Leaders in Cardiovascular Research: Peter Libby

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Watch the interview here: youtu.be/yOJds5B103U

Professor Tomasz Guzik, Editor-in-Chief of Cardiovascular Research, talks to Professor Peter Libby, Professor of Medicine at Harvard Medical School and President-elect of the International Atherosclerosis Society.

Q: What is your recipe for success in science—is it ambition or talent?

Hard work and persistence. Talent lies fallow without the word and persistence. You need to have an appetite for delayed gratification, some good luck, and to be in the right place at the right time. One of the things that’s worked for me is to never follow the pack.

Q: What drives you on in your pursuit of novel discoveries?

One is personal ambition. If you don’t have some ego, you won’t survive the difficulties of the challenging road of medical discovery. The other driver is, of course, for us as medical researchers is to learn something that advances scientific understanding and that’s of utility to help our patients and improve public health.

Q: If you were to define your greatest contribution to cardiovascular medicine, what would that be?

One of the most satisfying parts of my career has been furthering the understanding of the roles of inflammation in atherosclerosis. Being able to follow it from the very earliest pre-clinical stages through pilot clinical studies and, finally, on to a large-scale clinical trial showed that it was all worthwhile.

Q: What do you think is the way forward for immune-targeting in cardiovascular disease?

We’ve had a lot of disappointments despite pre-clinical data and even pilot clinical studies. For example, targeting p38 MAP kinase didn’t work out clinically and then with the lipophilic antioxidant drug succinobucol, which was effective in preventing oxidation of LDL, didn’t improve cardiovascular outcomes in a large trial. CANTOS did show that targeting a specific inflammatory cytokine could improve cardiovascular outcomes and that was important for me personally as I hypothesized a role for interleukin-1 in atherosclerosis in 1986.

Q: From your experience, can basic science compete with clinical research for the attention of patients?

We scientists are bad communicators with the public. We use an arcane vocabulary, speak in abbreviations, and do not advocate for the value of investment of society in basic research. We need to learn as a community to become better communicators about what value we add.

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Q: What are your future research plans?
We're very excited right now about the observation of clonal haematopoiesis. We have learned that these mutations not only carry a risk of eventual malignancy to leukaemia or myelodysplastic syndrome but also cause a heavy burden of cardiovascular disease.
That opens a huge field for us to understand new pathways and new targets; so, a large part of my laboratory is working on that now.

Q: Lastly, can you briefly tell us about your role in the International Atherosclerosis Society?
One of the reasons that leadership of the International Atherosclerosis Society appealed to me is because we need to widen the aperture. Atherosclerosis is more than lipids. LDL is causal in atherosclerosis, but even with excellent control of LDL, we have not cured the disease. For example, we need to broaden the perspective to include cardiometabolic disease.
The Society has a mission to provide a platform and a voice for people who are in emerging countries. There are large areas of the world where the greatest burden of cardiovascular risk exists today as we’ve conquered many communicable diseases, so people are now surviving to the age when they can develop cardiovascular disease.