ported were in the north. During the preceding 23 and subsequent 20 years, however, almost all were in the south. Farther out along Rubenson's chronology, other generalized shifts are visible. All told, the authors report, the data show a 106-year period, covering about 9½ solar cycles, during which the Swedish auroras underwent "a well-defined, systematic change" in their pattern of occurrence, first appearing in the south, then north, then south again and finally toward the mid-latitudes.

The shifting pattern is probably not due either to a change in observers ("there are no historical events which lend themselves to a change of observational patterns of the required scope") or of the earth's magnetic pole, whose movements over that period were too small and regular to have had much effect. But the auroral shifts do seem to have been real. Support comes from a compilation of sightings between 1780 and 1850 in New England, at a geomagnetic latitude similar to that of southern Sweden. When the Swedish auroras were southerly, others were conspicuous in New England; when the Swedish auroras moved north, above New England's geomagnetic latitude, the number of New England sightings dropped dramatically. There is even yet another catalog covering Scotland, Ireland and Great Britain from 1788 to 1835, says Siscoe, and with the appropriate latitudes figured in, all three compilations "fit like a glove."

"Since the change in auroral pattern took place simultaneously at two [or three] such widely separated locations," write Feynman and Silverman, "the cause must be in the driver of the aurora, the solar wind." Yet the pattern does not seem to follow the well-known 11-year cycle of another key solar variable, sunspots. Instead, the authors suggest, it seems more akin to an 80-to-100-year sunspot pattern known as the Gleissberg cycle or Gleissberg variation, inferred from three periods of reduced solar activity centered around 1710, 1810 and 1900.

More recently, however, there is some evidence for changes over shorter spans. In the 1958-59 International Geophysical Year, a time of maximum solar activity, about 40 percent of southern-hemisphere auroras appeared equatorward of 60° (corresponding to the dividing geomagnetic northern latitude in the Swedish catalog) and the rest between 60° and 70°. Five years later, during the 1963 International Year of the Quiet Sun, the equatorward auroras appeared only half as often. Still, the authors point out, the changes, though on a shorter timescale, "were not nearly so dramatic as that between, say, 1810 and 1850."

Variations in solar phenomena appear to take place over spans ranging from 2 to 100 years, due to a chain of relationships still being unraveled, and, the authors conclude, "we can expect that it will continue to do so in coming years." □

When is a disease not a disease?

With the vast number of biochemical reactions necessary to create the substances that maintain basic body functions, it sometimes seems a miracle that things work at all. In a condition called congenital adrenal hyperplasia (CAH), they don't work, and there are definite physical consequences, which can include such problems as early virilization and salt imbalance. But in a curious twist, researchers in New York City and Italy have identified people with the biochemical aspects of CAH who somehow escape the physical effects.

Researchers from the New York Hospital-Cornell Medical Center reported at the recent Endocrine Society meeting that they found the asymptomatic syndrome while studying the genetics of CAH. CAH is caused by the body's inability to produce the hormone cortisol, which plays an important role in normal body metabolism and maintenance of well-being. The syndrome is caused by the absence of any of a number of enzymes that catalyze chemical reactions necessary to the manufacture of cortisol.

As the body tries to produce enough cortisol to meet its demands, more and more of the precursors are manufactured. It is both the absence of cortisol and the overabundance of its precursors that lead to the physical problems.

Lenore Levine at New York Hospital started out to study the inheritance patterns of one form of CAH, in which the enzyme necessary for turning 17-hydroxyprogesterone into the immediate precursor of cortisol is absent. As the body keeps trying to produce cortisol, more and more of the 17-hydroxyprogesterone is produced. Some of it is converted to androgen, which causes early puberty and excessive virilization in affected males and ambiguous genitalia and virilization in females.

Persons with the syndrome have genetic deficits on both members of the chromosome pair that codes for the enzyme, and Levine and co-workers found a marker for this genetic anomaly. Brothers and sisters who have one of the faulty sets are carriers (as are both parents), and their carrier status can be confirmed by doing hormonal challenge tests. Since carriers only have one set of genes producing the enzyme instead of two, they will have a slight buildup of the precursor.

But when Levine and her co-workers looked at a little girl whose brother has CAH they had a surprise in store — the little girl had the excess 17-hydroxyprogesterone typical of a carrier, but she did not share the genetic marker with her brother. When Levine took a second look at the father, who she thought was a carrier, she found he had the biochemical aspects of the disease but not the physical manifestations. He actually had a "cryptic" disease expressed in his genes and hormone levels but not the short stature or early virilization typical of untreated sufferers of CAH. The daughter, who didn't share the affected genes of her brother, did have one of the cryptic genes from her father.

Levine, along with Maria New, Bo Dupont and others, working jointly with physicians in Italy, instigated a study of 600 persons in 124 families. They found eight families and a total of 16 asymptomatic "cryptics," suggesting, they say, that the enzyme deficiency may be more frequent than is now believed, and leaving them to puzzle out why the disease is evident in some genetically affected persons and not in others. □

Waiver goodbye to CO standard

A National Research Council panel recently stamped its seal of approval on the exemption of certain automobile models from the 1981 federal emission standard for carbon monoxide (CO). The panel concluded that although technology to lower CO emissions exists, it is not uniformly available to all manufacturers. The Environmental Protection Agency, therefore, is justified in granting specific waivers that allow manufacturers until 1982 or 1983 to develop emission controls.

The CO standard, the 1977 amendment to the Clean Air Act, states that by 1981 light-duty motor vehicles sold in the United States must meet a CO standard of 3.4 grams per mile (gpm), unless granted a waiver. (The standard for 1980 model cars is 7.0 gpm.) By mid-1979, the EPA had received such a diversity of waiver requests from several domestic and foreign automobile manufacturers that it asked the Council — an investigative agent of the National Academy of Sciences — to review the technological feasibility of compliance with the 1981 CO standard.

But consideration of technological feasibility has not been the basis for all EPA-granted waivers, says Clarence Ditlow of the Center for Auto Safety. "Look at the waiver they [EPA officials] gave Chrysler," says Ditlow. "They said, 'We find that the technology is available, but given Chrysler's financial straits, we don't feel we ought to put any more burden on them.'"

Ditlow says the Council's recent approval of certain EPA-granted exemptions underscores the political, rather than technological, basis for such waivers. He ways, "If this were 1977 or 1978 when car sales were still up and this weren't an election year, these [waivers] would, for the most part, be denied." □