time. The energetic swirling of Gulf Stream rings may protect their cold, fresh cores from dissipation for 2 to 3 years, but they are often resorbed by the Gulf Stream sooner than that.

If the Meddy actually did originate in the far eastern Atlantic from Mediterranean water, it had company. Laurence Armi of Scripps Institution of Oceanography returned in June from an oceanographic cruise to the eastern Atlantic during which he found not one but three eddies containing "relatively undiluted Mediterranean water." Their salinity was elevated by 0.80 part per thousand, "a huge anomaly oceanographically," Armi notes. About 80 kilometers wide, 900 meters thick, and centered about 1100 meters beneath the surface, these eddies were near the Meddy's proposed source area (the vicinity of 32°N, 23°W). But, because their salinity anomalies are so strong, Armi says, they must have formed even closer to the Mediterranean outfall itself.

Although the origin of the water of smaller eddies has been estimated with some certainty, how the eddies form remains a matter for speculation. One possibility for the Mediterranean eddies is that the narrow outfall current becomes unstable and spins them off, perhaps as it encounters the submarine ridges and seamounts and the islands west of Gibraltar. That mechanism seems to work in the eastern Caribbean Sea, according to Thomas Kinder and his group at the Naval Ocean Research and Development Activity, Bay St. Louis, Mississippi, and George Hepburn of Science Applications, Inc., Monterey. They found three 90-kilometer-wide eddies spinning clockwise just west of the island chain of the Lesser Antilles. Because the eddies were too large to have been carried through the island passages by the prevailing east-to-west current, Kinder and his colleagues believe that the current shed them after being narrowed to the width of the island passages. Their computer model of the process supports that idea.

Physical oceanographers also continue to be puzzled by a curious aspect of smaller eddies—their apparently irregular distribution in time and space. The LDE found six eddies, but the preceding Mid-Ocean Dynamics Experiment, which used 40-kilometer spacing of observations in an area centered 400 kilometers to the south, found no immediately obvious evidence of them, according to Taft. Armi notes that his survey turned up three eddies whereas four surveys in a region to the west found anomalous water at only one out of 150 sites.

A big help in sorting out possible sources, according to McDowell, would be greater use of other tracers in addition to salinity, temperature, and oxygen. He cites the case of a second, smaller eddy found near the Bahamas by himself and Rossby that, unlike the Meddy, had only a small salinity anomaly. In addition to salinity, they used the concentrations of dissolved oxygen, phosphate, silicate, and the radioactive tritium from nuclear testing to locate the water's origin within a relatively small area 3000 kilometers to the northeast. With such increased precision, observations of small eddy formation might not be the matter of chance that their discovery was.

—RICHARD A. KERR

Do Jumping Genes Make Evolutionary Leaps?

The genomes of higher organisms are in a state of dynamic change. This must have important consequences for evolutionary change

In the last analysis it is the genome, the package of genetic material, that is inherited through parent to progeny, and so on through succeeding generations. And it is the characteristics of the genome of one species that erect a biological barrier between it and all other species. It is therefore natural to scrutinize the various components of genome structure to determine their functions in an evolutionary context. Molecular and evolutionary biologists met recently in Cambridge, England, to do just this.*

The all-pervading message of the Cambridge meeting was that genomic DNA is in a surprisingly dynamic state. "Whenever you look at a plant genome you see indisputable evidence for sequence amplification and rearrangement," said Richard Flavell of the Plant Breeding Institute, Cambridge. "Everything you say about plants can be applied to sea urchins," responded Eric Davidson of the California Institute of Technology. Such comments were reiterated many times, embracing a wide range of organisms. The notion of a fluid genome, in which there is a constant flux of sequences, is now an accepted fact. The question is, what does it imply?

This question, particularly its evolutionary ramifications, applies to three major levels: chromosomal architecture, the status of repeated sequences, and the function of structural genes. Perhaps most intriguing of all, however, is whether the issue of fluidity is involved in the establishment of a biological barrier between species.

The most obvious comment to make about the genomes of higher organisms is that biologists understand the function of only a tiny proportion of the DNA in them: namely, the genes that code for proteins. In the human genome, for instance, these protein-coding genes constitute marginally more than 1 percent of all the DNA. The rest of the genome is the target of much speculation, but few secure answers.

The DNA in the human genome that does not code for proteins falls into three classes. Some 5 percent is made up of many families (sometimes with many millions of members) of short, simple repeats of nucleotide sequences known as satellite DNA. A quarter of the genome is formed of families of longer more complex repeat sequences, denoted intermediate repetitive DNA. The bulk of the genome is composed of unique sequence, or single copy, DNA that is interspersed with the intermediate repeats. The proportions of these classes, whose distinctions can often become blurred, can vary enormously between organisms.

The longer biologists searched in vain for functions for these three classes of noncoding DNA, the stronger grew the conviction that much of the DNA might well be "junk" that for some reason could be tolerated in the nuclei of higher organisms. The most recent intellectual

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assault on the origin of putative junk DNA was initiated 14 months ago with the publication of a brace of papers in *Nature* (vol. 284, pp. 601 and 604). Ford Doolittle and Carmen Sapienza of Dalhousie University, Nova Scotia, and Leslie Orgel and Francis Crick of the Salk Institute, California, sought in their papers to promulgate the notion that much of the noncoding DNA could be described as selfish DNA.

There followed a flurry of publications on the merits of the proposal. Scientific argument and semantic posturing admixed to produce a debate which, in Orgel's view at least, reached comic proportions. Reverberations of the debate could still be perceived at the Cambridge meeting, though John Maynard Smith, a population geneticist from the University of Sussex, England, cut through what confusion remained with this clear statement: "If there are elements in the genome that can multiply, and if certain structures of these elements can influence their replication, then it follows logically that there will be selfish DNA."

Gabriel Dover, of the Genetics Department, Cambridge, England, argued that the proportion of DNA that has the ability to influence its own replication might be small. Nevertheless, observation of families of repeated sequences in many genomes indicate that DNA is passively and accidentally multiplied by diverse mechanisms. The processes are stochastic and DNA may be said to be ignorant of what is happening to it. There was general agreement on the distinction between selfish and ignorant DNA.

Notwithstanding the origin of this DNA, the key question is, how important is it? Maynard Smith said that the selfish or ignorant origin of DNA does not necessarily imply that it has no effect on its host. Some sequences might become involved in signaling, and all of them, simply by increasing the amount of DNA in the nucleus, might influence the size and cycle time of the host cells.

Karyotype, that is, the number and overall patterns of chromosomes, differs widely throughout the living world, but there is a striking degree of consistency within groups. For instance, there is a generalized mammalian karyotype, and a separate one for reptiles, and so on. There is an even more surprising conservation of the disposition of structural genes over the chromosomes of even distantly related organisms. Mobility of sequences in the genome is mostly, but not exclusively, the prerogative of the repeated sequences. The genome can therefore be viewed as relatively stable islands of structural genes immersed in a steadily shifting tide of changing repeated sequences.

Discussion of the coordinated action of related genes frequently contains the very reasonable assumption that they will be arrayed as neighbors along a chromosome. Apparent exceptions to this, such as the separation of the α- and β-hemoglobin clusters on chromosomes 11 and 16 in humans, are a puzzle. However, if Michael Bennett, of the Plant Breeding Institute, Cambridge, is correct in his analysis of chromosome ordering in nuclei, this might not be a problem.

According to Bennett, the lengths of chromosome arms determine, in a stochastic way at least, the order in which a set of chromosomes will link together and organize themselves within the nucleus. An inevitable consequence of a preferred spatial disposition of chromosomes would be the establishment of certain groups of structural genes as close, and possibly functional, neighbors, even though they are on different chromosomes. "No, there is no direct evidence for this," admitted Bennett, "but it is a possibility worth considering." Alec Jeffreys, of Leicester University, welcomed the idea as potentially "extremely important." Others were more skeptical about the possibility of genes conversing across gaps between adjacent chromosomes.

If cross talk between genes in three dimensional space is real and important, then this would clearly place constraints on modifications of chromosome structure if the integrity of an organism is to be maintained. At least as important in this respect is the requirement that homologous pairs of chromosomes come together and engage in recombination during the formation of gametes.

It was therefore something of a surprise at the Cambridge meeting when Hugh Rees, of the University of Wales, Aberystwyth, described the successful production of fertile progeny from two species of rye that differed in DNA content by 40 percent. The chromosome number and pattern of the two species were the same, but a difference in DNA content of this magnitude is quite a challenge to normal homologous pairing. Flavell offered the suggestion that perhaps only certain sets of sequences along the length of the chromosome are required for effective pairing. Perhaps the DNA changes that have accumulated between these two species of rye do not involve these crucial sequences, he said.

When one moves from the first level of analysis, from chromosomes to repeated sequences, the data available become plentiful, as do the questions about them. Three properties unite satellite and intermediate repetitive DNA. First, it is clear that there are mechanisms in all genomes that can both amplify and delete repeated sequences to produce a steady turnover of such sequences. Second, there are also mechanisms that ensure a homogeneity of sequence within repeated sequence families. This phenomenon is known as concerted evolution. Last, part of the natural history of repeated sequences involved transposition both within the chromosome of origin and to other chromosomes.

The all-pervading message of the Cambridge meeting was that genomic DNA is in a surprisingly dynamic state.

Beyond these common properties of satellite and intermediate repetitive DNA, there are clear differences. The most striking being that, probably without important exception, satellite sequences are not transcribed into RNA copies. A strong indication emerged that satellite DNA may be involved principally with chromosomal architecture.

By contrast, as Roy Britten, Davidson, and their colleagues have abundantly demonstrated, many families of intermediate repetitive DNA are interspersed with protein-coding genes and transcribed, and in intriguing ways. More than any other macromolecule, these transcripts show consistent differences between tissues and between different stages of development. This observation has led Britten and Davidson to propose a model in which these repeated sequences play an important role in controling the expression of protein-coding genes. The case remains to be proved.

The apparent mobility of repeated sequences and of the larger, less common structures known as transposable elements must have important consequences. As Davidson commented, "From an evolutionary point of view there is a growing conviction that mobile sequences in the genome may have played an important part in moving

genes around, into and out of regulatory positions."

In Davidson's own contribution concerning the effect of genome rearrangements on the functioning of structural genes, he described a cogent example of their potential for inducing variation. Actin is a fibrous protein, or rather family of proteins, that performs various structural jobs in muscles and in the scaffold-like superstructure of cells. Organisms have a family of genes that code for the different actin variants, and each variant has several genes that are active at different times in development.

If genes move from one place to another in the genome, Davidson argued, they might find themselves in a discrete functional region in which they will be recruited to do a slightly different job. "Old genes in new contexts give new morphology," says Davidson. An indication of the truth of this aphorism comes from the discovery that the gene coding for cytoskeletal actin of vertebrates is the same one found in the muscles of mollusks.

The structural genes with the most closely charted evolutionary history are those coding for globin, the protein component of hemoglobin. As mentioned, human globin genes are arranged in two clusters, α and β, on two separate chromosomes. Jeffreys explained that by studying the structure of globin clusters in a wide range of organisms it has been possible to reconstruct the evolutionary path from a single primordial globin gene of some 500 million years ago to the most highly evolved arrangement found in humans. The path involved duplication of the gene to two closely linked genes which were the ancestors of the α-like and β-like clusters, the members of which came to be used in a coordinated way at different points of development. In addition the α and β clusters became separated onto different chromosomes, with further events creating greater divergence within them.

The five globin genes in the β cluster of humans are spread over a long stretch of DNA, measuring nearly 60 kilobases. The genes themselves occupy only 8 percent of the region. The question of what the rest of the DNA does has frequently been answered, nothing, it is junk. Jeffreys analyzed this DNA in detail throughout the primate order and found that the single copy and repeated sequence DNA that make up the region are strongly conserved. "We had set out to test the junk hypothesis," said Jeffreys, "and we were happy to rule it out provisionally in that these sequences have behaved in evolution as if they are functional." What this function is must still be determined.

Fascinating and intellectually satisfying though the story of globin gene evolution is, there are hints of a subplot that could have far-reaching importance. Legumes produce a protein called leghemoglobin, which is used in nitrogen fixation. The gene for this molecule has all the appearance of a primitive globin gene. The most obvious explanation for the presence of an animal's gene in the genome of a plant, suggested Jeffreys, is that it was translocated there relatively recently in evolution as a passenger on a virus. This possibility of horizontal transmission of genes is tremendously exciting, Jeffreys said. "Many people have a suspicion that this can happen," commented Davidson.

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Such a mechanism would of course circumvent the rules of classic Mendelian inheritance—a tantalizing possibility. Equally intriguing and almost as heterodox is Dover's proposal for the contribution of repeated sequences to the origin of new species. Dover's hypothesis rests on two points. First, members of individual families of repeated sequences tend to be structurally extremely similar to each other, through concerted evolution. Second, when a variant arises in a family through mutation there is a statistical probability, given the constant turnover of repeated sequences, that it will spread through the population until it has replaced the original form. This could be important for speciation, says Dover, if the presence of the new variant throughout the repeat family introduces reproductive incompatibility between one population of individuals and another.

An important aspect of the proposal is the concept of drive. A family of repeated sequences is in a dynamic state of turnover, through constant amplification and deletion of sequences. When a mutant arises it will either be eliminated or it will steadily become the dominant form through the turnover process underlying concerted evolution. The homogeneity eventually extends to all chromosomes, indicating that unequal exchange, direct transposition or mechanisms analogous to gene conversion are involved with the "horizontal" drive of the variant between chromosomes. An individual that begins life with the variant in just one of its pair of chromosomes will produce an excess of gametes carrying the variant, because copies of the sequence will have transposed between the chromosomes. The net result of this will be that the variant will increase in frequency in the population.

In classic population genetics the frequency of a gene may increase in a population if it confers greater fitness, thus giving rise to greater numbers of progeny bearing it. There is no question of such a gene duplicating and hopping between chromosomes to boost its rate of spread through the population. In Dover's hypothesis, the frequency and distribution of the new sequence in a population is determined by the rate of transfer between chromosomes, not necessarily by natural selection. The consequence, said Dover, would be the accidental genomic differentiation of a population which might produce "accidental speciation." Dover also suggested that the mechanism is compatible with the notion of long periods of evolutionary stasis, when repeat families are steadily turning over and being maintained by concerted evolution, interspersed with bursts of change, when a mutation is driven through an isolated population.

The hypothesis is audacious, but its force of logic and consistency with many experimental observations of concerted evolution of families of genes and noncoding sequences drew a good deal of sympathy. Maynard Smith gave it cautious approval on behalf of population geneticists when he said, "Dover may be right." He was anxious to emphasize, however, that there must be many modes of speciation that are determined by circumstance. Dover agreed.

The Cambridge meeting was timely in that it brought together two rapidly moving and converging lines of investigation. "Evolutionary biologists are going to have to take molecular biology seriously," said Maynard Smith in summary, "especially because of the demonstration of elements in the genome that can multiply outside the classic Mendelian framework. Molecular biologists must, however, realize that a lot of work has been done on evolutionary biology. Familiarize yourselves with it."

—ROGER LEWIN