Biographical Sketch

Provide the following information for each individual included in the Research & Related Senior/Key Person Profile (Expanded) Form.			
NAME ARI BARZILAI	POSITION TITLE PROFESSOR OF NEUROBIOLOGY		
EDUCATION/TRAINING (Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training).			
INSTITUTION AND LOCATION	DEGREE (IF APPLICABLE)	YEAR(S)	FIELD OF STUDY
The Hebrew University of Jerusalem, Israel	B.Sc.	1977-1980	Physics, Chemistry and Biology
The Hebrew University of Jerusalem, Israel	M.Sc.	1980-1982	Neurobiology, Cum laude
The Hebrew University of Jerusalem, Israel	Ph.D.	1982-1987	Neurobiology, Summa cum laude
Columbia University of NYC, NY, USA	Post-Dco	19871992	Neurobiology
Tel Aviv University, Tel Aviv, Israel	Lecturer	1992-1996	Neurobiology
Tel Aviv University, Tel Aviv, Israel	Senior lecturer	1996-2002	Neurobiology
Tel Aviv University, Tel Aviv, Israel	Associate Prof.	2002-2007	Neurobiology
Tel Aviv University, Tel Aviv, Israel	Professor	2007-Present	Neurobiology

Most Significant Contribution PIN: 265228 BARZILAI, Ari

1. **A. Cellular and molecular mechanisms underlying DA toxicity: Implication for Parkinson's disease (PD):** We were among the first to hypothesize and to validate that dopamine (DA) toxicity is a player in the etiology of PD. We showed DA toxicity involves p53 induction (Daily et al, Cell. Mol. Neurosci. 1999), as well as activation TCP-1δ is a subunit in the Chaperonin-containing TCP-1 (CCT) which is the cytosolic homologue of mitochondrial HSP60 (Zilkha-Falb et al, J. Biol. Chem. 2000).
2. **Semaphorin 3A is an apoptosis-induced molecule:** Sema3A belong to a large family of axonal guidance molecules which can confer attractive or repulsive cues. Neutralization of Sema3A or its receptor Neuropilin1 inhibited DA toxicity (Shirvan et al, J. Neurochem 1999). We also studied the involvement of semaphorins in *in vivo* experimental animal model of complete axotomy of the rat optic nerve. We demonstrated a marked inhibition of retinal ganglion cell (RGC) loss when axotomized eyes were co-treated by intra-vitreal injection of function-blocking antibodies against the Sema3A derived peptide (Shirvan et al. J. Biol. Chem. 2001). We further showed that sema3A was involved in the death of retinal ganglion cells in glaucomatous rabbits (Graefe's Arch. Clin. Exp. Ophthalmol. 2003) as well as in retinal detachment (Klebanov et al Graefe's Arch. Clin. Exp. Ophthalmol. 2009). Inactivation of Nbs1, which is an essential component of the DNA damage response altered the morphology and organization of the glial cells. We further showed that the levels semaphorin-3A and its receptor neuropilin-1 were up-regulated in the retina of the mutant mice, a typical injury response (Barranes et al Exp. Neurol. 2009). In addition, we demonstrated that exogenous administration of Sema-3A into the fish eye indirectly interferes with the regeneration process of the optic nerve. The findings corroborate our previous findings in mammals, and further validate Sema-3A as a key factor in the generation of a non-permissive environment after transection of the optic nerve (Rosenzweig et al Graefe's Arch. Clin. Exp. Ophthalmol. 2010). *Together, our studies open a new field and places Sema3A as a pro-apoptotic molecules that is a target for regenerative medicine as well as in anti-cancer therapy.*
3. **Cellular and molecular mechanisms of stress responses in ataxia telangiectasia:** We have shown that Atm deficiency led to the generation of oxidative stress specifically in the cerebellum (Kamsler et al, Cancer Res.

2001). We present evidence that ATM plays a key role in maintaining cellular homeostasis (Daily et al, J. Biol. Chem. 2001a,b, Stern et al, J. Biol. Chem. 2002; Weizman et al J. Biol. Chem. 2003). *This is now a widely accepted view of ATM functionality*. We found that cancer predisposition, which is typical of Atm-deficient animals was not enhanced in SOD1/Atm double null mice. In contrast, Mlh1/Atm double null mice did not display growth retardation or increased radioensitivitry compared to Atm-/- animals, but succumbed to lymphoma at significantly higher rate compared to Atm-deficient mice (Ziv et al, Hum. Mol. Genet. 2005). In contrast to the current dogma in the field, we found that ATM is essentially nuclear in these neuronal cells and that various readouts of the ATM-mediated damage response are similar to those seen in commonly used cell lines (Dar et al, J. Neurosci. 2006). Using MRI analysis we showed that malfunctioning DNA damage response severely affects the levels of white matter and its organization (Assaf et al, Exp. Neurol. 2008). We found that A-T-mutated protein deficiency was consistent with aberrant astrocytic morphology and alterations of the vasculature, often accompanied by reactive gliosis. Interestingly similar findings could also be reported in the case of other genetic disorders. (Raz-Prag et al, Am. J. Pathol. 2011; Mehsulam et al Front. Pharmacol. 2012). *These observations bolster the notion that astrocyte-specific pathologies, hampered vascularization and astrocyte-endothelium interactions in the CNS could play a crucial role in the etiology of genome instability brain disorders.*

4. **The role of ATM in the dynamics of neural-glial networks:** We discovered that Atm protein deficiency, which in humans leads to progressive motor impairment, leads to a reduced synchronization persistence compared to wild type synchronization, after chemically imposed DNA damage. Not only do these results suggest a role for DNA stability in neural network activity, they also establish an experimental paradigm for empirically determining the role a gene plays on the behavior of a neural network (Levine-Small et al, Front. Neurosci. 2011). We found that replacement of Atm-/- astrogliall cells with WT cells restores physiological neuronal network dynamics in *in-vitro* chimera neuron-glia networks extracted from Atm-deficient mice. These results support the notion that neuronal network failures in genetic brain degenerative diseases are strictly correlated with impairment of astroglial cell functionality (Kanner et al In Preparation 2015).
5. **Tools to study the dynamics of various types of neural-glial networks:** We designed the procedures for the preparation of modular neuronal networks composed of functionally inter-connected circuits (Kanner et al J. Vis. Exp. 2015).

List of publication

1. **Barzilai, A.,** and Rahamimoff, H., Inhibition of Ca^{++} transport ATPase from synaptosomal vesicles by flvonoides. Biochem Biophys. Acta. **730** 245-254 (1983).

2. **Barzilai, A.,** Spanier, R., and Rahamimoff, H., Isolation, purification and reconstitution of the Na^{+} gradient dependent Ca^{++} transport (Na^{+}-Ca^{++} exchanger) from brain synaptic plasma membrane vesicles. Proc. Natl. Acad. Sci. USA **81** 6521-6529 (1984).

3. Hermoni, M., **Barzilai, A**., and Rahamimoff, H., modulation of the Na^{+}-Ca^{++} antiport by its ionic environment: The effect of Li^{+}. Isr. J.Med. Sci. **23** 44-48 (1987).

4. **Barzilai, A.,** and Rahamimoff, R., The Stochiometry of the Na^{+}-Ca^{++} exchanger in nerve terminals. Biochemistry **26** 6113-18 (1987).

5. **Barzilai, A.,** Spanier, R., and Rahamimoff, H., Immunological identification of the synaptic plasma membrane Na^{+}-Ca^{++} exchanger. J. Biol. Chem. **262** 10315-10320 (1987).

6. Kennedy, T.E., Gawinowicz, M.A., **Barzilai, A.**, Kandel, E.R., and Sweatt., J.D., Sequencing of proteins from two-dimensional gels by using in situ digestion and transfer of peptides to PVDF membranes: Application to proteins associated with sensitization in .*Aplysia*. Proc. Natl. Acad. Sci. USA **85** 7008-7012 (1988).

7. Kennedy, T.E., Wager-Smith, K., **Barzilai, A.,** kandel, E.R., and Sweatt, J.D., Sequencing proteins from acrylamid gels. Nature **336** 499-500 (1988).

8. Sweatt, J.D., Wager-Smith, K., Gawinowicz-Koks, M.A., **Barzilai, A.,** Karl, K.A., and Kandel, E.R., Development of amino acids sequences for proteins identified and isolated on two dimension poly acrylamid gels Electrophoresis, **10** 152-157 (1989).

9. **Barzilai, A.,** Kennedy, T.E., Sweatt, J.D., and Kandel, E.R., 5-HT modulates protein synthesis and the expression of specific proteins during long term facilitation in *Aplysia* sensory neurons. Neuron **2** (1989) 1577-1586.

10. Schacher, S., Glanzman, D., **Barzilai, A.**, Dash, P., Grant, S.G.N., Keller, F., Mayford, M., and Kandel, E.R., Long term facilitaion in *Aplysia*: Possible phosphrylation and structural changes. Cold Spring Harbor Symp. Quant. Biol. **55** 187-200 (1990).

11. Mayford, M., **Barzilai, A.,** Keller, F., Schacher, S., and Kandel, E.R., modulation of an NCAM-related molecule with long term synaptic plasticity in *Aplysia*. Science **256** 638-644 (1992).

12. Kennedy, T.E., Kuhl, D., **Barzilai, A.,** Sweatt, J.D., and Kandel, E.R., long term sensitization training in *Aplysia* leads to an increase in calreticulin a major presynaptic calcium binding protein. Neuron **9** 1013-1024 (1992).

13. Khul, D., Kennedy, T.E., **Barzilai, A.,** and Kandel, E.R., Long term sensitization in *Aplysia* leads to increase in the expression of Bip, the major protein chaperon of the endoplasmic reticulum. J. cell. Biol. **119** 1069-1076 (1992).

14. Hu, Y.H., **Barzilai, A.,** Chen, M., Bailey, C.H., and Kandel, E.R., 5-HT and cAMP induce the formation of coated pits and vesicles and increase the expression of clathrin light chain in sensory neurons of *Aplysia*. Neuron **10** 921-929 (1993).

15. Soussan, L., **Barzilai, A.,** and Michaelson, D.M., Distinctly phosphorylated neurofilament in different classes of neurons. J. Neurochem. **62** 770-776 (1994).

16. Ziv, I., Melamed, E., Nardi, N., Luria, D., Achiron, A., Offen, D., and **Barzilai, A.,** Dopamine induces apoptosis like cell death in cultured chick embryo sympathetic neurons- A possible novel pathogenic mechanism in Parkinson's disease. Neuroscience Lett. **170** 136-140 (1994).

17. Offen, D., Ziv, I., Gorodin, S., **Barzilai, A.,** Malik, Z., and Melamed, E., Dopamine- induced programmed cell death in mouse thymocytes. Biophys. Biochem. Acta. **1268** 171-177 (1995).

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20. Ziv, I., Offen, D., **Barzilai, A.,** Haviv, R., Stein, R. ,Zilkha-Falb, R., Shirvan, A., and Melamed, E., Modulation of control mechanisms of dopamine induced apoptosis- A further approach to the treatment of Prkinson's disease. J. NeurL. Transm. [Suppl] **49** 195-201 (1997)

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23. Shirvan, A., Ziv, I., Michlin, T., Djaldeti, R., Zilkha-Falb, R., Melamed, E., and **Barzilai, A.,** Two waves of cell cycle related events are induced in chick embryo sympathetic neurons during dopamine triggered apoptosis. J. Neurochem. **69** 539-549 (1997).

24. Offen, D., Ziv, I., Panet, H., Wasserman, L., Stein, R., Melamed, E and **Barzilai, A.,** Dopamine-induced apoptosis is inhibited in PC12 cells expressing Bcl-2. Cell. Mol. Neurobiol. **17** 2289-304 (1997)

25. Offen, D., Ziv, I., **Barzilai, A.,** Gorodin, S., Glater, E., Hochman, A., and Melamed, E., Dopamine-melanin induces apoptosis in PC12 cells: Possible implication for the ethiliology of Parkinson's disease. Neurochem. Inter. **2** 206-217 (1997).

26. Ziv, I., Offen, D., Haviv, R., Stein, R., Achiron, A., Panet, H., **Barzilai, A.,** and Melamed, E., The protooncogene bcl-2 inhibits cellular toxicity of dopamine: Possible implication for Parkinson's disease. Apoptosis. **2** 149-155 (1997).

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28. Shirvan, A., Ziv, I., Zilkha-Falb, R., Machlin, T., **Barzilai, A.,** and Melamed, E., Expression of cell cycle related genes during neuronal apoptosis: Is there a distinct pattern? Neurochem. Res. **23** 767-777 (1998).

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33. Shirvan, A., Melamed, E., and **Barzilai, A.,** Induction of neuronal apoptosis by semaphorin 3A derived peptide. Brain Res. Mol. Brain Res. **83** 81-93 (2000).

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39. Daily, D., Vlamis, A., Offen, D., Holmgren, A., Melamed, E., Mittelmean, L., and **Barzilai, A.,** Glutaredoxin protects cerebellar granule neurons from dopamine-induced apoptosis by dual activation of the Ras-Phosphoinositide 3 Kinase and Jun N-terminal kinase pathways. J. Biol. Chem. **276** 21618-21626 (2001).

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42. Shirvan, A., Kimron, M., Holdengreber, V., Ziv, I., Melamed, S., Melamed, E., **Barzilai, A.,** and Solomon, A.S., Anti sema3A antibodies resuce retinal ganglion cells from cell death induced by optic nerve axotomy. J. Biol. Chem. **277** 49799-49807 (2002).

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Active participation in international scientific meetings

1. Sivan Kanner, Paolo Bonifaci, Eshel Ben Jacob and Ari Barzilai (2015). The role of ATM deficiency in cerebellar functionality. The 16th AT workshop, Beijing China, Beijing, China
   Invited?: Yes

2. Sivan Kanner, Paolo Bonifazi, Eshel Ben Jacob, Ronit Galron and Ari Barzilai (2015). The role of Glial cells in the etiology of A-T. XII European Meeting on Glial Cells in Heath and Disease, Bilbao Spain, Bilbao, Spain
   Invited?: Yes

3. Sivan Kanner, Paolo Bonifazi, Eshel Ben Jacob, Ronit Galron and Ari Barzilai (2015). The role of glial in the etiology of A-T. The Saarland University-Tel Aviv University meeting, Homburg, Germany
   Invited?: Yes

4. Sivan Kanner, Paolo Bonifazi, Ronit Galron, Eshel Ben Jacob and Ari Barzilai (2014). The role of Atm in the dynamics of neuro-glial networks. 2nd international Integrated Neuroscience Forum, Delhi, India
   Invited?: Yes

5. Sivan Kanner, Ronit Galron, Paolo Bonifazi, Eshel Ben Jacob and Ari Barzilai (2014). The role of malfunctioning DDR in the dynamics of neural-glial networks. GDN5 Genome Dynamics in Neuroscience, Copenhagen, Denmark
   Invited?: Yes

6. Sivan Kanner, Paolo Bonifazi, Ronit Galron, Eshel Ben Jacob and Ari Barzilai (2014). The role of Atm in the dynamics of neural-glial networks. Miami Winter meeting: Neurological diseases, Miami, United States
   Invited?: No

7. Sivan Kanner, Paolo Bonifaci, Eshel Ben Jacob and Ari Barzilai (2013). The role of malfunctioning DDR on the activity of neural networks. The 15th AT workshop, Birmingham UK, Birmingham, United Kingdom
Invited?: Yes

8. Noah Levine-Small, Paolo Bonifaci, Ziv Yekutieli, Eshel Ben Jacob. (2012). The role of malfunctioning DDR in neural network dynamics. The 14th AT workshop, Delhi, India
Invited?: Yes

9. Noah Levine-Small, Paolo Bonifaci, Ziv Yekutieli, Eshel Ben Jacob. (2012). Persistent synchronization in Atm proficient neural networks. Friedrich Schiller University, Jena, Germany
Invited?: Yes

10. Noah Levine-Small, Paolo Bonifaci, Ziv Yekutieli, Eshel Ben Jacob. (2012). Reduced synchronization persistence in Atm-deficient neural networks. University of Freiburg, Freiburg, Germany
Invited?: Yes

11. Sivan Kanner, Ronit Galron, Paolo Bonifazi, Eshel Ben Jacob. (2012). The effects of Atm deficiency on the dynamics of neural-glial networks. Fritz Lipmann Institute for Aging, Jena, Germany
Invited?: Yes

12. Noah Levine-Small, Paolo Bonifaci, Ziv Yekutieli, Eshel Ben Jacob. (2012). The effects of DNA damage on the dynamics of neural networks. BIT's 4th Annual World Congress of Molecular & Cell Biology, Beijing, China
Invited?: Yes