



## Annelise E. Barron

Associate Professor of Bioengineering

 Curriculum Vitae available Online

### Bio

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#### BIO

My research interests are in three areas:

(1) Molecular and cellular biophysics of human innate immunity, with a focus on the pleiotropic roles of host defense peptides and amyloidogenic peptides in human health and disease, including infectious disease, cancer, and plaque diseases, with a particular focus at present on Alzheimer's Disease.

(2) Design, synthesis, and biophysical studies of sequence-controlled, biomimetic oligomers (synthetic peptide mimics) with helical structures, for biomedical and biomaterial applications (mimicry of lung surfactant proteins, antimicrobial and anticancer innate immune peptides).

(3) Design, synthesis, and testing/demonstration of novel strategies and water-soluble polymeric materials for capillary and microchip electrophoresis (DNA sequencing and genotyping); creation of polymer-biomolecule conjugates.

#### ACADEMIC APPOINTMENTS

- Associate Professor, Bioengineering
- Member, Bio-X
- Member, Stanford Cancer Institute
- Member, Wu Tsai Neurosciences Institute

#### HONORS AND AWARDS

- National Hispanic Scholar, Univ. of Washington, Seattle (1986)
- Tektronix Foundation Merit Scholarship, Univ. of Washington, Seattle (1986)
- University of Washington Undergraduate Merit Scholarship (two consecutive years), Univ. of Washington, Seattle (1988, 1989)
- H.K. Benson Chemical Engineering Tuition Scholarship, Univ. of Washington, Seattle (1989)
- 1986-1990 National Merit Scholar and Recipient of Associated Four-Year Scholarship, National Merit Scholarship Corporation (1986-1990)

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#### PROFESSIONAL EDUCATION

- Postdoc, UCSF/Chiron Corporation , Biomimetic & Bioorganic Chemistry (1997)
- Postdoc, Soane BioSciences/ACLARA Biosciences Inc. , Molecular Biotechnology (1996)
- Ph.D., Univ. of California, Berkeley , Chemical Engineering (1995)

- B.S., Univ. of Washington, Seattle , Chemical Engineering (1990)

## LINKS

- Barron lab web page: <http://www.stanford.edu/group/barronlab/>

## Research & Scholarship

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### CURRENT RESEARCH AND SCHOLARLY INTERESTS

According to our recent findings, innate immune responses in humans and other mammals involving infection-, injury-, or stress-related dynamic imbalances between particular, potentially cytotoxic host defense peptides we study, and pro-amyloid / fibrillogenic peptides including ABeta and IAPP, may play a role in the poorly understood etiology of chronic / progressive plaque diseases, including psoriasis, lupus erythematosus, diabetes type II mellitus, atherosclerosis, and particularly, Alzheimers Disease. All of these diseases involve senescent/dystrophic cells, inflammation, and proteopathies with plaque accumulation; and can be complicated by infection.

The latter disease, Alzheimers, is in need of a major breakthrough in fundamental understanding, more than almost any human disease currently under study. Of a total of 415+ clinical trials initiated by Pharma towards the development of Alzheimer's treatments over the past 14 years, all of these trials have failed. There is no current effective treatment. Obviously, the most fundamental ideas for what drives Alzheimers must be flawed or incomplete.

Until recently Alzheimers disease was believed to be the sixth leading cause of death in the United States, according to the Centers for Disease Control and Prevention (CDC). But in March 2014, new research published in Neurology suggested that Alzheimers may actually be responsible for as many deaths each year as heart disease or cancer – the two leading causes of death in the U.S. – due to issues, in hospitals, of improper prior determinations of underlying cause of death in the elderly.

My lab is testing novel mechanistic hypotheses of Alzheimers etiology, based on recent, unique molecular biophysical observations of pro-amyloid and innate immune peptides. We are also looking at linkages to certain chronic infections.

Increasing numbers of epidemiological and co-morbidity studies indicate that multiple, progressive degenerative diseases, all involving plaque deposition in various body compartments, are linked. For instance, some researchers have begun to refer to Alzheimers Disease as "Diabetes Mellitus Type III". We seek, with current projects, to sleuth out the shared molecular biophysical bases for these emerging linkages.

(Note: Succinct, exemplary summaries of these fascinating epidemiological / comorbidity linkages are found, for instance, in the following papers: "The 'psoriatic march': a concept of how severe psoriasis may drive cardiovascular comorbidity", *Experimental Dermatology* (2011) 20, 303–307; "Circle of Willis atherosclerosis: association with Alzheimer's disease, neuritic plaques and neurofibrillary tangles", *Acta Neuropathol* (2007) 113:13–21; "Increased prevalence of the metabolic syndrome in patients with moderate to severe psoriasis", *Arch Dermatol Res* (2006) 298: 321–328; "Association of Alzheimer disease pathology with abnormal lipid metabolism", *Neurology* (2011) 77:1068).

## Teaching

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### COURSES

#### 2018-19

- Advances in Biotechnology: BIOE 450, CHEMENG 450 (Spr)

### GRADUATE AND FELLOWSHIP PROGRAM AFFILIATIONS

- Bioengineering (Phd Program)

- Biophysics (Phd Program)
- Medicine (Masters Program)

## Publications

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### PUBLICATIONS

- **Helical side chain chemistry of a peptoid-based SP-C analogue: Balancing structural rigidity and biomimicry.** *Biopolymers*  
Brown, N. J., Lin, J. S., Barron, A. E.  
2019; e23277
- **Role of Microbes in the Development of Alzheimer's Disease: State of the Art - An International Symposium Presented at the 2017 IAGG Congress in San Francisco** *FRONTIERS IN GENETICS*  
Fulop, T., Itzhaki, R. F., Balin, B. J., Miklossy, J., Barron, A. E.  
2018; 9
- **Effective in vivo treatment of acute lung injury with helical, amphipathic peptoid mimics of pulmonary surfactant proteins** *SCIENTIFIC REPORTS*  
Czyzewski, A. M., McCaig, L. M., Dohm, M. T., Broering, L. A., Yao, L., Brown, N. J., Didwania, M. K., Lin, J. S., Lewis, J. F., Veldhuizen, R., Barron, A. E.  
2018; 8: 6795
- **Intracellular biomass flocculation as a key mechanism of rapid bacterial killing by cationic, amphipathic antimicrobial peptides and peptoids.** *Scientific reports*  
Chongsirawatana, N. P., Lin, J. S., Kapoor, R., Wetzler, M., Rea, J. A., Didwania, M. K., Contag, C. H., Barron, A. E.  
2017; 7 (1): 16718
- **Evidence that the Human Innate Immune Peptide LL-37 may be a Binding Partner of Amyloid- $\beta$  and Inhibitor of Fibril Assembly** *Journal of Alzheimer's Disease*  
De Lorenzi, E., Chiari, M., Colombo, R., Cretich, M., Sola, L., Vanna, R., Gagni, P., Bisceglia, F., Morasso, C., Lin, J. S., Lee, M., McGeer, P. L., Barron, et al  
2017; 59 (4): 1213-1226

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