BIOGRAPHICAL SKETCH

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NAME: Lubov Nathanson

eRA COMMONS USER NAME (credential, e.g., agency login): LUBOVNATHANSON

POSITION TITLE: Assistant Professor

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

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Moscow State University, Moscow, Russia	M.S.	05/1985	Biology/Biophysics
Weizmann Institute of Science, Rehovot, Israel	Ph.D.	02/1998	Molecular Biology & Biochemistry
University of Miami, Miami, Florida	Post-Doctoral	05/2000	Biochemistry

A. Personal Statement

I earned my PhD degree in Molecular Biology and Biochemistry from the prestigious Weizmann Institute of Science, Israel. My Ph.D. and postdoctoral experience allowed me to gain better understanding of cellular processes. As a Manager and later Director of the Gene Expression and Microarray Core (2004 - 2010), I gained extensive hands-on experience of sample processing for microarray, Nanostring and NextGen sequencing analyses. As part of the Bioinformatics group (2010-2012) I used and evaluated many different bioinformatics software tools. I also gained experience in analyzing large genomics datasets as well as conducting systems biology analyses. Thus, I have combined expertise in bioinformatics, Molecular Biology, Biochemistry and Systems Biology. Much of my academic work in recent years involved analyses of gene expression data from Nanostring, microarrays and RNA-seq (see publications).

B. Positions and Honors

Positions and Employment

2000-2004 Senior Research Associate, Dept. of Biochemistry, University of Miami Miller School of Medicine, Miami, FL, USA

2004-2007 Assistant Scientist, Manager of Microarray Core Facility, University of Miami Miller School of Medicine, Miami, FL, USA

2007-2010 Director of Microarray and Gene Expression Core, Hussman Institute for Human Genomics, University of Miami Miller School of Medicine, Miami, FL, USA

2010-2012 Scientist, Bioinformatics group, Hussman Institute for Human Genomics, University of Miami Miller School of Medicine, Miami, FL, USA

2012 Research Consultant, Miami Veterans Affair Healthcare System, Miami, FL

2012-Present Assistant Professor, Dept. of Neuro-Immune Medicine, Nova Southeastern University, Fort Lauderdale, FL, USA

2012-Present Voluntary Assistant Professor, Dept. of Medicine, University of Miami Miller School of Medicine, Miami, FL, USA

Other Recent Relevant Experience and Professional Memberships

2008-Present American Society of Human Genomics

C. Contribution to Science

1. My earlier research contributed to the understanding of the structure and function of the protein synthesis apparatus in mammalian cells. Publications from this period showed significance of the protein complexes and their interaction with the cytoskeleton in the mammalian cells.
   a. **Nathanson L**, Deutscher MP. Active aminoacyl-tRNA synthetases are present in nuclei as a high molecular multienzyme complex. *J Biol Chem*, 2000, 275(41:31559-62
   b. **Nathanson L**, Xia T, Deutscher MP. Nuclear protein synthesis: a re-evaluation. *RNA*, 2003, 9(1):9-13
   c. Hudder A, **Nathanson L**, Deutscher MP. Organization of mammalian cytoplasm. *Mol Cell Biol*, 2003, 23(24):9318-26

2. I was actively involved in the projects that investigated development of cancer and helped to evaluate the effects of anti-cancer grugs.
   a. Ramachandran K, Speer C, **Nathanson L**, Claros M, Singal R. Role of DNA methylation in cabazitaxel resistance in prostate cancer. *Anticancer Res*, 2016, 36(1):161 - 168
   b. Subbarayan PR, Sarkar M, **Nathanson L**, Doshi N, Lokeshwar B, Ardalan B. In vitro global gene expression analyses support the ethno-pharmacological use of *Achyranthes aspera*, Evid Based Complementary Altern Med. Volume 2013, Article ID 471739, 13 pages, 2013 http://dx.doi.org/10.1155/2013/471739.
   c. Piña Y, Houston SK, Murray TG, Koru-Sengul T, Decatur C, Scott WK, **Nathanson L**, Clarke J, Lampidis TJ. Retinoblastoma treatment: impact of the glycolytic inhibitor 2-deoxy-d-glucose on molecular genomics expression in LH(BETA) T(AG) retinal tumors. *Clin Ophthalmol*, 2012, 6:817-830. PMID: 22701083.
   d. Houston SK, Pina Y, Clarke J, Koru-Sengul T, Scott WK, **Nathanson L**, Schefler AC, Murray TG. Regional and temporal differences in gene expression of LHBETATAG retinoblastoma tumors. *Invest. Ophthalmol. Vis. Sci.* 2011, 52(8): 5359 – 5368. PMID: 21571674.

3. As a manager, and later as a Director of the Microarray and Gene Expression Core, I participated in multiple projects and was responsible for the analysis of the large genomic datasets.
   a. Humphries CE, Kohli MA, **Nathanson L**, Whitehead P, Beecham G, Martin E, Mash DC, Pericak-Vance MA, Gilbert J. Integrated Whole Transcriptome and DNA Methylation Analysis Identifies Gene Networks Specific to Late-Onset Alzheimer's Disease. *J Alzheimers Dis*. 2015 Jan; 44(3):977-987.
   b. McElroy JP, Krupp LB, Johnson BA, McCauley JL, Qi Z, Caillier SJ, Gourraud PA, Yu J, **Nathanson L**, Belman AL, Hauser SL, Waubant E, Hedges DJ, Oksenberg JR. Copy number variation in pediatric multiple sclerosis. Mult Scler. 2013 Jul;19(8):1014-21
   c. Hedges DJ, Hamilton-Nelson KL, Sacharow SJ, Nations L, Beecham GW, Kozhekbaeva ZM, Butler BL, Cukier HN, Whitehead PL, Ma D, Jaworski JM, **Nathanson L**, Lee JM, Hauser SL, Oksenberg JR, Cuccaro ML, Haines JL, Gilbert JR, Pericak-Vance MA. Evidence of novel fine-scale structural variation at autism spectrum disorder candidate loci. Mol Autism. 2012 Apr 2;3:2

4. Lately I participated in the genomic research in order to find new therapeutic strategies for the multi symptoms diseases such as Chronic Fatigue Syndrome and Gulf War Illness.
   a. Broderick G, Ben-Hamo R, Vashishtha S, Efroni S, **Nathanson L**, Barnes Z, Fletcher MA, Klimas N. Altered immune pathway activity under exercise challenge in Gulf War Illness: an exploratory analysis. *Brain Behav Immun*. 2013, Feb;28:159-69
   b. Craddock TJ, Harvey JM, **Nathanson L**, Barnes ZM, Klimas NG, Fletcher MA, Broderick G (2015) *BMC Med Genomics*. 2015, Jul 9;8:36

Complete List of Published Work in MyBibliography:

http://www.ncbi.nlm.nih.gov/myncbi/browse/collection/47397055/?sort=date&direction=ascending

D. Research Support

Ongoing Research Support

R15 NS087604-01A1 (PI Nathanson) 04/15/2015 – 03/31/2018
NIH NINDS
Genomic approach to find novel biomarkers and mechanisms of CFS/ME.
The employment of advanced technologies (RNA-seq, Copy Number Variation and DNA methylation) and a well-rounded research approach to identifying regulators of transcription that result in characteristic symptomatology associated with CFS/ME, will enable us to provide clinicians with novel biomarkers within regulatory systems that can improve CFS/ME diagnosis and potentially management.
Role: PI

R15 NS087604-01A1S1 (PI Nathanson) 08/03/2015 – 03/31/2018
NIH NINDS
Genomic approach to find novel biomarkers and mechanisms of CFS/ME.
Research Supplement to Promote Diversity in Health-Related Research (Admin. Supp.).
Role: PI

R15 NS087604-01A1S2 (PI Nathanson) 08/03/2015 – 03/31/2018
NIH NINDS
Genomic approach to find novel biomarkers and mechanisms of CFS/ME.
Research Supplement to Promote Diversity in Health-Related Research (Admin. Supp.).
Role: PI

R15 NS087604-01A1S3 (PI Nathanson) 08/01/2016 – 07/31/2017
NIH NINDS
Genomic approach to find novel biomarkers and mechanisms of CFS/ME.
Administrative Supplement to Existing NIH Grants and Cooperative Agreements.
Role: PI

R21AI124187-01 (PI Nathanson) 04/15/2016 – 03/31/2018
NIH NIAID
Sex-specific genomic mechanisms of transcriptional regulation in ME/CFS/SEID.
In an effort to provide insight into the key biological targets involved in sex-specific ME/CFS/SEID presentation, the main objective of this research proposal is to identify male-specific biomarkers and therapeutic targets of ME/CFS/SEID and provide insight into sex-specific disease onset and progression, which will lead to the better therapeutic intervention.
Role: PI

R21AI124187-01S1 (PI Nathanson)
NIH NIAID
Sex-specific genomic mechanisms of transcriptional regulation in ME/CFS/SEID.
Research Supplement to Promote Diversity in Health-Related Research (Admin. Supp.).
Role: PI

GW140077 (PI Waziry) 07/01/2015 – 06/30/2018
DoD GWIRP
An integrated genomics and cell biology approach to correlate novel GWI indicators of infections and neuroinflammatory mechanisms with targeted drug therapy.
The goal of this study is to investigate changes in the transcriptome, possible regulation mechanisms of these changes and possible viral influence on the cellular distribution of the transcripts. This study will reveal novel

biomarkers for development of better diagnostics, and provide clear targets for therapeutic intervention in Gulf War Illness.
Role: Co-PI

R01 NS090200-01 (PI Fletcher) 9/01/2014 – 4/30/2018
NIH
Gender Differences in Myalgic Encephalomyelitis/Chronic Fatigue Syndrome.
We aim to understand the mediators of persistence and relapse in men with ME/CFS, as we have in women. We will approach this by: (i) integration across several of the body's regulatory systems of data and knowledge collected from disparate sources, and (ii) mapping of the coordinated interactions between these physiologic systems and the potential for dysfunctional signaling networks. This project will extend this modeling of immune regulatory pathways and pathways that regulate latent viral expression in a way that will enable us to compare gender differences in illness mechanisms and explore gender-specific therapeutic targets. My role is to design, conduct and interpret experiments involving genomics, such as microarrays and Nanostring. Results of these projects will help to identify the mediators of persistence and relapse in men with ME/CFS at the level of transcription.
Role: Co-Investigator

W81XWH13-2-0072 (PI Sullivan) 09/30/13-09/29/2017
DoD USAMRAA
Brain-Immune Interactions as the Basis of Gulf War Illness: Gulf War Illness Consortium (GWIC).
My role is to provide genomic lab support to the investigators and to help with the interpretation of results.
Role: Co-Investigator

Completed Research Support

PFRDG14 (PI Nathanson) 07/01/2013-01/01/2015
NSU
Regulation of the Metabolic Changes in Chronic Fatigue Syndrome: Role of CNV
The goal of this project is to establish Nanostring assays of changes in copy number variations of the genomic regions of the differentially expressed genes in the Chronic Fatigue Syndrome patients.
Role: PI

R56 AI065723-06A1 (PI Fletcher) 12/15/2006 – 8/31/2014 (ext. 8/31/2015)
NIH
Immunologic Mechanisms, Biomarkers and Subsets in CFS/ME.
In this project extension, we will further investigate the potential illness mechanisms that drive the altered patterns of immune signaling that we observed and published, with the objective of designing a robust multiplex ELISA-based assay that captures the most clinically relevant interactions linking markers of immune, endocrine and nervous system function. My effort includes design of the Nanostring panel for the further investigation of the potential illness mechanisms.
Role: Co-Investigator

PFRDG15 (PI Nathanson) 07/01/2014 – 06/30/2015
NSU
Explorative gene expression and pathway analysis for innovative cancer treatment.
We propose an extensive investigation of cancer cell gene expression upon statin treatment and viral infection. In order to accomplish this, we will measure expression of all cellular transcripts using RNAseq, which is an unbiased discovery tool. Furthermore, we will use bioinformatics tools to correlate gene expression for identification of specific pathways that can be further targeted for controlled oncolytic virotherapy.
Role: PI

PFRDG15 (PI Waziry) 07/01/2014 – 06/30/2015
NSU

Epigenetic modulation of viral infection: role of DNA methylation in recovery.
We propose to study methylation patterns by comparing statin-treated, infected and non-treated cells, followed by correlation of such changes in methylation with statins' modulation of gene expression.
Role: Co-PI