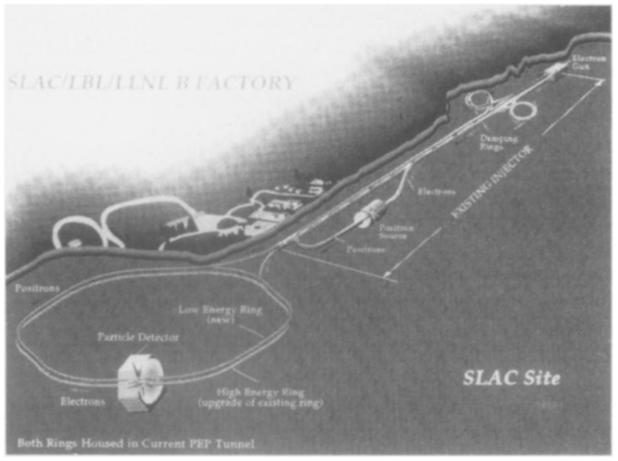
Particle physics: Stanford wins a B Factory

Mothballed since 1990, the Positron-Electron Project (PEP) particle accelerator at the Stanford Linear Accelerator Center (SLAC) had outlived its usefulness. But last week, its fortunes were revived when the Department of Energy selected SLAC as the site of a new research facility known as the B Factory.

Designed to mass-produce large quantities of subatomic particles known as B mesons via collisions between electrons and positrons (the oppositely charged, antimatter counterparts of electrons), the B Factory will be installed in the existing tunnel occupied by the PEP colliding-beam storage ring.

"This facility will be a crucial element in a balanced U.S. high-energy physics program and will ensure continued U.S. leadership on the electron frontier," says Burton Richter, SLAC director.



SLAC

In SLAC's B Factory, beams of electrons and positrons circulate in opposite directions at different energies along separate rings in a tunnel 2.2 kilometers in circumference. Inside the detector, the beams are brought together with a total collision energy of 10.6 billion electron-volts.

Construction of the B Factory would allow particle physicists to study in great detail a subatomic process—known as CP violation — that may be responsible for the overwhelming preponderance of matter over antimatter in the universe. Theorists suggest that the universe started out with equal amounts of matter and antimatter and that somehow a minuscule anomaly at the very beginning skewed this distribution toward matter.

Discovered in 1977, B mesons consist of a bottom quark paired with an anti-up or an anti-down quark. When these particles decay into other particles, a small fraction doesn't follow the usual rule that leaves parity, a characteristic of particle interactions, unchanged. Thus, it's possible in these interactions to distinguish between our world and its mirror image, even when particles are replaced by their antiparticles. By measuring the decays of large numbers of B mesons, physicists can study these rare, CP-violating events more readily.

In the B Factory, collisions between electrons and positrons in beams having a total energy of 10.6 billion electron-volts produce copious quantities of subatomic particles called upsilons. These particles, in turn, decay to produce B and anti-B meson pairs. By using electron and positron beams of unequal energies, researchers can examine the decays of B and anti-B mesons separately.

Two groups vied to host the B Factory. SLAC worked with the Lawrence Berkeley Laboratory and the Lawrence Livermore National Laboratory on a proposal to upgrade and modify its PEP accelerator to hunt for B mesons. Cornell University, which already has an accelerator with a first-rate detector for tracking B mesons, submitted its proposal at the same time. After several years of reviews by various panels and committees, SLAC won the competition.

With the cost of its PEP upgrade put at $177 million, the SLAC proposal also calls for an additional $60 million to build a detector. A significant proportion of the funding for this detector may come from foreign sources. If construction starts this year, the SLAC B Factory should be open for research in 1998.

Aimed at the elucidation of CP violation and matter-antimatter asymmetry, the B Factory complements several other particle physics projects. At the Fermi National Accelerator Laboratory in Batavia, Ill., physicists are searching for the top quark — the only one of the six types of quarks that remains undetected (SN: 4/24/93, p.264). At the Superconducting Super Collider (SSC), under construction in Texas, researchers will study high-energy collisions between protons and antiprotons to gain insights into the origin of mass.

Construction of the B Factory can start this year if Congress appropriates $36 million for this purpose in the fiscal 1994 budget. A conference committee of House and Senate members must also decide the fate of the SSC. Last June, the House voted by a large margin to kill the project (SN: 7/17/93, p.45). Late last month, however, the Senate decided by a 57-42 vote to continue funding the SSC. *—I. Peterson*

Pair wins Nobel for 'split-gene' finding

Two men whose discoveries underlie today's burgeoning genetic-engineering industry will share this year's Nobel Prize for Physiology or Medicine, the Karolinska Institute and the Swedish Academy of Sciences announced this week. Phillip A. Sharp of the Massachusetts Institute of Technology and Richard J. Roberts of New England Biolabs in Beverly, Mass., will split the $825,000 award.

In 1977, the researchers shook the genetics community with related reports based on their independent studies of a common cold virus. They found that contrary to the prevailing view — which had been based on work with bacteria — individual genes do not always reside as continuous bands of information along a strand of DNA. Rather, surplus "nonsense" segments of DNA can lie between the useful bits of each gene in nonbacterial organisms.

To activate a gene — the process by which proteins are made — a cell must copy each fragment of the gene that codes for the desired protein. Then the cell must splice them into a continuous band of RNA, which contains the information for making that protein.

Shortly after publication of Sharp's and Roberts' reports on adenovirus DNA, other researchers showed that "split genes" occur throughout higher organisms. These studies also showed that functional gene segments vary depending on the tissue in which the gene is activated or on the stage in an organism's development. In other words, the same DNA can be spliced into patterns that produce different proteins — and perform different functions.

Such observations have given rise to a new theory about the nature of evolutionary change.

Previously, biologists suspected evolution occurred as a series of accumulated, small alterations in an organism's DNA. But the split-gene finding immediately suggested that each segment of a gene might correspond to a particular subfunction of a protein. By merely shuffling these relatively large segments of DNA into new combinations, or swapping segments between genes, proteins with new functions could emerge.

Understanding the role of gene splicing also has helped researchers identify the mechanisms responsible for many genetic disorders. Beta-thalassemia, a potentially life-threatening anemia, for instance, traces to inherited gene-splicing errors. The faulty protein produced by the errors shortens the lifespan of red blood cells. *—J. Raloff*