Stanford

Peter Sarnow

Professor of Microbiology and Immunology

Microbiology & Immunology

Curriculum Vitae available Online

Bio

ACADEMIC APPOINTMENTS

- Professor, Microbiology & Immunology
- Member, Bio-X
- Member, Maternal & Child Health Research Institute (MCHRI)
- Member, Stanford Cancer Institute

ADMINISTRATIVE APPOINTMENTS

- Director of Graduate Program, Dept. Microbiology and Immunology, Stanford University School of Medicine, (2002- present)

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HONORS AND AWARDS

- Predoctoral Fellowship, Studienstiftung des Deutschen Volkes (1979-1982)

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PROFESSIONAL EDUCATION

- Ph.D., SUNY at Stony Brook , Molecular Virology (1982)

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LINKS

- http://cmgm.stanford.edu/micro/sarnow_lab/index.html: http://cmgm.stanford.edu/micro/sarnow_lab/index.html
- Personal Web site: http://cmgm.stanford.edu/micro/sarnow_lab/index.html

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Research & Scholarship

CURRENT RESEARCH AND SCHOLARLY INTERESTS

Our laboratory has been been studying the mechanism by which a liver-specific microRNA, miR-122, regulates the amplification of the hepatitis C virus (HCV) genome in cultured cells. Specifically, we have found that miR-122 interacts with the 5' end of the viral RNA and is essential for viral replication. Consequently, sequestration of miR-122 by antisense-oligonucleotides results in rapid loss of viral RNA. We are currently examining the mechanism by which miR-122 helps HCV RNA replication and are searching for cellular targets of miR-122 and their regulation by miR-122. These lines of investigations will lead to new insights how these small noncoding RNAs regulate expression of cellular and viral mRNAs and may point to new venues for antiviral therapeutics against HCV.

In a second line of investigation, we are studying the unusual mechanism of translation initiation by internal ribosome entry in certain viral (i.e. HCV, picornavirsues and some insect viruses) and cellular mRNA molecules. In the conventional scanning mechanism of translation initiation, which operates on most mRNA molecules, 40S subunits are recruited at or near the 5' end of the mRNA. Subsequently, the 40S ribosomal subunits are predicted to scan the mRNA in a 5' to 3' direction until the first AUG codon is encountered as start site for protein synthesis. However, certain viral and cellular mRNAs, notably encoding proto-oncogenes and regulatory genes, contain long 5' noncoding regions with multiple AUG codons. Thus, the translation initiation rate in these mRNAs is predicted to be low according to the scanning model; alternatively, other translation initiation mechanisms may operate to ensure efficient translation. Indeed, some of such mRNAs with long leaders contain internal ribosome entry sites which can bind ribosomes directly. Much of our work has been focussing on the mechanism and prevalence of internal ribosome binding. Specifically, we are addressing the following questions: Which cellular and viral mRNAs can be translated by internal ribosome binding? What are the cellular gene products that mediate internal ribosome binding? Is internal initiation regulated in the cell? What is the molecular basis for designating a given AUG codon as start site codon?

Teaching

COURSES

2018-19

- Principles of Biological Technologies: MI 215 (Spr)

2017-18

- Principles of Biological Technologies: MI 215 (Spr)

2016-17

- Principles of Biological Technologies: IMMUNOL 215, MI 215 (Spr)

2015-16

- Principles of Biological Technologies: IMMUNOL 215, MI 215 (Spr)

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STANFORD ADVISEES

Doctoral Dissertation Reader (AC)

Alex Johnson, Makeda Robinson

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GRADUATE AND FELLOWSHIP PROGRAM AFFILIATIONS

- Microbiology and Immunology (Phd Program)

Publications

PUBLICATIONS

- **Precursor microRNA-122 inhibits synthesis of Insig1 isoform mRNA by modulating polyadenylation site usage** *RNA*
  Norman, K. L., Chen, T., Zeiner, G., Sarnow, P.
  2017; 23 (12): 1886–93

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