# Tim Hunt

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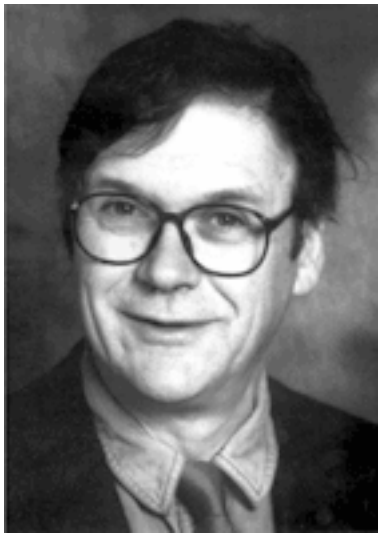

Read more: Tim Hunt in school

Tim Hunt is a born storyteller. Whenever the Nobel Prize-winner gets the chance to talk to young people about his work at Cancer Research UK he will fascinate them or throw them off balance them with sentences like: "I can't see any ethical problem in cloning human beings. I think it wouldn't be worse. If I lost my daughter by accident, it would be great if I could create her again from one of her cells".

Tim Hunt, born on the 19th of February 1943 at Neston near Liverpool, always expresses what he is thinking. His father, Richard Hunt, was a lecturer in paleography in Liverpool University, but the family moved to Oxford when the war was over, so he grew up in post-war time. " My earliest memories are of the winter of 1947/48, pushing a pram down to the coal depot about a mile away near the railway station as the delivery trucks were stuck in snow drifts. Food rationing also made a deep impression on me, and the habit of breaking eggs separately into a cup lest one should be bad and the week's omelette be spoiled persisted for many years. I was impressed by the food-parcels, which kind Americans sent us. Maybe those parcels are one reason why I liked the USA since I was a young boy", Hunt says.

The young German Gerd Sommerhoff, who conducted science lessons at his school, aroused his interest on biology. "It is hard to remember back, when I decided to be a biologist. I think it was when I did particularly well in a school exam when I was eleven years old", Hunt says and adds: "Honestly speaking, I had no choice. I was bad at physics and mathematic, hopeless at history, fanatical about cricket, though not terribly good."

1961 Tim Hunt took up his studies at Clare College in Cambridge. "It was quite a shock, when I went to university because I met so many clever people there, who knew much more

than me", Hunt remembers. Only three years later he started his proper scientific career in the Department of Biochemistry of the University of Cambridge (UK). He worked with Asher Korner, who encouraged a great deal of freedom among his students to work on any aspect they chose of DNA, RNA or protein synthesis. Asher Korner was the first Professor of Biochemistry at the University of Sussex. He died in 1971 in his 40s, only four years after moving to Sussex from Cambridge.

"Very early, I decided that I wanted to work on the control of translation of mRNA, and thanks to Louis Reichardt (who spent a year in Cambridge in the same bay as me before going to Stanford) I learned about the use of rabbit reticulocytes for studies of haemoglobin synthesis, and began to appreciate the advantages of simple model systems. Together with Tony Hunter, a fellow student, I became interested in the question of when the haem was inserted into globin to make haemoglobin, and the question of whether ribosomes had to queue up under conditions where iron (and hence haem) was limiting", Tim Hunt tells. This Research topic was the reason that Tim Hunt gave lectures at the Wilsede Meeting in 1973 and 1975 ? twenty-six years before he got the Nobel Prize in Medicine.

from left: Fred Stohlmann, Ron Mc Caffery, Rolf Neth, Robert C. Gallo,Tim Hunt (Wilsede 1973)

In 1982 - to be precise on the 22nd of July - Tim Hunt accidentally stumbled on cyclin, a key protein that controls the cell cycle. He told his friends about his discovery and "they looked at me, worrying whether I was crazy. It seemed to be too simple", Hunt remembers and smiles. Well, it took him and others ten years to prove the theory essentially correct. "But not well-organized experiments are often the ones that bring breakthroughs. One has to have luck. Look at my first discovery, which enabled me to write my first paper in Nature. We were trying to find out whether ribosomes were uniformly distributed on messenger RNA. And each experiment took about two to four hours. One day, we went out for lunch and didn't return in time, and the centrifuge ran too long. Nonetheless we decided to analyse the results ? and so discovered that there were fewer ribosomes on the messenger RNA making alpha chains than on the ones making beta chains. We never suspected this, and got the result by accident. Serendipity plays a major role in discoveries, but it usually only tells you that there is something going on that you didn't suspect!" But the interpretation of this discovery, which Tim Hunt and his friends presented in Nature 1968, was wrong. Harvey Lodish later corrected them.

There is one piece of advice that Tim Hunt likes to give young people: Find out what you like doing - what interests you ? and then do it! If you want to become a scientist, you need a lot of curiosity and a lot of enjoyment from finding things out. It is not an ordinary career." he warns. "I never really had to make any decisions, I was always doing what I liked and found fascinating. Education to me is finding out what you like and what you are good at. So it doesn't feel like work, but like fun." With this fun, Tim Hunt sometimes could not buy enough to eat. "There was always money, but not very much. There was only one exception, when I worked as a postdoc in USA. I was paid $11,500 per year, and in 1968 that was quite a lot of money! Back in England, I earned five times less, which was hard. I was poor, had hardly enough money to buy food. But I felt lucky, we had lots of fun, because my friends and me were doing what we liked to do." In Cambridge he found himself reunited with two old friends from the Korner laboratory, Richard Jackson and Tony Hunter. Sometimes Tim Hunt was worried about his own future, and then he tried to get a job. But he failed. In retrospect that was the chance of his life. "The freedom to have a grant, just to do research is a wonderful blessing", Hunt says. "We weren't sure if we good be successful researches, but we had good models. In Cambridge there were lots of Nobel Prize-winners and you could always talk to them, discuss with them. " For my generation, Francis Crick was probably the most obviously influential presence. He was often at lunch in the canteen of the Laboratory of Molecular Biology, where he liked to explain what he was thinking about, and he was always careful to make sure that everyone round the table really understood. He also went to lots of seminars, and almost always asked questions at the end. Sometimes, I remember thinking, they seemed slightly ignorant questions to which a man of his extraordinary range and ability ought to have known the answers. Only slowly did it dawn on me that he only and always asked questions when he was unclear or unsure, a great lesson."

Tim Hunt is also interested in science policy. When he got the Nobel Prize in 2001 he interrupted his scientific work. "I thought, maybe it's a good time to stop. So I engaged myself more in European science policy, especially helping to lobby for the European Research Council, which is now established. But soon I realized that I could do only one thing really well: experiments. And I like doing it till now. Maybe I will retire in a few years." The question of what controls the cell cycle is still a challenge. "We discovered that cell cycle transitions are catalysed by protein kinases. But we don't know, how many proteins these kinases have to phosphorylate to make the cell enter mitosis. And what about the control of the phosphatases that reverse the process?" And even the discovery Tim Hunt got the Nobel Prize for is still not completely understood. "Cyclins are still a bit mysterious. We don't really understand how substrates are recognized and what the mechanism is. Working at this molecule is still great fun."

He is sceptically whether genomics, proteomics and other "omics" will lead science to better understanding of nature. "I don't find these global approaches very satisfying. And a lot of the work can't be judged by the standards that we normally use, so it's hard to tell if its right or not. I think we have to do proper work as molecular physiologists. That will help us to learn how to treat cancer and other illnesses that occur because the cell cycle is not working properly anymore", Hunt judges.

And there is one question that moves him: Can biologists help towards the resolution of human conflict? He would like to do so!

Hamburg 10.4.2007, Angela Grosse

## 2 Curriculum Vitae

I was born in 1943 at Neston in the Wirral, not far from Liverpool where my father, Richard William Hunt was a lecturer in paleography, the study of mediaeval manuscripts. Richard's father was a doctor, and there is still a chemist's shop (i.e. pharmacy) in Winchester that bears the family name. My mother's father, Harry Rowland was a businessman, a timber merchant who imported wood from South America. Actually, as I discovered from letters to my mother, Kit, which I found after they were both dead, Richard was working in London at Bush House, presumably in some kind of intelligence role although he never spoke of it, and I was always afraid to ask. He was the kind of man who, having signed the Official Secrets Act, would have felt bound by it to the grave. Immediately the war was over, Richard accepted a position as Keeper of the Western Manuscripts in the Bodleian Library in Oxford, a post he held for the next 32 years. My earliest memories are of the winter of 1947-8, pushing a pram down to the coal depot about a mile away near the railway station as the delivery trucks having been stuck in snow drifts. Food rationing also made a deep impression on me, and the habit of breaking eggs separately into a cup lest one should be bad and the

week's omelette be spoiled persisted for many years. Kind American mediaevalists would send food parcels, which prejudiced me strongly in favour of the USA at an early age.

My education started with latin taught at home by a governess, I can't imagine why, and for some reason I attended the Infants Department of the Oxford High School for Girls before moving to the Dragon School at the dangerous age of 8 or so. All I can remember of the girls is their bossy mothers, who liked to organise parties that I detested because of the competitive spirit that I detected in games like musical chairs. There was also knitting squares for the refugees' blankets, at which I was hopeless - the girls produced neat squares, myself ragged trapezoids. And there was the matter of the garden. My small patch was so barren that I was told to describe an imaginary garden when it came time to write Nature notes. The Dragon was much better, much less regimented, at the same time much more playful and more serious. Woe betide you for grammatical errors. Although mornings were largely devoted to latin and greek, at which I got worse and worse as time went by, there was a science lesson every week conducted by a young German called Gerd Sommerhoff. It was he who showed me that biology was an easy subject, and from then on I really never had to make any more career decisions. I also liked English, was bad at maths and hopeless at history, and fanatical about cricket, though not terribly good. My hero was Denis Compton.

At the age of 14, I moved across town to Magdalen College School, Oxford, where science played a much larger role in the curriculum. I loved Chemistry in particular, largely because the teacher, Colonel Simmons was much more concerned with principles than facts, although a thoroughly practical man himself. We were allowed considerable freedom, and on more than one occasion started fires from distilling volatile flammable solvents. One became adept at avoiding injury. Later on, biology again came to the fore when a young teacher called Terence Doherty took just three of us for Zoology. We dissected my brother's pet rabbit when it died, which was a treat after all the formalin-fixed dogfish. Terence wanted to be a painter, really, and later worked as an assistant with the sculptor, Michael Ayrton. I was also introduced by Terence to the Craftsman Potters' Shop in London, where I still love to browse. Very important during these years were Extramural Lectures given by the University of Oxford and the Christmas lectures in the Oxford Museum. For me, the University appeared to be largely devoted to the classics and history, and our house had a steady flow of mediaevalists of various stripes, but here in corners were revealed real treasures: the shrunken heads of the Pitt Rivers Collection, the dinosaur skeletons, lectures on the Theory of Evolution (on the occasion of the centenary of the publication of the Origin of Species) and on the Krebs Cycle. We also used to visit local factories and research institutes; at Alcan Aluminum they were trying to develop a Coca-Cola resistant lining for the cans, and somewhere we watched the zone refinement of an enormous silicon crystal. Balances that could weigh a human hair were pretty impressive, and even the telephone exchange was fascinating behind the scenes. George Beadle spent a year as Eastman Professor and

came to give a lecture to the Scientific Society (his son Redmond attended the school), and I remember asking him to explain the work that led him to win the Nobel Prize.

In the fall of 1961 I went up to Clare College, Cambridge to read Natural Sciences, with the intention of becoming a biochemist in the end. The courses were mostly excellent, and I did much better in the practical exams than in the theory. We were worked extremely hard, with certain practical classes falling on Saturday afternoons. Sydney Brenner organised seminars in his college rooms, and gave lectures that were officially off-limits to biochemists. It was all tremendously exciting, and we were very conscious, I think, of being surrounded by Very Great Men as well as very much in awe of the weight of the history of scientific discovery, especially in physics.

I started my scientific career in 1964 in the Department of Biochemistry at Cambridge, under the supervision of Asher Korner, who encouraged a great deal of freedom among his students to work on any aspect they chose of DNA, RNA or protein synthesis! Very early on, I decided that I wanted to work on the control of translation of mRNA, and thanks to Louis Reichardt (who spent a year in Cambridge in the same bay as me before going to Stanford to do his Ph.D. with Dale Kaiser) I learned about the use of rabbit reticulocytes for studies of haemoglobin synthesis, and began to appreciate the advantages of simple model systems. Together with Tony Hunter, a fellow student, I became interested in the question of when the haem was inserted into globin to make haemoglobin, and the question of whether ribosomes had to queue up under conditions where iron (and hence haem) was limiting. This was inspired by a talk we hear in 1965 by Vernon Ingram at a meeting in Cambridge on the subject of haemoglobin, at which I also listened to Henry Borsook's talk comparing protein synthesis in sea urchin eggs with protein synthesis in red cells. I thought very little about this at the time, but it planted an important seed. Tony and I found that the ribosomes were evenly spaced along the mRNA, and never formed a queue unless we forced them by other means. In 1966, I went to another meeting about haemoglobin, this time in Thessaloniki in northern Greece, where I met Irving London. I persuaded him that it would be fun to come and work in his lab during the summer, and by the time I was back in Cambridge (the train through Yugoslavia went very slowly), tickets to New York awaited me. I spent July through October 1966 living in a very hot dormitory out in the far east Bronx, and thoroughly enjoyed myself. The lab was the coolest place to be.

When I finished my Ph.D. in 1968, I went to New York to work with Irving, who had long been interested in the haem question. These were turbulent times, but I was fortunate in having a number of collaborators at the Albert Einstein College of Medicine. In particular, with Nechama Kosower and her husband Ed, we discovered that tiny amounts of oxidized glutathione were extremely inhibitory to protein synthesis in reticulocytes, and with Ellie Ehrenfeld that even tinier amounts of double-stranded RNA killed protein synthesis. Most striking was the finding that depletion of haem, and addition of GSSG or dsRNA all seemed to have similar effects on cell-free protein synthesis in the reticulocyte lysate, as though these

disparate agents all acted in a similar manner. I also discovered that simple gel-filtration of the lysate caused a drastic fall in its activity. All these effects needed several more years to work out. One other thing I learned about during the studies on the inhibition of protein synthesis by dsRNA was that micrococcal nuclease, which requires Ca2+ for activity, did not inhibit protein synthesis in the reticulocyte lysate provided no Ca2+ was present.

On my return to Cambridge, I found myself reunited with two friends from the Korner laboratory, Richard Jackson and Tony Hunter. They had recently made the important discovery that Met-tRNAf was used to initiate haemoglobin synthesis, and this enabled us to probe the process of initiation in much more detail, leading to a set of very surprising results: that the initiator tRNA bound to ribosomes before the mRNA, and that all the inhibitory effects I had been studying in New York blocked the very first step, the binding of Met-tRNAf to 40S ribosomal subunits. It took three or four years to realise that this inhibition was due to the action of at least two inhibitors of the process, one activated by the absence of haem, the other by the presence of dsRNA, and that these inhibitors were protein kinases, which phosphorylated eIF-2, the key initiation factor that catalysed the binding of Met-tRNAf to ribosomes. Actually, it turned out to be rather complicated, because phosphorylation did not block the reaction the first time round, but rather prevented nucleotide recycling on the initiation factor, so that its re-use was inhibited.

By 1977, the outlines of all this were pretty clear, and we had a very good understanding of the reticulocyte lysate and its properties. The cause of the gel-filtration problem proved to be loss of polyamines, and the GSSG inhibition was a problem with thioredoxin and thioredoxin reductase which were needed to keep something in the reduced state. The micrococcal nuclease calcium dependence was put to good use by Hugh Pelham, a graduate student in the lab, who developed the use of nuclease treatment of the lysate to assay heterologous mRNAs. An important factor in this development was a ready source of messenger RNA, for which we used tobacco mosaic virus. Working with David Zimmern, a student of Aaron Klug's, and John Knowland, who was in John Gurdon's laboratory, it became clear that the RNA in the virions did not direct the synthesis of coat protein. Tony Hunter and I therefore ground up som TMV-infected tobacco leaves in liquid nitrogen and found to our great joy that a small mRNA was present that efficiently directed the synthesis of authentic TMV coat protein.

I therefore turned to new systems for studying translational control, and took the opportunity of teaching summer courses at the Marine Biological Laboratory, Woods Hole, to look at changes in protein synthesis in sea urchin and clam eggs after fertilization. This opened new horizons, not only in learning to deal with new systems, but in the breadth of approaches and interests of the scientists who passed through Woods Hole during the summers. I learned a tremendous amount of developmental and cell biology over the years. In 1979, Joan Ruderman and her student Eric Rosenthal were teaching the embryology course, and I helped them with experiments on the translational control of maternal mRNA; the major mRNAs concerned later turned out to be the A and B-type cyclins and the small subunit of

ribonucleotide reductase. We carried out quite a lot of work together on the translational control, and Nancy Standart worked as a postdoc first with Joan and then with me in Cambridge. I believe that these clam mRNAs were the first really well-authenticated case of specific translational control. Together with Nancy, we later identified the key regulatory regions in the 3' untranslated region of ribonucleotide reductase and cyclin A mRNAs that are necessary for translational masking.

By 1982, work on the control of protein synthesis in sea urchin eggs had almost ground to a halt; every idea that my students and I tested proved to be false, and the very basis of the system was essentially flawed in that we were studying a system in which the ribosomes changed from having almost no activity to one in which about 2% of them were active, leaving the 98% majority of them still inert. The one question we did not ask up to that point, because the answer was supposed to be known already, was as to why an increase in protein synthesis occurred at fertilization, although it had been shown many years earlier that inhibiting protein synthesis inhibited cell division. One day in July 1982, with teaching over, I did a very simple experiment that was actually designed to answer the question of whether the proteins made after fertilisation were the same as the proteins made after parthenogenetic activation of the eggs. The eggs were divided into two batches, one of which was fertilized, the other treated with calcium ionophore, A23187. Radiolabelled methionine was added to both, and samples taken until after the fertilized eggs had divided into two-cell embryos. When the autoradiograph of the 1-dimensional SDS slab gel was developed, I noticed a very strange thing in the fertilized samples, which was a labelled band that went away, unlike all the others which, as expected, got stronger with time. Scarcely was the film dry when I ran into John Gerhart at the traditional Friday night lecture wine and cheese party, who told me about experiments he and Marc Kirschner had been doing on the oscillations in frog MPF activity during meiotic maturation of Xenopus oocytes. They found that MPF activity went away transiently between meiosis I and meiosis II, but if protein synthesis was inhibited, the MPF never returned. The behaviour of the band I had just seen fitted in beautifully with their physiological findings, and I was sure that this band, which we called cyclin, must have something to do with MPF. The rest of that summer was spent looking for cyclins in other species, and describing the basic behaviour of the strange disappearance, which turned out to occur about 10 minutes before each cell division. The oscillations went on for several hours after fertilization. Clam eggs did the same thing, except that two bands showed this behaviour. It is very surprising that nobody had spotted this before; it could have been done any time after the invention of the slab SDS polyacrylamide gel in about 1970.

It was impossible to work on cyclins back in Cambridge, because there were no clams or sea urchins, and by the time the next summer arrived I thought perhaps the suddenly-disappearing proteins had been a complete fantasy. Fortunately, on return to Woods Hole next summer the results proved quite reproducible, but it was clearly going to be an uphill struggle to find out what was going on, and whether it had any general application. Progress

was slow for various accidental reasons, like the fact that Tom Evans who spent the summer of 1982 as my undergraduate assistant had already signed up with David Secher at the MRC Molecular Biology Laboratory as his Ph.D. supervisor and it did not seem right to alter the arrangements. Jon Pines joined me a year later, but his first year was a terrible one for sea urchin eggs, delaying library construction by a whole year! Not until Christmas 1986 did we succeed in cloning and sequencing sea urchin cyclin B. After that, the pace began to accelerate, and it did not take long to isolate clones for Xenopus cyclins A and B, which was very exciting, because it made it clear that these proteins were not restricted to marine invertebrates. We developed the use of oligodeoxynucleotides in conjunction with RNase H as a way of specifically cutting particular mRNAs, and were able to show thereby that B-type cyclin synthesis was necessary for Xenopus extracts to enter mitosis, and that Xenopus MPF contained B-type cyclins as well as p34cdc2.

Today, cyclins and cyclin-dependent protein kinases are recognised as key elements in the regulation of cell cycle transitions, and the family of proteins has grown considerably, thanks to work in many laboratories. I well recall the scepticism in the early days, however, when most people did not believe things could be so simple as making an enzyme to catalyse mitosis, and then destroying the enzyme to leave mitosis. Moreover, in retrospect, we were very slow to realise that cyclins were regulatory and activating subunits of Cdc2 and its relatives. I do not know why this penny took so long to drop.

Moving to ICRF allowed better access to Xenopus than had been possible in Cambridge, and important achievements have been the identification of c-mos as the activator of MAP kinase with Angel Nebreda, which also led to the discovery of a new member of the MAP kinase family called p38; the identification of John Shuttleworth's MO15 kinase as the CDK activating kinase with Randy Poon, and the purification of soluble and active fragments of cyclin A that allowed Jane Endicott, Martin Noble and Nick Brown to solve its crystal structure. We have defined a very large family of cyclins in Xenopus, and tested the importance of different members of the cyclin B family by making "knockout" mice. Right now, major interests of the laboratory lie in understanding how cyclins are targeted for proteolysis by their "destruction boxes", and in seeking substrates for cyclin dependent kinases. This is no easy matter, and our successes in the past, finding the salient substrates for c-mos and MO15 owed more to luck, and keeping our eyes open while doing other things, than any rational approach.

Postscript

Of the many letters of congratulation that arrived after the announcement of the Nobel Prize, the one that gave me the purest pleasure was from a Swedish woman called Kerstin Westin in Uppsala, enquiring if I was, by any chance, the Tim Hunt she had known in Oxford in 1950 when she worked as an au pair girl with my family - and I was. Unlike my brother Sandy, who was only 5 at the time, I could clearly recall the young girl who looked after us all those

years ago, and we had a happy reunion 51 years later in Stockholm. Apparently, nothing about the 7-year old boy gave any hint of his future. I would say that I was lucky to grow up in a loving family with wonderful teachers. Later, as a young scientist, I had the further good fortune to witness many of the founders of molecular biology at close hand. They set the standards of what was and could be expected. For my generation, Francis Crick was probably the most obviously influential presence. He was often at lunch in the canteen of the Laboratory of Molecular Biology where he liked to explain what he was thinking about, and he was always careful to make sure that everyone round the table really understood. He was a frequent presence at talks in and around Cambridge, where he liked to ask questions. Sometimes, I remember thinking, they seemed slightly ignorant questions to which a man of his extraordinary range and ability ought to have known the answers. Only slowly did it dawn on me that he only and always asked questions when he was unclear or unsure, a great lesson.

For many years, I used to supervise students in Cambridge, preferring either to teach the introductory course in Cell Biology or third year students in their biochemistry laboratory projects. Here again, I had the good fortune to meet a succession of clever people, many much cleverer than I. Supervisions worked best when the group (typically of two or three students and myself) was engaged on a mutual course of discovery, for at the time, not so much was known about how cells worked and one could profitably muse on how things might work. Very different, as a friend pointed out, from teaching, say, crystallography where the principles had been worked out years and years before and your job was to clarify them for the newcomer. As more and more became known, I found teaching became less fun, whereas learning became more pleasurable. In particular, I learned a tremendous amount at the Marine Biological Laboratory during the summers I taught and researched there, and when that phase of my life was over, I started working with John Wilson on the problems book that accompanies Molecular Biology of the Cell by Alberts et al. The ferreting around trying to make sense of unfamiliar territory and the discussions with the other authors provided an even more intense pleasure of learning new things.

But none of these pleasures, great and satisfying though they are, match the joy of discovery.

From Les Prix Nobel 2001.

http://www.nobel.se/medicine/laureates/2001/hunt-autobio.html

R. Timothy (Tim) Hunt, born February 19, 1943

Address:

Imperial Cancer Research Fund,
Clare Hall Laboratories,
South Mimms,
Herts EN6 3LD,
UK

Academic Education and Appointments:
1964 B.A. University of Cambridge
1968 Ph.D. University of Cambridge
1991- Principal Scientist, ICRF Clare Hall Laboratories
Selected Honours and Awards:
1991 Fellow of the Royal Society
1991 Foreign Associate of the US National Academy of Sciences
1993 Abraham White Scientific Achievement Award of the George Washington University

## 3 Publications

## 4 Multimedia

Videos and Audio files

**Tim Hunt: Getting in and out of mitosis (Wilsede Meeting 2008)** DOI: 10.3205/wsc-2008-en-000014.05

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## 5 Contact

email: Tim Hunt