Prof. Steven Patierno: perseverance, creativity and collaboration are the keys to successful research

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Expert's introduction

Prof. Steven Patierno is the Deputy Director of Duke Cancer Institute (DCI), Duke University Medical Center, where he also serves as Professor of Medicine, Professor of Pharmacology and Cancer Biology, and Professor of Community and Family Medicine under the School of Medicine.

Prof. Patierno is world-recognized for his expertise in cancer causation and molecular oncology. His research covers a broad spectrum of laboratory, population level, and health services research. His carcinogenesis research lays emphasis on molecular mechanisms of DNA damage and repair, and cellular signaling responses at the nexus between cell death and survival. In his translational research, he works extensively on the genomics of cancer disparities, cancer biology, molecular pharmacology and targeted experimental therapeutics to govern the aggressiveness of tumors.

Editor's note

From 9–10 December 2017, the 2017 Annual Meeting of China Anti-Cancer Association-Genitourinary Cancer Committee and the 7th Shanghai Genitourinary Oncology International Symposium took place on piercingly cold days with shivery wintry breeze. Yet, a thaw had set in due to the intense academic passion and warmth of a team of experts coming from all corners of the world. At the symposium, we had the honor to invite the Deputy Director of Duke Cancer Institute, Prof. Steven Patierno, to share his insights into the genomics, genetic mutations, pathogenesis and prospects of prostate cancer (*Figure 1*).

Interview

TAU: Would you introduce us to the talk you presented today at the symposium?

Prof. Patierno: My talk today was on research I conducted in my former role as Director of the George Washington University Cancer Center in Washington DC, and in my current laboratory at the Duke Cancer Institute. We studied the genomics of prostate cancer across different races by conducting comparative genomics research on Caucasians, African Americans, and now Asians. We discovered a novel change in something called Alternative RNA Splicing (ARS). By comparing splicing variation between races, we were able to identify new genes involved in driving the aggressiveness of prostate cancer or protection against the cancer. We think these new genes can be served both as biomarkers for aggressive disease or point towards new interventions for treatment or prevention of prostate cancer.

Figure 1 Prof. Steven Patierno and AME Editor Miss Cora W. Xu.

TAU: What are the differences in genetic mutations between Eastern and Western prostate cancers?

Prof. Patierno: Genetic mutations between Eastern and Western prostate cancers have not yet been looked at extensively. However, my laboratory is beginning to do so. We know a lot about the genes that are mutated in prostate cancer in general, e.g., *p53*, *TP63* and *PTEN*. However, in our laboratory, we believe the major difference between Eastern and Western prostate cancers lies in RNA splicing because it is regulated in the germline and is affected by

ancestral genetics, which is essentially the biology that makes us different as our ancestors spread around the planet. We have begun to find major changes between races in splicing in prostate cancer. At the DNA mutation level, we know there are different frequencies of specific gene mutations between different races. Yet, we do not know whether those have biological significance. At the RNA splicing level, we know that many of the differences are actually affecting the biology of the cancer.

TAU: What is the molecular mechanism of tumor pathogenesis of prostate cancer?

Prof. Patierno: The molecular pathogenesis of prostate cancer is very complicated. It is a complex tumor that evolves over a long period of time. We know that different genes affect the risk for getting early stage disease and most of those are very different from the genes that actually drive the aggressive and lethal form of late stage disease. We know that there are combinations of gene mutations and epigenetic changes that are occurring during the evolution of that cancer. However, what my laboratory has discovered is that a large part of that biology is actually being driven by ARS. We have identified what we call the "tumor splicing burden", and have found more than 2000 splicing changes between aggressive prostate cancer and low-grade prostate cancer, between prostate cancers in different races, and comparing prostate cancer to normal adjacent tissue. We think the splicing differences are contributing tremendously to the biology of the disease.

TAU: How do you see the future of prostate cancer genomics?

Prof. Patierno: The future of prostate cancer genomics is going to revolve around finding new ways to handle large datasets, and to combine datasets between DNA mutation, epigenetic changes, RNA splicing, metabolomics, and possibly microbiome work. One of the most exciting developments right now is called immunogenomics, which looks at the immunobiology of both the tumor and the host, i.e., the patient, and explores whether patient's cancer is treatable with immunotherapy or is resistant to immunotherapy.

TAU: Who have been posing greatest impact on you throughout your career?

Prof. Patierno: There is one person that I can trace my scientific career back to. It was one of my college professors—the dean of the University of Connecticut School of Pharmacy where I attended as an undergraduate. I was a good student academically, but at that time, I did not fit in well because I did not want to go into the field of Pharmacy. It was the dean who walked me into a different part of the building where the laboratories were and introduced me to research. In those laboratories, I found many people like me who were not really interested in the practice of Pharmacy, but instead in the research aspect of pharmacology. And that is where I officially launched my research career.

TAU: How do you overcome the challenges you met during research?

Prof. Patierno: How to move forward in research is an important question. Many graduate students, fellows, or medical students who newly engage in research very often overlook how difficult research is and mainly look at the positive sides of it. As a matter of fact, there are plenty of questions that you try to answer where the answer is negative, which requires lots of perseverance, determination, a strong will to keep going, and the ability to learn from the experiments that give you negative answers. Instead of just disregarding the negative experience, you must learn from it. And with hard work and creativity you will gradually make discoveries and get positive results. To me, perseverance, creativity and collaboration are three key elements to successful research.

TAU editor: What motivates you to keep moving forward in your research career?

Prof. Patierno: What provides encouragement is the hope of making discoveries that will impact patients, that will change the way we treat patients, and that will help us determine who need, or do not need, to be treated with precise therapies that minimize toxicity and maximize positive outcomes. That is what keeps us going and what we get up every day in the morning trying to accomplish. We want to take care of our patients. We want to minimize the overall burden of cancer in our population. We want to help people live healthier and with productive lives. And if they do get diagnosed with a disease, we want it to be cured.

TAU: Thank you.

For more details, please check out the interview video below (*Figure 2*).

Figure 2 Interview with Prof. Steven Patierno: perseverance, creativity and collaboration are the keys to successful research (1). Available online: http://www.asvide.com/article/view/23348

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Footnote

Conflicts of Interest: The author has no conflicts of interest to declare.

References

1. Li B, Xu CW, Zhou SL. Interview with Prof. Steven Patierno: perseverance, creativity and collaboration are the keys to successful research. Asvide 2018;5:120. Available online: Available online: http://www.asvide.com/article/view/23348

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