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The Power of Symbiosis in Life and Science

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Keywords

Rhizobium–legume symbiosis, nitrogen-fixing nodule development, *nod* genes, symbiotic signaling, plant peptides, NCRs, nodGRPs, cell cycle, cell differentiation, terminal bacteroid differentiation, autobiography

Abstract

This article traces more than four decades of Eva Kondorosi's personal life and scientific journey in symbiotic nitrogen fixation, from early insights into nitrogenase structure to the molecular mechanisms governing root nodule development and symbiotic cell differentiation in the *Medicago*–*Sinorhizobium meliloti* symbiosis. Effective symbiosis depends on precise molecular communication between the partners, beginning in the soil and continuing through a highly coordinated, progressive differentiation program. A defining feature of symbiotic cell development is endoreduplication in both host plant cells and their *Rhizobium* partners. Host-induced bacterial endoreduplication results in the formation of large, polyploid, noncultivable nitrogen-fixing bacteroids. This terminal differentiation is orchestrated by plant-derived effector peptides, notably nodule-specific cysteine-rich (NCR) and nodulin glycine-rich (nodGRP) peptides, which act sequentially to reprogram bacterial physiology. Together, these findings establish symbiotic nitrogen fixation as a model for cross-kingdom cellular differentiation and highlight NCR and nodGRP peptides as a vast, largely unexplored resource with promising applications in agriculture and medicine.

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EARLY YEARS

Family Background

I was born in 1948 in Budapest, Hungary, just a few years after the Second World War. The city was still in ruins, and people queued for food that could only be obtained with ration tickets. Meat was available once a week, chocolate was unheard of, and in winter the only fruit available was apples. Yet, despite the hardships, I had a happy childhood.

I grew up in my grandmother’s house, built by my grandfather at the beginning of the war in the outer suburbs of Budapest, far from the bombing and fighting. My grandfather had owned a successful construction company, so my grandmother did not have to work outside the home and instead devoted herself to the household. My mother was their only child. She admired her father deeply and dreamed of becoming an architect, but that dream was never fulfilled.

My grandfather developed leukemia during the war. While my grandmother traveled constantly to the countryside in search of food, my teenage mother stayed by his side. He died in 1945, when my mother was just 18 and my grandmother was only 40. They lost almost everything except their house.

My mother, beautiful and intelligent, could not continue her studies. Instead, she married my father, Dezső Kuzsel. His family had lived in Transcarpathia, but after the war, due to the Czechoslovak–Hungarian population exchange, they were forced to move. His parents eventually settled in Cheb, Czechoslovakia. Because of the Iron Curtain and strict travel restrictions, I first met my paternal grandparents when I was eight years old.

Although my parents initially moved to Czechoslovakia after marrying, my mother returned to Hungary in her ninth month of pregnancy, and I was born in Budapest. My father began working in the office of a large car repair factory. His language skills and academic talent later brought him a university degree and a PhD in economics, and eventually he became the head of the international department at the Ministry of Transport. My mother stayed at home with me until I was a year and a half and later worked as a secretary in various factories, finishing her career at a watch and jewelry company. Since both of my parents worked, my grandmother—only 43 years old when I

was born—became my main caregiver. She gave me a harmonious and loving childhood, always supporting me.

In 1956, during the Hungarian Revolution, we hid with neighbors in the basement of my grandmother's house. Because we lived far from the city center, we were spared the worst of the fighting. I did not attend kindergarten but started my education in a small local primary school. Later, I moved to a larger elementary school, where I spent the next four years.

When I was 11, my parents divorced—something still rare at the time—and I felt deeply stigmatized. A year later, my mother married Andor Tarnai, a literary historian. At first I struggled to accept him, but he became a wonderful stepfather, giving me love, guidance, and encouragement. I was his only daughter.

My mother was determined that I should attend the best secondary school in Budapest. I was enrolled at Fazekas Mihály High School in the humanities program, inspired by my stepfather's work as a literary historian. I read voraciously, studied Latin, and dreamed of becoming a scholar of literature. But after a year, the humanities track was abolished and the school specialized in mathematics. They offered me a place in the math class, but the students there were exceptionally talented: Many went on to become world-famous mathematicians, including László Lovász, later awarded the prestigious Abel Prize. I felt like I could not compete with these math geniuses, so I turned down the opportunity, and our entire class was transferred to the renowned Eötvös József High School.

This high school, in the heart of the city, had excellent teachers and a spirit of freedom and curiosity. With eight parallel classes—some for boys, some for girls, and some mixed—it offered plenty of opportunities to make new friends. My class was all girls, but we mingled widely, and I loved being there. School finished early in the afternoon, leaving time for cinema, theater, and, above all, reading.

I had the best grades, but I was uncertain about which path to take at university. At that time, admission was very competitive, and a degree was the only way to open the world for us. In my final year of high school, I drove my parents to distraction with my indecision. I had only two months left before applications were due when some classmates mentioned a special molecular biology course for secondary school students, organized by Bruno F. Straub, head of the Institute of Medical Chemistry, Medical University, in Budapest at the campus of the Eötvös Loránd University (ELTE). Straub had been a research assistant to the Nobel Prize laureate Albert Szent-Györgyi and later discovered actin and proposed the theory of conformational selection in 1964.

ELTE was only a five-minute walk from my high school, so I began attending. During the course, I heard lectures by brilliant scientists such as Pál Venetianer and Vilmos Csányi. After just three lectures, I knew I had found the defining passion of my life. During the communist era, our official curriculum barely mentioned DNA as the hereditary material. These lectures opened an entirely new world, revealing the excitement and possibilities of scientific research. Within three weeks, I submitted my application to study biology at ELTE.

University Studies and the Start of Scientific Research

A biology course had been introduced only two years earlier on Straub's initiative, designed to train a new generation of researchers in modern biology. Out of more than 250 applicants, just 17 of us were accepted into this pioneering program at ELTE. We were given individual training by scientists and attended special courses, each supervisor guiding only four or five students.

At that time, everyone in Hungary was poor. But thanks to my good academic results, I received a scholarship that allowed me to live in somewhat more comfortable conditions. I could buy beautiful Italian shoes; go to restaurants; and attend the theater, opera, and concerts—all of

which were still inexpensive then. I also had the time and freedom to continue my passion for reading. Those five university years remain among the happiest of my life.

I began research in my first year at the university's Institute of Evolution and Heredity, later renamed the Institute of Genetics. The head of the institute, Béla Faludi, was a loyal Party member and staunch supporter of the government, and the shadow of Lysenko's doctrines still lingered. Yet, in certain laboratories, real genetic research had already begun. I joined the group of Imre Fedorcsák, who held a double affiliation: one research team at ELTE and another at Stockholm University in Sweden. This unique arrangement fostered exchanges and collaborations between young scientists from both groups. It was an exceptional opportunity, and I was thrilled to join.

In his laboratory, I learned to work with microbes and phages, often guided by senior students. One of them was Adam Kondorosi—brilliant in both academics and sports, admired by many, and very handsome. Adam and I shared a mutual admiration, keeping a polite “three-step distance”; still, deep inside, I already felt he might be my ideal husband. After graduation, Adam went to the Stockholm laboratory for a year, while I took over his projects at the university. When he returned, he joined the Institute of Genetics at the Hungarian Academy of Sciences (HAS) in Budapest.

The Creation of the Biological Research Center in Szeged, Hungary: My Future Host Institute and the Birth of Modern Biology

Under Straub's leadership with the support of HAS, the Biological Research Centre (BRC) in Szeged was being built. Straub, as general director, envisioned it as the cradle of modern biological sciences in Central Europe—bringing together genetics, molecular biology, plant sciences, and biophysics. He appointed top scientists as directors and actively recruited the most talented young graduates to start their careers there. Szeged also offered apartments for rent to newcomers, a rare and attractive opportunity for young people. When the center opened, the average age of the staff members was under 30 years old.

Straub secured United Nations Development Programme (UNDP) support for the new center and brought world-class scientists to Szeged for seminars and mentoring. Sydney Brenner came, as did Rollin Hotchkiss, who had an especially decisive role. He spent several extended visits there, offering guidance, sharp criticism, and encouragement—particularly to young researchers from the Institute of Genetics. These discussions were invaluable and transformative. Straub also initiated an international training program for young students from all over the world. Since the borders were closed, this program and the presence of international mentors created an international atmosphere at the BRC.

Many of the recognized scientists and promising young researchers, including Adam, moved from Budapest to Szeged in 1971, when the first half of the BRC was opened. Adam joined the Institute of Genetics, led by director Lajos Alföldi, who had a strong interest in *Rhizobium* bacteria, which induce the formation of nitrogen-fixing root nodules in legume plants. Alföldi instructed Adam to investigate the genetics of *Rhizobium* (later renamed *Sinorhizobium*) *meliloti*, the symbiotic partner of *Medicago sativa* (alfalfa). At that time, in 1971, virtually nothing was known about the genetics or molecular biology of rhizobia.

I was still completing my degree at ELTE, and my project inherited from Adam required regular discussions between us, in both Budapest and Szeged. I vividly remember my first visits to Szeged: the corridors buzzing with debates late into the night, the tea rooms and staircases alive with arguments and ideas. The lights of the building shone until dawn, filled with the energy of young researchers competing to solve problems and outdo each other in cleverness. It was electrifying—something I had never experienced at university.

A BUMPY START

Although I had initially not intended to move to Szeged—I had good job offers in Budapest—I gradually realized that Szeged was the right place for me, and not only for the science. Frequent meetings with Adam drew us ever closer and bound us to each other for life. We married in 1973, and I received a two-year scholarship from HAS, enabling me to begin work in Szeged under the supervision of Lajos Alföldi. He had returned a few years before from the Pasteur Institute in Paris, France, where he had worked with Nobel laureates François Jacob, Jacques Monod, and André Lwoff. It would have been the perfect beginning for my scientific career. However, it did not happen. When Alföldi found out that I had married Adam, he immediately told me that I could not work under any circumstances at the Institute of Genetics. He considered Adam the most talented of all the young people, who had already published three papers by 1973, including one in *Nature New Biology* (26).

He was afraid that a young wife would hinder Adam's work, so he directed me to the biochemistry institute. Although I was disappointed and disagreed with him, I never harbored negative feelings toward Alföldi for my inability to remain at the Institute of Genetics, and I should add that a lifelong friendship developed between him and us.

At the Institute of Biochemistry, my supervisor was supposed to be Pál Venetianer. I had admired him ever since I heard his lecture as a secondary school student. He was the pioneer of genetic engineering in Hungary, and I looked forward to working with him with great enthusiasm. But only three weeks later, he told me that he had been invited to the United States for a year, and he would leave with his family.

Instead, I was assigned to Géza Dénes, a respected biochemist who had just started a new immunology project in Szeged—an idea proposed by his young and talented but rather eccentric wife. At first, things went well. I worked on developing a protocol for the isolation of T lymphocytes. My results progressed smoothly, although I disliked working with animals, especially the act of killing them.

At that time, Adam received a one-year fellowship to join John Postgate's Unit of Nitrogen Fixation at Sussex University in Brighton, United Kingdom (UK)—the global center of nitrogen fixation research, with outstanding biochemists and geneticists. It was an extraordinary opportunity, and, naturally, we wanted to go together. But in Hungary this was almost impossible. Authorities feared young scientists would stay abroad. So Adam was allowed to go, but I had to remain in Szeged.

When I told my supervisor that I wanted to join Adam, his attitude toward me changed overnight. Until then, he had been pleased with my work. But once he knew I might leave the group, he decided to punish me. He assigned me a new project I considered absurd: measuring the daily variation in the weight of internal organs in Specific Pathogen-Free (SPF) mice and rats. Every day, I had to kill 40 animals. My tears fell while he smiled behind my back. At that moment, I seriously considered leaving biology forever.

My passport was blocked, and I could not leave the country. I suspected that Géza Dénes, who had joined the communist party at that time, was behind it. One morning I told him plainly that if I did not receive a passport and visa to follow Adam, I would resign. By 2 PM that same day, I had my passport in hand, and soon I was on my way to England.

My First Acquaintance with Nitrogen Fixation: Evidence for Different Subunits of the Molybdenum-Iron Protein

Although I had no work permit or fellowship, the director of the unit, Joseph Chatt, kindly allowed me to work in a small laboratory that I shared with Adam. He was amused to discover that our first

names were Adam and Eva and promptly nicknamed our little room the Eden Lab. The unit was filled with brilliant young scientists—geneticists such as Ray Dixon, Frank Cannon, and Christina Kennedy; biochemists such as Bob Eady and Roger Thornley; and a steady stream of international visitors including Fred Ausubel and Claudine Elmerich.

I began analyzing nitrogenase enzyme complexes purified from free-living nitrogen-fixing bacteria using sodium dodecyl sulfate (SDS) gel electrophoresis. To my surprise, I found that the molybdenum-iron protein consisted of two nonidentical subunits, which appeared as separate bands on the gel. At first, nobody believed me. The biochemists had seen only one band. I had to prove my case. I collected every batch of SDS available in the institute, from different suppliers, and ran the gels again. It turned out that the resolution of the two subunits depended on the quality of the SDS.

After three months, I received a six-month fellowship to work in immunology at the School of Biological Sciences at Sussex. During this time, the work on the molybdenum-iron protein was completed and later published in the *Biochemical Journal* (22). Adam's work at the Unit progressed very well and culminated in a publication in *Nature* (7).

My Return to Hungary and Liberty by the St. George's Day Rule

Returning to Hungary from the UK, I was forced back into the Dénes group. Unlike the general open spirit at the BRC, none of us in Dénes's group was allowed to talk about our experiments at the Center, and the atmosphere was deeply depressing. Fortunately, Straub had introduced a unique rule: On St. George's Day each year, young scientists were allowed to change their supervisor. I seized the opportunity and moved to the group of Ernő Duda, who was brave enough to accept me despite possible disadvantageous consequences.

Working with Ernő was a revelation. He was full of ideas and an inspiring mentor, launching the careers of several brilliant scientists, including Katalin Karikó, Zsuzsanna Izsvák, and Kornélia Polyák. The atmosphere in his laboratory was lively, stimulating, and cheerful. My project was to develop liposome-mediated DNA transfer into human cells. Research funding was scarce, so we had to make much of our own material. Since phospholipids were unavailable to purchase, we isolated them ourselves. We extracted lipids from calf brains in the sweltering summer heat amid the fumes of liters of organic solvents. It was tough work, but we were able to introduce DNA into mammalian cells (58).

But it wasn't only lipids we lacked: Almost every chemical was in short supply. There was no money, and sometimes the compounds we obtained were of poor quality, so we had to purify them ourselves through repeated crystallization. If we wanted to do research, we had to invent solutions.

For cloning experiments, laboratories needed large amounts of restriction enzymes, which at the time were prohibitively expensive. We decided to produce them ourselves and then sell or exchange them for other essential chemicals. By then, Pál Venetianer was the director of the Biochemical Institute, and during his trips abroad he arranged business deals involving these enzymes.

Alongside this work, I was deeply engaged with Adam's research. I often joined his group's five o'clock tea discussions at the Institute of Genetics, where lively debates took place. And in the evenings, at our own dinner table, we continued the conversations—sharing ideas, weighing possibilities, and exploring solutions. In this way, I became fully aware of the challenges in his field and the scientific problems waiting to be solved. Their group published in *Nature* the circular linkage map of the *R. meliloti* chromosome (27) and later mapped the nodulation and nitrogen fixation genes to a high-molecular-weight plasmid of *R. meliloti* (3).

But the key question remained: Which *Rhizobium* genes are responsible for inducing nodule development in the host plant root?

A LIFELONG COMMITMENT TO SYMBIOTIC NITROGEN FIXATION

Act 1: Identification of Nodulation Genes

In 1980, Adam received an invitation from Jeff Schell, director of the Max Planck Institute for Plant Breeding Research (MPIZ) in Cologne, Germany, to establish a *Rhizobium* research laboratory at his institute. Jeff Schell was a renowned plant scientist, celebrated for his groundbreaking work in plant molecular biology and biotechnology. Together with Marc Van Montagu, he had developed the method of *Agrobacterium* T-DNA-mediated plant transformation.

To deliver the invitation, Jeff sent Bill Broughton as a messenger to Hungary. Both Jeff and Bill had imagined Adam to be a senior scientist and were astonished to discover how young he was. Adam did not immediately give a positive answer. Shortly afterward, Jeff Schell and his wife Betsy traveled to Budapest to persuade him in person. Eventually, Adam accepted the offer, on the condition that I could also accompany him to Cologne and work there. I was awarded a Max Planck fellowship and received permission from both the BRC and my supervisor, Ernő Duda, to spend a year in Germany.

Since I knew I would be working on the identification of *R. meliloti* nodulation genes, I first learned all the routine molecular biology techniques from my colleagues in Venetianer's group before leaving for Cologne. At that time, no handbooks or commercial kits existed; molecular biology was in its infancy.

At the Max Planck Institute, I shared a laboratory with young researchers from the United States who brought with them a variety of methods and expertise. The institute itself had just been established under the leadership of Jeff Schell, who had recently moved from Ghent, Belgium, to Cologne to build up the new research groups. When we arrived at the end of 1981, only a relatively small number of scientists were there, but every one of them was outstanding.

The atmosphere was warm and collegial—almost like a family. Each day we gathered for lunch at a long table in the canteen, where the menu was always a variation of eintopf, a hearty vegetable soup with a bit of meat or sausage. Despite these modest surroundings, our research ambitions could not have been higher.

What defined the institute above all was Jeff's intellectual leadership: his brilliant ideas, sharp critiques, and the stimulating scientific discussions he fostered. Together with Adam, he was a source of constant inspiration, and over time we developed a deep and lasting friendship.

To localize the nodulation genes on the symbiotic megaplasmid, I constructed a cosmid library carrying 30–45-kb fragments of the *R. meliloti* genome and identified the clones containing the *nif* genes. By hybridizing these clones to symbiotic plasmid (pSymA) deletion mutants, to R' plasmids carrying large regions of pSymA, and to the cosmid library itself, we were able to construct a physical map of the *nod–nif* region.

My daily routine was rigorous: Each day I prepared 96 minipreps, digested them, ran them on agarose gels, and in the evening began the Southern blotting. Adam prepared the radioactive probes and carried out the hybridizations, and together we assembled the physical map.

Of course, we were not the only ones working on the identification of *nod* genes. Among the key figures were John Beringer, Andy Johnston, and Allan Downie in the UK; Fred Ausubel, Sharon Long, Ann Hirsch, and Gary Ruvkun in the United States; Pierre Boistard, Georges Truchet, and Jean Dénarié in France; Barry Rolfe, John Shine, Peter Gresshoff, and Michael Djordjevic in Australia; and Alf Pühler in Germany. Although the competition was intense, the atmosphere among us was also remarkably friendly and supportive.

A major milestone came when Alf Pühler organized the first International Symposium on the Molecular Genetics of the Bacteria–Plant Interaction, held in Bielefeld, Germany, on October 11–14, 1982. This meeting brought together all the leading scientists working on *Agrobacterium*



Figure 1

Participants of the first International Symposium on the Molecular Genetics of the Bacteria-Plant Interaction, held in Bielefeld, Germany, from 11 to 14 October 1982. (a) Jeff Schell, (b) Graham Walker, (c) Fred Ausubel, (d) Adam Kondorosi, (e) Jonathan Jones, (f) John Beringer, (g) Peter Gresshoff, (h) Eva Kondorosi, (i) Alf Pühler, (j) Desh Pal Verma, (k) Sharon Long, (l) Tön Bisseling, (m) Clive Ronson, (n) Allan Downie, (o) Andy Johnston, (p) Marc Van Montagu, (q) Barry Rolfe, and (r) Bill Broughton.

and *Rhizobium*-legume symbiosis (**Figure 1**). Since then, the symposium has grown into the long-running International Society for Molecular Plant-Microbe Interactions (IS-MPMI) conference series.

Back in Hungary, at the Institute of Biochemistry, I was able to continue my work on the identification of nodulation genes. I carried out directed Tn5 mutagenesis of *nod* regions located on 8.5-kb and 6.8-kb fragments of pSymA, mapped the insertions, and analyzed the symbiotic phenotypes of the mutants. By the end of 1983, we had identified the nodulation genes of *R. meliloti* together with Zsófia Bánfalvi, Adam's PhD student, who contributed to this work by constructing R' plasmids and deletion mutants.

The second IS-MPMI conference was in 1984 at Cornell University, Ithaca, United States, and we were all eager to know the advancements made by other groups. I still remember our conversation with Andy Johnston, Allan Downie, and Gerd Hombrecher before the official opening, with Andy asking tricky questions and with a friendly smile wanting to know our unpublished data. Then at the conference I had to present our results. I was in the same session as Sharon Long, and I was terribly nervous. Sharon always gave beautiful talks, and with my poor English, even

with solid results, I felt greatly disadvantaged. We had different results that we discussed after our presentations, and Sharon agreed with our results.

We had the physical and genetic map of a 135-kb segment of pSymA and demonstrated that one set of *nod* genes (*nodABC* and *nodD*) is common across rhizobia, while another set determines host specificity (28). Around this time, nodulation genes were also localized on DNA regions and identified from other *Rhizobium* species and strains (see, for example, 1, 8–10, 20, 48, 55, 64), and Ausubel's laboratory gave significant contributions and generous help to these studies (16, 52).

In the years that followed, we had a fruitful collaboration with Jeff Schell's institute. Jeff reserved a laboratory and an office for us that we could use any time, allowing frequent visits for us and other group members from Szeged to Cologne. We could stay in the guest house and for longer times in the apartment at MPIZ. We obtained financial support from the Deutsche Forschungsgemeinschaft (DFG) and had numerous students from Germany who obtained their PhD in Hungary.

During this period, I focused on the regulation of *nod* genes. We sequenced the *R. meliloti* nodulation genes (63) and identified a conserved regulatory *cis* element, the *nod-box* in the promoters of *nod* operons (49). We wanted to know which plant signals induce the expression of *nod* genes that are not expressed in bacteria cultivated in media. I created a *nodABC* promoter–*lacZ* fusion in our *R. meliloti* strain 41 and added seed and root exudate to the bacteria but did not observe an increase in β -galactosidase activity. This line of research also began in Sharon Long's laboratory at Stanford, California, United States, where it was shown that luteolin induces the expression of *R. meliloti nod* genes (47). It was intriguing and unexplainable until I realized that *nod* gene expression is under tight regulation, controlled both by transcriptional activators (NodDs) and by a repressor (NolR) (29, 31). We showed that NodD and NolR bind adjacent sites within the *nod-box*; however, in the presence of luteolin, only NodD remains bound, while NolR dissociates. Binding of NolR prevents RNA polymerase from accessing the promoter, thereby repressing the transcription of *nod* genes. Later, we examined the *nolR* gene in *R. meliloti* strain 1021—the strain used by Sharon—and we discovered a mutation that abolished repressor function (5), resulting in the activation of nodulation genes in the presence of luteolin.

In addition to *nod* gene regulation, we also sought to identify host-specific nodulation (*bsn*) genes and, more broadly, to understand the function of nodulation genes. The organization, structure, and symbiotic function of *R. meliloti* nodulation genes were elucidated through collaboration with colleagues from the MPIZ and published in *Cell* (19), followed by several other joint publications.

Act 2: A New Life and Plant-Oriented Studies at the CNRS Institute of Plant Sciences in France

In 1987, an unexpected offer came from the French National Centre for Scientific Research (CNRS), the largest basic science agency in Europe. The Division of Life Sciences of CNRS had decided to create a modern plant science institute at their Gif-sur-Yvette campus in the region southwest of Paris. An international search committee, chaired by Peter Albersheim, director of the Carbohydrate Research Center at the University of Georgia, nominated three candidates for the directorship, including Adam. Ultimately, CNRS selected Adam to lead the new Plant Science Institute.

This was both good and difficult news for us. Our work in Szeged was thriving, and the collaboration with Jeff Schell was ideal. Starting a new life—and even more so, a new institute—in the shell of a long-closed facility, the giant old Phytotron building (**Figure 2a**), while navigating the complexities of French administration, was a daunting challenge we did not necessarily have to accept. Adam was well aware of the risks and did not immediately give a positive answer. He

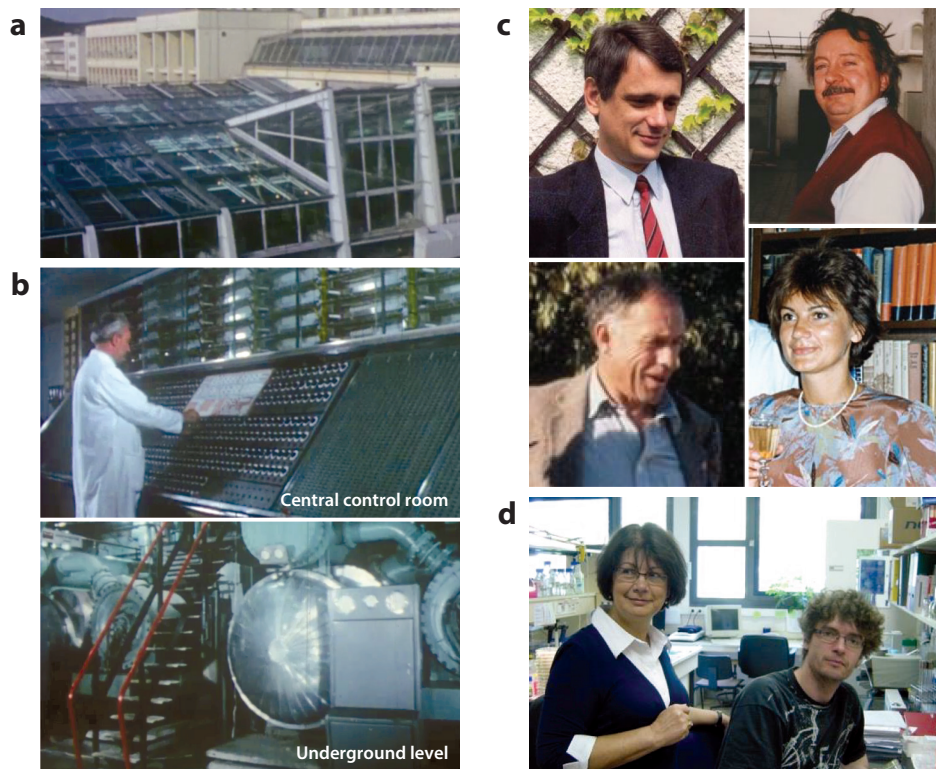


Figure 2

Creation of the Institute of Plant Sciences on the CNRS campus in Gif-sur-Yvette. (a) The original Phytotron building. (b) The central operations room and an underground level of the Phytotron building. (c) Founding members of the Institute of Plant Sciences (clockwise from top left): Adam Kondorosi, Bruno Gronenborn, Eva Kondorosi, and Jacques Tempé. (d) Working with Peter Mergaert. Panels a and b adapted with permission from *Le Phytotron* d'Eric DUVIVIER; copyright CNRS/Sciencefilm – 1969 (CC BY-NC-SA 4.0). <https://images.cnrs.fr/en/video/1342>.

wished to maintain his laboratory and work with the people at the BRC and hesitated to jump into such uncertainty.

At this point, Jacques Demaille, head of the Life Sciences Division of CNRS, traveled to Budapest to negotiate with us, with the leadership of HAS, and with Lajos Alföldi, director of the Institute of Genetics. Demaille presented an extraordinary offer: an apartment in the CNRS château on the Gif campus, a car with a driver, 20 postdoctoral positions, and many other benefits. Both HAS and Alföldi advised us that this opportunity could not be refused. Still, Adam requested financial support from CNRS to maintain his group in Szeged, and CNRS agreed.

Friends from France came to warn us not to believe such incredible promises. I did not speak any French, while Adam, although he held a high-level certificate, had not practiced the language in years.

That summer, while staying at Lake Balaton, we carefully weighed the pros and cons (Figure 3). In the end, Adam accepted the position and was officially appointed director on January 1, 1988. I applied for a permanent CNRS research position at both the junior (*chargé de recherche*) and senior (*directeur de recherche*) levels. The latter was rarely granted to external scientists, yet, to my great surprise, I was awarded it.



Figure 3

The decision has been made: We are moving to France. Eva and Adam Kondorosi in August 1987. Photo by our friend Károly Gink.

Adam asked for a general director to manage administrative and financial matters, and Roger Guillet was appointed. He was professional, fair, supportive, and kind, though his English was limited. Adam's most urgent task was to recruit top, internationally recognized group leaders. In some respects, this was easy: Bruno Gronenborn, an excellent plant virologist from MPIZ, and Jacques Tempé, renowned for his work on *Agrobacterium*, both agreed to join as founding group leaders. Thus, we were four (**Figure 2c**).

The building itself had to be completely renovated. The old Phytotron's operating control room with its retro-futuristic design looked like something from an early James Bond film, complete with mysterious underground levels (**Figure 2b**). I drafted the new layout on graph paper—walls, windows, offices, laboratories, instrument rooms, chemical fume hoods, and sterile hoods—and presented my plans to the CNRS architect, Mr. Mongé. He approved them, and the institute was rebuilt accordingly, opening in 1989. Meanwhile, Adam, Bruno, and Jacques worked on recruitment, interviewing candidates, and deciding which staff members from the former Phytotron should join. Since the institute did not yet exist, we met regularly in Gif, Cologne, or sometimes at Jacques's house to discuss scientific plans, staffing, and organization.

Our daughter, Fanny, was born in March 1988. I had long awaited this moment, and in Hungary it was customary for mothers to spend three years at home with their baby. I visited Gif in May 1989 to look around, planning to spend another six months settling in, learning French, and caring for Fanny. I requested unpaid leave, but CNRS informed me that if I did not start by May 31, I would lose my position. This left me just one week to rent a house, buy furniture, and arrange childcare. As always, I could rely on my family. My grandmother moved with us to France and cared for Fanny and the household. During the day I ran back and forth between the institute and our home so I could walk or play with Fanny, spending evenings at home until she slept, and then returning to the laboratory at night to keep my work progressing.

A further difficulty was that Hungarian visas were valid for only three months. My grandmother therefore had to be replaced regularly by my mother and occasionally by other babysitters. At that time, my father was a professor at the Universities of Vienna and Berlin, so my mother was constantly traveling between those cities and Budapest. Adam and I also shared the childcare duties,

and weekends were usually reserved for family time. With this support, I could still focus on my research and students.

Together with Adam, I codirected the *Rhizobium*–legume group, and we divided responsibilities. In the early years, Adam was fully occupied with setting up the institute, inviting and recruiting internationally recognized scientists, and expanding the scope of research. The 20 postdoctoral positions originally promised by Demaille never materialized—by then, he was no longer in his post. Instead, CNRS honored its commitment by establishing a twinning (*jumelage*) program with the Szeged laboratory, which provided annual 12-month fellowships for young Hungarian researchers to spend several months at the Institut des Sciences Végétales (ISV). Soon, Jean Guern and his team working on auxin signaling joined ISV, followed by Jérôme Giraudat, who established a group on abscisic acid signaling. Gradually the institute filled with French and international students and postdocs. On July 12, 1990, *Nature* (Vol. 346) acknowledged the successful start of ISV in an article titled “Hungarian Sowing Seeds.”

One of the first postdocs to join our group was Michael Schultze from Basel, Switzerland, who became a key contributor to our studies in the 1990s and a supportive, kind friend. We wanted to discover the nature of the bacterial signals produced by the *nod* genes and how *Rhizobium* communicates with its host plant to trigger infection and nodule organogenesis.

To address this, I constructed an *R. meliloti* strain with a constitutively high expression of *nod* genes. Michael, together with chemists from the CNRS campus, worked intensively on identifying the bacterial signals. Meanwhile, similar research was underway in Jean Dénarié’s laboratory in Toulouse, France, which was the first group to characterize the Nod factors (33). Our own study, published in 1992, revealed that *R. meliloti* does not produce a single signal molecule but rather a family of sulfated lipooligosaccharides, each exhibiting different degrees of host specificity (56).

We then turned our attention to plant responses evoked by the Nod factors. These included studies on ion fluxes in root hairs (13) and the identification and role of early nodulins such as *enod40* (6). While Adam supervised most of these studies and was eager to develop *Medicago truncatula* as a model plant for symbiosis research, I started to work in a new direction, and cell cycle regulation emerged as the dominant research line in my laboratory.

The integration of the cell cycle in nodule development. In response to Nod factors, the cell cycle is reactivated in the root cortical cells and results in the formation of a persistent meristem. In the indeterminate root nodules, there is an age and differentiation gradient, extending from the apical meristem to the nodule base (Figure 4a,b). *Rhizobium* infection occurs in the postmitotic cells, and the symbiotic cells then develop gradually into huge nodule cells filled with thousands of nitrogen-fixing bacteroids (Figure 4b,c). Our research focused on the reactivation of the cell cycle in *Medicago* roots and on the differentiation of symbiotic cells.

In the early 1990s, plant cell cycle regulation was in its initial state, but Dénes Dudits’s group at BRC had already worked on the *Medicago* cell cycle and helped us to initiate this new research line.

We described several cell cycle genes in *Medicago* (14, 50, 54), but the real breakthrough came with the identification of the cell cycle switch protein CCS52A. With major contributions from two exceptionally talented Spanish postdocs, we demonstrated that the development and growth of symbiotic nodule cells are achieved through endoreduplication cycles, which lead to high ploidy levels and large cell volumes and are essential for the development of nitrogen-fixing root nodules (4, 32, 69) (Figure 4b). During the identification of CCS52A, Peter Mergaert, a fresh postdoc from Marcelle Holsters’s group in Ghent, joined my laboratory, while Michael Schultze moved to York, UK. Working with Peter was a joy, and we formed an effective team (Figure 2d).

Because CCS52A is conserved across plants, we extended this research to *Arabidopsis*, where cell cycle control was already intensively studied in many laboratories. Our work showed that CCS52A

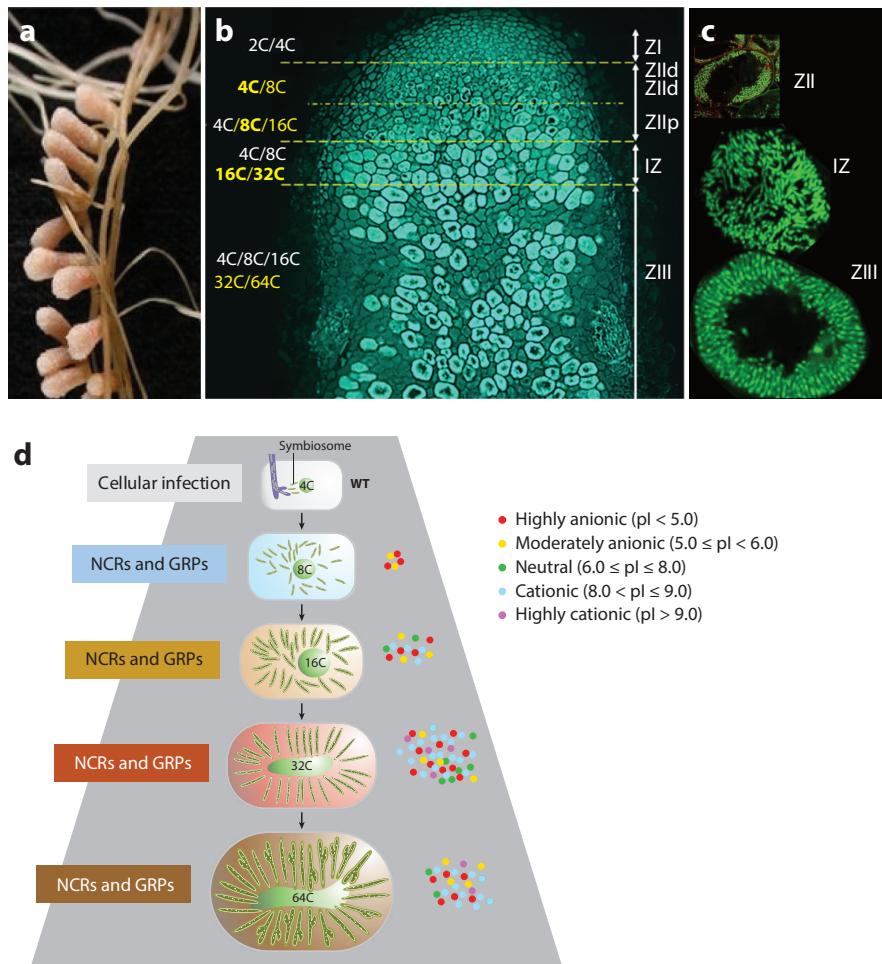


Figure 4

Development of nitrogen-fixing root nodules. (a) Nitrogen-fixing nodules formed on *Medicago truncatula* roots. (b) Longitudinal section of a nodule showing the gradual growth of symbiotic cells, tightly coupled to successive endoreduplication cycles. DNA content of uninfecting, non-symbiotic cells is shown in white, whereas symbiotic cells are shown in yellow; dominant ploidy levels are indicated in bold. ZI represents the meristem containing 2C/4C nuclei (C, haploid DNA content), ZII represents the infection zone with 4C/8C nuclei in the distal ZIIId cell layers and predominantly 8C nuclei in the proximal ZIIp cells, IZ represents the interzone with 16C/32C nuclei, and ZIII represents the nitrogen-fixing zone with 32C/64C nuclei. Panel adapted from Reference 43. (c) Multiplication of rhizobia within ZII symbiotic cells, followed by their elongation driven by irreversible loss of cell division capacity and continued endoreduplication in the IZ. This process results in the formation of large, polyploid, noncultivable, nitrogen-fixing bacteroids in ZIII. (d) Progressive bacteroid differentiation is directed by stage-specific "cocktails" of NCR and GRP peptides produced in symbiotic cells. (Left) NCR and GRP peptides shown on different background colors represent distinct peptide cocktails acting at successive stages of symbiotic cell differentiation. (Right) Colored dots represent peptides with different physicochemical properties; dot number reflects peptide abundance (from a few tens at early stages to several hundreds, up to ~400, at later stages). Dot colors indicate peptide charge classes based on pI. Abbreviations: GRP, nodule-specific glycine-rich peptide (nodGRP elsewhere); NCR, nodule-specific cysteine-rich; pI, isoelectric point; WT, wild type. Panel adapted from Reference 30; copyright 2013 Annual Reviews.

is essential for cell cycle exit, cell differentiation, and the induction of endoreduplication cycles, thereby enabling the formation of polyploid cells (2, 32, 61, 68). This positioned our group within a broader European context, and I became a member of the European Cell Cycle Organization (ECCO) in the EU framework program FP7, where I led the work package on endoreduplication. In 2006, I was also elected to the European Molecular Biology Organization (EMBO).

Rhizobium bacteria can differentiate like eukaryotic cells. The *Rhizobium* partners in indeterminate nodules of *Medicago* and related galeoid legumes undergo drastic morphological changes in the symbiotic nodule cells, becoming huge, elongated, and often Y-shaped nitrogen-fixing bacteroids (30) (**Figure 4c**). As in eukaryotic cells, there is a correlation between nuclear and cell volumes; I hypothesized that similar changes might also occur in bacteria. We demonstrated that the genome of bacteroids is indeed amplified through repeated endoreduplication cycles, reaching an ~20-fold increase in DNA content and producing cells 5 to 10 times longer (40). This confirmed a positive correlation in prokaryotes between DNA content and cell size. However, these bacteroids were uncultivable, and we showed that their irreversible, terminal differentiation is controlled by the host plant (40).

While the science progressed very well, we faced a tragedy in our family life. Adam—struggling with sleep problems and an overwhelming workload—was prescribed a neuroleptic medication. Instead of helping, it provoked severe anxiety. The doctor assured me this was normal and would improve within weeks. During the winter school holiday, Adam and Fanny traveled to Hungary for a relaxing week of theater and rest. The day after their arrival, Fanny called me in a panic: Adam had a very high fever and was paralyzed in a V-shape. In retrospect, I know he was suffering from neuroleptic malignant syndrome. Years later, I learned that Adam's mother had also had the same problem and was misdiagnosed with Parkinson's disease. Likewise, Adam was misdiagnosed and treated with escalating doses of antiepileptic drugs. Later, a specialist admitted that Adam had no epilepsy but warned that stopping the treatment would cause epilepsy. For seven hopeless years I gave all my attention and love to Adam. During many sleepless nights I was thinking about solving scientific questions, and this period became one of the most productive of my life. In many ways, science itself saved me from falling into deep depression.

Novel plant peptide families driving bacteroid differentiation. Terminal bacteroid differentiation—characterized by altered morphology, genome amplification, and loss of cell division ability—occurs in only a subset of legumes. This phenomenon is found in *Medicago*, pea, vetch, and related species belonging to the inverted repeat-lacking clade (IRLC) but is absent in *Lotus japonicus* (the model for determinate nodules), as well as in bean, soybean, and many other legumes where the fate of bacteroids is reversible between the free-living and symbiotic lifestyle. Based on these differences, we hypothesized that specific genes required for terminal bacteroid differentiation are expressed in nodules of *Medicago* but are absent from *Lotus*. Comparative transcriptomic analysis supported this hypothesis by revealing hundreds of peptide-coding genes expressed in *Medicago* nodules that were not only missing from the *Lotus* transcriptome but also completely absent from its genome.

This work led to the discovery of the nodule-specific cysteine-rich (NCR) peptides. Initially, more than 300 NCR peptides were identified in *Medicago* (39), and subsequent sequencing of the *M. truncatula* genome revealed the presence of over 700 NCR genes. NCRs share a conserved signal peptide but differ in their mature peptide sequences, which remain highly variable apart from the conserved positions of four or six cysteine residues. Representing a completely new family of plant peptides, NCRs are expressed exclusively in symbiotic nodule cells. Different subsets of NCRs are activated sequentially during the stages of bacteroid development, tightly linking their expression with the progression of symbiotic cell differentiation.

In parallel, we identified another peptide family consisting of 38 members that encode nodule-specific glycine-rich peptides (nodGRPs) (24). Like NCRs, nodGRPs possess a conserved signal peptide, but their mature proteins, which are between 100 and 200 amino acids long, differ structurally from classical glycine-rich proteins. Importantly, nodGRPs were found only in legumes where bacteroids undergo terminal differentiation, reinforcing the idea that they, together with NCRs, act as plant effectors of this process.

In nodule cells, rhizobia are present in the form of symbiosomes, where each bacteroid is surrounded by a plant-derived membrane, giving it an organelle-like character. We showed that both NCRs and nodGRPs are targeted to the symbiosomes and that blocking their delivery prevents bacteroid differentiation (67). These findings provided direct evidence that secreted plant peptides determine the developmental fate of rhizobia within nodules (30, 42, 67) (**Figure 4d**). These findings transformed our symbiotic research, and similar peptides were later also discovered in insect–bacterium symbioses (36, 65).

The identification and functional analysis of NCRs and nodGRPs were made by the combined efforts of my French laboratory and coworkers from Hungary either working at ISV or in Szeged at BAYGEN in my newly created twinning institute.

Act 3: Return to Hungary

During Jacques Chirac's presidency, an agreement was signed between France and Hungary to establish a French–Hungarian Biotechnology Institute in Szeged. As the end of Chirac's term approached, the agreement needed to be finalized, and the director position was announced in 2006. I had not considered applying, but the French Ministry of Research encouraged me, noting my familiarity with both the French and Hungarian systems. CNRS also supported me, allowing me to share my time between France and Hungary.

Given our family situation, I felt Adam would receive much better care in Hungary, surrounded by relatives, friends, and professional nursing. I applied and was selected from among three candidates. Hungary provided the financial support (€5.3 million), and the BAYGEN Institute was inaugurated on March 15, 2007. I commuted weekly: one week with my daughter and ISV in France and one week with Adam and BAYGEN in Hungary. I recruited two young scientists—one from the United States and another from France—to lead divisions of the institute, and invited Adam's former student, Attila Kereszt, from Australia to collaborate on symbiosis. The enthusiasm of the young researchers, combined with a stimulating and constructive atmosphere, quickly made BAYGEN internationally visible. Intensive collaboration between BAYGEN and ISV resulted in several joint publications.

Funding for BAYGEN, however, was guaranteed for only five years. As part of the BAY Zoltán Foundation for Applied Research, I was required to prepare monthly business plans—something I disliked. To secure long-term research, I submitted a European Research Council (ERC) Advanced Grant application, and in December 2010 I was thrilled to learn it had been awarded.

But soon after, the Hungarian government decided to close all foundations, including BAY Zoltán. With the ERC grant in hand, I turned to HAS and my former affiliation BRC. At that time, no life science ERC Advanced Grants existed within HAS, so its president, József Pálkás, was eager to host us at BRC. Although financial and administrative obstacles caused a long delay, we were eventually reintegrated into my original institute, and finally my ERC grant “SYMBIOTICS” started on July 1, 2012.

By the end of 2010, Adam was in very poor condition and was hospitalized with a pulmonary infection in Szeged. He passed away in January 2011. I was paralyzed by grief, on the verge of

collapse myself, but I knew I had to survive for my daughter's sake. I wanted to preserve Adam's memory—for the scientific community and for all who loved, admired, and respected him for his remarkable contribution. I also felt a strong responsibility not to let our research—and the results Adam and I had built together—be lost.

One spring day in 2010, the telephone rang in my office with an unexpected call from the United States: I had been elected as a foreign associate of the National Academy of Sciences. The news was overwhelming—exciting, humbling, and truly fantastic. I could hardly find the words to respond. Later, I learned that Sharon Long and Bob Haselkorn may have been my nominators. I remain especially grateful to Sharon, as we were competitors, although we maintained a collegial and friendly relationship.

My path to membership in HAS was far from straightforward. Nominations are possible only once every three years, and a candidate may be proposed only three times. I was nominated in 2004, 2007, and finally in 2010. At the time, the Academy was overwhelmingly male dominated, and female academicians were as rare as white ravens. My first two nominations were declined for reasons that felt deeply unfair: (a) I was Adam's wife, so my individual contribution was questioned; (b) one academician was considered "enough" for a family; and (c) I had spent too much of my career abroad.

By 2010, however, the situation was different. I was directing an institute in Hungary, Adam had long ceased to be involved in my work, and I had already been a member of EMBO since 2006. Still, my chances remained slim—until the biology class realized that I was also a member of the US National Academy of Sciences, the only Hungarian scientist to hold that honor at the time. That recognition helped open the door.

In the same year, I was also elected to Academia Europaea without any obstacles. The following year I became a foreign member of the French Agricultural Academy, and a year later of the German National Academy of Sciences Leopoldina. In 2012, the Hungarian state awarded me the Széchenyi Prize for outstanding scientific contributions, and I received together with Adam—posthumously—the IS-MPMI Prize for the highest impact in the field.

NCR peptide functions in symbiosis and beyond. In March 2013, I left CNRS. Peter Mergaert became the group leader at ISV, and I continued my research entirely in Hungary. At that time, most laboratories in the field were focusing on the Nod signal transduction pathway. Within the ERC SYMBIOTICS project, our challenge was to uncover the role of NCR peptides (23). A central question was where to begin—how to select one peptide from the ~700 members of the NCR family?

Nodule transcriptome analyses (37, 51) revealed that distinct subsets of NCRs are expressed at early, intermediate, and late stages of symbiosis. However, experimental work was constrained by technical limitations: Purification of NCRs from heterologous expression systems proved unsuccessful, and isolation from nodules was not feasible due to their high complexity and low abundance. To overcome this, we turned to chemical peptide synthesis, selecting candidates based on expression pattern, size, and physicochemical properties.

Our first study focused on NCR247, a cationic peptide of only 22 amino acids, the smallest member of the family, which also displayed antimicrobial activity *in vitro*. NCR247 is expressed in symbiotic cells at the transition when bacterial cell division ceases and cell elongation begins. We demonstrated that NCR247 acts through a multihit mechanism, interacting both with bacterial membranes (11, 12, 41) and with numerous intracellular targets, thereby modulating diverse microbial pathways, including transcription and translation (11). One of its key targets was FtsZ: binding of NCR247 to FtsZ abolished septum formation and thereby blocked bacterial cell division.

Parallel studies in other laboratories investigated related aspects of NCR247: transcriptional responses controlling the bacterial cell cycle during symbiosis (46), the differential activity of oxidized versus reduced NCR247 isoforms (15, 57), and its role in promoting iron uptake (53).

But how many NCRs are essential for nitrogen-fixing nodule development? While this remains an open question, we know that NCR169 is essential in *Medicago* species (17), as are NCR211 (25) and NCR-new35 (18). Interestingly, although these genes are expressed at different stages of symbiotic cell development, their absence triggered a common plant response: premature senescence caused by impaired symbiotic differentiation and failure to sustain bacteroid viability. This type of senescence is distinct from the regulated aging process of developmental senescence (45).

Evolutionary analyses of NCRs across 10 legume species representing different IRLC subclades and bacteroid morphotypes revealed striking variation in family complexity, with a positive correlation between NCR diversity and their physiological and morphological effects on bacteria (42).

In 2018, the Balzan Prize in Life Sciences was announced in the field of chemical ecology. *Rhizobium*–legume symbiosis is an exceptionally rich example of chemical ecology, characterized by molecular dialogs between eukaryotic and prokaryotic partners and, in particular, the involvement of hundreds of plant peptides. I never learned who nominated me, but receiving the news that I was awarded the Balzan Prize was once again unexpected, wonderful, and deeply moving. Beyond the honor itself, the prize provided exceptionally generous support. Over the years, it has helped me not only to advance our scientific research but also to support students and young researchers in my laboratory and to create opportunities for mentoring programs, symposia, and conference participation.

The regulation of *NCR* genes—whose expression is restricted to symbiotic cells and specific developmental stages—was another major puzzle. Together with Peter's group in Gif, we investigated gene expression in symbiotic cells (38) and showed that ploidy-dependent changes in the epigenome correlate with specific patterns of gene expression (43). Because NCRs are found only in IRLC legumes, I initially hypothesized that they coevolved with specific transcription factors. Surprisingly, this was not the case. When we introduced an NCR169 promoter–GUS fusion into soybean—which lacks NCRs and terminal bacteroid differentiation—we found that the NCR169 promoter was still active (70). This demonstrated that the activation of NCR169 does not require NCR-specific transcription factors. Moreover, promoters of other NCRs coexpressed with NCR169 in *M. truncatula* nodules were also active in soybean. By identifying transcription factors binding to the NCR169 promoter, we demonstrated that the expression of *NCR* genes is activated by widely conserved AT-Hook Motif Nuclear Localized (AHL) transcription factors (70).

The remarkable diversity in the physicochemical properties of NCR peptides represents a vast, untapped reservoir of biological activities. Since many NCRs are produced in nodule cells at the stage when bacteria irreversibly lose their ability to divide, we began to explore their antimicrobial potential, considering the global health challenge posed by antibiotic resistance and the scarcity of novel antibiotics.

We tested the killing activity of more than 100 peptides—including mature NCRs, truncated and substituted variants, and chimeric derivatives—against some of the most difficult-to-treat, clinically relevant pathogens. These included the ESKAPE bacteria (*Enterococcus faecium*, *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and *Enterobacter* spp.) (21, 34, 35, 62), as well as fungal pathogens such as *Candida* species (44, 59, 60). Antimicrobial activity was strongly associated with positive charge, cationic isoelectric point, and specific amino acid sequences. While a few peptides displayed broad-spectrum activity and killed all tested pathogens, others acted selectively against only certain bacteria or unique species.

The most active, engineered NCR peptides eliminated pathogens within minutes, inhibited biofilm formation, and degraded already-formed biofilms, all while showing no cytotoxicity toward human cells (21, 34). These peptides act through a multitarget mechanism, simultaneously disrupting bacterial membranes and interfering with intracellular processes via interactions with nucleic acids and diverse proteins. Such a broad and multifaceted mode of action greatly reduces the likelihood of resistance development. Taken together, NCR peptides represent an underexplored but highly promising resource for the development of novel antimicrobial agents.

At the border of science and policy: scientific recommendations. The ERC grant provided excellent working conditions, and, together with my memberships in prestigious academies, it opened unforeseen opportunities. Until then, my life had been devoted almost entirely to scientific research. In 2012, I was nominated to the Scientific Advisory Board of the United Nations Secretary-General. This was a completely new experience that showed me how science can influence policy, change lives, and shape our shared future.

One opportunity led to another. A year later, I joined the Scientific Council of the ERC and eventually became its vice president responsible for life sciences. I also became part of the Board of Directors of Academia Europaea, where I now serve as chair of the life sciences class. More recently, I had the privilege to serve as a member of the group of Chief Scientific Advisors to the European Commission, where my work focused on updating cancer screening guidelines. These experiences taught me that science does not end with discovery: It can also guide decisions that affect society at large.

FUTURE PERSPECTIVES

We are still at the early stages of uncovering the roles of symbiotic peptides. So far, only a handful of NCRs have been investigated in depth, leaving the vast majority—and the related nodGRPs—largely uncharted. How dozens or even hundreds of peptides function together within symbiotic cells is a fundamental question. Protein binding assays already hint at the formation of heteromeric complexes between NCRs and between NCRs and nodGRPs, suggesting a sophisticated peptide–protein interaction network whose functions are still unknown.

While NCRs can be activated by general transcription factors, their spatiotemporal regulation is far from understood. How does the plant cell detect the precise stage of bacteroid differentiation to trigger the next suite of peptides required for development? How do bacteroids communicate back to the host? What are the bacterial signals that fine-tune host gene expression, physiology, and ultimately the success of symbiosis?

As this research will unfold over decades, I will not be part of its full journey. Yet I hope that the questions raised here—and the possibilities they point toward—will inspire and guide the scientific community. The next generations of researchers will be the ones to unlock the full potential of symbiotic peptides, and in doing so, may not only transform our understanding of plant–microbe interactions but also create innovations with lasting impact on agriculture, medicine, and beyond.

Adam meant everything to me. He was a loving husband, a wonderful father to our daughter, and my friend and partner in life and research. We were truly made for each other. Together, we experienced the excitement of discovery, faced and solved countless challenges, and always complemented and supported one another. In science, we always strove for the maximum. Adam founded a school and left a lasting mark, and his loss was mourned by our entire scientific community. In his memory, we established the Academia Europaea Adam Kondorosi Prize, awarded in two categories: one recognizing the life's work of the most outstanding researchers in our field and the other honoring breakthrough achievements by young scientists (Figure 5).

Since Adam's illness, Agnes Ullmann stood by my side. I always held Agnes in the highest regard; she was one of the most remarkable microbiologists at the Pasteur Institute and in the



Figure 5

Winners of the Adam Kondorosí Academia Europaea Awards. Advanced Research Award (a) winners: (b) Allan Downie (2017), (c) Ray Dixon (2019), (d) Paola Bonfante (2021), (e) Michael Udvardi (2023), and (f) Jens Stougaard (2025). Early-Career Investigator Award (g) winners: (h) Simona Radutoiu (2016), (i) Peter Mergaert (2018), (j) Caroline Gutjahr (2021), (k) Pierre-Marc Delaux (2023), and (l) Myriam Charpentier (2025).

world (66). Her support, her honest criticism, and her wise advice were invaluable to me, and over the years we became very close. Our bond went far beyond science: It felt as if she were my sister or I were her daughter.

Sadly, they are no longer with us, but their legacies continue. I am deeply grateful to all who have worked with me over the years, and to those who stood by me and offered support during difficult times—especially Peter Mergaert, Claudine Elmerich, Attila Kereszt, Gabriella Endre, and Bruno Gronenborn.

This year, Adam would have turned 80. To honor his memory, I am organizing a symposium on April 30, 2026, part of the 200th anniversary of HAS. Friends and colleagues from all over the world will come together from our field to remember the beginnings of research on symbiotic nitrogen fixation, to celebrate the progress we have made, and to look ahead to the challenges that still await us. It will be both a tribute to Adam's pioneering work and a celebration of the vibrant community that has grown from those foundations.

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