Caveat emptor: if you have PKD, be careful of what you drink?

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IN A RECENT ISSUE OF THE AMERICAN JOURNAL OF PHYSIOLOGY-RENAL PHYSIOLOGY, work by Vincent Gattone (4) examines a potential confounding variable in the treatment of autosomal dominant polycystic kidney disease (PKD). Dr. Gattone was the first to describe a role for vasopressin V2 receptor antagonist as a way to decrease cyst growth in the setting of PKD. His original work was performed in the pcy murine model of PKD, a nonorthologous model of PKD (3). Subsequent work extended this original observation to an orthologous model, and, subsequently, V2 receptor antagonism showed some clinical efficacy in the TEMPO study (6, 7). Since patients with autosomal dominant PKD have defective urinary concentrating ability, these patients are prone to elevated vasopressin levels and have increased urinary cAMP (1). Based on these observations, it has been suggested that increasing water intake can suppress vasopressin (5). However, water is seldom pure, and, in some cases, contaminants at a level considered safe may unduly affect at risk populations.

The recent study (4) examines whether a common potable water contaminant, dichloroacetate (DCA), can increase cyst growth in the PCK rat model of PKD. DCA is formed when chlorine covalently combines with acetate in solution. The Environmental Protection Agency has a maximum allowable concentration of 60 μg/l DCA in drinking water (2), so this is a common chemical component of drinking water, and it could possibly affect people with subtle alterations in metabolism. Clearly, further work is needed to fully identify the risks of permissible water contaminants to people with underlying genetic disorders.

This work is also the final scientific effort of Vincent H. Gattone. Dr. Gattone passed away on January 21, 2014, finally succumbing to cancer. Vince earned a Bachelor of Science degree in Chemistry from Ursinus College followed by a Masters degree in Academic Pathology from George Washington University. Subsequently, Vince earned his PhD in Medical Sciences from the Medical College of Ohio in 1980. In late 1980, he came to Indiana University. Following his postdoctoral experience, he took a faculty position at Pennsylvania State University and then joined the Department of Anatomy and Cell Biology at the University of Kansas University Medical School. It was at Kansas University Medical School where Vince began the work that, in many respects, became the focus of his scientific career. While he was at Kansas University Medical School, Vince demonstrated that a vasopressin V2 receptor antagonist could ameliorate cystic disease in a nonorthologous model of murine PKD. Subsequent work demonstrated similar efficacy in an orthologous model, and these agents are now being tested in human studies.

In 2000, Dr. Gattone joined the faculty of Cell Biology and Anatomy at Indiana University. While I had known Vince from our conversations at annual American Society of Nephrology meetings, when he came to Indiana University we began to work closely together, spurred by a common interest in PKD, medical education, and Italian food. Like many fellow scientists working on PKD, I found Vince to be unflaggingly supportive and helpful. He had a unique ability to connect people and ideas. Vince was especially dedicated to encouraging young investigators.

The report published in this issue of the AMERICAN JOURNAL OF PHYSIOLOGY-RENAL PHYSIOLOGY in many respects demonstrates many aspects of Vince’s research focus. In clinical medicine, we speak of patient-centered care; Vince conducted patient-centered research. It is this feature of his work that was noted by the committee awarding the Lillian Kaplan Award, an international award given for excellence in scientific research on PKD. The award acknowledged a lifetime of pioneering work on the pathogenesis and treatment of PKD.

Dr. Gattone is survived by his wife and five children. Every member of his family had fond memories of being swept up in Vince’s scientific quests. All were introduced early to the laboratory and data collection. No holiday was complete without a visit to the laboratory. Vince’s dedication to his scientific work was shared by his family and reflected his view that science is a communal affair.

When Vince was diagnosed with cancer, he discovered that our medical school curriculum lacked coursework addressing the needs of patients facing terminal illness. Vince contacted the curriculum committee and worked with our palliative care group to create course content. During a time in which many people would withdraw, Vince embraced the insights he could relate to others about his condition. He allowed researchers unfettered access to his life and reflections as a terminally ill colleague. His humanity, grace, and fellowship will be greatly missed.

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REFERENCES


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