

CURRICULUM VITAE

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Education

<u>Year</u>	<u>Degree</u>	<u>Field</u>	<u>Institution</u>
1989	B.A.	Biology	Division of Biology, Moscow State University, Moscow, USSR
1992	M.S.	Genetics	Department of Genetics, Moscow State University, Russia
1995	Ph.D.	Biochemistry	Institute of Molecular Genetics, Russian Academy of Sciences

Postdoctoral Training

<u>Dates</u>	<u>Specialty</u>	<u>Mentor</u>	<u>Place of Training</u>
1995-1997	Molecular Biology	Dr. A. Goldfarb	Public Health Research Institute, New York

Academic Appointments

1997-2003	Assistant Professor, New York University School of Medicine, New York, NY
2003-2005	Associate Professor (tenured), New York University School of Medicine
2005-2008	Professor, Dept. of Biochemistry, NYU School of Medicine
2008-	Julie Wilson Anderson Professor, NYU School of Medicine
2013-	Investigator, Howard Hughes Medical Institute

Awards/Honors

1997	The Helen Hay Whitney Foundation research fellowship (declined)
1998	The Searle Scholar Award
2001	The Irma T. Hirschl Career Scientist Award
2002	The Edward Mallinckrodt, Jr. Foundation Research Award
2002	Fogarty International Research Collaboration Award
2004	Keynote Lecture: The RNA Biochemistry Symposia. Blaubeuren, Germany
2004	United States-Israel Bi-national Science Foundation Award
2006	The Dynasty Foundation Award
2006	NIH Director's Pioneer Award
2008	Crain's "40 Under 40" Alumni
2008	Julie Wilson Anderson Professorship (endowment chair)
2009	The Vilcek Foundation Finalist Prize
2010	Keynote Lecture: The Harden Conference on Transcription. Cambridge, UK
2010	The Blavatnik Award, New York Academy of Science
2011	The Robertson Foundation Award
2012	Biogerontology Research Foundation Award
2013	Investigator, Howard Hughes Medical Institute
2013	Honorary Professor, Moscow Institute of Physics and Technology

2013	Master Scientist, NYU Langone Medical Center
2014	Dean's Honor Lecture, NYU School of Medicine
2016	The Neil Welker Memorial Award
2016	Engelhardt Memorial Lecture, IMB, Russian Academy of Science
2016	Elected Foreign Member, Russian Academy of Sciences
2017	Elected Fellow, American Academy of Arts and Sciences
2018	Glenn Award for Research in Biological Mechanisms of Aging
2019	Blavatnik Family Foundation Award

Research Statement

Our most significant contributions to science include:

1. Discovery and implications of RNA polymerase (RNAP) “backtracking” and “ratcheting”: In 1997 we described back-and-forth sliding of RNAP along DNA and RNA (Nudler et al., *Cell* 1997). Our group then showed that this universal phenomenon plays a key role in controlling gene expression (e.g. Proshkin et al., *Science* 2010), genome instability (e.g. Dutta et al., *Cell* 2011), and DNA repair (Epshtein et al., *Nature* 2014). We were also the first to demonstrate that RNAP is a Brownian ratchet machine (Bar-Nahum et al., *Cell* 2005). Our findings explained in mechanistical detail how RNAP translocates, how it responds to regulatory signals and factors, and how it terminates transcription.

2. Uncovering the mechanisms of transcription termination and antitermination: We have uncovered the general mechanistical principles of the termination process in bacteria and formulated models explaining the intricate molecular pathways leading to both intrinsic and Rho-dependent transcription termination (Gusarov et al., *Mol Cell*, 1999; Epshtein et al., *Mol Cell* 2007; Epshtein et al., *Nature* 2010). We also studied how these processes are regulated in the cell and formulated the mechanistical models of factor-dependent (antitermination) (Gusarov et al., *Cell* 2001) and factor-independent (riboswitches - Mironov et al., *Cell* 2002; RAPs – Sedlyarova et al., *Mol Cell* 2017) modes of regulation. Our studies uncovered novel functions of Rho, such as its role in silencing of horizontally transferred (and potentially toxic) genes (Cardinal et al., *Science* 2008) and in preserving genomic integrity (Dutta et al., *Cell* 2011). We found that Rho functions as the global regulator of transcription acting at the 5'UTRs of hundreds of bacterial genes, and that sRNAs control Rho-dependent termination genome-wide, thus establishing sRNAs as transcription elongation factors (Sedlyarova et al., *Cell* 2016).

3. Discovery of a new mechanism of transcription-coupled DNA repair (TCR): In 2014 we uncovered a general mechanism of TCR that relies on active RNAP backtracking (Epshtein et al., *Nature* 2014). He found that in bacteria, the key DNA repair factor UvrD binds RNAP during transcription elongation and, using its helicase activity, forces RNAP to slide backward along DNA. By inducing backtracking, UvrD exposes DNA lesions shielded by blocked RNAP, allowing the repair enzymes to gain access to sites of damage. The active recruitment of nucleotide excision repair machinery to backtracked RNAP greatly expedites the DNA repair process. We also showed that the small molecule bacterial alarmone ppGpp and general elongation factor NusA contribute to UvrD-mediated TCR (Kamarthapu et al., *Science* 2016). Because backtracking is a shared feature of all cellular RNAP, this mechanism enables RNAPs to function as global DNA damage scanners in bacteria and eukaryotes.

4. Discovery of riboswitches: In 2002, we discovered first ligand-sensing mRNAs that regulate biosynthetic genes in *B. subtilis* (Mironov et al., *Cell* 2002). Simultaneously, Breaker and colleagues reported similar findings in *E. coli*. Since then, dozens of riboswitches have been described in bacteria and eukaryotes where they control numerous genes. We have shown that riboswitches can activate and suppress gene expression acting at the level of transcription termination, translation initiation, and modulating alternative splicing and mRNA stability

(reviewed in Serganov and Nudler, *Cell* 2013).

5. Discovery of eukaryotic RNA thermosensor: In 2006 we isolated a complex composed of the translation elongation factor eEF1A1 and a novel non-coding RNA (HSR1) that is required for activation of heat shock genes in mammals (Shamovsky et al., *Nature* 2006). We have shown that HSR1 serves as a molecular thermosensor. We also showed that eEF1A1 orchestrates the whole process of heat shock response, from transcription activation to mRNA stabilization, transport, and translation (Vera et al., *eLife* 2014). These findings provide a new paradigm of cellular adaptation to stress, with far-reaching clinical implications in neurodegeneration and cancer.

6. Discovery of a gas defense system in bacteria: We showed that endogenously produced gases NO and H₂S protect bacteria from oxidative stress, immune attack, and numerous antibiotics (Gusarov et al., *Science* 2009; Shatalin et al., *Science* 2011). These results support the emerging concept of antibiotic killing, which relies in part on oxidative damage, and establish NO- and H₂S-producing enzymes as promising new targets for antimicrobial therapy. We also showed that NO produced by bacteria inside their host (*C. elegans*) diffuses into animal's tissues where it activates a defined set of genes that protect animals from environmental stress and extend their lifespan (Gusarov et al., *Cell* 2013).

Bibliography:

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