2017 Spring
Life Sciences & IBB Seminar

"Reimagining Alzheimer's disease: glimpses of three new disease models"

[Prof. Karl Herrup]

Hong Kong University of Science and Technology

Medical research has put a great deal of effort into trying to understand how deposits of amyloid are formed in the brain and how they trigger the complex cellular and behavioral symptoms we recognize as Alzheimer's disease (AD). In the one hundred years since Alzheimer's discovery, however, enough data have accumulated that the case can be made amyloid deposits are not the prime causative of AD; it is even plausible to propose that despite initial optimism amyloid deposits have nothing to do with the initiation or progress of AD. The problem faced by the field at the moment is that anti-amyloid strategies have so dominated our thinking that there are few alternative disease models available to guide our clinical research and future drug trials. Taking a fresh look at all of the data leads quite naturally to a model built on the premise that age is the primary 'causative' agent. And the meaning of "age" at a cellular level is finally receiving the attention is deserves. The aging process involves many changes, but among the most important is the slow accumulation of DNA damage and the cellular responses to this damage. These responses include a deregulated cell cycle program and the associated imposition of a senescent phenotype. The secondary damage caused by the senescent phenotype may actually be the more significant than previously appreciated. Additional data suggest that nutritional factors such as those associated with adult onset diabetes interact with the DNA damage response resulting in an increasingly irreversible disease process. The new models are not complete, but they already suggest a number of new strategies by which to approach the prevention and treatment of Alzheimer's disease.

- **Date: 4:30PM/Apr. 26(Wed.)/2017**
- **Place: Auditorium(1F), Postech Biotech Center**
- **Inquiry: Prof. Kee Hoon Sohn (279-2357)**

*** This seminar will be given in English.**
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