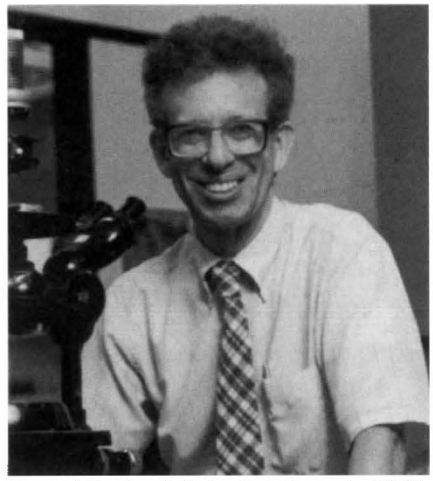
OBITUARY

Howard M. Temin (1934–94)

On 9 February, Howard Temin, recipient of the Nobel Prize in Physiology or Medicine in 1975, virologist and professor of oncology at the University of Wisconsin, died of metastatic adenocarcinoma of the lung.

Temin's many scientific contributions are crowned by his concept, developed in the mid-1960s, that RNA tumour viruses reproduce by a process involving an integrated DNA intermediate. He called this DNA intermediate the provirus, by analogy with the prophage of a bacterial virus; in retrospect, his idea of a provirus, based at first only on indirect laboratory results, was one of those rare moments in science when one man's insight drags a whole field into a new realm of thought and pushes aside bottlenecks in several other areas. But for several years he was constrained by disbelief and criticism, until the dramatic and independent discoveries in 1970 by Temin and Mizutani (*Nature* 226, 1211; 1970) and by David Baltimore (*Nature* 226, 1209; 1970) of the enzyme reverse transcriptase, a singular DNA polymerase that provides the catalytic activity for transcribing viral RNA into DNA. The enzyme of Temin and Mizutani was from a chicken retrovirus whereas Baltimore's was from a mouse leukaemia retrovirus, but it was soon learned that reverse transcriptase is carried by every infectious RNA tumour virus.

The way was now open to determine the replication cycle of these cancer-causing animal viruses and the strategies they use to transform cells, but this was only the start of the impact of reverse transcriptase on molecular biology. The unidirectional flow of genetic information, DNA to RNA to protein, had until then been taken for granted. Further, reverse transcriptase became a key tool in biotechnology by virtue of its ability to synthesize DNA copies from any messenger RNA template. Finally, measuring the activity of the enzyme soon became a sensitive and specific way to assay for a retrovirus and was one of two crucial methods that led to the discovery of human retroviruses, the leukaemia viruses (HTLV-I and HTLV-II) and the human immunodeficiency virus responsible for AIDS.



Howard Temin, Nobel prize winner in 1975.

Temin began his postdoctoral fellowship at Cal Tech with Harry Rubin and Renato Dulbecco in 1956 (Dulbecco and Baltimore shared the 1975 Nobel prize with Temin). He started studying the transformation of cells by the Rous (avian) sarcoma retrovirus when he realized that this *in vitro* phenomenon, then just described by scientists at Rutgers University, was an opening to understanding cellular alteration, an interest he maintained throughout his career. In 1960 he moved to Madison to join the cancer research centre at the McArdle Laboratory in the University of Wisconsin, where he remained a member of this renowned group. In recent years he turned his attention to mechanisms of generating variation in retroviruses, central to the problem of drug resistance and vaccine development for AIDS. He continued to be active as a teacher, researcher and advisor to the National Cancer Institute (as a member of the National Cancer Advisory Board) until only weeks before his untimely death.

Howard Temin's mind held a rare combination of creativity and penetrating, analytical insights, and his judgement was a rare combination of compassion and independent objectivity. In an era when biological laboratories have grown in size, needs and complexity, his team maintained a focus on individual creativity. Despite the modern pressures on families, he was a devoted family man — to his wife, Rayla Greenberg Temin (geneticist at the University of Wisconsin), and to his daughters, Sarah and Miriam. Finally, at a time when biomedical scientists in the United States are more and more involved in entrepreneurial adventures, Temin remained where he began, the pure academician. Once when I asked him why, his reply was a typical Teminism: "With all those experiments going on out there, there must be at least one control!".

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in which the ratio of fluorinated polymeric surfactant to crosslinking agent is varied between 0.3:1 and 3:1. As the films become more highly crosslinked, the concentration of R_F groups at the surface decreases, and these groups are less able to reorient themselves. The surface free energy lies between 11 and 16 mJ m^{-2}, indicating that the surface is indeed composed of a preponderance of R_F groups. X-ray data are consistent with layers of R_F groups about 8 nm thick.

The new synthetic scheme is versatile enough to allow production of surfaces with a range of energies. A series of coatings with tailored surface energies could be made by varying the monomer ratios in the reactants, perhaps using non-fluorinated monomers when higher surface energy is desired, and used to isolate and explore the influence of surface free energy, which is widely believed to be the primary determinant of adhesion. Although synthetic adhesives show a reasonable (but not perfect) inverse relationship between bonding strength and surface energy, natural glycoproteins and polysaccharides show the least adhesion within a range of surface energy which is near, but does not include, the lowest values. Baier and Meyer[6] recommend 20 to 30 mJ m^{-2} as the optimum range for the free energy of a surface designed to shed biofouling and say that efforts to reduce the surface energy below this range are counterproductive.

Before we conclude that the ultimate non-stick coating is at hand, silicone and urethane chemists must have their innings. Compared with a carbon backbone, the O–Si–O backbone has a more open structure, with a flatter angle and a longer bond length; available surface-active groups on a silicone backbone adopt the lowest surface-energy configuration much more readily. Silicone coatings will need improved mechanical strength for some applications. Elastomeric urethane coatings, however, combine backbone flexibility with good mechanical properties. My view is that the consummate non-stick coating will have a silicone backbone, but I'm not yet prepared to bet whether it will contain fluorine. □

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