

Serendipity

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As I look back, I realize that serendipity has played a major role in my life. I grew up in Santiago, Chile. As far back as I can remember I was interested in plants and animals, enjoying my Aunt Olga's farm, where I could observe the reproduction of rabbits and crossed plants of different colors. When I was a teenager a devastating earthquake in the South killed an aunt and two infant cousins who happened to be at the epicenter just for one night. That unfair tragedy convinced me that life had been created by natural forces, and the way to prove it was by synthesizing a living cell *in vitro*. I wanted to be a scientist and decided that I would direct my efforts toward that aim. My bad grades in high school on all the subjects that required memory, except biology and math, almost prevented me from being accepted to the Faculty of Chemistry and Pharmacy. The Faculty of Biology and Medicine was out of my reach. During my college years I read everything I could find about the biology of cells and decided that I should first study the simplest of all self-reproducing organisms, namely viruses. To get my degree I had to work on a research problem, and by luck I read a paper that W. M. Stanley had published on the crystallization of tobacco mosaic virus (TMV) (1). I asked the Director of the Department of Viruses at the Instituto Bacteriologico de Chile that was dedicated to growing viruses and producing vaccines if I could work on my thesis there and in return help with the work. I repeated Stanley's procedures, growing *Nicotiana tabacum* for infection and isolation of TMV and *Nicotiana glutinosa*, which produces single lesions, for quantifying the virus, and obtained beautiful pictures of the crystals (2). At that time, the fact that a self-reproducing entity could be just a molecule was of great philosophical discussion and reassuring to me.

In 1946, I applied to the Institute of International Education for a scholarship to continue my studies on the chemical nature of viruses in the United States. Serendipity led me to Washington University in Saint Louis, Missouri where the Bacteriology Department had an instructor, A. D. Hershey, who was teaching such a course (Fig. 1). When I arrived, the Director, Dr. Bronfenbrenner, informed me that because of post-war constraints the course had not been scheduled. He suggested that I should take the Medical Bacteriology course instead and perform research with him. The project consisted of purifying bacteriophage to determine its phosphorus content. I was the third student that was assigned to that task because the previous ones had found phosphorus and according to Bronfenbrenner the phage had only protein and the presence of phosphorus indicated contamination. I worked hard to purify the phage through more procedures than necessary and, of course, I found phosphorus. I was truly desolate thinking that my year in the United States was a flop. Then, Hershey took pity on me and decided to give a special course on phage the next semester for the 7 graduate students. I will never forget that course because he used phage as units of infection that could demonstrate the quantitative aspects of biological problems. In 1950 when Hershey moved to the Carnegie Institute in Cold Spring Harbor he became famous for the "phage course" that he continued to give every summer. Hershey received the Nobel Prize in 1969, together with Delbruck and Luria, for showing that only DNA is introduced into the cell upon infection. At the end of the academic year Hershey asked me if I wanted to be his assistant for a project he was just starting. I was delighted and asked permission from the Institute of International Education to stay longer, which was approved. This opportunity was invaluable to me because Hershey had a unique mind for performing research such as I have never seen in other



FIGURE 1. Al Hershey, Raquel Sussman, and Al's son Peter. Phage meeting, Cold Spring Harbor, NY, 1976.

scientists. For example, when I asked him a question about the results that I was getting in phage crosses he did not respond right away, but the next day he gave me 3 type-written pages with an analysis of the question. He described the different results that were possible to get from that cross, how each result should be interpreted, and what experiments could be performed to discriminate among the possible interpretations. His ego was completely detached from the importance of the experiments that we were doing, namely showing for the first time that there is genetic exchange (DNA recombination) of the virus when it is reproducing inside the bacterium (3, 4). Unfortunately, after 5 months of work the Immigration Department sent me a serious letter informing me that if I did not leave the United States immediately they would deport me in 3 weeks because I had a student visa and was not supposed to work as an assistant. I was forced then to leave in February.

Another serendipitous event that year was meeting Maurice Sussman, who was Sol Spiegelman's graduate student. He was very intelligent and totally engrossed in research, and we became inseparable. In 1948 I came back to the United States to marry him. I also became Spiegelman's graduate student.

At that time, performing science with Sol was another unique experience. He was only a few years older than us with not many more years of scientific experience but talked as an old pro. He always had one problem dominating his mind and thought about it 18 h a day. We had coffee breaks every 4 h to talk about experiments and we worked until 11 p.m. The problem that he assigned to me was *Saccharomyces chevalieri*'s "long term adaptation" to galactose utilization (5). This strain of yeast required days of contact with galactose before the population became positive, in contrast with other strains of yeast that adapted in a few hours. I was supposed to find out its

mechanism. Sol already had a working hypothesis that this strain had "plasmagenes" that controlled the synthesis of the new enzymes, and he expected me to prove it. The first possibility was that a few cells in the growing population spontaneously mutated to gal⁺, followed by selection. I used the statistical analysis of Luria and Delbruck (6) to determine whether this was the mechanism. My results clearly showed that it was a massive population change requiring the continuous presence of galactose. At that time there were no available techniques to determine whether it was a cytoplasmic or a nuclear phenomenon. Nevertheless, Sol published my results as: "On the cytoplasmic nature of long term adaptation in yeast" (7). It took 47 years to shed light on that phenomenon; a paper published in 1997 (8) showed the "two-step model" of *gal* operon induction, involving three specific interacting proteins necessary to start transcription of the *gal* promoter in the nucleus. Later Sol contributed significantly to science, always in a pioneering way, for example studying viruses that induce cancer. One example of his creative mind was demonstrating the *in vitro* evolution of an RNA virus (9).

In 1950 I had my first son, Paul, and we moved to Evanston, Illinois because Maurice had accepted an appointment as an instructor and established a laboratory in the biology department at Northwestern University. Maurice wanted to apply the powerful approaches of the newly constituted discipline of molecular biology to phenomena of cell differentiation and morphogenesis that attend the genesis of multicellular organization. He chose to study the cellular slime molds, primitive amoebae that displayed two distinct and separate phases, namely growth and differentiation. He established controlled conditions to study their development in sufficient number and synchronously to be able to identify and study the kinetics and logistics of key biochemical events, particularly the synthesis of new proteins that attend differentiation into fruiting body (10).

I wanted children, and although taking care of them has always been my first priority, I did not want to quit science completely in doing so. However, at that time, no laboratory would hire a woman with a child and able to work only part-time, so I started work in Maurice's laboratory for 2 h a day. Two more sons came along 3 and 4 years apart. Our income was very low and we could not afford baby-sitters. Moreover, in those days fathers did not participate in the daily care of the offspring. My mother came to my rescue because she understood my passion for science, and so with the last two babies she left the family in Chile and came to help me for 8 months each trip. With her overseeing the children I was able to increase the hours I spent



FIGURE 2. Gordon Sato and Raquel Sussman. Brandeis University, 1970.

in the laboratory. The focus of Maurice's research at that time was to isolate mutants that grow normally but have aberrant morphogenesis (11–14).

In 1958 we moved to the biology department of Brandeis University in Waltham, Massachusetts (with a professorship for Maurice but no academic appointment for me) where we found excellent graduate students and an exciting scientific atmosphere. The chairman of biochemistry, Nathan Kaplan, was a genius in recruiting young scientists with creative minds. He arranged to have our laboratory adjacent to the biochemistry laboratories. People that happened to meet in the hall started to talk about their latest scientific results, which led to open discussions. I would not have been able to learn the latest techniques in molecular biology and apply them to slime molds without the daily contact with Julius Marmur (15) (who was so generous in sharing the latest information), Larry Grossman (16), and Gordon Sato (17) among others (Figs. 2 and 3). I continued to collaborate with the ongoing research in our laboratory, which had more students and post-docs (18–21) and was able to show that vegetative amoebae fused producing heterozygotes that segregated in a Mendelian fashion (22).

In 1961, we took a sabbatical year to work at the Pasteur Institute in Paris. I chose to work with François Jacob and upon arrival told him that I was interested in studying repressors of lysogenic viruses. These bacterial viruses, upon infection, had a choice of either expressing most of their genes, thereby reproducing, lysing the host, and producing clear plaques on the bacterial lawn or expressing only two genes (C_I and C_{II}), which repressed all the lytic genes and produced turbid plaques. At that time the repressors were inferred from clear plaque mutations in these two loci that complemented each other. Their



FIGURE 3. Larry Grossman, Maurice Sussman, and Julius Marmur. Brandeis University, 1970.

chemical nature was still unknown because they had not been isolated yet. François went to his stock of lambda phages and gave me vials of several mutants that according to him were “bizarre.” I plated them; they all had the clear phenotype, but their complementation to the C_I group was peculiar. I kept plating them but had no clue why they behaved differently. Serendipity again occurred one day when I removed the agar plates from the 37 °C incubator and all the plaques of the λC_I 857 mutant were turbid. I immediately looked at the temperature of the incubator, and it was 1 degree lower, which meant that the mutation produced a temperature-sensitive repressor, implying that it was a protein. After determining all the parameters affecting its induction (the denaturation curve was very sharp with a T_m at 36–37 °C) we published the paper in *Comptes Rendus* (23). One day François told me that Jacques Monod had suggested that if repressors are proteins, I should be able to isolate mutants that are clear on the regular *Escherichia coli* indicator strain but turbid in suppressor strains of *E. coli*, because these strains contain mutant tRNAs that insert the right amino acid in response to the mutated codon, producing active repressor. I succeeded in isolating several mutants, and we published these results again in *Comptes Rendus* (24). These mutants were very useful later in constructs of plasmids with genes inducible by shifts in temperature. They also allowed Mark Ptashne to isolate and purify lambda repressor (25). My detailed data on the properties of these phages were never published in an English journal, but upon my return I was invited to present the work at the Department of Genetics of Yale University and at Harvard Medical School.

Back at Brandeis, Maurice started to focus on the appearance and disappearance of proteins during morphogenesis (26), and I analyzed the RNA metabolism, comparing the growing cells to the differentiating ones (27). For this project I had to use *Polysphondelium pallidum*, a slime mold that was able to grow axenically, finding

that ribosomal RNA was synthesized continually, including through the process of differentiation, at a time when not only the nutrients were absent but when the cells were degrading the existing RNA and protein (26). The common knowledge at the time was that “shift-down” conditions were correlated with inhibition of ribosomal RNA and ribosomal protein synthesis. We speculated that the mRNA synthesized during differentiation required specialized ribosomes coded from different genes than the ones used in the vegetative state. To answer that question, I took advantage of a hybridization procedure that had just been designed by Gillespie and Spiegelman (28) based on competition hybridization of labeled rRNAs from vegetative and differentiating cells to *P. pallidum* DNA. We showed that both ribosomal messages competed equally well for the complementary DNA sequences, indicating that the same genes coded them. As a control we used *Dictyostelium discoideum* DNA, which only allowed partial competition as expected from a different rRNA sequence (27). The other hypothesis that new messenger RNA has to be transported from the nucleus to the cytoplasm by nascent ribosomes remains untested. While we were speculating about ribosomes, Alexander Rich at MIT showed that proteins were synthesized in large ribosomal aggregates, which he called polyribosomes (29). I collaborated with his post-doc, W. D. Phillips, showing that the primitive slime molds also synthesized proteins in polyribosomes (30).

During those 22 years that I worked on *Dictyostelium* I never received any remuneration. Maurice and I had agreed that it was not ethical to ask a granting agency to pay the wife of the principal investigator. I am reflecting on this only to stress how unfair the treatment of women in science was in those times and in our case how brainwashed scientists could be.

In 1973 I became independent to pursue my own choice of scientific problems when we accepted an invitation to work at the Hebrew University in Jerusalem. I was appointed “Martze Bahir” (equivalent to Associate Professor) in the Department of Molecular Biology at Hadassah Medical School. I had my own laboratory and started to work on a problem that interested me greatly: the phenomenon of “SOS repair” in bacteria. The publication of Evelyn Witkin (31) had inspired me, and even before I had the possibility of working on that problem I already had a hypothesis to explain why damage to the bacterial chromosome or inhibition of DNA replication elicited a number of seemingly unrelated phenomena: cessation of cell division, inhibition of septum formation leading to filamentation, increased mutagenesis, production of colicin

and de-repression of lysogenic viruses. I had learned a great deal about DNA repair from Larry Grossman and proposed that the affected genes were controlled by repressors such as those in lambda phage that were present at a very low concentration sufficient to inhibit transcription of its operon. During repair of the chromosome several intermediates of the DNA helix are produced and if the repressors had affinity for a specific DNA repair product they would bind to it when present, thus decreasing the effective concentration needed to silence their own operators.

At Hebrew University I finally had a chance to test the hypothesis, and serendipity brought me Hanna BenZeev, who applied to be my technician. She was extremely competent and intelligent. To prove the hypothesis, we used a competition filter binding assay with extracts of lambda repressor and radioactive DNA and showed that *E. coli* DNA did not compete in the binding of repressor to lambda DNA, whereas DNA extracted from bacteria undergoing SOS repair did compete. Moreover, the non-inducible repressor (ind^-), used as control, did not compete. Also, increasing the DNA damage of the bacteria resulted in DNA with increased interference in the competition assay (32). Only two and a half years later, when we moved to the University of Pittsburgh, I had a chance to purify repressor and to prepare *E. coli* DNA containing different specific lesions in Larry Grossman’s laboratory. We proved that lambda repressor had a greater affinity for double-stranded DNA containing single strand gaps (33). Nevertheless, it turned out that my hypothesis was wrong; Roberts, Roberts, and Mount (34) established that RecA was the protein that bound to ssDNA and was activated to cleave the repressors. I still have the intuition that the affinity of repressors for gapped ssDNA is necessary to increase their local concentration at the RecA/ssDNA catalytic site.

Serendipity continued at the University of Pittsburgh, where two of the brightest graduate students applied to work in my laboratory: Jim Resnick and John Baluch. We established many important parameters of the SOS repair mechanism (35–39). At that time I had to work very hard to get tenure: teaching new courses every semester, getting good evaluations from students, publishing enough articles, and of course receiving continued support from NIH and NSF. The grants lasted long after I got tenure, took early retirement from the University of Pittsburgh at 66, and became an Associate Scientist at the Marine Biological Laboratory in Woods Hole. The move was motivated by our desire to be close to our children, who lived in Massachusetts.

I am fascinated by RecA, a mere 37.8-kDa protein that is the physiological equivalent of the vertebrate p53, because it exhibits a great number of sophisticated enzymatic activities necessary to protect the bacterial cell from diverse injuries (for review see Ref. 40). This protein forms a helical complex with ssDNA and NTP for all these activities. One subset of the activities requires a high RecA/DNA ratio and no hydrolysis of NTP (the co-protease activity and D loop formation) whereas the other subset requires NTPase activity necessary for recombination of two homologous duplexes. The work that I consider my best is postulating that RecA undergoes an allosteric change in structure/function dictated by the number of nucleotides available when forming the required ternary complex (41). The dynamics envisioned for a cell that undergoes a DNA lesion is as follows. When pol III encounters a distorted chromosome, it cannot proceed replicating the DNA, giving rise to small ssDNA gaps. SSB present in a large concentration in the cell binds to the ssDNA to protect it from DNases. Chrysogelos and Griffith (42) established that SSB organizes the ssDNA into nucleosome-like loops linked by 30 bases free of protein. We have shown that RecA binds to these linkers with a stoichiometry of 8 RecA monomers per linker, that is 3–4 nucleotides/monomer (39). This turns out to be the saturated complex, exhibiting the highest co-protease activity, designated state “a” (RecA^{*}). Thus, the complex proceeds to catalyze the cleavage of LexA thereby derepressing the genes under its control, including *recA*, *sfiA*, and *umuD*, which have to be cleaved by RecA^{*} to become a subunit of pol V. This polymerase is competent to replicate DNA containing lesions, which is probably repressed under normal cell life because it is error prone. Pol V proceeds to enlarge the ssDNA gap, thus allowing RecA to contact more nucleotide phosphates. RecA then changes to state “b,” which is proficient as an ATPase, thereby becoming a recombinase. If this were correct, it would be the first example of a protein responding to the specific temporal requirements of the cell by changing catalytic activities.

Despite the excellent studies performed analyzing complexes of RecA with ssDNA and dsDNA, we still do not know its atomic interactions with DNA, which requires crystallization. Until now that task was unattainable because of the dynamism of the system. Our contribution to this problem is to show that small oligos, in the presence of ATP γ S, retain enzymatic activities and are very stable; the RecA/dT₁₆ behaves like a saturated complex catalyzing the self-cleavage of repressors, whereas RecA/dT₂₄ has ATPase activity necessary for ssDNA recombination (40). I believe that these stable

complexes will be easy to crystallize, thus helping to establish their structure/function.

I am very fortunate to have witnessed the birth and development of molecular biology.

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