BIOGRAPHICAL SKETCH

Provide the following information for the key personnel and other significant contributors in the order listed on Form Page 2.
Follow this format for each person. **DO NOT EXCEED FOUR PAGES.**

NAME	POSITION TITLE
Kurt Giles	Assistant Adjunct Professor
eRA COMMONS USER NAME KGILES	

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
University of Oxford, United Kingdom	BA	1991	Chemistry
University of Oxford, United Kingdom	MA	1994	
University of Oxford, United Kingdom	D.Phil.	1995	Pharmacology
Weizmann Institute of Science, Israel	Post-doc	1995-1998	Structural Biology

A. Positions and Honors

Employment

1998-1999 Training Manager, Israeli National Node, Weizmann Institute of Science, Israel
1999-2002 Departmental Lecturer, Departments of Zoology and Experimental Psychology, University of Oxford, United Kingdom
2002- Assistant Adjunct Professor, Institute for Neurodegenerative Diseases, University of California, San Francisco, CA

Other Experience

1995-1999 Consultant, Protein Data Bank, Brookhaven National Laboratory, NY
2000-2002 Consultant, Synaptica Ltd., United Kingdom
2000- Editorial Board member, Biochemistry and Molecular Biology Education
2004- Consultant, InPro Biotechnology, South San Francisco, CA

B. Selected peer-reviewed publications (in chronological order)

1. Webb, C.P., Nedergaard, S., Giles, K. & Greenfield, S.A. (1996). Involvement of the NMDA receptor in a non-cholinergic action of acetylcholinesterase in guinea-pig substantia nigra pars compacta neurons. *European Journal of Neuroscience*, 8, 837-841
2. Cousin, X., Hotelier, T., Giles, K., Lievin, P., Toutant, J.P. & Chatonnet, A. (1997). The α/β fold family of proteins database and the cholinesterase gene server ESTHER. *Nucleic Acids Research*, 25, 143-146
3. Giles, K. (1997). Interactions underlying subunit association in cholinesterases. *Protein Engineering*, 10, 677-685
4. Cousin, X., Hotelier, T., Giles, K., Toutant, J.P. & Chatonnet, A. (1998). aCHEdb: the database system for ESTHER, the α/β fold family of proteins and the Cholinesterase gene server. *Nucleic Acids Research*, 26, 226-228
5. Navaratnam D.S., Fernando F.S., Priddle J.D., Giles K., Clegg S.M., Pappin D.J., Craig I. & Smith, A.D. (2000) Hydrophobic protein that co-purifies with human brain acetylcholinesterase: amino acid sequence, genomic organization and chromosomal localization *Journal of Neurochemistry* 74, 2146-2153
6. Kryger G., Harel M., Giles K., Toker L., Velan B., Lazar A., Kronman C., Barak D., Ariel N., Shafferman A., Silman I. & Sussman J.L. (2000) Structures of recombinant native and E202Q mutant human acetylcholinesterase complexed with the snake-venom toxin fasciculin-II *Acta Crystallographica D* 56, 1385-1394

7. Safar, J., Geschwind, M.D., Deering, C., Didorenko, S., Sattavat, M., Sanchez, H., Serban, A., Vey, M., Baron, H., Giles, K., Miller, B.L., DeArmond, S.J. & Prusiner, S.B. (2005). Diagnosis of human prion diseases. *Proceedings of the National Academy of Sciences USA* 102, 3501-3506.

8. Peretz, D.[†], Supattapone, S.[†], Giles, K.[†], Vergara, J., Freyman, Y., Lessard, P., Safar, J.G., Glidden, D., McCulloch, C., Nguyen, H.-O., Scott, M., DeArmond, S. & Prusiner, S.B. (2006) Inactivation of prions by acidic sodium dodecyl sulfate. *Journal of Virology* 80, 322-331.
[†]These authors contributed equally to the work

9. Tamgüney, G., Giles, K., Nelken, P., Yang, J., Bosque, P.J., Miller, M.W., Safar, J., DeArmond, S.J. & Prusiner, S.B. (2006) Transmission of elk and deer prions to transgenic mice. *Journal of Virology* 80, 9104-9114.

10. Philipp, W.J., Groth, D., Giles, K., Vodrazka, P., Schimmel, H., Feyssaguet, M., Miyagawa, E., Toomik, R., Schacher, P., Osman, A., Lachmann, I., Wear, A., Arsac, J.-N. & Prusiner S.B. (2007) Transgenic mouse brains for evaluation and quality control of BSE tests. *Chemical Biology*, *in press*.

11. Phuan, P.-W., Zorn, J.A., Safar, J., Giles, K., Prusiner, S.B., Cohen, F. & May, B.C.H. (2007) Discriminating cellular and misfolded prion protein using affinity to 9-aminoacridine compounds. *Journal of General Virology, in press*

12. Karpuj, M.V., Giles, K., Gelibter, S., Scott, M.R., Lingappa, V.R., Szoka, F.C., Peretz, D., Denetclaw, W. & Prusiner, S.B. (2007) Phosphorothioate oligonucleotides reduce PrP^{Sc} levels and prion infectivity in cultured cells. *Molecular Medicine, in press.*

C. Research Support

1 R01 AI064709 Prusiner (PI) 04/01/06 – 03/31/11 NIH-NIADS
Preventing Human Prion Disease – Inactivation of Prions
The major aim of this project is to develop methods to inactivate prions, particularly those bound to the surface of surgical instruments, in order to reduce the risk of iatrogenic transmission of Creutzfeldt-Jakob disease.
Role: Co-Principal Investigator

5 P01 AG002132 Prusiner (PI) 01/01/04 – 12/31/08 NIH/NIA
Degenerative and Dementing Diseases of Aging
Project 4 of this program project focuses on determining the amino acid residues important in controlling transmission between species and between different human prion strains. The science core (Core B) provides screening and quality control of the large number of novel transgenic lines developed in Project 4, and also screening of mice for Projects 1 and 3, in addition to other reagents.
Role: Investigator, Project 4; Investigator, Core B

5 P01 AG010770 Prusiner (PI) 07/15/05 – 06/30/10 NIH/NIA
Molecular Pathogenesis of Age-Dependent CNS Degeneration
This program uses synthetic prions produced from recombinant protein to understand the pathogenesis of prion diseases. Core B (science) supplies the recombinant protein and screening of transgenic mice for the constituent projects. Mice are required for infectivity (Project 1), structural (Project 3) and neuropathological (Project 4) studies.
Role: Co-Principal Investigator, Core B

5 P01 AG21601 Prusiner (PI) 06/01/03 – 05/31/08 NIH-NIA
Novel Therapeutics for Prion Diseases
A multi-disciplinary approach is required to develop new therapeutic for the treatment of prion diseases. The science core (core C) provides reagents and services for all projects, including DNA screening, cell based assays for lead compounds, recombinant proteins and antibodies, protein interaction analysis and screening for transgenic mice.
Role: Co-Principal Investigator, Core C