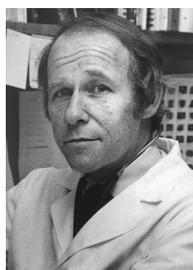




A tribute to Michael L. Steer (1939–2019)



Dr. Michael Lewis Steer (born June 26th 1939) died of complications related to colon cancer on April 18th 2019 at the age of 79. He was a prominent surgeon/scientist in the pancreas field and a pioneer in the cell biology of pancreatitis. Mike Steer was born in Brooklyn, NY, the son of Esther Steer and Arthur Steer, a Pathologist in the US Army. His father's career enabled him to travel to many locations throughout the United States and overseas, including Japan. He received his undergraduate and medical school training at Duke University, where he graduated with an MD in 1964. After an internship at King County Hospital in Seattle he served as a flight surgeon in the 3rd armored division in Hanau, Germany from 1965 to 1967. While working in the Kibbutz Sarid during a visit to Israel, he met Vera Paz, his lifelong soulmate and future wife of 47 years. On his return to the US at the peak of the "Hippie" movement, they both relocated to California, albeit for a residency at UCSF, before moving to Boston's Beth Israel Hospital where Mike completed his surgical training.

In 1972 he and Vera moved back to Israel on an NIH academic training grant and he joined the group of Alex Levitzki (whose work on protein kinase inhibitors lead to the development of Gleevec®) at the Biophysics Department of the Weizmann Institute of Science. Here, Mike not only experienced the changing fortunes of the Yom Kippur War but also his first exposure to basic science, protein chemistry and enzymology. At the time, the Levitzki lab studied the adrenergic regulation and signal transduction via adenylate cyclase [1]. Mike investigated these mechanisms initially in erythrocytes [2] and later, upon his return to Boston, in turkey platelets, sometimes in collaboration with Ed Salzman [3], who occupied the adjacent lab at the Beth Israel Hospital. Platelet signaling kept him interested into the early eighties, although it became increasingly less relevant to his surgical practice and clinical interests.

An offhand remark of his mentor Al Hall and the fact that the early seventies had witnessed the development of several non-invasive animal models of pancreatitis made him change his scientific focus. For the first time these new disease models permitted

the study of the pathophysiology and cell biology of pancreatitis before organ destruction develops and to specifically address the early events that precede cellular injury. Mike would not regret his decision to engage in this new field, quickly became a prominent Pancreatologist and accepted numerous invitations to 6 continents, which suited his love for travelling.

He started by delving into an ongoing controversy at the time. Since 1964, George Palade had established by EM-autoradiography that the exocrine pancreas secretes its digestive enzymes via discrete, hormone-stimulated, parallel exocytosis from the apical cell surface [4]. In 1967 Stephen Rothman proposed that, alternatively, a non-parallel, constitutive pathway of enzyme secretion operates in the pancreas [5]. The outcome of this debate is history: Palade received the Nobel Prize for his discovery in 1974 and Steve Rothman's theory quickly lost attention. Mike's initiation into the field of pancreas research began with his attempt to reproduce these findings in the rabbit pancreas and he confirmed parallel, bulk secretion [6]. These experiments, however, made him realize that state of the art cell biology in those days required sophisticated electron microscopy, a technique not yet available to him. He therefore entered into a collaboration with Jacopo Meldolesi in Milan, a student of George Palade. With their combined expertise (and that of their respective students) they discovered a number of cellular mechanisms with relevance to pancreatitis such as the formation of autophagy and crinophagy [7], the role of lysosomes [8,9], dysregulation of endocytosis [10], and disruption of the acinar cell cytoskeleton [11].

In addition to his busy practice as a pancreatic surgeon, Mike managed to obtain continuous NIH funding including an NIH MERIT award from 1988 to 1997. He staffed his lab initially with surgical fellows from the Beth Israel Hospital and, when his reputation grew, with fellows from Japan [12], Germany [13] and other parts of Europe [14]. Mike quickly realized that rotating residents or overseas fellows would not maintain the techniques, skills and expertise required for his ambitious projects and he therefore added basic scientists to his group as long-term collaborators. The first of these was Ashok Saluja who ran his lab for exactly 20 years and who introduced new concepts and methods and trained the junior staff and fellows. Ashok was then followed by George Perides as lab director, who continued this legacy and greatly facilitated the transfer of the lab when Mike ultimately moved his group to Tufts New England Medical Center in 2002.

The collaborations with Ashok and George also brought new techniques and ideas to the group and the lab not only expanded its models to include biliary pancreatitis [15,16] but also shifted its focus to signal transduction events such as calcium release [17], the CCK receptor cascade [18,19] and protease-activated receptor-2 [20], all of which are involved in premature digestive protease

activation and acinar cell injury [21]. While making these discoveries and becoming an expert in the search for early cellular events in pancreatitis, Mike rose through the ranks at Beth Israel Hospital and Harvard University and achieved the rank of full Professor in 1984 and later followed Dr William Silen as interim chief of surgery. Mike not only regarded Bill Silen as his most important mentor [22], but Bill also became the surgeon who removed part of Mike's pancreas, a shared experience both of them would rather have done without! It made Mike even more determined to ultimately identify treatment targets for pancreatitis.

Mike didn't much believe in serendipity in science or in Popper's conjecture and refutation concept. His philosophy was rather to 'drill down upon a selected phenomenon, peel back its layers, and address its underlying mechanistic questions' to use his own words [22]. Those of us who trained with him were made to drill mightily, were challenged by his inquisitive mind at weekly lab meetings and were not easily forgiven for being messy, sluggish or – worst – imprecise. With his sharp intellect, Mike always spotted the inconsistencies in your argument and he didn't suffer fools lightly. It helped to bring back Cuban cigars from overseas visits, which raised his spirits when he revised your latest manuscript on a porch in Newton, at Lake Winnepesaukee, or later, on Cape Cod. Not surprisingly many of his trainees and fellows decided to stay in academic medicine or surgery and several followed his role model and dedicated their research careers to the pancreas, an organ system and disease universe with a lot of drilling-down still to do. After his (semi)retirement at Tufts, he became Director of Surgery with Partners in Health and the Program for Global Surgery and Social Change. Here he generously dedicated his time and surgical skills to the truly needy in Haiti after the 2010 earthquake [23]. His eyes always lit up with enthusiasm when he reported back from Cange, 36 miles out of Port-au-Prince.

Having lost his wife Vera in 2014, Michael Steer leaves behind his children Dylan, Karen and David Steer, his four grandchildren, Sienna, Talia, Olive and Ella and his younger brother Paul. To them he was the loving father, grandfather and brother. The pancreas community will remember Mike Steer not only as the past president of the American Pancreatic Association (1996) and the APA lifetime achievement award recipient in 2013 but as a pioneer in the cell biology and pathophysiology of pancreatitis, a rigorous mentor, a role model scientist, and the most authentic of friends.

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