

Outstanding Muslim Researcher

Dr. Hina Chaudhry

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Dr.

Chaudhry is a cardiologist, and in addition to clinical patient care and teaching, she runs an NIH-funded research program investigating mechanisms of cardiac regeneration. Her findings regarding key genetic regulators of cell division in the heart and the use of stem cells will help pave the way for growing new heart muscle cells in patients after a heart attack. She has several patents pending for methods to prevent degeneration of heart tissue after heart attack or during heart failure. Some of her ongoing projects are designed to test clinical delivery systems for the gene products she has characterized as part of a long term commercialization strategy. She has published in numerous medical and scientific journals and has been interviewed for NBC news and Business Week regarding cardiac repair. Prior to joining the faculty at Columbia, she was pursuing her fellowship training at the Hospital of the University of Pennsylvania, where she received the American Heart Association's highest award for cardiology fellows, the Clinician-Scientist Award, one of ten awarded

nationally. She was also the top-ranked fellow in the nation for the NIH's National Research Service Award. Since arriving to Columbia, she has been named an Irving Scholar, Columbia University's highest award for its young medical faculty. Her most recent work was selected as one of the "Highlights" of the European Society of Cardiology Congress in September 2005.

Dr. Chaudhry holds a B.S. in Chemistry and a B.S. in Biology with a thesis in Physics from the Massachusetts Institute of Technology, and obtained her M.D. with Honors from Harvard Medical School. She is board-certified by the American Board of Medicine in both Internal Medicine and Cardiovascular Disease.

In addition to her professional endeavors within medicine, Dr. Chaudhry serves on a 15-member advisory panel to the U.S. Department of State on US-Pakistan relations through the auspices of the Aspen Institute. She also founded the North American Medical Alumni (NAMA) chapter of Association of Pakistani Physicians of North America (APPNA), and served as its first president for two years. She is also on the board of advisors for the Organization of Pakistani Entrepreneurs (OPEN).

The editors asked Dr. Chaudhry to describe her research:

My research focus is cardiac regeneration. When I was completing my clinical cardiology training, I realized the most important questions facing clinical management of cardiovascular disease needed to be answered using molecular and cellular approaches. I then obtained further research fellowship training in cell and developmental biology and genetics. I was

compelled to study the embryonic development of the heart, as I felt this process held important clues to the treatment of adult acquired heart disease. The morbidity and mortality associated with cardiovascular disease stems from the fact that human or mammalian cardiac tissue cannot regenerate after injury such as myocardial infarction (MI). In the neonatal heart, DNA replication and mitosis declines quickly and cardiomyocyte division ceases. I felt that a thorough understanding of the mechanisms of this process may potentiate therapeutic strategies for cardiomyocyte regeneration. I initially chose to focus my research efforts on the family of genes responsible for the cell division cycle in cardiomyocytes. I found that the critical regulator of the mitotic process in cardiomyocytes is cyclin A2. I had engineered a mouse model which continued to express cyclin A2 well after birth, at which time this gene normally undergoes "silencing". These cardiomyocytes in these mice continued to undergo mitosis and cell division well after birth. These results were published in August, 2004 in the *Journal of Biological Chemistry*.

I then wished to determine whether this gene could mediate cardiac regeneration after an MI. My team then induced heart attacks in these mice, and we were stunned with the results. There was an 86% improvement in measures of contractility as assessed by MRI scanning of the experimental group over the control group. We also noted cellular evidence of mitoses in small, immature-appearing myocytes in the infarct zones of our experimental group only, and not in the controls. We also noted re-entry into the cell cycle and evidence of mitoses in the healthy, undamaged myocytes outside of the infarct zones. These results are pending publication, and I was invited to present these results at the American Heart Association in 2004, and this year as a "Highlights" speaker at the European Society of Cardiology Congress in Stockholm, Sweden in September, 2005.

Since the first presentation of these results, we had also completed making an adenovirus that carries

the gene encoding cyclin A2, and administered it to rats that had undergone an MI. The control group of rats received the "null" adenovirus that did not carry the gene. We have seen dramatic improvement in the experimental group of rats that received the cyclin A2 compared to controls. This data is being presented at the upcoming meeting of the American Heart Association in November, 2005.

I have also been approaching the question of whether cardiac regeneration is possible utilizing a novel class of adult stem cells, "side-population" or SP cells. We have some pretty dramatic findings after performing cell transplantation experiments using this class of stem cells in infarcted mice. These results will be ready for presentation next year in 2006. Based on the research described above, which has been funded by the NIH, the American Heart Association, and the Irving Scholars Program at Columbia (the Irving program has specifically funded my research studies in human heart failure patients), I now have three patents pending through Columbia University for cardiac repair after an MI and during heart failure. This is critical research as the statistics are chilling: 5 million patients in the U.S. alone have heart failure, heart failure is the leading cause of hospitalization in the U.S., and close to \$400 billion is spent on treatment of cardiovascular disease in the U.S. The only definitive cure for heart failure is heart transplant, and the number of donors in the U.S. has leveled off at approximately 2200/year. That does not even begin to address the issue of heart failure in third world countries, where there is no access to heart transplantation.

Submitted by Dr. Faroque Ahmed Khan, Professor of Medicine, State University of New York, Stony Brook; Master, American College of Physicians; member, IMANA Board of Regents; member, Majlis Al-Shura, Islamic Society of North America; President, Islamic Center of Long Island; Associate Editor, JIMA.