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Raising a Bacterium to the Rank of a Model System: The *Listeria* Paradigm

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Abstract

My scientific career has resulted from key decisions and reorientations, sometimes taken rapidly but not always, guided by discussions or collaborations with amazing individuals from whom I learnt a lot scientifically and humanly. I had never anticipated that I would accomplish so much in what appeared as terra incognita when I started to interrogate the mechanisms underlying the virulence of the bacterium *Listeria monocytogenes*. All this has been possible thanks to a number of talented team members who ultimately became friends.

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BEGINNINGS

I am from the north of France and grew up in a city called Arras, where my father's family had been living for centuries, not far from Cambrai, my mother's family's city. Both my parents had ten siblings! I was the oldest of five. My father was a flour miller who extended the family business by creating a factory of food for cattle, hens, fishes, pheasants, etc. My mother took care of the family at home. As I was born in 1948 after the war, I belong to the so-called baby boom generation, characterized by women who wanted to study, work, and be independent. I went to school in Arras and decided to study chemistry the day I received my first book of chemistry, at the age of 13. It was a revelation! This was unexpected, as I was in a class with Greek and Latin as majors. I thus switched toward a more scientific education.

CHEMISTRY TO BIOCHEMISTRY

University of Lille (1965–1970)

After my baccalaureate, I left the family for the University of Lille. The university campus was split into two parts, one in the city where Louis Pasteur himself had been teaching and a newer one in the countryside. It was a fantastic period in all aspects, with a lot of new friends, parties, and courses I enjoyed a lot. In 1968, after the Mai 68 revolution, exams were canceled in June

and pushed back to September. Soon after, I started my first year of research in a chemistry lab where smells of all kinds were striking. Serendipitously, a few weeks later, I attended the opening lecture of the biochemistry course. I had never attended a biochemistry lecture, and I had another revelation! All I had studied in chemistry suddenly was making sense. At the end of the lecture I went to see the professor, Jean Montreuil, discussed with him, and finally changed labs.

People in the Montreuil lab were enthusiastic about their work on sugars and glycoconjugates. I started to analyze thiols and disulfide bonds under the mentorship of Michel Monsigny. A new IUT (Institut de Technologie Universitaire) in applied biology was created in Lille, and I was offered a teaching position, which I enjoyed very much. I was reading a lot and started to question my research project and my current lifestyle. When M. Monsigny accepted a professorship in the French city of Orleans, I decided to radically change and go to the United States: I applied for fellowships advertised in *Chemical and Engineering News*. I was accepted at several universities and chose to go to Georgetown University, in Washington, DC, as a university fellow in the Department of Chemistry.

Georgetown University, Washington, DC (1970–1971)

The campus was beautiful. I enthusiastically discovered a way of teaching and learning so different from the French system, with smaller and more interactive classes. I chose to perform my research project in the lab of Jacinto Steinhardt, but before leaving for the United States, I had met the father of my future children! While I still wanted to obtain the master of science diploma, I knew I wanted to go back to France. I thus worked like crazy!

BIOCHEMISTRY TO MICROBIOLOGY: EARLY YEARS AT THE PASTEUR INSTITUTE (1971–1985)

PhD: Protein Sequencing, Paris (1971–1977)

A Georgetown professor once told me: "There is only one place to go back to in France: Institut Pasteur." Remember that a Nobel Prize had been awarded to André Lwoff, François Jacob, and Jacques Monod from Institut Pasteur (IP) shortly before, in 1965. IP then had numerous bright people and was very attractive for Americans. I was easily accepted into the laboratory of Georges Cohen, to whom I had said, fascinated by the work of the Nobel Prize laureate Fred Sanger, that I would love to sequence a protein. His lab was working on a bifunctional protein, the *Escherichia coli* aspartokinase I-homoserine dehydrogenase I, and by using protein chemistry had just shown that it was tetrameric. Georges Cohen liked my proposal to sequence the protein completely, which fitted with the lab's plan to determine the 3D structure of the protein.

As soon as I arrived at the lab and long before obtaining my PhD, to get a permanent post I applied to CNRS, the main research agency in France, and at Paris University—unsuccessfully! Monsieur Cohen thus organized an interview with Jacques Monod, who had just become the IP director. After an hour and a half of questions, comments on women in science, etc., Monod told me, "I'll give you the Roux fellowship from the Pasteur Institute." One year later, my position in Pasteur became permanent! Yet there was one condition: I had to do the microbiology, virology, and immunology courses. I did the first two, where I met Olivier Raibaud, Patrick Berche, and Christine Petit, who became close friends. Because I was pregnant with my first child, I never did the immunology course. Note that a permanent Pasteur position is great because it is permanent, but it does not allow you to change places to somewhere else in France like a CNRS position or an INSERM position. I never regretted having this type of position, which nevertheless meant doing some teaching and organizing some courses.

To sequence a bacterial protein, you first need to grow the bacteria and produce the protein in high amounts. I rapidly learnt how to inoculate a liquid medium. Bacteria were then grown in 300-L fermenters, and after, I was given a pellet from which I could purify the enzyme. I decided to cleave the protein with cyanogen bromide (BrCN), which cuts at methionine residues, which are rare amino acids. A Japanese postdoc had tried before me and reported that the resulting peptides were insoluble. I citraconylated the BrCN peptides, which were then soluble. I was able to separate them and started to sequence the largest one, CB1. I was really happy, but this was a very ambitious PhD project!

Fourteen months in Laos (1974–75). In France in the 1970s, men could perform their military service in former colonies. My husband was offered a professorship in Laos. Upon my request, I was offered a professorship in medical biochemistry at the Royal School of Medicine in Vientiane. Jacques Monod allowed me to leave for 14 months and put my peptides in the freezer. My second daughter was born in Laos!

When I came back, I went on to study the tryptic and chymotryptic peptides of CB1 until a special day in the autumn of 1976. Moshe Yaniv (Nesh) from the IP molecular biology department came to visit Georges Cohen and told him that at Harvard, Alan Maxam and Wally Gilbert were sequencing DNA. Nesh had with him the protocol, which was not published yet. We discussed the idea of cloning and then sequencing *thrA*, the gene encoding the enzyme I was working on. I said to Nesh: "Even though the CB1 amino acid sequence is not finished, I will write my thesis and come to your lab to set up the DNA sequencing of *thrA*." Soon after, Wally Gilbert gave a fantastic seminar at IP on their new technique and all the possibilities that it opened. A new period was starting for me.

Postdoctorate in Paris: DNA Sequencing (1978–1985)

I defended my thesis in December 1977 and crossed the street (rue du Docteur Roux!) to perform my postdoctorate in Yaniv's lab, in the "Jacques Monod" building.

***thrA*, *malA*, *Tn5*, and *crp*.** I was lucky that in the Cohen lab, Isabelle Saint-Girons was studying the regulation of threonine biosynthesis and had lambda phages carrying the *thrA* gene. She extracted the DNA from one of those phages, and we succeeded in inserting a fragment carrying both *thrA* and *thrB* into pBR322 (21). Then the big adventure started with Mikael Katinka, a student in Yaniv's lab. Sequencing meant 5′-end P^{32} labeling of DNA fragments with radioactive ATP that we were producing or buying. Then the nice partial cleavage products appeared as bands on the autoradiographs in the different lanes (G, A+G, C, C+T). This technique and this period have been important for me. Via the study of DNA, I was entering into a field that seemed so lively and rich: biology!

It had been such a pleasure when the first readable DNA sequence happened to encode "my" CB1 peptide (21)! During my third pregnancy leave, Mikael continued sequencing, and we finalized the project after a little more than two years of work. This was the first gene sequenced at IP (53). We were very proud. The *lamB* gene under study in another IP lab was published one year later. I sequenced the *thrB* gene and then moved back from Yaniv's to Georges Cohen's unit.

The label "sequencer" seemed to appear on my forehead! I helped Olivier Raibaud with the sequencing of the *malA* regulatory region, which encodes two divergent operons positively regulated by MalT (25). With Philippe Mazodier I then sequenced the central part of *Tn5*. We showed that in addition to the known kanamycin gene there were a bleomycin resistance gene and a streptomycin resistance gene silent in *E. coli* (75). I started to have the feeling that my contribution was mostly technical!

The last project for which I was contacted in the late 1970s concerned the cyclic AMP (cAMP) receptor protein CAP, encoded by *crp*. CAP is a positive regulator of genes involved in metabolism of nonglucose sugars, such as lactose. When glucose decreases, cAMP increases and binds CAP. The cAMP-CAP complex then binds DNA, attracting RNA polymerase at promoter sites for transcription of genes under its control, such as the *lac* operon. Brigitte Gicquel-Sanzey in Agnès Ullmann's lab had studied *crp* for several years and was interested to know the structure of the protein and understand the structure of a mutant whose activity did not require cAMP. I agreed to collaborate with her but was rapidly confronted by something new: competition, with Japanese scientist Hiroji Aiba. We contacted him and agreed to try to publish back-to-back. It worked (1, 20). In addition, our sequences were identical. What a relief! It was also fun to see the protein sequence fit so well with the X-ray structure predetermined by Tom Steitz and colleagues, at Yale.

DNA-protein interactions: the CAP adventure. When the *crp* sequence was completed, Brigitte Gicquel was interested in continuing our collaboration. She was a geneticist, and I was a "sequencer." We were forming a strong team. Soon after we agreed to pursue our study of the CAP protein and the regulation of its encoding gene. During a poster presentation in Boston, I was approached by Richard Ebright, a student from the Beckwith lab. Richard had isolated altered-specificity *crp* mutants and wanted to identify the mutations responsible for the altered specificity. This resulted in a productive collaboration, with a series of long phone calls! Our joint papers revealing the mutated loci and why the mutants had lost their specificity were internationally applauded (34).

The field of DNA-binding regulatory proteins was hot. The helix-turn-helix motif was established as a classic DNA-binding site in repressors. Brigitte and I contributed to this concept by reporting our analysis of DNA-binding regulatory proteins, repressors, and activators and their binding sites, in the first issue of the *EMBO Journal* (38)! We were proud of our discovery of the TVxR motif found in many of these proteins.

We were willing to extend our work on DNA-protein interactions, but the IP asked us to reorient our research toward infectious diseases. Brigitte had worked with viruses and was ready for a switch. For me, once again, this was going to be a significant change!

INFECTION BIOLOGY: INSTITUT PASTEUR (1986–2021)

As both Brigitte and I had permanent positions at the Pasteur Institute, pressure was not high to find a new orientation to our research! We spent six months visiting labs and discussing medical biology, pathogenesis, and possible research projects. I have been particularly interested in discussions with IP professor Hugo David about tuberculosis and leprosy. Both diseases were fascinating, but the bacteria seemed hopeless: greasy with complex compounds on their surface and growing slowly on strange media! One day after a seminar, we had a long chat with Patrick Berche, who was working at the Necker hospital and had studied *L. monocytogenes* during his postdoctoral work with the immunologist Robert North. Patrick told us how *Listeria* causes an interesting disease with clinical features ranging from gastroenteritis to brain infections, and maternofetal infections; that it grows fast, is not too pathogenic, and can be studied under "normal" laboratory restrictions, and that interestingly, it is intracellular and can be inoculated into mammalian cell cultures. Finally, he told us that he was trying to isolate a mutant unable to produce the classical halo detectable around *Listeria* colonies on blood agar plates. He said it would be fantastic to collaborate with us: We would bring the molecular biology and sequencing competence he did not have.

After months of discussing and thinking, Brigitte and I decided to focus on both mycobacteria and *Listeria* with the goal of understanding the mechanisms underlying their virulence and intracellular nature. We were then offered lab space by Julian Davies, who was arriving at IP in the new

building that was named “biotechno” at the time and is now called Building Fernbach. In 1986 we accepted and moved in, after obtaining funds from CNRS. Brigitte was interested to set up tools for mycobacteria and started to sequence a plasmid from *Mycobacterium fortuitum* to be used as a cloning tool. I undertook to determine the transposon insertion site in the *Listeria* nonhemolytic mutant isolated by Jean-Louis Gaillard in the Berche lab. We then formally separated our projects and I focused on *Listeria*.

The Birth of Cellular Microbiology: Listeriolysin O, Internalins, ActA

The term cellular microbiology, which we coined in 1996 (18), “describes the discipline that bridged the disciplines of cell biology and microbiology. Questions of bacterial pathogenesis started to be addressed by exploring cell biology tools while microbial pathogens started to solve questions in cell biology,” says Stanley Falkow in his preface to the book *Cellular Microbiology*, published by the American Society for Microbiology (19).

Listeriolysin O. The identification of the transposon insertion site in the nonhemolytic mutant isolated by Jean-Louis Gaillard revealed that *Listeria* expressed a hemolysin similar to that of *Streptococcus pyogenes* and that of *Streptococcus pneumoniae*. Competition (again!) started with a group in Wurzburg, Germany: that of Werner Goebel, with his postdoc Trinad Chakraborty, and to a lesser extent with another one in the United States, the group of Dan Portnoy, who at the time was in Philadelphia. A student, Jérôme Mengaud, arrived at the lab, and we succeeded in being first to determine the sequence of *hlyA*, the gene encoding listeriolysin O (LLO), and showing that *hlyA* was sufficient for the phenotype, because we could complement the mutant strain with a plasmid bearing the wild-type *hlyA* gene (81). But it was clear that the three groups had contributed to showing that the gene and the encoded protein were critical for virulence in mice and that LLO was critical for the escape of the phagocytic vacuole. I took care to always mention in my talks the names of the three groups, inasmuch as Dan soon acquired an international reputation for his discovery of *Listeria* actin-based motility and the description of the successive steps of the infection: entry, lysis of the phagocytic vacuole, actin-based motility, and cell-to-cell spread (114). Dan was giving great talks, and the pictures of the actin tails were amazing!

Together with Janet Chenevert, Eammon Gormley, and Edith Gouin, we then continued to analyze the *hlyA* region (16). We were sequencers and had noticed open reading frames upstream and downstream of *hlyA* (82). We identified upstream and in opposite orientation to *hlyA* a gene encoding a phosphatidylinositol-specific phospholipase C (PI-PLC) essential for virulence. We named it *plcA* (78). Jérôme then showed that downstream of *plcA* there was a gene responsible for expression of LLO (79). Our paper was submitted when an article describing the same results came out from the Goebel group (69). Our two groups had identified the gene encoding the major regulator of virulence, PrfA (69, 79). Interestingly, PrfA belongs to the same family of dimeric transcriptional activators with a helix-turn-helix motif as in CAP (108). Another regulator of virulence genes, VirR, was identified several years later by Pierre Mandin and Hafida Fsihi (72). The region downstream of *hlyA* encodes a metalloprotease whose substrate still remains mysterious. Later, a postdoc in the lab, Jose Vasquez Boland, sequenced this whole region (116) (**Figure 1**).

Internalins. Soon after completing the LLO story, Jean-Louis became interested in deciphering the mechanism underlying entry of *Listeria* into cells and asked to join my lab as a postdoc. I was delighted because I had in mind the beautiful paper published by Ralph Isberg and Stanley Falkow describing the role of invasin in the invasiveness of *Yersinia* (50). Jean-Louis isolated a noninvasive *Listeria* mutant, and we performed the same strategy as previously for LLO: determination of the site of the transposon insertion, cloning of the wild-type gene, and complementation of the

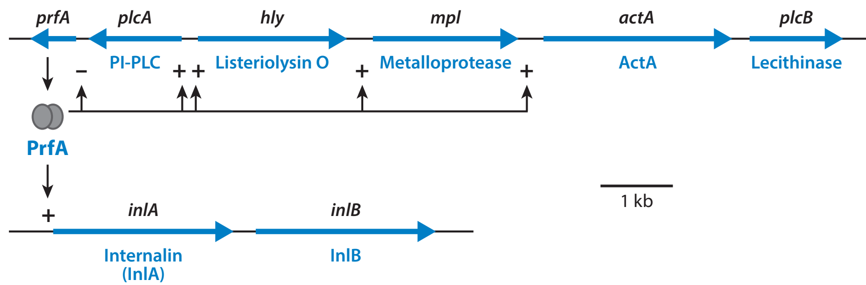

Figure 1

Schematic representation of the two *Listeria* main virulence gene loci regulated by PrfA (adapted with permission from Reference 64). Abbreviation: PI-PLC, phosphatidylinositol-specific phospholipase C.

noninvasive mutant to restore the invasive phenotype. In an elegant experiment that was key for the paper, we expressed the internalin gene in the noninvasive species *Listeria innocua* and found that *L. innocua* became invasive (37). Internalin is a protein with a series of leucine-rich repeats. I had noticed a hydrophobic tail and proposed it would help anchor the protein in the bacterial membrane. Interestingly at the American Society for Microbiology's general meeting, an abstract from the Fischetti group on the M protein of *Streptococcus pyogenes* later published in *Molecular Microbiology* (36) described a hydrophobic region preceded by an LPXTG motif in the C terminus of the M protein. Strikingly, internalin also had an LPXTG motif. I visited the Fischetti lab and discussed this with postdoctoral fellow Olaf Schneewind. Clearly M protein and internalin had a similar anchor in the cell wall, allowing them to be present on the bacterial surface, a property important for their function, as shown by Maryse Lebrun in the case of internalin (63). Later we collaborated with Olaf Schneewind on the role of sortases in the cell wall anchoring process (8).

Then emerged the fact that *inlA*, the internalin gene, is part of a family of genes that encodes proteins containing leucine-rich repeats (30). The gene next to *inlA*, *inlB*, also encodes a leucine-rich repeat protein of the internalin family that was shown by Shaynoor Dramsi and Laurence Braun to be critical for entry into hepatocytes (29). It does not have the LPXTG motif, but large repeats allow binding to the cell wall (11) (**Figure 1**). A third internalin protein named InlC, which is secreted inside cells, was later shown by Edith Gouin to interact with the kinase IKKα (which normally phosphorylates the inhibitor of NF-κB) and to prevent the activation of the NF-κB transcription factor–mediated innate immune response (40, 41). InlC, as shown by Keith Ireton, also promotes cell-to-cell spread by interacting with Tuba and relieving cortical tension (94). Other internalin proteins present in *L. monocytogenes* and absent in *L. innocua* were studied later (see below).

ActA. When Christine Kocks joined the lab, I proposed to her to analyze whether internalin was involved in cell-to-cell spread by testing the internalin mutant in a cell-to-cell spread assay. I had together with Edith Gouin identified, in a bank of mutants that we had constructed, a mutant that had a phospholipase-negative phenotype: In contrast to the wild type, this mutant did not produce a halo on egg yolk agar. I asked Christine to test this mutant also. Two days later, she asked me to come to the microscope. The phospholipase mutant was making intracellular colonies while the wild-type bacteria were present at the tip of long protrusions invading the neighboring cells (**Figure 2**). We were really excited. To make a long story short, we had identified the gene responsible for actin-based motility, which we named *actA*, that was upstream from a gene encoding a protein easily identified as a lecithinase on the basis of protein sequence homology (55). A polar effect of the transposon inserted in *actA* was inhibiting expression of the lecithinase gene that we

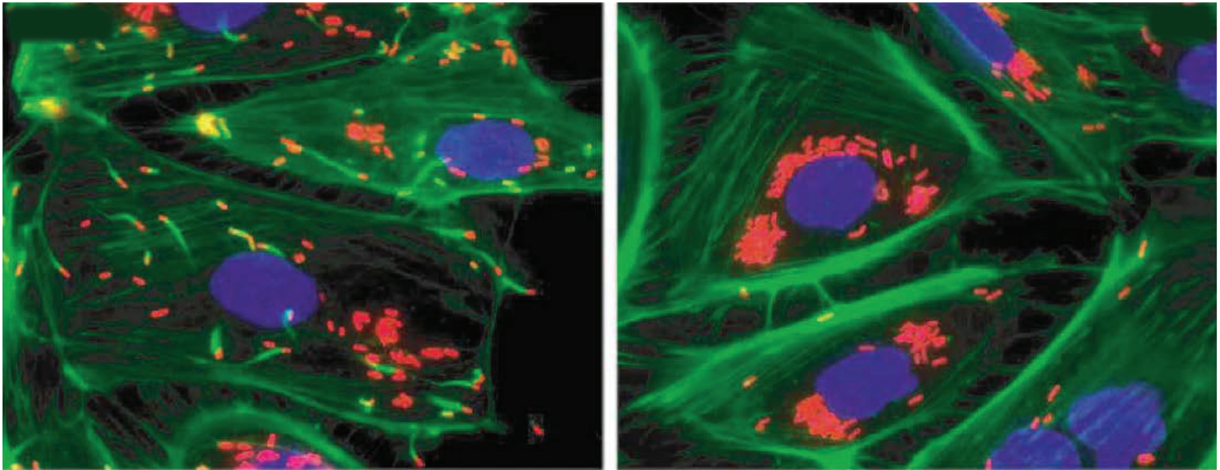

Figure 2

Mammalian cells infected by *Listeria* wild type (*left*) or the *actA* deletion mutant (*right*). Infected cells were fixed and labeled with DAPI (*blue*) to label DNA in the nucleus and in bacteria, FITC phalloidin (*green*) to label actin and anti-*Listeria* antibodies (*red*) for fluorescence analysis. The *actA* mutant forms microcolonies inside cells. Adapted with permission from Reference 44.

named *plcB*. We showed that a *plcB* mutant still made actin tails and was involved in the lysis of the two-membrane vacuole after cell-to-cell spread (55) (**Figure 3**).

The discovery of ActA was like a tsunami propelling *Listeria* (and myself as a speaker!) in many cell biology and cytoskeleton meetings. Yet the way ActA induced motility remained unknown. That it was polarly distributed on the bacterial surface made sense (56), but the next step in understanding the phenomenon was reached only when Matt Welch used ActA to search for potential ligands and identified the Arp2/3 complex (119). This complex is made of seven proteins, two of them being closely related to actin (Arp is named for actin related proteins). The association of Arp2/3 complex with ActA was critical for actin-based propulsion. We expressed *actA* in *L. innocua*, which was then able to make actin tails in cell extracts or in macrophages where entry was possible without internalin (57). We performed this experiment in parallel to expressing in *E. coli* the gene *icsA*/*virG*, which had been identified as responsible for the actin-based motility of *Shigella* by two groups, that of Sasakawa (71) and that of Sansonetti (5). Iñigo Lasa meticulously dissected ActA and identified its critical parts for the whole process (60, 61). The discovery of ActA paved the way for many studies on actin-based propulsion of bacteria (44) and of other cells as well.

Reading about actin led me to suspect that vaccinia virus could make comet tails. I asked Pasteurian virologists to infect cells with the virus. Labeling the infected cells with FITC phalloidin revealed that vaccinia virus produced actin tails. I was not planning to extend my research to viruses and contacted Gareth Griffiths at the European Molecular Biology Laboratory (EMBL), Heidelberg. Gareth and his postdoc Michael Way enthusiastically decided to work on vaccinia virus: This has been a success (23). I was at the time interested by another bacterium, *Rickettsia conorii*, described as making long actin tails (46). We labeled the actin filaments with myosin S1 to analyze the tails and the actin filaments in the tails by electron microscopy. The paper comparing *Listeria*, *Shigella*, and *Rickettsia* tails was well received (43). It demonstrated that different types of actin polymerization can take place, since *Rickettsia* produces longer filaments than *Listeria* and *Shigella*. Later we showed that *Rickettsia* has an ActA homolog called RickA (42), but as shown by Matt Welch, RickA is necessary for the first phase of the process, and a formin-like protein then induces formation of the long actin filaments (97).

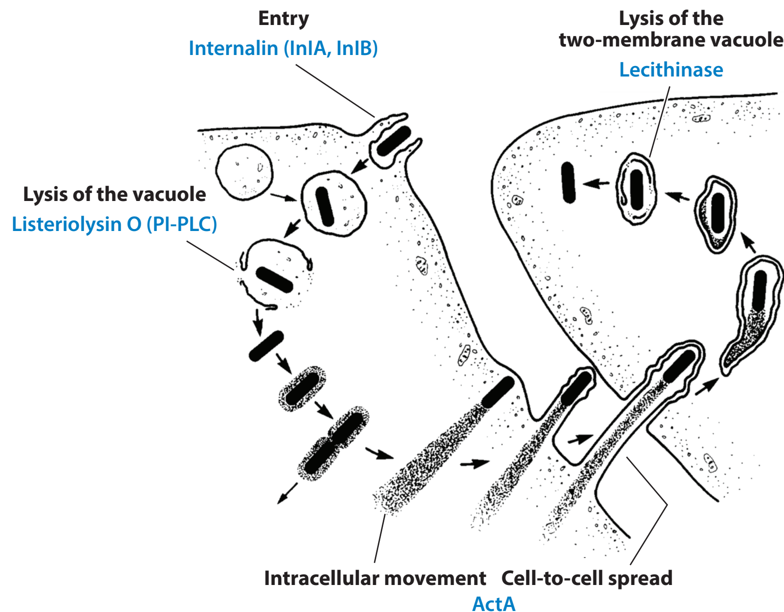

Figure 3

Schematic representation of the cell infectious process by *Listeria monocytogenes*. The bacterial factors involved are indicated in blue (adapted with permission from Reference 114). Abbreviation: PI-PLC, phosphatidylinositol-specific phospholipase C.

Javier Pizarro-Cerdá showed that septins were associated with InlB-phagosomes but not InlA-phagosomes (90). With Serge Mostowy, we proposed that this particular class of GTPases should be considered the fourth component of the cytoskeleton, since similar to actin, tubulin and intermediate filament septins polymerize into filaments (85). Serge demonstrated that some septin isoforms are involved in entry but, more interestingly, that they are in most cases associated with actin and, spectacularly, they cage *Shigella* actin tails—but not *Listeria* actin tails—and prevent them from spreading from cell to cell (84). Septins contribute to many processes including mitochondrial apoptosis and protection against infections.

Internalin Receptors and Signaling Pathways Leading to Entry

Jérôme made an important discovery when he came back from his postdoc work. Using an affinity approach, he identified the internalin receptor as E-cadherin, a transmembrane protein well-suited to allow entry into cells. In particular, the intracellular part of E-cadherin binds catenins, which are connected to the cell cytoskeleton. By using *L. innocua* expressing internalin, Jérôme showed that fibroblasts that express E-cadherin are permissive while those which do not express E-cadherin or express another cadherin are not permissive (80). Interestingly one or two years prior to the discovery of the receptor, I attended a seminar by Rolf Kemler and I remember telling him, "This would be a perfect receptor for *Listeria*!"

A receptor for InlB, gC1qR/p32, which is the receptor for the globular part of the complement component C1q, was identified by Laurence Braun (12). When gC1qR is expressed in cells that are not permissive to *Listeria* via the InlB pathway, it allows entry with tyrosine phosphorylation of Cbl, Gab1, and Shc, as in permissive cells. A second receptor was identified by Keith Ireton. It is the HGF receptor, or Met, a protein present in many cell types (109). This result was in line with

E-cadherin: transmembrane protein expressed on the surface of epithelial cells that allows homophilic E-cadherin–E-cadherin interactions and formation of an epithelium

the discovery made by Keith when he arrived at the lab, i.e., that the PI3 kinase pathway—which is known to be triggered by growth factors—is activated by InlB for entry into cells (49).

The lab spent years deciphering the numerous main actors involved in entry, the critical cytoskeletal proteins and signaling events taking place, including posttranslational modifications (PTMs) of the receptors or associated proteins. A series of postdocs and students, in particular Keith Ireton (49), Hélène Bierne (8), Javier Pizarro-Cerdá and Andreas Kühbacher (58, 59), Matteo Bonazzi (9, 10), Stephanie Seveau (106, 107), Sandra Sousa (110), and Esteban Veiga (117, 118), contributed to understanding and visualizing how the bacterium *Listeria* takes advantage of cellular pathways for efficient entry. Esteban and Matteo demonstrated that clathrin is involved in phagocytosis, which was against the dogma that clathrin is solely used for internalization of tiny particles or viruses (9, 117, 118). Cell biologists were really surprised!

Genome-wide small interfering RNA screens performed by Javier and Andreas identified complementary signaling pathways involved in infection and revealed different actin nucleation mechanisms during cell invasion and comet tail formation. Moreover, unexpectedly, different Arp2/3 variant complexes regulate these different processes (59, 89).

Ascel Samba-Louaka demonstrated that during infection, *Listeria* dampens the DNA damage response (104). Together with Juan Martinez, we started to investigate the early signaling events of the entry of *R. conorii* into mammalian cells and identified one receptor (73, 74).

Crossing of Host Barriers by *Listeria*: The Gastrointestinal and Fetoplacental Barriers

Marc Lecuit discovered that while *Listeria* enters into human epithelial cells, it does not enter into mouse epithelial cells, which have an E-cadherin similar to that of human cells. Marc then showed that one amino acid at position 18 is responsible for this specificity (65). Later, Nadia Khelef together with Marc showed that InlB also displays species specificity (54).

Generation of a transgenic mouse line expressing human E-cadherin in the intestine in collaboration with Charles Babinet allowed us to demonstrate that in this mouse line, *Listeria* can cross the intestinal barrier and eventually kill, providing for the first time an oral model for the human disease, which also was very instrumental to deciphering how bacteria target goblet cell E-cadherin to cross the intestinal barrier (68, 86).

Understanding the crossing of the fetoplacental barrier required another animal model closer to humans for the placenta. As shown by Marc, gerbils are such animals and have an E-cadherin that allows entry of *Listeria* into epithelial cells. By a series of experiments Marc showed that crossing of the fetoplacental barrier requires both the InlA and the InlB pathways (27, 66).

The Genomes of *L. monocytogenes* and *L. innocua*: Genomics and Postgenomics Studies

In 2001, we published the sequences of *L. monocytogenes* and *L. innocua* (39). In that aim, I coordinated the consortium financially supported by the European commission. I had planned to sequence the *Listeria* genome for many years (83). It was obvious for me that this would be necessary and possible. I had wanted to sequence the genomes of both species, the pathogenic and the nonpathogenic, in order to make comparisons and detect genes critical for virulence. We had in the past shown that the *hlyA* locus (16), which is surrounded by the *prfA*, *plcA* operon and the *mpl*, *actA*, *plcB* operon, and the *inlA*, *inlB* locus are absent in *L. innocua*, predicting that there might be other loci contributing to virulence that would be present in *L. monocytogenes* and absent in *L. innocua*. This has been the case (39). Philippe Glaser and Carmen Buchrieser played a key role in this project!

The postgenomics period started in the lab. Olivier Dussurget first showed that a bile salt hydrolase gene is present solely in *L. monocytogenes* and contributes to the persistence of *L. monocytogenes* in the gut by counteracting the action of bile (32). We and others showed that InlC is a secreted protein of the internalin family absent in *L. innocua* and induced intracellularly via PrfA (40). Several surface proteins present only in *L. monocytogenes* were then identified by Hélène Bierne, Didier Cabanes, Laurent Dortet, Marie-Anne Nahori, Nicolas Personnic, and Christophe Sabet, and were shown to contribute to virulence, e.g., InlH, InlJ, InlK, Vip, Auto (13, 14, 28, 88, 102, 103).

One of the most unexpected factors identified by comparative genomics, i.e., present only in *L. monocytogenes*, and contributing to virulence was discovered by Hélène Bierne and Alice Lebreton (62). It is a secreted protein named LntA that targets the nucleus and interacts with a protein named BAHD1, which we showed is involved in heterochromatin formation. LntA desequesters BAHD1 and allows expression of type III interferon and of interferon-stimulated genes (ISGs). Interferons are secreted proteins of the cytokine family that regulate innate and adaptive immune responses to infection. They are produced by infected cells, bind to receptors on many other human cells, and trigger signaling cascades that lead to the production of many ISGs involved in anti-infection functions. The importance of interferons and ISGs in antiviral response has long been appreciated. However, their role in bacterial infections, and in particular in *Listeria* infections, is more complex and is currently a major focus of investigation (31).

As other proteins from human or plant bacterial pathogens, such as LntA, have been shown to target the nucleus and interfere with gene expression, we proposed in a review to call this family of proteins nucleomodulins (6). Olivier Dussurget identified the second *Listeria* nucleomodulin: it is OrfX encoded downstream of *plcB* (91). It targets RybP in the nucleus and decreases its level and dampens the oxidative stress response of infected macrophages, allowing evasion of macrophage oxidative stress and bacterial multiplication.

The second *L. monocytogenes* protein absent in *L. innocua* with a quite novel type of function was recently discovered by Alessandro Pagliuso, who identified in *Listeria* the first known secreted bacterial RNA-binding protein and named it Zea, after a Greek goddess (87). Zea associates extracellularly with a subset of RNAs. During infection, Zea binds RIG-I and potentiates the type I interferon response. This study demonstrated that bacterial RNAs can act as social RNAs that trigger a host response during infection.

Transcriptomics, RNA-Mediated Regulation, Riboswitches, and Small Open Reading Frames

The regulation of most *Listeria* virulence factors is coordinated by PrfA. We realized that virulence factor expression is maximal at body temperature but low at low temperatures. What about PrfA itself? Adriana Renzoni figured out that at low temperatures the mRNA is expressed but the protein is not made (98). Jörgen Johansson and Pierre Mandin then demonstrated that an RNA thermosensor is responsible for lack of translation at low temperatures and high expression at high temperatures. This result and the published paper reporting a series of complementary elegant experiments were well received (52).

After the genome determination and the discovery of the thermosensor, I became increasingly interested in small RNAs and RNA regulation. In a study led by Jörgen Johansson and Edmund Loh, we showed that the small transcript resulting from an arrest in transcription in riboswitches can act as a noncoding RNA and inhibit gene expression by hybridizing to an RNA region critical for expression (70).

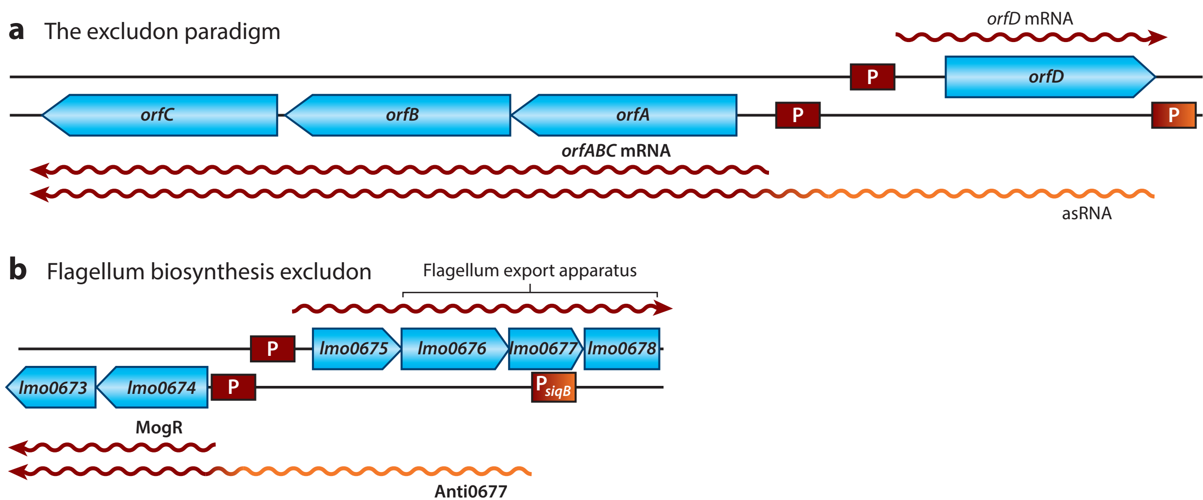

Figure 4

Schematic representation of an excludon. (*a*) The excludon paradigm. (*b*) The flagellum biosynthesis excludon. Abbreviation: asRNA, antisense RNA. Adapted with permission from Reference 105.

We then had Affymetrix arrays produced for us. With Alejandro Toledo-Arana, we analyzed the complete *Listeria* transcriptional landscape after bacterial growth in a variety of conditions and with several mutants (115). Based on this analysis, we drew the operon map of the genome (the first ever published for a bacterium) and identified a series of small RNAs. We have identified open reading frames for some and noticed that others could be part of riboswitches.

A productive collaboration with Rotem Sorek started in 2010. We mapped all the transcriptional start sites after bacterial growth in conditions identical to those used in the array study (121). This study led to new discoveries: (*a*) Together with Nina Sesto we discovered what I named excludons (I love this neologism!), a new concept in bacterial antisense regulation (105). An excludon defines a genomic locus encoding an unusually long antisense RNA (asRNA) that spans divergent genes or operons with related or opposing functions. Because these asRNAs can inhibit the expression of one operon while functioning as an mRNA for the adjacent operon, they act as fine-tuning regulatory switches in bacteria (**Figure 4**). (*b*) With Jeff Mellin we discovered that noncoding RNAs can be regulated by riboswitches. The first B12 riboswitch–regulated noncoding RNA identified was an asRNA precluding the expression of a pleiotropic regulator of propanediol utilization in the gut (77). The second was a small RNA, also B12 riboswitch–regulated, able to sequester a two-component response regulator (76). Then Rotem's group invented a technique to map precisely the 3′ end of RNAs. This technique coupled with the transcriptional start site determination technique allowed our groups to identify several riboregulators controlling antibiotic resistance (24). In the first example the gene involved in antibiotic resistance encoded a special type of ABC protein involved in desequestering the antibiotic from the ribosome. In the second, we highlighted with Mélodie Duval a new mechanism in which the antibiotic resistance relies on the protein HflX, which is able to dissociate and recycle ribosome subunits (33). The same mechanism was later discovered in mycobacteria (101).

After the transcriptome, the translatome! Using N-terminomics, Francis Impens identified a large number of N termini and a never described small protein called prli42 present in other gram-positive bacteria (48). prli42 acts as a link between external stresses, the stressosome and the σ^B

regulon. The stressosome is a big nanomachine that Allison Williams analyzed by cryo–electron microscopy (120). Precisely how signaling occurs still requires investigation.

All the omics studies in the lab increasingly required bioinformatic analyses, which were performed by Christophe Bécavin (3, 4, 48, 77). Pierre Dehoux also contributed (11, 13, 30, 32, 42) and unexpectedly discovered that salmolysin from *Salmonella* is in fact a regulatory protein (26).

Histone Modifications and Epigenetic Modifications Upon Infection

Infections reprogram gene expression in infected cells, in vitro as well as in vivo. Marc Lecuit first showed using the transgenic mice expressing human E-cadherin that in the intestine, LLO is the key determinant of the host response to *Listeria* (67). How this occurs is unknown. Changes in gene expression involve transcriptional regulators and chromatin modifications. As viruses manipulate histones for infection, I was interested in analyzing what happens during the course of a *Listeria* infection and when histone modifications also occur. Mélanie Hamon accepted this new project for the lab. She showed that LLO induces dephosphorylation of histone H3 Ser-10 as well as deacetylation of H4 during the early phases of infection, leading to downregulation of genes involved in the immune response of the host (45). She next demonstrated that H3 at Lys-18 is deacetylated by Sirtuin 2, which shuttles to the nucleus and associates with chromatin once dephosphorylated on Ser-25. This mechanism relies on the InlB Met interaction and leads to repression of a number of genes. Interestingly SIRT2-deficient animals show decreased bacterial loads in liver and spleen, revealing that *Listeria* requires SIRT2 activity for full virulence (35). Both Melanie's and Hélène's projects on LntA and histone modification opened the field of epigenetics and bacterial infections (7). The field of epitranscriptomic modifications in mammalian cells upon bacterial interactions was opened by Sabrina Jabs (51). Indeed Sabrina showed that presence of the microbiota or bacteria in the gut leads to mRNA m^6A modifications in the cecum and in the liver. *Akkermansia muciniphila* and *Lactobacillus plantarum* affected specific m^6A modifications in mono-associated mice. Whether pathogens induce specific modifications is a field fully open for investigations!

Mitochondria and Infection

Mitochondria are essential organelles, providing most cellular ATP and several biosynthetic intermediates. They constantly undergo fusion and fission and are important integrators of signaling cascades. Fusion and fission of mitochondria regulate their size and subcellular distribution and reflect their functional state. When I discovered this dynamic behavior, at a conference, I was fascinated and wanted to investigate this phenomenon during infection. Esteban Veiga performed a preliminary experiment showing that *Listeria* induces rapid mitochondrial fragmentation upon infection. Fabrizia Stavru accepted this postdoc project and demonstrated that fragmentation is transient, mediated by LLO and critical for infection (112). She then showed with Amy Palmer that this fragmentation is atypical and does not depend on the classical fission player Drp1 but relies on mitochondrial contact points with the endoplasmic reticulum and actin polymerization (113). Further experiments carried out with Filipe Carvalho highlighted a role for Mic10, a component of the mitochondrial contact site and cristae organizing system (MICOS) complex, which is significantly enriched in mitochondria isolated from cells infected with wild-type but not LLO-deficient mutant. Mic10 is necessary for fragmentation independently of MICOS proteins Mic13, Mic26, and Mic27. This study was the first to show a role for Mic10 in mitochondrial fission (15). Fabrizia's student Anna Spier then showed that mitochondrial respiration modulates *L. monocytogenes* entry by limiting endocytic recycling of receptors such as c-Met back to the plasma membrane, leading to decreased bacterial load in cells with high respiratory activity (111). Many groups are now interested in microbes-mitochondria interactions.

Posttranslational Modifications During Infection: SUMOylation, ISGylation

PTMs tune protein functions and in most cases are reversible. Olivier Dussurget and Cristel Archambaud showed that activity of *Listeria* superoxide dismutase is downregulated by phosphorylation and that the active nonphosphorylated form of superoxide dismutase is secreted via the secA2 pathway, a mechanism that deserves more investigation (2). Phosphorylation modifies many sites in cells. However, while phosphorylation and also ubiquitination successively modify the *Listeria* receptors as a prelude for bacterial entry, as shown by Matteo Bonazzi (10), other PTMs also contribute to the host response to infection. Counteracting these PTMs may be a way for bacteria to control infection.

SUMOylation is involved in transcription regulation, stress, and other responses. Similar to the case of ubiquitin, conjugation of SUMO on a target protein involves an E1, an E2, and an E3 enzyme. David Ribet showed that *Listeria* infection leads to the degradation of Ubc9, the unique SUMO E2 enzyme, as well as that of several SUMOylated proteins (including PML, the promyelocytic leukemia protein), leading to a global decrease in the levels of SUMO-conjugated host cellular proteins (47, 99). Deletion of PML leads to extreme susceptibility to infection. Overexpressing SUMO counteracts infection, revealing that SUMOylation is critical for infection. Other investigations of SUMO and bacterial pathogens were then undertaken.

ISG15 is an interferon-stimulated linear di-ubiquitin-like protein with antiviral activity. Its role in bacterial infection had never been investigated. Liliana Radoshevich showed that ISG15 expression in nonphagocytic cells is dramatically induced upon infection (96). It is type I interferon independent and depends on the cytosolic surveillance pathway that senses bacterial DNA and signals through STING. Most importantly ISG15 expression restricts *Listeria* infection in vitro and in vivo. Endoplasmic reticulum and Golgi proteins are ISGylated upon infection, which correlates with increased secretion of cytokines known to counteract infection. This study reinforced the view that ISG15 is a key component of the innate immune response.

Bacteriocins and the Entry of *Listeria* in the Microbiome Era

Listeria is a foodborne pathogen, and it is expected that *Listeria* infection is influenced by the gut microbiota or probiotics present in dairy products. Cristel Archambaud performed a comprehensive analysis of the impact of two *Lactobacillus* species on *Listeria* infection in a gnotobiotic humanized mouse model and showed that treatment with each *Lactobacillus* species induced a significant decrease in infection and in host gene expression, in particular decrease in expression of ISGs and of several microRNAs (3). It reshaped the *L. monocytogenes* transcriptome and upregulated transcription of genes encoding enzymes allowing utilization of intestinal carbon and nitrogen sources, e.g., genes involved in propanediol and ethanolamine catabolism and cobalamin biosynthesis. Interestingly, as shown by Cristel, the intestinal microbiota interferes with the response of microRNAs to oral *Listeria* infection (4).

Among the three identified *L. monocytogenes* evolutionary lineages, lineage I strains are overrepresented in listeriosis outbreaks, but the mechanisms underlying the higher virulence potential of strains of this lineage had remained elusive. Javier Pizarro-Cerdá revisited a result from Colin Hill and discovered that listeriolysin S (LLS), a virulence factor present only in a subset of lineage I strains, is a bacteriocin highly expressed in the intestine of orally infected mice that alters the host intestinal microbiota and promotes intestinal colonization by *L. monocytogenes* as well as deeper organ infection (92, 93). These results identified LLS as a bacteriocin in *L. monocytogenes*, the first to be described, and associated modulation of host microbiota by *L. monocytogenes* epidemic strains with increased virulence.

Nathalie Rolhion identified a second bacteriocin while studying a gene present in *L. monocytogenes* and absent in *L. innocua* (100). This 107–amino acid protein is similar to lactococcin 972 secreted by *Lactococcus lactis* and putative bacteriocins of pathogenic bacteria, including *Streptococcus iniae*, *Streptococcus pneumoniae*, and *Staphylococcus aureus*. Nathalie showed that deleting the gene affected mice that were orally infected but not if the mice were germ-free, clearly demonstrating a role of the bacteriocin in the presence of a microbiota. By analyzing the composition of the intestinal contents after oral infection with wild-type *Listeria* or the deletion mutant, Nathalie showed a specific effect of the bacteriocin on *Prevotella copri*, a result confirmed by incubating a series of commensals with a synthetic bacteriocin. Only *P. copri* was targeted by the bacteriocin. The mechanism underlying this specificity is still unknown!

COMMENTS

Competition

I had two episodes of strong competition, but in each case, thanks to discussions, we came to an agreement. In the end my competitors became friends. In the first case, we agreed with Hiroji Aiba to send our *crp* sequences to the same journal on the same day, and our papers were published back-to-back (1, 20)! When sequencing the *hlyA* gene of *Listeria*, we agreed with T. Chakraborty that the first group ready to publish would inform the other—which I did!

I think it is important to give credit to those who have obtained results in closely related areas. I have always tried to do that, in particular when giving talks.

Organizing Meetings and Courses

I have always believed that live discussions between scientists lead to the emergence of new ideas. I also think that it is easier (at least for me) to listen to a talk on a new topic than to read about it. Therefore, I have been involved in organizing many unit and departmental seminars and meetings, in particular EMBO workshops, and even meeting series. The INSERM Philippe Laudat conference I organized in Aix les Bains with Rino Rappuoli, Patrice Boquet, and Staffan Normark in 1989 to gather microbiologists and cell biologists was particularly important. The *Science* editor who was present asked us to write a report on it and to coin a name for the emerging discipline that was the topic of the meeting (18). "Cellular microbiology" was immediately accepted, and the American Society for Microbiology then asked us to edit a book, which became popular for teaching!

Every year, P. Sansonetti and I, from Institut Pasteur, and colleagues from the Max Planck Institute for Infection Biology, Berlin (T. Meyer, Stefan Kaufmann, and Arturo Zychlinsky); from Imperial College London (David Holden); from GSK, Siena (Rino Rappuoli); and from the Karolinska Institute, Stockholm (Birgitta Henriques-Normark and Staffan Normark), gathered with young scientists from our five research centers in the framework of what we called the European Initiative for Basic Research in Microbiology and Infectious Diseases (EIMID). Meetings were every year in a different country. It has been a lot of fun and important for promoting European infection biology, including cellular microbiology!

Personally, I went to many other meetings: I particularly liked Gordon Research Conferences, where I could meet my transatlantic friends Jorge Galan, Brett Finlay, Ralph Isberg, John Mekalanos, Stanley Falkow, Jeff Miller, Sam Miller, Pam Small, and Dan Portnoy, among many others, and also Europeans like Agnès Labigne, Philippe Sansonetti, Gisou van der Goot, Staffan and Birgitta Normark, David Holden, Guy Cornelis, Rino Rappuoli, Cesare Montecucco. . . .

Taking over the organization of the famous international summer course previously led by Marianne Grunberg-Manago on the island of Spetses, Greece, has been great: During 20 years

my friend Roberto Kolter and I have had the immense pleasure to host students and lecturers in an unusual and magical format: enthusiasm of both students and lecturers never decreased (22)!

Being a Woman in Science

Being a woman in science (and a mother of three) in France, at the Pasteur Institute, has not been particularly difficult. My children went to the crèche the whole day when they were very young! I was living close to the Institut Pasteur. I also had some help from an aunt living close by. In fact I had my children when I was relatively young and started to travel a lot when they were already quite grown up.

It is important to note that I early on accepted giving some of my time for the community and taking positions of responsibility, including chair of the *commission de classement*, director of department, and president of the scientific council (twice). I sat on many international scientific advisory boards, panels, and important awards committees or trustee boards, where I learnt a lot. Ironically though, I was never asked to be a consultant for a big pharmaceutical company or even a start-up!

FINAL THOUGHTS AND ACKNOWLEDGEMENTS

I have accomplished my scientific career at a fantastic time. A new phase in the study of host-microbe interactions was exploding when I started to work on *Listeria* in the late 1980s, and I have tried to contribute as much as possible. I do think that we have raised *Listeria* to the rank of a model system (17, 95) and that several of our initiatives or new research themes have inspired scientists working with other microbial pathogens.

Of course, all this would not have been possible without all the fantastic collaborators I have had through the years. I could not cite all of them—in particular students who were working under the supervision of postdocs and senior scientists in the lab. I would like to thank all of my postdocs and students for their hard work and dedication. I truly enjoyed working with each and every one of them. I have tried to provide them with a well-funded lab, thus facilitating expensive or risky experiments. It has been a real pleasure and honor to become an international scholar of HHMI (Howard Hughes Medical Institute) and then to receive two successive ERC (European Research Council) grants.

I also thank my secretary, Marie-Thérèse Vicente, who has done so much work for the Unité des Interactions Bactéries-Cellules.

I am happy that most of my former collaborators are in academic positions, in most cases as group leaders at IP-Paris, or elsewhere in France (e.g., Montpellier, Rouen, Grenoble, Jouy en Josas, Poitiers), or in Spain, Portugal, Belgium, the United States, the United Kingdom, New Zealand, or elsewhere in the world. It was fun to welcome Gunnar Lindahl, Joel Swanson, Nicole King, and Amy Palmer for short sabbaticals. I wish we had more senior visitors. I give a special thanks to Daniel Louvard for his enthusiasm and his support to microbiologists interested in cell biology in the early nineties.

I also thank all those (editors and authors) who contributed to the two special issues of *Cellular Microbiology* and *Molecular Microbiology* published in my honor in 2020. It was so nice and so unexpected!

I would also like to acknowledge those who supported me and nominated me for elections or awards. I am aware that I have been the laureate of many distinctions, again because I had fantastic collaborators.

Closing my lab in 2020 when I reached the mandatory retirement age was terrible! I had thought that being elected at the prestigious, important, and interesting position of Secrétaire

Perpétuel at the French Academy of Sciences in 2016 would compensate my loss of a lab; it did not. I thus decided to complete my 6-year mandate as Secrétaire Perpétuel and to finish my scientific career as a visitor at EMBL, Heidelberg. I truly thank Nassos Typas and Edith Heard!

In finishing this article, I have very special thoughts for two extremely bright colleagues, Fabrizia Stavru and Hélène Bierne, who both tragically passed away much too early, in May 2021 and August 2022, respectively. As do all of their family members, friends, and colleagues, I miss them a lot.

Finally, I sincerely thank Mélanie Hamon, who agreed to read this article and provide me with comments and advice that I followed in most cases but not all!

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