	2019-2024
18.	Psychological, social and biological predictors of child mental health and development: a longitudinal study of shared and distinctive risk and protective factors in UK & India. **(As Co-I).** **International grant (MRC, UK; in collaboration with Liverpool University).**	Medical Research Council, UK.	£2.59 Million	2020-2025.
19.	Analysis of the role of IL-33/ST2 pathway in Guillain Barre Syndrome: A biochemical, genetic and gene expression study. **(As Co-I)**	NIMHANS Intra-mural grant	Rs. 10,00,000/-	2020-2022
20.	A study of interactions between the gut microbiome and the human host biology to elucidate novel aspects of the pathophysiology and pathogenesis of schizophrenia. **(As Co-I)**	NIMHANS Intra-mural grant	Rs. 10,00,000/-	2020-2022
21.	Neuropathy in Alcohol Use Disorder: A study of prevalence, profile, determinants and	SERB	Rs. 46,67,360/-	2020-2023

	pathobiological substrates. **(As Co-I)**			
22.	Clinical Research Centre for Neuromodulation in Psychiatry **(As Co-I)**	Wellcome Trust/DBT India Alliance	Rs. 10.0 crore	2020-2025
23.	Clinical, pathological and genetic studies of patients with tropical ataxic neuropathy. **(As Co-I)**	ICMR	Rs. 30.0 lakhs	2020-2023
24.	Evaluating the efficacy of Ayurvedic intervention as add on to conventional treatment and explore the interaction of epigenetics, neuro/gut biomarkers and neuroimaging in paediatric ADHD. **(As Co-I)**	Central Council for Research in Ayurvedic Sciences	Rs. 99.50 lakhs	2021-2024
25.	An open-labeled controlled clinical study to evaluate the efficacy of Ayurveda Medicines to evaluate the efficacy of Ayurveda Medicines Sudarshana Churna, Yashtimadhu Churna and Amrutarishta as add on therapy on Symptomatology, Inflammatory Markers and RT PCR in positive cases of COVID-19 – A Collaborative Study **(As Co-I)**	AYUSH	Rs. 9,26,100/-	2021-2022
26.	Evidence based validation of a Tele-Yoga intervention in positive cases of COVID-19-A controlled pilot study on inflammatory markers, T cell function. **(As Co-I)**	DST-SATYAM	Rs. 8,50,000/- +	2021-2022
27	Understanding the interactions and impact of Gut Microbiota and Mucosal Associated Invariant T (MAIT) cells in the pathobiology of Inflammatory Nodopathies.	SERB, GOI **(As PI)**	Rs. 57,78,360/-	2022-2025
28	Centre for Excellence on "Yoga and Ayurveda in Neuroscience Translational Research Accelerator Programme" (YANTRA) **(As Co-I)**	AYUSH	Rs. 10.0 crore	2022-2025
29	'Understanding the role of circadian, inflammatory and sleep homeostatic pathway-related genes in sleep dysregulation in children with Autism Spectrum Disorder. **(As Co-I)**	NIMHANS Intra-mural grant	Rs. 10.0 lakhs	2023-2025
30	Deciphering the change in sleep architecture and its relation to circulating melatonin level, and circadian rhythm gene expression in Parkinson's disease following deep brain stimulation surgery: A longitudinal video-polysomnography-based cohort study. **(As Co-I)**	ICMR, GOI	Rs. 80.0 lakhs	2024-2027

Research Guidance (Ph.D./DM/MD):

Sl no.	Name	Course	Thrust Area	Remarks
1	Ms. Geetanjali Murari	M.Phil. (Neuroscience)	Immunogenetics of Guillain Barre Syndrome	Awarded (2016)
2	Ms Ashwini R.	PhD	Psychiatric Immunogenetics	Awarded (2017)
3	Dr. Sri Mahavir Agarwal	PhD	Psychiatric Genetics	Awarded (2017)
4	Ms Deepthi Venugopal	PhD	Psychiatric Immunogenetics	Awarded (2018)
5	Dr. Rahul Wahatule	DM (Neurology)	Immunobiology of Guillain Barre Syndrome	Awarded (2017)
6	Dr. Ramesh Siram	DM (Neurology)	Genetics of Inherited Peripheral Neuropathy	Awarded (2018)
7	Dr.Kiran Polavarapu	PhD	Genetics of Muscular Dystrophy	Awarded (2019)
8	Ms. Harleen Chhabra	PhD	Psychiatric Genetics	Awarded (2020)
9	Ms Manjula Subbanna	PhD	Psychiatric Immunogenetics	Awarded (2020)
10	Dr. Jayakrishnan Menon	DM (Addiction Psychiatry)	Immunobiology of Addiction	Awarded (2021)
11	Ms. Pinku Mani Talukder	PhD	Animal Model of Schizophrenia	Awarded (2021)
12	Dr. Praveen Sharma	DM (Neurology)	Immunogenetics of Guillain Barre Syndrome	Awarded (2021)
13	Mr Debprasad Dutta	PhD	Immunogenetics of Guillain Barre Syndrome	Awarded (2022)
14	Dr. Sowmya Selvaraj	PhD	Psychiatric Genetics	Awarded (2022)
15	Dr. Nibu Varghese	DM (Neurology)	Immunobiology of Myasthenia Gravis	Awarded (2022)
16	Dr. Ram Chandra	DM (Neurology)	Neurobiology of Supranuclear Palsy	Awarded (2022)
17	Dr. Srinath	DM (Neurology)	Role of inflammasome pathway in GBS	Awarded (2023)
18	Dr. Pradeep Shetty	DM (Neurology)	Role of Node-paranode antibodies and NF-kB pathway in inflammatory neuropathies	Submitted (2023)
19	Mr. Trinath M	PhD	Psychiatric Immunogenetics	Submitted (2024)
20	Dr. Preethi Reddy	PhD	Immunobiology of Bipolar	Submitted

			Disorder	(2023)
21	Dr. Swarnabudha Nayok	PhD	VNS and immunobiology of Schizophrenia	Submitted (2023)
22	Ms. Nikhita Sreenivas	PhD	Immunobiology of alcohol induced neuropathy	On-going
23	Mr. Saikat Dey	PhD	Genetic and epigenetic understanding of Supranuclear Palsy.	On-going
24	Ms. Kavya Vp	PhD	Impact of maternal stress on the mental illnesses of the offspring	On-going
25	Mr. Sandipan Mondal	PhD	Role of gut microbiota, MAIT and Treg cells in inflammatory neuropathies.	On-going
26	Mr. Aishwarya Kumar	PhD	PET-MRI based study in PD, MSA and PSP	On-going
27	Ms. Twinkle Moothedan	PhD	Statistical Methods to Assess the Stability of Reference Genes in Gene Expression Studies	On-going
28	Dr. Jayaram	DM (Neurology)	Immunobiology of ASD	On-going
29	Dr. Akhil P.	DM (Neurology)	Immune checkpoint molecules in inflammatory neuropathies	On-going
30	Dr. Subhajit Roy	DM (Neurology)	Genetics of PD	On-going

Research Guidance (Under and post- graduate thesis/ Research Interns)

Sl no.	Name	Course & Name of the Institute	Thrust Area	Year
1	Ms. Sushmita Sastry	M.Sc. Vellore Institute of Technology, Tamil Nadu, India.	Immunogenetics of Schizophrenia	2018
2	Ms. Shreya Shetty	B.Tech. Dr. D Y Patil University, Mumbai, India	Immunobiology of Guillain Barre Syndrome	2019
3	Ms. Renu Balaji	B.Tech.	Immunobiology of	2018 & 2019

		Dayanand Sagar University, Bangalore, India.	Schizophrenia	
4	Ms. Shruti Bahukudumbi	B.Tech. Anna University, Tamil Nadu, India	Immunogenetics	2019
5	Mr. Abdul Fazal	B. Tech. SRM University Chennai, India.	Animal Model of Schizophrenia	2019
6	Aria Sarkar	The Village School, Houston, USA	Immunobiology of GBS	2023
7	Nisha Prakash	Azim Premji University, Bangalore.	NA	2023
8	Saiesha Bade	The International School f Bangalore	NA	2023
9	Shristi Jain	Christ University, Bangalore	NA	2023

Awards received by the Ph.D. students:

- Mr. Debprasad Dutta (Ph.D. student) has received **Best Poster Award (2nd position)**, on the paper "Epimolecular affairs at the cross road of natural selection and psychological adaptation with reference to mind-body axis reprogramming" in the Onde day symposium on "Yoga and Indian Psychology", June 25, 2017, NIMHANS, Bangalore, India.

- Mr. Debprasad Dutta (Ph.D. student) has been awarded **"Commonwealth Split-Site Scholarship"** for the year 2019 by Commonwealth Commission, UK to work in the Liverpool University, UK for one year.

- Ms. Manjula Subbanna (Ph.D student) received **"Young Investigator award"** from World Congress of Biological Psychiatry, World Federation of the Societies of Biological Psychiatry (WFSBP) 2-6 June 2019, Vancouver, Canada.

- Ms. Manjula Subbanna (Ph.D) student has received **"Dr. T.S. Agarwal Inspiring Scientist Sushrata Award"** for best paper presentation in the 3rd Annual meeting of International Society of Gene and Cell Therapy (ISGCT)-2019, November 10-12, 2019, Bangalore, India.

- Mr. Debprasad Dutta (PhD student) received **AWSAR Award for Best Ph.D stories (2019)** from Department of Science & Technology, Government of India.

- Dr. Preethi Reddy (PhD student) received first prize in oral presentation competition in "Yoga and Neurosciences: Traditions and Research Approaches (YANTRA)" conference, during 9th-10th and 16th - 17th October, 2020, NIMHANS, Bangalore.

- Mr. Thrinath M. (PhD Student) received Best Poster Award, 10th International Conference on Schizophrenia, Schizophrenia Research Foundation (SCARF), Chennai, 25 August 2022.

- Mr. Saikat Dey (PhD student) received travel grant to attend International Congress of Parkinson's Disease and Movement Disorders, Movement Disorder Society (MDS), Madrid, Spain, 15-18 September 2022.

- Mr. Saikat Dey (PhD student) received 2nd Best Paper Award (oral presentation), MDSICON 2022, Mumbai, 13-15 May 2022.

- Mr. Saikat Dey (PhD student) received C. U. Velmurugendran – U Meenakshisundaram Award for Movement Disorders" at IANCON 2023, held at Madurai, India, from September 14th to 17th 2023. (Best oral presentation award).

Универзитет у Крагујевцу
Факултет медицинских наука
Декану проф. др Владимиру Јаковљевићу

СТРУЧНА САРАДЊА СА ПРОФ. МОНОЈИТ ДЕБНАТОМ

Поштовани Декане,

Обраћам се молбом да размотрите интензивирање сарадње са проф. Монојит Дебнатом (енгл. *Monojit Debnath*, Department of Human Genetics, National Institute of Mental Health & Neurosciences, Hosur Road, Bangalore – 560029, INDIA).

До сада смо сарадњу остварили у стручној публикацији високог ранга (*World Journal of Diabetes*, M22, IF 3.1 Q3) (публикација у прилогу), а могућности су за даљу сарадњу у областима психијатрије, неурологије и имуногенетике у заједничким међународним истраживачким пројектима, што оцењујемо посебно важним због бројних студената из Индије који сада похађају различите смерове нашег Факултета.

Проф. Монојит Дебнат је планирао учешће у годишњем конгресу Европске психијатријске асоцијације (32nd European Congress of Psychiatry, Budapest, 6–9 April, 2024), па је изразио интересовање да у том периоду посети и наш Факултет и одржи предавање по позиву, а предлажем да се размотри и његов избор у звање гостујућег професора (детаљна биографија у прилогу).

У нади да ћете молбу размотрити,

С поштовањем,

проф. др Милица М. Боровчанин

У Крагујевцу, 11. 01. 2024. године

World Journal of Psychiatry

Submit a Manuscript: https://www.f6publishing.com

World J Psychiatry 2022 September 19; 12(9): 1183-1193

DOI: 10.5498/wjp.v12.i9.1183

ISSN 2220-3206 (online)

ORIGINAL ARTICLE

Case Control Study

Galectin-3 mediated risk of inflammation in stable schizophrenia, with only possible secondary consequences for cognition

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Specialty type: Psychiatry

Provenance and peer review: Invited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's scientific quality classification
Grade A (Excellent): A
Grade B (Very good): B
Grade C (Good): 0
Grade D (Fair): D, D
Grade E (Poor): 0

P-Reviewer: Khosravi M, Iran; Radhakrishnan R, New Zealand; Shu Liu, China

Received: April 20, 2022
Peer-review started: April 20, 2022
First decision: May 30, 2022
Revised: June 14, 2022
Accepted: August 10, 2022
Article in press: August 10, 2022
Published online: September 19, 2022

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Abstract

BACKGROUND

Evidence suggests that cytokines cause immune disturbances, shape immunological sequelae later in life, and modulate the risk of schizophrenia (SC). Galectin-3 (Gal-3), a multifaceted molecule of the glycan family, is involved in the formation of the immunological synapse and modulates the signalling pathway and effector functions of T lymphocytes, which are major producers of cytokines. We have previously reported elevated serum Gal-3 levels in stable SC patients. However, Gal-3 as a link between cognitive functioning and inflammation has not yet been investigated in SC.

AIM

To investigate the relationship between serum Gal-3 levels and cognitive performance, serum cytokines, and white blood cell count in three-month stably treated SC patients.

METHODS

Twenty-seven patients with SC in remission and 18 healthy volunteers participated in this case-control and correlational study. Clinical assessment was performed using the Positive and Negative Syndrome Scale and the Montreal-Cognitive Assessment. The results of previously measured serum levels of Gal-3, interleukin (IL)-33, soluble suppression of tumorigenicity 2 (sST2), tumor necrosis factor-alpha (TNF-α), IL-6 and IL-17 were used for further statistical analyses, and IL-4, IL-23, IL-1β and transforming growth factor-beta (TGF-β) were now additionally measured with a sensitive enzyme-linked immunosorbent assay. The number of leukocytes in the blood and the percentage of neutrophils, lymphocytes, and monocytes were determined with a standardized routine measurement procedure (Sysmex Technology). Statistical analyses were performed using SPSS 20.0 software.

RESULTS

We found no correlation between serum Gal-3 levels and cognitive functioning in SC patients. A positive correlation was found between the levels of Gal-3 and TNF-α (r = 0.476; P = 0.012), Gal-3 and IL-23 (r = 0.417; P = 0.031), and Gal-3 and sST2 (r = 0.402; P = 0.038). The binary logistic model, which included all nine cytokines measured in this patient sample, indicated the particular role of Gal-3 and TGF-β in the duration of SC. In the stabilization phase of SC, we observed a moderate and negative correlation between serum Gal-3 levels and leukocytes (r = -0.449; P < 0.019). Additional linear regression analysis showed a positive correlation between Gal-3 expression and risperidone dose (F: 4.467; P < 0.045; r^2 = 0.396).

CONCLUSION

The combined activity of Gal-3 and proinflammatory cytokines, TGF-β downregulation and lower counts of leukocytes influence the SC duration. Gal-3 likely manifests indirect immunometabolic regulation of cognition in SC.

Key Words: Schizophrenia; Galectin-3; Cytokines; Leukocytes; Antipsychotics

Core Tip: In clinical sampling, there is an urge to place the results of biological measurements in a much broader context. Elevated serum galectin-3 (Gal-3) levels in schizophrenia (SC) have not been studied in relation to other peripheral biomarkers and subsequent neuroinflammation. We found that Gal-3 contributes to ongoing peripheral systemic inflammation and disease duration in patients with SC. All of this may be an underlying indirect immunometabolic mechanism for cognitive performance in patients with SC.

Citation: Minic Janicijevic S, Jovanovic IP, Gajovic NM, Jurisevic MM, Debnath M, Arsenijevic NN, Borovcanin MM. Galectin-3 mediated risk of inflammation in stable schizophrenia, with only possible secondary consequences for cognition. *World J Psychiatry* 2022; 12(9): 1183-1193

URL: https://www.wjgnet.com/2220-3206/full/v12/i9/1183.htm

DOI: https://dx.doi.org/10.5498/wjp.v12.i9.1183

INTRODUCTION

Immune dysregulations during prenatal and postnatal life are increasingly associated with neurodevelopmental disorders and have also recently been shown to be an important etiological construct of schizophrenia (SC)[1,2]. Multiple post-mortem brain and neuroimaging studies have also provided evidence for neuroinflammation in SC[3,4]. One of the best-known hypotheses, proposed by Bechter, links SC to mild and localized encephalitis[5]. There is strong evidence that cytokines cause these immune disturbances, shape immunological sequelae later in life, and modulate SC risk. In particular, T lymphocytes are one of the major producers of cytokines, and it has been reported that blood levels of cytokines derived from various lineages of T lymphocytes such as T helper 1 (Th1), Th2, Th17 and regulatory T cells (Treg) are altered in SC[6-8]. Studies have shown that patients with SC have increased serum concentrations of proinflammatory cytokines, including interleukin (IL)-1β, IL-6, and tumor necrosis factor-alpha (TNF-α)[9,10].

Studies have also shown that Gal-3, a multifaceted molecule in the glycan family, is directly involved in the formation of the immunological synapse and appears to play a pivotal role in modulating the signalling pathway and effector functions of T lymphocytes[11]. It is noteworthy that Gal-3 has both

immune and non-immune functions in the brain. Gal-3 appears to play a neuroprotective role in neuronal tissue and is involved in the reparative processes of brain lesions and ischemia. In contrast, Gal-3 may promote microglia-mediated neuroinflammation and contribute to neuroprogression[12]. Gal-3 increases the secretion of proinflammatory cytokines from microglia and astrocytes[13] and is also required for leukocyte recruitment during an acute inflammatory response[14].

Biomarkers that can be conveniently measured in blood may also reflect changes in the central nervous system and dysfunction of the blood-brain barrier (BBB). There is evidence of BBB dysfunction in brain disorders, including SC. Brain microvascular endothelial cells (BMECs) are a key element of the microvasculature that forms the BBB and shields the brain from toxins and reactive immune cells. However, it is not known whether BMECs themselves are functionally compromised and lead to BBB dysfunction in brain disorders[15]. An increased ratio of cerebrospinal fluid to serum albumin in patients with SC suggests increased permeability of the BBB[16]. Given the important role of galectins in cell adhesion, migration, polarity, and chemotaxis, it is likely that modulation of galectin levels in BMECs that form the BBB could compromise BBB integrity and consequently contribute to neuroinflammation[17]. Plasma levels of Gal-3 have been shown to be increased after aneurysmal subarachnoid hemorrhage (SAH), and a Gal-3 inhibitor could potentially prevent post-SAH BBB disruption by inhibiting Gal-3[18].

We have previously reported elevated serum Gal-3 levels in patients with SC who received stable 3-mo antipsychotic therapy[19]. We wanted to go further in exploring Gal-3 interactions and not only measure serum levels during stabilisation of SC. Recently, such an association between Gal-3 and cognition was found in Alzheimer's disease[20]. In this additional analysis, we tested the hypothesis that serum Gal-3 levels in patients with stable SC might be related to cognitive functioning and different white blood cell counts and types of cytokines in stable SC patients. In this way, we aimed to investigate the possible involvement of this glycan in peripheral systemic inflammation and disease duration, but also its position as a link between cognitive functioning and inflammation, which has not yet been investigated in SC.

MATERIALS AND METHODS

Participants

Patients with SC in remission (SC in remission) were recruited in 2016 in the Psychiatric Day Hospital of the Kragujevac Clinical Centre. Participants were between 18 and 65 years old. Diagnoses were made using the International Statistical Classification of Diseases and Related Health Problems, Tenth Revision (ICD-10) criteria[21] for SC (F20). The major inclusion criterion was stable mental functioning and adherence to three months of stable antipsychotic depot therapy with risperidone or paliperidone. Add-on therapy for patients included anxiolytics or hypnotics only. A complete medical history was obtained from each patient.

Exclusion criteria were current infections during the three-month remission period, allergies or autoimmune disorders, current anti-inflammatory or antiviral medications, or dual diagnoses of other mental illnesses. Healthy controls (HCs) were recruited during blood donation at the Blood and Blood Products Service of the Kragujevac Clinical Centre, and controls with a family history of psychosis were excluded. All laboratory measurements and immunoassays were performed at the Centre for Molecular Medicine and Stem Cell Research, Faculty of Medical Sciences, University of Kragujevac. The study was conducted after the Ethics Committee of the Kragujevac Clinical Centre gave its approval. Participants were able to give informed consent, and each patient signed the informed consent form before participating in the study.

The study sample was estimated considering the first type error (α) of 0.05 and the power of the study of 0.8 for the two-tailed t-test for two independent samples using the statistical softer G* Power 3.1.9.2. Considering previous studies and similar methods for measuring serum cytokine levels[22], the minimum number of participants required in each group was estimated to be 14.

Clinical assessment

Psychological assessment was performed by trained raters. Psychopathology was assessed using the Positive and Negative Syndrome Scale (PANSS)[23]. Cognition was assessed using the cognitive factor of the PANSS (consisting of items P2-N5-G11)[24], which primarily refers to sustained attention, and executive functioning such as mental flexibility and problem-solving as components of executive functioning[25]. In addition, cognitive impairment was assessed using the Montreal-Cognitive Assessment (MoCA)[26], a cognitive screening tool for older population with mild cognitive impairment and dementia that has also been shown to be useful in patients with psychosis[27]. The MoCA test assesses multiple cognitive domains including attention, concentration, executive functions, memory, language, visual-constructive skills, conceptualization, and orientation, with a maximum total score of 30 and a lower limit for normal cognition of 26.

Blood collection and cytokine measurements

Blood samples were taken in the morning (approximately 8 am) after overnight fasting. The blood clot was cut and then centrifuged. After separation, serum samples were stored at -20° until analysis. The results of previously measured serum levels of Gal-3, IL-33, soluble suppression of tumorigenicity 2 (sST2), TNF-α, IL-6 and IL-17[19,28] were used for further statistical analyses, and IL-4, IL-23, IL-1β and transforming growth factor-beta (TGF-β) were now additionally measured using sensitive Enzyme-Linked Immuno-Sorbent Assay kits specific for the human cytokines according to the manufacturer's instructions (R&D System, Minneapolis, MB). The procedure has been described in detail previously [19]. Briefly, 96-well plates coated with capture antibody and incubated overnight were washed with wash buffer and incubated with blocking buffer for 1 h at room temperature. Serum samples or standard recombinant IL-4/IL-23/IL-1β/TGF-β were added to the plates for 2 h before a biotinylated detection antibody and streptavidin peroxidase were applied for 1 h each at room temperature. The plates were developed with substrate reagent for 20 min, and the reaction was stopped by addition of 4 mol/L sulfuric acid. The absorbance was read at 495 nm using a microplate reader. The exact concentration of the above biomarkers was measured by interpolating a standard curve with a series of known concentrations according to the manufacturer's instructions. The values of the measured cytokines are expressed in pg/mL. Blood cell populations were determined using a standardized routine laboratory procedure (Sysmex Technology).

Statistical analysis

Demographic and clinical data were presented descriptively. Various covariates were included in linear and multiple linear regression models to examine the effects of these variables on the results. Pearson's or Spearman's correlation analysis was used to examine the significance of the correlation between serum Gal-3 levels and blood cell counts, serum cytokine levels, and clinical scores and subscores of PANSS and MoCA. To determine the best prediction of serum cytokine levels for the presence of illness, binary logistic regression analysis was performed. A P-value of ≤ 0.05 was considered statistically significant. Statistical analyses were performed using IBM SPSS Statistics for Windows, version 20.0. Armonk, NY: IBM Corp.

RESULTS

Demographic and clinical characteristics

There were no statistically significant differences in age (P = 0.886) and sex (P = 0.851) between patients (n = 27) and HC subjects (n = 18). The demographic and clinical characteristics of the patients were the same as those presented previously[19,28] and are listed in Table 1. Among patients with SC, the duration of illness was 9.95 ± 7.71 years, with 2.18 ± 1.92 years as multiple previous hospitalizations. Most patients were individuals with high school education (n = 22). The mean PANSS total score and subscores, MoCA total score and subscores, and medications taken in the SC group are shown in Table 1.

Differentiation of serum cytokine levels between groups

In this study, lower TGF-β levels (272.09 ± 101.59 *vs* 360.41 ± 45.13, P = 0.003) were observed in patients with SC (Figure 1A), with no difference in serum IL-4, IL-23 and IL-1β levels (data not shown). The binary logistic model, which included the presence of illness as a dependent variable and all measured cytokine serum levels as covariates in a stepwise Backward-Wald method, highlighted the particular role of Gal-3 and TGF-β in SC, both of which have an impact on disease presentation with an odds ratio for Gal-3: 1.002 (95%CI: 1.000-1.004; P = 0.022) and TGF-β: 0.982 (95%CI: 0.9968-0.997; P = 0.015) (Figure 1B), suggesting that higher Gal-3 levels are associated with stabilization in later phases of SC.

Serum Gal-3 levels correlate significantly with proinflammatory mediators and risperidone dosing

The correlation between Gal-3 serum levels and cognitive functioning considering MoCA total score, subscores, and PANSS Cog was not significant (data not shown). In addition, we now examined the relationship between systemic Gal-3 levels and cytokines with divergent immune properties. A positive and moderate correlation was observed between Gal-3 and TNF-α (r = 0.476; P = 0.012), Gal-3 and IL-23 (r = 0.417; P = 0.031), and Gal-3 and sST2 (r = 0.402; P = 0.038) levels (Figure 2).

Moreover, linear regression analysis revealed a positive correlation between Gal-3 and risperidone dose (F: 4.467; P < 0.045; r^2 = 0.396).

Serum levels of Gal-3 inversely correlate with leukocyte count

We also examined the correlation between Gal-3 and the number of leukocytes (neutrophils, lymphocytes, and monocytes) involved in the immune response. A negative correlation was found between Gal-3 and total leukocyte count (r = -0.449, P < 0.019), with no other significant correlations with the percentages of specific populations.

Table 1 Demographic and clinical characteristics of subjects

Characteristics	SC in remission (n = 27)	Healthy control (n = 18)	P value
Age (yr), mean ± SD	36.18 ± 9.27	37.67 ± 9.96	0.862
Sex (male/female)	16/11	12/6	0.851
Duration of illness (yr), mean ± SD	9.95 ± 7.71	-	-
Number of previous hospitalizations	2.18 ± 1.92	-	-
PANSS			
PANSS total score	99.22 ± 18.2	-	-
Positive syndrome scale	22.26 ± 5.97	-	-
Negative syndrome scale	27.52 ± 6.09	-	-
General psychopathology scale	49.44 ± 7.83	-	-
MoCA			
MoCA total score	22.74 ± 4.76	-	-
Visuospatial/Executive	4.11 ± 1.25	-	-
Naming	2.78 ± 0.69	-	-
Attention	5.07 ± 1.21	-	-
Language	1.89 ± 0.69	-	-
Abstraction	1.41 ± 0.84	-	-
Delayed recall	1.81 ± 1.62	-	-
Orientation	5.74 ± 0.81	-	-
Medications			
Long-acting risperidone/paliperidone	22/5	-	-
Long-acting risperidone dosage 25/37.5/50 mg	3/9/13	-	-
Cell counts			
Leukocytes ($\times 10^9$/L)	6.67 ± 2.06	-	-
Neutrophils (%)	0.61 ± 0.07	-	-
Lymphocytes (%)	0.31 ± 0.07	-	-
Monocytes (%)	0.08 ± 0.02	-	-

PANSS: Positive and Negative Syndrome Scale of Schizophrenia; MoCA: Montreal-Cognitive Assessment; SC: Schizophrenia.

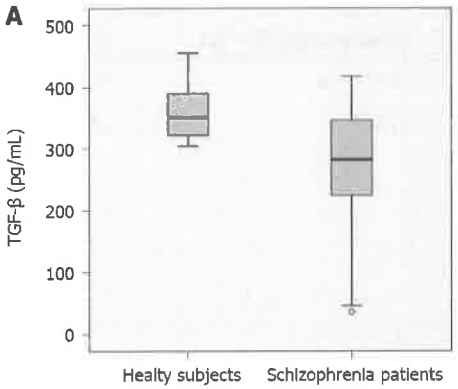
DISCUSSION

The current study contains several new and interesting findings. One of the salient findings was a significant correlation between serum Gal-3 levels and levels of proinflammatory cytokines in a stable phase of SC. Serum Gal-3 correlated positively with TNF-α, IL-23, and soluble ST2 in SC in remission (Figure 2) and was associated with downregulation of the counterregulatory cytokine TGF-β and appears to play a role in disrupting leukocyte migration. In addition, the increase in Gal-3 might be influenced by risperidone dosing.

This study was the first to investigate a possible relationship between Gal-3 and cognitive functioning in SC patients. No correlation was found between serum Gal-3 levels and cognitive performance, suggesting a more indirect immunometabolic regulation of cognition in SC, as we have recently discussed[12]. It has been demonstrated that proinflammatory cytokines and mediators of oxidative stress could influence serum Gal-3 levels, and a reciprocal role of Gal-3 in these cascades could not be excluded[29]. Recently, Dal Lin *et al*[30] (2020) pointed out the close relationship and regulatory effect of cognitive functioning on some molecular processes in the human body, including acute attenuation of oxidative stress and inflammation, which inversely affect Gal-3 levels. Based on these findings, Gal-3 may prove to be a potential therapeutic target in SC.

Currently, there are no studies on the correlation between Gal-3 and proinflammatory cytokine levels in SC patients. In our previous study on the same cohort, we found higher systemic Gal-3 levels[19] and

A



B

Cytokine impact on illness[1]	B	SE	Wald	df	Sig.	Exp(B)	95%CI	
							Lower	Upper
TGF-β	-0.018	0.007	5.902	1	0.015	0.982	0.968	0.997
Gal-3	0.002	0.001	5.279	1	0.022	1.022	1.000	1.004

[1]Binary logistic regression, 9 steps analysis included cytokines representing different types of immune responses [galectin-3, interleukin (IL)-33, soluble suppression of tumorigenesis, tumor necrosis factor-alpha, IL-4, IL-6, IL-23, IL-1β and transforming growth factor-beta]

DOI: 10.5498/wjp.v12.i9.1183

Figure 1 Transforming growth factor-beta and galectin-3 levels impact the illness. A: Lower transforming growth factor-beta (TGF-β) levels (272.09 ± 101.59 *vs* 360.41 ± 45.13 pg/mL, $P = 0.003$) were measured in patients; B: These parameters of serum concentrations of galectin-3 and TGF-β both had an impact on disease presentation. TGF-β: Transforming growth factor-beta; IL: Interleukin; Gal-3: Galectin-3.

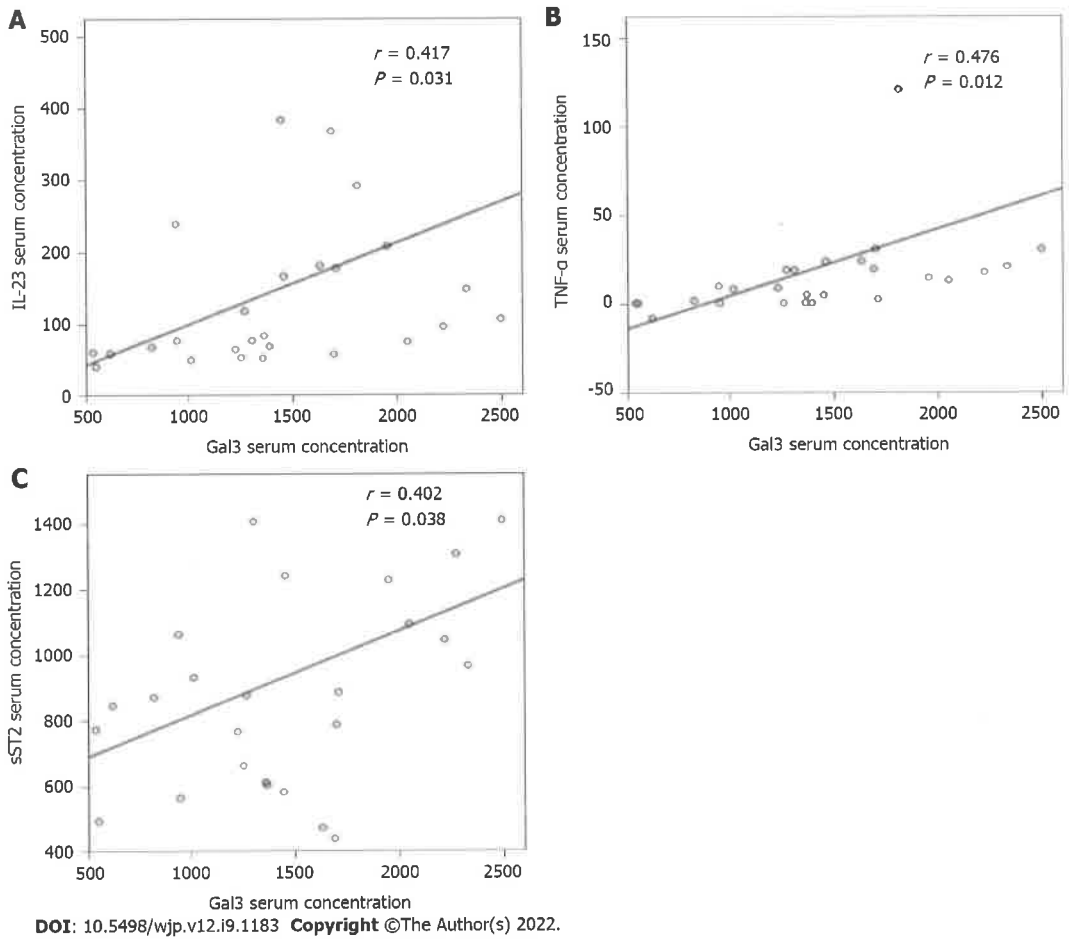
TNF-α[24]. In addition to our study, Kajitani *et al*[31] (2017) also reported elevated serum Gal-3 levels in a stable phase of SC. In one study, Gal-3 was tested for its capacity to induce proinflammatory cytokines such as TNF-α and IL-6 from plasmacytoid dendritic and form myeloid dendritic cells isolated from blood. This lectin was found to activate both, TNF-α and IL-6[32]. In addition, a pre-clinical model of intracerebral haemorrhage (ICH) also demonstrated increased expression of Gal-3 in perihematomal brain regions after ICH and Gal-3-induced release of IL-6, suggesting a role for Gal-3 in inflammatory responses after ICH[33]. These findings suggest the hypothesis that neuronal damage could be followed by inflammation involving Gal-3. The elevated serum Gal-3 levels observed in SC patients in the current study could lead to BBB disruption and contribute to the persistence of mild chronic neuroinflammation suspected in SC.

In particular, somatic comorbidities common in SC, such as obesity, hyperlipidaemia, dyslipidaemia and type 2 diabetes, could be monitored by measuring Gal-3[34]. Gal-3 correlates positively with obesity and inflammation, as measured by the inflammatory markers IL-6 and C-reactive protein (CRP)[35]. Contrary to this finding, the IL-6 axis was not active in this phase and in the specific subpopulation of patients, but rather overweighted type-1 immune response with representative TNF-α. Taken together, these findings suggest potential systemic inflammatory properties of Gal-3 through its interactions with proinflammatory markers in SC that contribute to immunometabolic processes in SC.

The association of Gal-3 and sST2 and their changes at follow-up with the development of heart failure in patients with ST-segment elevation myocardial infarction showed that the levels of Gal-3 and sST2 were significantly increased at one-year follow-up[36]. Interestingly, the increased serum Gal-3 concentration correlated with the production of IL-17 and exhibited a significant correlation with neutrophil/lymphocyte ratio, white blood cell count, and CRP, but inversely correlated with the production of IL-10 and IL-12 in patients with untreated colorectal cancer[37]. Some findings suggest that Gal-3 is required to efficiently recruit leukocytes during an acute inflammatory response[14]. These findings may indicate the diverse role of Gal-3 in this SC chronic inflammation, as we have previously discussed that Gal-3 plays a predominant role in the resolution of inflammation[12]. In chronic SC, our studies have shown that serum Gal-3 levels are elevated and that Gal-3 is negatively correlated with leukocyte count. This lower leukocyte count may be related to the decline in immunity of patients with SC in later stable phases and their greater susceptibility to infection.

Although the Gal-3 signalling pathway is not well understood, Gal-3 can be secreted into the extracellular space, where it can interact with different structures such as cell surface and extracellular matrix glycoproteins[38]. In autoimmune neuroinflammation, endogenous Gal-3 may potentiate its severity by decreasing the frequency of Treg cells, controlling IL-10 production, and modulating Notch activation[39]. The Notch and TGF-β signalling crosstalk, which plays an important role in regulating endothelial and neural development[40], could also be influenced by Gal-3. Our findings might shed important light on the Notch-TGF-β axis in SC (Figure 1B). As for TGF-β, our previous data indicate that serum levels of TGF-β are significantly increased in patients with SC in relapse and first-episode psychosis compared to healthy subjects[41,42]. However, in the current study, significantly lower TGF-β levels were observed in SC patients in remission compared to a group of HC subjects (Figure 1A), suggesting that TGF-β levels vary during the course of SC.

Regarding the possible influence of antipsychotics, a recent *in vitro* study reported that the atypical antipsychotic risperidone reduced the production of proinflammatory cytokines by lipopolysaccharide-stimulated glial cells but had no effect on IL-10[43]. However, paliperidone increased TGF-β and IL-10 during acute stress and during prolonged chronic stress[44]. Our recent hypotheses about the



DOI: 10.5498/wjp.v12.i9.1183

Figure 2 Correlations of serum concentrations of galectin-3 with proinflammatory mediators. A positive and moderate correlation was observed between serum concentrations of galectin-3 (Gal-3) and interleukin-23 (IL-23), Gal-3 and tumor necrosis factor-alpha (TNF-α), and Gal-3 and soluble suppression of tumorigenicity 2 (sST2). A: IL-23 serum concentration; B: TNF-α serum concentration; C: sST2 serum concentration. IL: Interleukin; TNF-α: Tumor necrosis factor-alpha; sST2: Soluble suppression of tumorigenicity 2.

involvement of antipsychotics in the processes of glycosylation can be explained by the effects of their higher doses on serum Gal-3 levels. The findings of the current study suggest that higher doses of prescribed risperidone may lead to an increase in Gal-3 levels. Whole-serum proteins show increased glycosylation after antipsychotic use, indicating the usefulness of these processes for understanding the pathogenesis and monitoring the treatment of patients with SC[34,45].

A higher percentage of Gal-3-expressing innate and adaptive immune cells in the lamina propria was observed in patients with comorbid ulcerative colitis and metabolic syndrome[46]; this encouraged us to explore other immune biomarkers in patients with SC. N-acetylcysteine (NAC) has been proposed for the adjunctive treatment of SC and ulcerative colitis[47]. Oral intake of NAC was shown to lower inflammatory biomarkers, CRP and Gal-3 in patients with acute myocardial infarction receiving fibrinolytic therapy[48]. Preliminary results indicated the usefulness of NAC in improving all domains of SC functioning[49].

As a limitation of our study in terms of cognitive assessment, we must consider that only specific domains of cognitive functioning were assessed, using available validated and brief instruments to detect cognitive impairment in SC in our population. Although we tried to exclude all somatic states, we should be aware that comorbidity and psychotropic medication could influence the results of both cognitive functioning and serum measurements. We believe that it is necessary to investigate these issues further in a larger sample with a much more thorough analysis of confounding factors, which has not been done within the scope of this manuscript, but these results are valuable to guide us in the future.

CONCLUSION

In clinical sampling, there is an urge to place the results of biological measurements into a much wider concept. Higher serum levels of Gal-3 in SC have not been explored in interaction with other peripheral biomarkers reflecting possible inflammatory changes. We observed that Gal-3 contributes to ongoing peripheral systemic inflammation and disease duration in patients with SC. Moreover, its influence on BBB permeability and consequent neuroinflammation should be explored. Our data revealed some new complex roles of Gal-3, such as its possible involvement in neuroinflammation and cognitive processing, contributing to a better understanding of the specific immune profile in patients with SC. Inflammation also appears to be the potential pathway by which Gal-3 may affect cognitive functioning in SC. The efficacy of antipsychotics could be improved and their adverse effects corrected if the role of Gal-3 in glycosylation processes were considered. These findings provide a rationale for further strategies targeting Gal-3 for therapeutic intervention in SC.

ARTICLE HIGHLIGHTS

Research background

Galectin-3 (Gal-3), a multifaceted molecule of the glycan family, modulates T lymphocytes' signalling pathway and effector functions. We have previously reported elevated serum Gal-3 levels in stable schizophrenia (SC) patients, but Gal-3 as a link between cognitive functioning and inflammation has not yet been investigated in SC.

Research motivation

Elevated serum Gal-3 levels in SC have not been studied in relation to other peripheral biomarkers and subsequent neuroinflammation. All of this may be an underlying indirect immunometabolic mechanism for cognitive performance in patients with SC.

Research objectives

Investigating the relationship between serum Gal-3 levels and cognitive performance, serum cytokines, and white blood cell count in three-month stably treated SC patients could contribute to a better understanding of the specific immune profile in patients with SC.

Research methods

Twenty-seven patients with SC in remission and 18 healthy volunteers participated in this case-control and correlational study. Clinical assessment was performed using the Positive and Negative Syndrome Scale and the Montreal-Cognitive Assessment. The results of previously measured serum levels of Gal-3, interleukin (IL)-33, soluble suppression of tumorigenicity 2 (sST2), tumor necrosis factor-alpha (TNF-α), IL-6 and IL-17 were used for further statistical analyses, and IL-4, IL-23, IL-1β and transforming growth factor-beta (TGF-β) were now additionally measured with a sensitive enzyme-linked immunosorbent assay. The number of leukocytes in the blood and the percentage of neutrophils, lymphocytes, and monocytes were determined with a standardized routine measurement procedure. Statistical analyses were performed using SPSS 20.0 software.

Research results

Serum Gal-3 correlated positively with TNF-α, IL-23, and soluble sST2 in SC in remission and was associated with downregulation of the counterregulatory cytokine TGF-β and appears to play a role in disrupting leukocyte migration. The increase in Gal-3 might be influenced by risperidone dosing.

Research conclusions

The combined activity of Gal-3 and proinflammatory cytokines, TGF-β downregulation and lower counts of leukocytes influence the SC duration. Gal-3 likely manifests indirect immunometabolic regulation of cognition in SC.

Research perspectives

We observed that Gal-3 contributes to ongoing peripheral systemic inflammation and disease duration in patients with SC. Moreover, its influence on blood-brain barrier permeability and consequent neuroinflammation should be explored. Inflammation also appears to be the potential pathway by which Gal-3 may affect cognitive functioning in SC.

ACKNOWLEDGEMENTS

We thank Aleksandar Ilic for excellent technical assistance and Bojana Mircetic for language editing.

FOOTNOTES

Author contributions: Minic Janicijevic S and Borovcanin MM presented the design of this project, recruited the participants, performed the psychological and somatic assessment, collected the samples for laboratory measurements, structured the manuscript and incorporated all parts of the manuscript; Jovanovic IP, Gajovic NM and Arsenijevic NN performed the cytokine measurements; Jurisevic MM and Borovcanin MM did the statistical analysis and prepared tables and figures; All authors, especially Debnath M, additionally searched the literature and provided new insights into specific areas of their expertise, made a final revision of the manuscript, and corrected the figures; All authors read, discussed, and approved the final version of the manuscript.

Supported by Ministry of Science and Technological Development of the Republic of Serbia, No. 175069; and Faculty of Medical Sciences, University of Kragujevac, No. JP15-05.

Institutional review board statement: The study was reviewed and approved by the Ethics Committee of the Kragujevac Clinical Centre.

Informed consent statement: All study participants provided informed written consent prior to study enrollment.

Conflict-of-interest statement: Minic Janicijevic S, Jovanovic IP, Gajovic NM, Jurisevic MM, Arsenijevic NN and Borovcanin MM has received research funding from Ministry of Science and Technological Development of the Republic of Serbia, No. 175069 and Faculty of Medical Sciences, University of Kragujevac, No. JP15-05. Debnath M declared no conflict of interest.

Data sharing statement: No additional data are available.

Country/Territory of origin: Serbia

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S-Editor: Fan JR
L-Editor: A
P-Editor: Fan JR

REFERENCES

1 **Debnath M**, Venkatasubramanian G, Berk M. Fetal programming of schizophrenia: select mechanisms. *Neurosci Biobehav Rev* 2015; **49**: 90-104 [PMID: 25496904 DOI: 10.1016/j.neubiorev.2014.12.003]
2 **Zengeler KE**, Lukens JR. Innate immunity at the crossroads of healthy brain maturation and neurodevelopmental disorders. *Nat Rev Immunol* 2021; **21**: 454-468 [PMID: 33479477 DOI: 10.1038/s41577-020-00487-7]
3 **Trépanier MO**, Hopperton KE, Mizrahi R, Mechawar N, Bazinet RP. Postmortem evidence of cerebral inflammation in schizophrenia: a systematic review. *Mol Psychiatry* 2016; **21**: 1009-1026 [PMID: 27271499 DOI: 10.1038/mp.2016.90]
4 **Dong Y**, Yong VW. When encephalitogenic T cells collaborate with microglia in multiple sclerosis. *Nat Rev Neurol* 2019; **15**: 704-717 [PMID: 31527807 DOI: 10.1038/s41582-019-0253-6]
5 **Bechter K**. Updating the mild encephalitis hypothesis of schizophrenia. *Prog Neuropsychopharmacol Biol Psychiatry* 2013; **42**: 71-91 [PMID: 22765923 DOI: 10.1016/j.pnpbp.2012.06.019]
6 **Subbanna M**, Shivakumar V, Talukdar PM, Narayanaswamy JC, Venugopal D, Berk M, Varambally S, Venkatasubramanian G, Debnath M. Role of IL-6/RORC/IL-22 axis in driving Th17 pathway mediated immunopathogenesis of schizophrenia. *Cytokine* 2018; **111**: 112-118 [PMID: 30138899 DOI: 10.1016/j.cyto.2018.08.016]
7 **Sahbaz C**, Zibandey N, Kurtulmus A, Duran Y, Gokalp M, Kırpınar I, Sahin F, Guloksuz S, Akkoc T. Reduced regulatory T cells with increased proinflammatory response in patients with schizophrenia. *Psychopharmacology (Berl)* 2020; **237**: 1861-1871 [PMID: 32221694 DOI: 10.1007/s00213-020-05504-0]
8 **Kim YK**, Myint AM, Lee BH, Han CS, Lee HJ, Kim DJ, Leonard BE. Th1, Th2 and Th3 cytokine alteration in

schizophrenia. *Prog Neuropsychopharmacol Biol Psychiatry* 2004; **28**: 1129-1134 [PMID: 15610925 DOI: 10.1016/j.pnpbp.2004.05.047]
9 **Upthegrove R**, Manzanares-Teson N, Barnes NM. Cytokine function in medication-naive first episode psychosis: a systematic review and meta-analysis. *Schizophr Res* 2014; **155**: 101-108 [PMID: 24704219 DOI: 10.1016/j.schres.2014.03.005]
10 **Dickerson F**, Stallings C, Origoni A, Schroeder J, Katsafanas E, Schweinfurth L, Savage C, Khushalani S, Yolken R. Inflammatory Markers in Recent Onset Psychosis and Chronic Schizophrenia. *Schizophr Bull* 2016; **42**: 134-141 [PMID: 26294704 DOI: 10.1093/schbul/sbv108]
11 **Chen HY**, Fermin A, Vardhana S, Weng IC, Lo KF, Chang EY, Maverakis E, Yang RY, Hsu DK, Dustin ML, Liu FT. Galectin-3 negatively regulates TCR-mediated CD4+ T-cell activation at the immunological synapse. *Proc Natl Acad Sci U S A* 2009; **106**: 14496-14501 [PMID: 19706535 DOI: 10.1073/pnas.0903497106]
12 **Borovcanin MM**, Radosavljevic GD, Pantic J, Milovanovic J, Mijailovic NR, Arsenijevic AN, Arsenijevic NN. Contrasting Roles of the Galectin-3 in the Schizophrenia Onset, Clinical Presentation, and Somatic Comorbidity. *Curr Top Med Chem* 2021; **21**: 1471-1487 [PMID: 34126898 DOI: 10.2174/1568026621666210611162420]
13 **Jeon SB**, Yoon HJ, Chang CY, Koh HS, Jeon SH, Park EJ. Galectin-3 exerts cytokine-like regulatory actions through the JAK-STAT pathway. *J Immunol* 2010; **185**: 7037-7046 [PMID: 20980634 DOI: 10.4049/jimmunol.1000154]
14 **Gittens BR**, Bodkin JV, Nourshargh S, Perretti M, Cooper D. Galectin-3: A Positive Regulator of Leukocyte Recruitment in the Inflamed Microcirculation. *J Immunol* 2017; **198**: 4458-4469 [PMID: 28438899 DOI: 10.4049/jimmunol.1600709]
15 **Pong S**, Karmacharya R, Sofman M, Bishop JR, Lizano P. The Role of Brain Microvascular Endothelial Cell and Blood-Brain Barrier Dysfunction in Schizophrenia. *Complex Psychiatry* 2020; **6**: 30-46 [PMID: 34883503 DOI: 10.1159/000511552]
16 **Bechter K**, Reiber H, Herzog S, Fuchs D, Tumani H, Maxeiner HG. Cerebrospinal fluid analysis in affective and schizophrenic spectrum disorders: identification of subgroups with immune responses and blood-CSF barrier dysfunction. *J Psychiatr Res* 2010; **44**: 321-330 [PMID: 19796773 DOI: 10.1016/j.jpsychires.2009.08.008]
17 **Parikh NU**, Aalinkeel R, Reynolds JL, Nair BB, Sykes DE, Mammen MJ, Schwartz SA, Mahajan SD. Galectin-1 suppresses methamphetamine induced neuroinflammation in human brain microvascular endothelial cells: Neuroprotective role in maintaining blood brain barrier integrity. *Brain Res* 2015; **1624**: 175-187 [PMID: 26236024 DOI: 10.1016/j.brainres.2015.07.033]
18 **Nishikawa H**, Liu L, Nakano F, Kawakita F, Kanamaru H, Nakatsuka Y, Okada T, Suzuki H. Modified Citrus Pectin Prevents Blood-Brain Barrier Disruption in Mouse Subarachnoid Hemorrhage by Inhibiting Galectin-3. *Stroke* 2018; **49**: 2743-2751 [PMID: 30355205 DOI: 10.1161/STROKEAHA.118.021757]
19 **Borovcanin MM**, Janicijevic SM, Jovanovic IP, Gajovic N, Arsenijevic NN, Lukic ML. IL-33/ST2 Pathway and Galectin-3 as a New Analytes in Pathogenesis and Cardiometabolic Risk Evaluation in Psychosis. *Front Psychiatry* 2018; **9**: 271 [PMID: 29988422 DOI: 10.3389/fpsyt.2018.00271]
20 **Ashraf GM**, Baeesa SS. Investigation of Gal-3 Expression Pattern in Serum and Cerebrospinal Fluid of Patients Suffering From Neurodegenerative Disorders. *Front Neurosci* 2018; **12**: 430 [PMID: 30008660 DOI: 10.3389/fnins.2018.00430]
21 **World Health Organization**. International Statistical Classification of Diseases and Related Health Problems Tenth Revision. Geneva: World Health Organization; 1992
22 **Liu J**, Xing Y, Gao Y, Zhou C. Changes in serum interleukin-33 levels in patients with acute cerebral infarction. *J Clin Neurosci* 2014; **21**: 298-300 [PMID: 24210798 DOI: 10.1016/j.jocn.2013.04.036]
23 **Kay SR**, Opler LA, Fiszbein A. Positive and Negative Syndrome Scale Manual. North Tonawanda, NY: Multi-Health Systems; 1994
24 **Rodriguez-Jimenez R**, Bagney A, Mezquita L, Martinez-Gras I, Sanchez-Morla EM, Mesa N, Ibañez MI, Diez-Martin J, Jimenez-Arriero MA, Lobo A, Santos JL, Palomo T; PARG. Cognition and the five-factor model of the positive and negative syndrome scale in schizophrenia. *Schizophr Res* 2013; **143**: 77-83 [PMID: 23201306 DOI: 10.1016/j.schres.2012.10.020]
25 **Ehmann TS**, Khanbhai I, Macewan GW, Smith GN, Honer WG, Flynn S, Altman S. Neuropsychological correlates of the PANSS Cognitive Factor. *Psychopathology* 2004; **37**: 253-258 [PMID: 15452413 DOI: 10.1159/000081022]
26 **Kljajevic V**. Montreal Cognitive Assessment: Scrb`s Version. Aktuelnosti iz neurologije, psihijatrije i granicnih podrucja, 2009; 17: 31–39
27 **Gil-Berrozpe GJ**, Sánchez-Torres AM, García de Jalón E, Moreno-Izco L, Fañanás L, Peralta V, Cuesta MJ; SEGPEPs group. Utility of the MoCA for cognitive impairment screening in long-term psychosis patients. *Schizophr Res* 2020; **216**: 429-434 [PMID: 31801676 DOI: 10.1016/j.schres.2019.10.054]
28 **Borovcanin MM**, Minic Janicijevic S, Jovanovic IP, Gajovic NM, Jurisevic MM, Arsenijevic NN. Type 17 Immune Response Facilitates Progression of Inflammation and Correlates with Cognition in Stable Schizophrenia. *Diagnostics (Basel)* 2020; **10** [PMID: 33182582 DOI: 10.3390/diagnostics10110926]
29 **Kumric M**, Ticinovic Kurir T, Borovac JA, Bozic J. Role of novel biomarkers in diabetic cardiomyopathy. *World J Diabetes* 2021; **12**: 685-705 [PMID: 34168722 DOI: 10.4239/wjd.v12.i6.685]
30 **Dal Lin C**, Brugnolo L, Marinova M, Plebani M, Iliceto S, Tona F, Vitiello G. Toward a Unified View of Cognitive and Biochemical Activity: Meditation and Linguistic Self-Reconstructing May Lead to Inflammation and Oxidative Stress Improvement. *Entropy (Basel)* 2020; **22** [PMID: 33286589 DOI: 10.3390/e22080818]
31 **Kajitani K**, Yanagimoto K, Nakabeppu Y. Serum galectin-3, but not galectin-1, levels are elevated in schizophrenia: implications for the role of inflammation. *Psychopharmacology (Berl)* 2017; **234**: 2919-2927 [PMID: 28698921 DOI: 10.1007/s00213-017-4683-9]
32 **Schroeder JT**, Adcosun AA, Bieneman AP. Epithelial Cell-Associated Galectin-3 Activates Human Dendritic Cell Subtypes for Pro-Inflammatory Cytokines. *Front Immunol* 2020; **11**: 524826 [PMID: 33154744 DOI: 10.3389/fimmu.2020.524826]
33 **Bonsack F**, Sukumari-Ramesh S. Differential Cellular Expression of Galectin-1 and Galectin-3 After Intracerebral Hemorrhage. *Front Cell Neurosci* 2019; **13**: 157 [PMID: 31156388 DOI: 10.3389/fncel.2019.00157]

34 **Borovcanin MM**, Vesic K, Jovanovic M, Mijailovic NR. Galectin-3 possible involvement in antipsychotic-induced metabolic changes of schizophrenia: A minireview. *World J Diabetes* 2021; **12**: 1731-1739 [PMID: 34754374 DOI: 10.4239/wjd.v12.i10.1731]

35 **Pang J**, Nguyen VT, Rhodes DH, Sullivan ME, Braunschweig C, Fantuzzi G. Relationship of galectin-3 with obesity, IL-6, and CRP in women. *J Endocrinol Invest* 2016; **39**: 1435-1443 [PMID: 27444618 DOI: 10.1007/s40618-016-0515-8]

36 **Tymińska A**, Kapłon-Cieślicka A, Ozierański K, Budnik M, Wancerz A, Sypień P, Peller M, Balsam P, Opolski G, Filipiak KJ. Association of Galectin-3 and Soluble ST2, and Their Changes, with Echocardiographic Parameters and Development of Heart Failure after ST-Segment Elevation Myocardial Infarction. *Dis Markers* 2019; **2019**: 9529053 [PMID: 31687050 DOI: 10.1155/2019/9529053]

37 **Shimura T**, Shibata M, Gonda K, Nakajima T, Chida S, Noda M, Suzuki S, Nakamura I, Ohki S, Takenoshita S. Association between circulating galectin-3 levels and the immunological, inflammatory and nutritional parameters in patients with colorectal cancer. *Biomed Rep* 2016; **5**: 203-207 [PMID: 27446542 DOI: 10.3892/br.2016.696]

38 **Le Mercier M**, Fortin S, Mathieu V, Kiss R, Lefranc F. Galectins and gliomas. *Brain Pathol* 2010; **20**: 17-27 [PMID: 19371355 DOI: 10.1111/j.1750-3639.2009.00270.x]

39 **Fermino ML**, Dias FC, Lopes CD, Souza MA, Cruz ÂK, Liu FT, Chammas R, Roque-Barreira MC, Rabinovich GA, Bernardes ES. Galectin-3 negatively regulates the frequency and function of CD4(+) CD25(+) Foxp3(+) regulatory T cells and influences the course of Leishmania major infection. *Eur J Immunol* 2013; **43**: 1806-1817 [PMID: 23592449 DOI: 10.1002/eji.201343381]

40 **Blokzijl A**, Dahlqvist C, Reissmann E, Falk A, Moliner A, Lendahl U, Ibáñez CF. Cross-talk between the Notch and TGF-beta signaling pathways mediated by interaction of the Notch intracellular domain with Smad3. *J Cell Biol* 2003; **163**: 723-728 [PMID: 14638857 DOI: 10.1083/jcb.200305112]

41 **Miller BJ**, Buckley P, Seabolt W, Mellor A, Kirkpatrick B. Meta-analysis of cytokine alterations in schizophrenia: clinical status and antipsychotic effects. *Biol Psychiatry* 2011; **70**: 663-671 [PMID: 21641581 DOI: 10.1016/j.biopsych.2011.04.013]

42 **Meyer U**, Schwarz MJ, Müller N. Inflammatory processes in schizophrenia: a promising neuroimmunological target for the treatment of negative/cognitive symptoms and beyond. *Pharmacol Ther* 2011; **132**: 96-110 [PMID: 21704074 DOI: 10.1016/j.pharmthera.2011.06.003]

43 **Obuchowicz E**, Bielecka-Wajdman AM, Paul-Samojedny M, Nowacka M. Different influence of antipsychotics on the balance between pro- and anti-inflammatory cytokines depends on glia activation: An in vitro study. *Cytokine* 2017; **94**: 37-44 [PMID: 28411046 DOI: 10.1016/j.cyto.2017.04.004]

44 **MacDowell KS**, Caso JR, Martín-Hernández D, Moreno BM, Madrigal JLM, Micó JA, Leza JC, García-Bueno B. The Atypical Antipsychotic Paliperidone Regulates Endogenous Antioxidant/Anti-Inflammatory Pathways in Rat Models of Acute and Chronic Restraint Stress. *Neurotherapeutics* 2016; **13**: 833-843 [PMID: 27233514 DOI: 10.1007/s13311-016-0438-2]

45 **Telford JE**, Bones J, McManus C, Saldova R, Manning G, Doherty M, Lewcke FM, Rothermundt M, Guest PC, Rahmoune H, Bahn S, Rudd PM. Antipsychotic treatment of acute paranoid schizophrenia patients with olanzapine results in altered glycosylation of serum glycoproteins. *J Proteome Res* 2012; **11**: 3743-3752 [PMID: 22594947 DOI: 10.1021/pr300218h]

46 **Jovanovic M**, Simovic Markovic B, Gajovic N, Jurisevic M, Djukic A, Jovanovic I, Arsenijevic N, Lukic A, Zdravkovic N. Metabolic syndrome attenuates ulcerative colitis: Correlation with interleukin-10 and galectin-3 expression. *World J Gastroenterol* 2019; **25**: 6465-6482 [PMID: 31798282 DOI: 10.3748/wjg.v25.i43.6465]

47 **Rind L**, Ahmad M, Khan MI, Badruddeen, Akhtar J, Ahmad U, Yadav C, Owais M. An insight on safety, efficacy, and molecular docking study reports of N-acetylcysteine and its compound formulations. *J Basic Clin Physiol Pharmacol* 2021; **33**: 223-233 [PMID: 33638319 DOI: 10.1515/jbcpp-2020-0099]

48 **Wasyanto T**, Yasa' A, Jalaludinsyah A. Effect of Oral N-Acetylcysteine Supplementation on the Immunity System in Patients with Acute Myocardial Infarction. *Acta Med Indones* 2019; **51**: 311-317 [PMID: 32041914]

49 **Pyatoykina AS**, Zhilyaeva TV, Semennov IV, Mishanov GA, Blagonravova AS, Mazo GE. [The double-blind randomized placebo-controlled trial of N-acetylcysteine use in schizophrenia: preliminary results]. *Zh Nevrol Psikhiatr Im S S Korsakova* 2020; **120**: 66-71 [PMID: 33081449 DOI: 10.17116/jnevro202012009166]

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