In the questing spirit of cancer research, the 1979 discovery of the gene named p53 was the equivalent of finding the Holy Grail. One of its discoverers, the oncologist David Lane, dubbed it the “Guardian of the Genome.” The gene, it turned out, is a kind of master switch that orchestrates life-and-death signals in every cell in our bodies.

In normal circumstances, p53 acts to protect us from cancer, killing out dangerous cells and preventing tumors. But sometimes it malfunctions and turns deadly in a different way. In more than half of all human tumors, mutations corrupt p53, and the gene starts to propagate cancer rather than prevent it.

Sue Armstrong, a science writer based in Edinburgh, Scotland, recounts the fascinating story of p53’s discovery, an effort that drew in some of the best-known names and institutions in the U.S. and Europe and involved researchers heading down blind alleys and confronting the skepticism of colleagues. To conduct her research, she says that she received financial help from the Pathological Society of Great Britain and Ireland and credits the institution for supporting “the proposition that even the toughest science deserves a popular audience.”

But explaining tough science is no easy task, and Ms. Armstrong doesn’t always succeed, especially if “p53: The Gene That Cracked the Cancer Code” is compared with equally complex but more accessible works, such as Siddhartha Mukherjee’s “The Emperor of All Maladies: A Biography of Cancer” (2010). The detail in “p53” can be daunting as Ms. Armstrong describes the small victories or telling defeats that nudge the gene quest forward. One researcher, like others, “found that her efforts to separate Abl protein from everything else in cancerous cells brought along another protein for the ride with a
molecular weight close to 53 kilodaltons.” Even so, Ms. Armstrong clearly grasps the science, and she makes a valiant effort to guide the reader through the maze, starting every chapter with a synopsis, such as: “In which we peer into the machinery of the cells to see how the genes make the proteins that do virtually all the work in our bodies.”

Scientists stumbled on p53, Ms. Armstrong tells us, while investigating a monkey virus. The two men most credited are Arnold Levine, then at Princeton, and Dr. Lane at the Imperial Cancer Research Fund in London. But dozens of other scientists worked together in a rare collaborative effort. At first, p53 was dismissed as an unimportant molecule. One researcher even warned a young colleague not to work “on this bullshit protein.”

But as the era of gene sequencing unfolded in the 1980s, Dr. Levine and others began investigating whether p53 was an oncogene—an abnormal gene that predisposes cells to develop into cancers. Researchers at the time were beginning to see cancer as a battle between oncogenes and tumor-suppressor genes—i.e., genes that prevent cancer when they are working properly but allow it to develop when they are knocked out.

Evidence began to accumulate that, in fact, p53 was a tumor suppressor. But it wasn’t easy to figure out just how it worked or what caused it to go wrong. Researchers eventually discovered that, when p53 is functioning normally, it attaches itself to the DNA in a damaged cell and takes control of other genes, switching them on and off as necessary and disabling cells that might threaten an organism. One of the most important weapons in p53’s armory: It can induce a state of permanent arrest and instruct a seriously damaged cell to commit suicide.

By the early 1990s, though, scientists were discovering that p53 mutants sometimes cooperate with oncogenes to drive the growth of tumors and develop malignancy, rather than just losing their ability to act as suppressors. The result of this discovery was a search for the causes of p53 mutation—not only so-called somatic mutations that occur by chance due to carcinogens like tobacco or the ultraviolet rays in sunlight but also “germline” mutations, which happen to cells in sperm or eggs and thus can be passed on to future generations.

Ms. Armstrong’s book comes alive in the sections where she explores cancer’s human
toll, including the devastating experience of families with rare genetic mutations, such as Li-Fraumeni syndrome, which leaves children of parents with a faulty gene vulnerable to cancer at almost any age. She also captures the excitement of researchers as they come upon eureka moments. Pediatric oncologist Michael Kastan, while treating a child with Down syndrome and cancer, began to uncover how p53 stops cell growth after reading a paper about, of all things, yeast. One of nature’s most simple organisms, yeast stops at a checkpoint in its life cycle if its DNA is damaged by radiation. This set up a challenge for Dr. Kastan: Could he identify genes and proteins that might be doing a similar job in humans? His findings helped uncover the mechanism by which p53 suppresses tumors. Though he was an unknown in the field—“a nobody with a no-technology lab,” he says—he had “happened to ask an important question.”

Today, as Ms. Armstrong makes clear, harnessing what is known about p53 to cure disease remains among the most critical, and challenging, goals in medicine. Assuming that we can replicate p53’s targeted killing of malign cells—as well as identify p53 mutations and inhibit their activity—there is the potential to prevent cancer and find new therapies. The gene may also play a role in stemming the insults of aging, as scientists explore whether p53, normally a driver of cell loss as the body ages, can be suppressed to keep aging at bay. Dr. Lane notes that he is paid to worry about p53 but that “perhaps we all should,” since the correct functioning of this single gene “is apparently all that lies between us and an early death from cancer.”

Ms. Landro writes the Informed Patient column for the Journal.