Alan M. Jones

How did you spend your career?

The year I was born, the Soviets launched the first satellite, Sputnik, awakening Americans to the importance of science for their dominant rank in the world. Growing up at this time inspired me to become a scientist, and I knew at the age of eight that that was what I wanted to be. I had an essential requisite, namely a great curiosity. Growing up in Florida, I could have been destined to become a marine biologist, but the allure of plants and their tractability for observation and discovery drew me to botany. This attraction was nurtured by a few academic family friends. Until my last semester as an undergraduate at the University of Florida, when I took a course on plant physiology, I was on course to become a card-carrying classical botanist. The last plant physiology lecture, which was on phytochrome, was particularly exciting; I'll come back to that later. The quantitative nature of physiology, in contrast with anatomy and systematics, appealed to me. Physiology, with its associated statistics, provided a level of confidence to experimental data. Molecular biology would do the same, and it was just around the corner in the mid-1970s.

So, what can you do with a BS in botany? Well, for one thing, you can go to graduate school, and that's what I decided to do. I went to the University of Illinois at Urbana–Champaign to do a PhD in plant biology because UIUC had some of the top scientists in several aspects of plant physiology, and I had not yet settled on one particular topic. The only thing I was sure of was that I did not want to work on auxin because it was too controversial and campy at the time. In those days, graduate schools did not recruit or offer rotations, as there were too many Sputnik-motivated baby boomers, like me; they simply assigned you a lab. I was assigned an auxin lab! It was run by the head of the department, Larry Vanderhoef, who told me he chose me for his lab because he believed in the underdog.

Soon thereafter, just as Tony Trewavas arrived at UIUC to do a sabbatical year in the Vanderhoef lab, Larry moved to the University of Maryland as provost. We were all surprised. Tony was my mentor that year, and 2D electrophoresis was the state of the art. When Tony returned to Edinburgh University, I joined the lab of Fred Meins. Fred also left UIUC, but I followed him to the Friedrich Miescher Institute in Basel, Switzerland. The FMI, and Fred's lab in particular, was intellectually stimulating. This time was the genesis of plant transformation, with power labs including Meins, Potrykus, King, and the Hohns, giving one a sense of scientific purpose, although I was still stuck on auxin research at the time. I pressed on until it was time to return to UIUC and wrap things up. I spent the last year and a half with David Ho, who, by the way, also soon left UIUC, making me wonder about the effect was I having on my PhD advisers!

I learned a great deal from each of these mentors, and I am indebted to them for the diversity of my training. They didn't work on auxin, and one might wonder why I stuck with auxin the whole time. It's because my PhD project was based on a preexisting collaboration with one of the top 10 organic chemists in the world, Nelson Leonard. I did not want to let him down; indeed, no one did. I spent my first year acting like I was a chemist, synthesizing azido auxins to develop photoaffinity labels to identify auxin receptors. After four years and five published papers, I described a 22-kDa maize protein that labeled with [^{3}H] 5-azidoindole-3-acetic acid; I later showed it to be Auxin-Binding Protein 1 (ABP1).

My choice for postdoctoral training goes back to that exciting undergraduate lecture on phytochrome. I persuaded Peter Quail at the University of Wisconsin to take me into his lab, which contained a robust group of researchers divided into phytochrome structure and

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phytochrome gene-cloning teams. This began my structural biology period, which I have continued. Over two and a half years, I purified many milligrams of phytochrome, each prep of which required over 24 nonstop hours working in green light. This research resulted in six publications describing the subdomain function and native oligomeric state of phytochrome (a homodimer).

Peter Quail left UW (again I wondered if I was too difficult to work with), and I had a powerful urge to return to the Southeast. So I did not follow Peter to Berkeley, perhaps to his relief. Luckily, the University of North Carolina at Chapel Hill was seeking young, "rising-star" plant biologists, as they told me, "to get on the map for modern plant biology." At least I had the "young" part right; I was barely 29. I continued working on phytochrome structure with my first student, Mike Edgerton (Monsanto, now retired), but NSF was more interested in my ideas on ABP1, so once again I found myself working on auxin. By the mid-1990s, programmed cell death (PCD) was becoming a hot topic in cell biology, and I was the first to jump into PCD in plants with my student Andrew Groover (now a professor at UC Davis). Unfortunately, the great advantages that *C. elegans* brought to PCD research were not afforded by plants, and I realized that without the genetics of PCD, my work would continue to be descriptive at best. I needed a change.

My research took a sharp turn when a student, Hemayet Ullah (now a professor at Howard University), wanted to work on the heterotrimeric G protein pathway. This coincided with the application of reverse genetics to plant research, and we were one of the first labs to describe T-DNA insertion mutants in Arabidopsis. Our Arabidopsis mutants of G protein core components captured the attention of NIH, and I am grateful to have subsequently had continuous NIH funding to work on G protein signaling in diverse organisms. Since 1999, my lab has been entirely focused on G protein activation mechanisms.

What do you consider to be your most important contributions to plant science?

When it comes to G protein signaling, it is pretty clear—my lab established that plants are not just green animals. In 1999, many of my pharmacology colleagues wondered what I would teach them about G protein signaling using a weed called Arabidopsis. That was a fair criticism, because by then there were already eight Nobel Laureates (and two more by 2012) recognized for their work on G protein signaling; indeed, this pathway is one of the most studied and best understood pathways in animals. But what Arabidopsis taught us is remarkable. We showed several things that plants and protists do differently: (1) they do not use G protein coupled receptors to activate signaling, (2) they use receptor-like repressors of signaling to modulate signaling in dynamic environments, (3) they use receptor-like kinases to modulate G protein signaling, and (4) they use both duration and dosage of signaling (i.e., dose–duration reciprocity vs. a threshold-based model). Understanding the full plasticity of G protein activation through evolution-based approaches opened up a whole world of ideas about how to regulate cell and organism behavior. Among important contributors from my lab were Jay Chen, Yan Fu, Jan Jones, Meral Tunc-Ozdemir, and Daisuke Urano, but many others played important roles.

I am also proud of our work on PCD in plants. We introduced the idea of a "functional corpse," or at least we introduced the idea for vascular cells. Of course, animals, too, have cell corpses that are functional, including squamous cells and corneal cells, but the idea that cell death creates different types of functional corpses through different combinations of hydrolases in the lytic vacuole originated from our work.

My lab also contributed a large body of work on ABP1 that was useful. We brought rigor to the field of plant hormone "receptor-ology," but in the end we failed to show that ABP1 is essential for auxin action. Auxin research taught me that scientists must be humble, because any single body of work is only part of a long journey toward understanding an aspect of life; this journey takes more than the lifetime of one scientist. Someday, someone will figure out what ABP1 does and why it is highly conserved in plants.

When did you become a member of ASPP/ASPB?

The Society was called ASPP when I joined in 1978. Without a doubt, it was Larry Vanderhoef who suggested (one might say, insisted) that I choose a professional society early,

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pay my dues each year, contribute to its health and mission, and stick with it my whole career.

How did the Society impact your career, and what motivated you to become a Founding Member of the Legacy Society?

The annual meeting has been and still is a great place for me to network and to share my discoveries. I gave talks at every annual meeting I attended (about 30, I think), because it is important to be heard and understood. Maarten Chrispeels, the sixth editor-in-chief of *Plant Physiology* and a devoted attendee of the annual meetings, must have heard and understood me because he invited me to serve on his editorial board early in my career. This began my opportunities to serve the Society, and I followed with service on the Constitution and Bylaws Committee, Program Committee, Executive Committee as an elected representative, Science Policy Committee, Board of Trustees, and Plantae Steering Committee and as president. I am honored to be among the Founding Members of the Legacy Society, and considering how much I have received from the Society, I feel I still have more to do.

What important advice would you give to individuals at the start of their career in plant science?

Focus, focus, focus, and find ways to be heard and understood. You do not have to figure this out alone. ASPB has so much to offer in career development, and ASPB is ready to serve you.

Academic Family Tree
https://academictree.org/plantbio/tree.php?pid=800883