



## Suzanne Cory



**Date of Birth** 11 March 1942

**Place** Melbourne (Australia)

**Nomination** 27 January 2004

**Field** Molecular Biology

**Title** Professor

### Professional address

The Walter and Eliza Hall Institute of Medical Research  
1G Royal Parade  
Parkville, Victoria (Australia)

### Most important awards, prizes and academies

**Awards:** David Syme Prize, University of Melbourne (1982); Avon Australia 'Spirit of Achievement' Award (1992); Lemberg Medal, Australian Society for Biochemistry & Molecular Biology (1995); Burnet Medal, Australian Academy of Science (1997); Australia Prize (shared) (1998); Charles S. Mott Prize (shared), General Motors Cancer Research Foundation (1998); L'Oreal – UNESCO Women in Science Award (2001); Royal Medal, Royal Society (2002); Centenary of Federation Medal, Australia (2003). **Academies:** Australian Academy of Sciences (1986); Royal Society (1992); American Association for Immunology (1993); Royal Society of Victoria (1996); US National Academy of Science (1997); Asia-Pacific International Molecular Biology Network (1998); American Academy of Arts and Sciences (2001); French Academy of Sciences (2002); Australian Society for Biochemistry and Molecular Biology; American Association for Cancer Research.

### Summary of scientific research

Suzanne Cory's research has had a major impact on the understanding of immunology and the development of cancer. After pioneering Ph.D. studies determining the sequence of methionine transfer RNA, using the sequencing methods that had just been developed by Fred Sanger, her post-doctoral studies at the University of Geneva focused on sequence analysis of R17 phage RNA a model messenger RNA. Cory and Adams returned to Melbourne in 1971 to The Walter and Eliza Hall Institute. During the first few years, they discovered 5' caps on mammalian messenger RNAs, helped to introduce gene cloning technology in Australia, and addressed a central puzzle regarding the immune response: how does the body make the myriad antibodies needed to fight diverse infectious agents? Their laboratory helped uncover the astonishing solution: antibody genes are encoded as bits and pieces which can combine in a myriad ways, thereby creating much greater diversity with which to fight infection. In 1981, their attention turned to the nature of the genetic accidents that cause cancer. Their laboratory showed that damage to chromosomes can activate cancer-promoting genes. They tracked down the mutation which activates the oncogene *myc* and leads to Burkitt's lymphoma, a malignancy of antibody-producing cells. In collaboration with Alan Harris, they then engineered novel lines of lymphoma-prone mice, to study the early stages of disease and test for synergistic mutations. The current focus of their research is how cells decide whether to live or die. This program was launched in 1988 by the seminal finding of David Vaux in their laboratory that *bcl-2*, the gene responsible for follicular lymphoma, promotes cell survival. This discovery opened an entirely new way of thinking about cancer development, since all other oncogenes (cancer-causing genes) had been found to promote cell proliferation. The *bcl-2* gene proved to have numerous relatives, and some actually promote cell death (apoptosis) rather than cell survival. Today, a major program at the Hall Institute, led by Adams, Cory, Harris, Strasser, Huang, Vaux, Gerondakis and Colman is directed to understanding how apoptosis is controlled, influences normal development and contributes to cancer and other diseases. This knowledge will lead to the development of more effective therapeutics for cancer and degenerative diseases.

### Main publications

Adams, J.M. and Cory, S., Modified nucleosides and bizarre 5'-termini in mouse myeloma mRNA, *Nature*, 255, pp. 28-33 (1975); Cory, S. and Adams, J.M., Deletions are associated with somatic rearrangement of immunoglobulin heavy chain genes, *Cell*, 19, pp. 37-51 (1980); Adams, J.M., Gerondakis, S., Webb, E., Corcoran, L.M. and Cory, S., Cellular *myc* oncogene is altered by chromosome translocation to an immunoglobulin locus in murine plasmacytomas and rearranged similarly in Burkitt lymphomas of man, *Proc. Natl. Acad. Sci. USA*, 80, pp. 1982-6 (1983); Corcoran, L.M., Adams, J.M., Dunn, A.R. and Cory, S., Murine T lymphomas in which the cellular *myc* oncogene has been activated by retroviral insertion, *Cell*, 37, pp. 113-22 (1984); Adams, J.M., Harris, A.W., Pinkert, C.A., Corcoran, L.M., Alexander, W.S., Cory, S., *et al.*, The *c-myc* oncogene driven by immunoglobulin enhancers induces lymphoid malignancy in transgenic mice, *Nature*, 318, pp. 533-8 (1985); Vaux, D., Cory, S. and Adams, J.M., *Bcl-2* gene promotes haematopoietic cell survival and cooperates with *c-myc* to immortalize pre-B cells, *Nature*, 335, pp. 440-2 (1988); Johnson, G.R., Gonda, T.J., Metcalf, D., Hariharan, J.K. and Cory, S., A lethal myeloproliferative syndrome in mice transplanted with bone marrow cells infected with a retrovirus expressing granulocyte-macrophage colony stimulating factor, *EMBO J.*, 8, pp. 441-8 (1989); Strasser, A., Harris, A.W., Bath, M.L. and Cory, S., Novel primitive lymphoid tumours induced in transgenic mice by cooperation between *myc* and *bcl-2*, *Nature*, 348, pp. 331-3 (1990); Rosenbaum, H., Harris, A.W., Bath, M.L., McNeall, J., Webb, E., Adams, J.M. and Cory, S., An *Eμ-v-abl* transgene elicits plasmacytomas in concert with an activated *myc* gene, *EMBO J.*, 9, pp. 897-905 (1990); Elefanti, A.G., Hariharan, J.K. and Cory, S., *bcr-abl*, the hallmark of chronic myeloid leukaemia in man, induces multiple haematopoietic neoplasms in mice, *EMBO J.*, 9, pp. 1069-78 (1990); Perkins, A., Kongsuwan, K., Visvader, J., Adams, J.M. and Cory, S., Homeobox gene expression plus autocrine growth factor production elicits myeloid leukemia, *Proc. Natl. Acad. Sci. USA*, 87, pp. 8398-8402 (1990); Strasser, A., Harris, A.W. and Cory, S., *bcl-2* transgene inhibits T cell death and perturbs thymic self-censorship, *Cell*, 67, pp. 889-99 (1991); Adams, J.M. and Cory, S., The *Bcl-2* protein family: arbiters of cell survival, *Science*, 281, pp. 1322-26 (1998); Print, C.G., Loveland, K.L., Gibson, L., Meehan, T., Stylianou, A., Wreford, N., de Kretser D., Metcalf, D., Kontgen, F., Adams, J.M. and Cory, S., Apoptosis regulator *Bcl-w* is essential for spermatogenesis but appears otherwise redundant, *Proc. Natl. Acad. Sci. USA*, 95, pp. 12424-31 (1998); Bouillet, P., Purton, J.F., Godfrey, D.I., Zhang, L.C., Coultas, L., Puthalakath, H., Pellegrini, M., Cory, *et al.*, *BH3*-only *Bcl-2* family member Bim is required for apoptosis of autoreactive thymocytes, *Nature*, 415, pp. 922-6 (2002); Cory, S., Adams, J.M., The *Bcl2* family: regulators of the cellular life-or-death switch, *Nat. Rev. Cancer*, 2(9), pp. 647-56 (2002); Adams, J.M., Cory, S., Apoptosomes: engines for caspase activation, *Curr. Opin. Cell. Biol.* 14(6), pp. 715-20 (2002); Cory, S., *et al.*, The *Bcl-2* family: roles in cell survival and oncogenesis, *Oncogene*, 22(53), pp. 8590-607 (2003); Egle, A., Harris, A.W., Bath, M.L., O'Reilly, L., Cory, S., *VavP-Bcl2* transgenic mice develop follicular lymphoma preceded by germinal center hyperplasia, *Blood*, 103(6), pp. 2276-83 (2004); Egle, A., Harris, A.W., Bouillet, P., Cory, S., Bim is a suppressor of *Myc*-induced mouse B cell leukaemia, *Proc. Natl. Acad. Sci. USA*, 101(16), pp. 6164-9 (2004); Smith, D.P., Bath, M.L., Metcalf, D., Harris, A.W., Cory, S., *MYC* levels govern hematopoietic tumor type and latency in transgenic mice, *Blood*, 108(2), pp. 653-61 (2006); van Delft, M.F., Wei, A.H., Mason, K.D., Vandenberg, C.J., Chen, L., Czabotar, P.E., Willis, S.N., Scott, C.L., Day, C.L., Cory, S., *et al.*, The *BH3* mimetic ABT-737 targets selective *Bcl-2* proteins and efficiently induces apoptosis via Bak/Bax if *Mcl-1* is neutralized, *Cancer Cell.*, 10(5), pp. 389-99 (2006).