pendent national efforts, as happened in the IGY? Or will it be necessary to set up a new international body? And not incidentally, who will manage the global data archive?

No one should have any illusion about how long this will take. The Global Atmospheric Research Project was first proposed by President John F. Kennedy in 1961; the first experiment was begun in 1978.

• Government support. IGBP has not yet gained much visibility in policy circles, so it is hard to say how enthusiastic the various national governments will be. On the positive side there is an honest scientific rationale to the program, there is a potential prestige value in participating, and there is the painful fact that in many countries, problems like acid rain and deforestation are of real practical concern.

On the other hand, there is the matter of money. The cost of IGBP is still nebulous, although the proponents have tried to be reassuring on at least one point: IGBP will *not* come on top of the existing international programs. Nor will it come at their expense. "IGBP will be a focus for their interaction," says Francis P. Bretherton of the National Center for Atmospheric Research, chairman of NASA's new Earth Systems Science Committee. "It will complement them."

• The U.S. program. "A U.S. national program doesn't make sense unless it's embedded in a global program," adds Bretherton. "The problems are so big, both conceptually and observationally, that the United States simply cannot do it alone.

"But to get a strong world program you need effective leadership," he adds, "and the United States is the *only* country able to take strong leadership. We have such a large fraction of the world's scientists that if this country can't get its act together, then the rest of the world can't."

Most scientists would probably agree with that statement. In essence, the Eddy committee is trying to formulate such a U.S. program. Congress seems receptive to having the United States take a lead. The science adviser's office at the White House likes the idea. But then, no one has asked for any money yet, either.

In the last analysis, of course, the question is really one of political will. "Scientists are more than willing to join forces," said Friedman in his keynote address at the ICSU meeting. "Governments must be persuaded that it is in their interests to support international cooperation."—**M. Mitchell Waldrop**

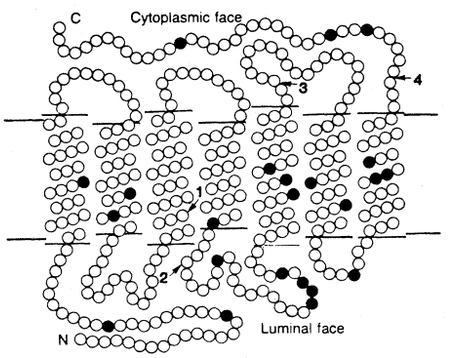
Pigment Gene Scrutinized

Jeremy Nathans and David Hogness, of Stanford University, have isolated and characterized the gene that codes for the protein component of human rhodopsin, the purple pigment that underlies high sensitivity, or night vision (*1*). Their analysis of the implied structure of the protein, opsin, gives interesting insights into its detailed conformational arrangement within the photoreceptor cells, which are known as rods. The Stanford researchers' interest in the rhodopsin gene is, however, only a stepping stone to a better understanding of the curious problem of color vision.

Abnormalities in color vision represent one of the most common and widely known genetic defects among human populations. Although it is usually assumed that the genetic aberration in color blindness affects the protein structure of the visual pigment located in one or more of the three classes of photoreceptors employed in color vision—red, green, and blue cones—this has not been unequivocally demonstrated. One very good reason for this is that no one has unequivocally demonstrated that the different absorption spectra of the cones are the result of differences in protein structure of the pigments.

Pigment profile

The rhodopsin molecule is folded and embedded in the disc membrane of the rod cell. Open circles represent amino acids in the human protein, with filled circles showing substitutions compared with bovine rhodopsin. Numbers 1 to 4 indicate intron positions.



Nathans and Hogness therefore plan to use the rhodopsin gene as a hook with which to go fishing for the putative three cone pigment genes. Analysis of the DNA sequence of these genes will give some indication of structural differences in the encoded proteins. In addition, they hope to synthesize enough of the proteins in vitro to be able to assemble the suite of cone pigments and thereby test the effect of differences in primary structure of the proteins on their light absorption properties.

Rhodopsin, like the cone pigments, is embedded within membranes, or discs, stacked within the photoreceptor cell. Drawing on a model for the disposition of a related protein, the much-studied bacteriorhodopsin within its membrane, Nathans and Hogness describe the folding of the 348-amino acid-long rhodopsin protein chain within the disc membrane. The protein passes through the membrane seven times, leaving the N-terminal region on the luminal face and the C-terminal on the cytoplasmic face. Comparing this arrangement with that for bovine rhodopsin, the gene for which the Stanford team had isolated as the initial phase of their program (*2*), shows that the three protein loops on the cytoplasmic face of the membrane are perfectly conserved. The reasonable inference is that these regions are important in initiating nerve impulse generation once the pigment has absorbed light.

Analysis of the human gene structure reveals that the coding sequences, exons, are interrupted by four noncoding regions, introns, a pattern that is repeated precisely in the bovine gene. Moreover, three of the introns (2, 3, and 4) interrupt the gene at the junction between membrane and extramembrane segments. The pattern, details of which are to be seen in membrane-anchoring segments of surface immunoglobulin and histocompatibility antigens, is nicely consistent with the idea that at least some genes are assembled from structural or functional domains.—**Roger Lewin**

References

1. J. Nathans and D. S. Hogness, *Proc. Natl. Acad. Sci. U.S.A.* **81**, 4851 (1984).
2. ———, *Cell* **34**, 807 (1983).