Breeding Butterflies and Culling Cuckoos: How Climate Change Affects Biodiversity

By Pien Huang

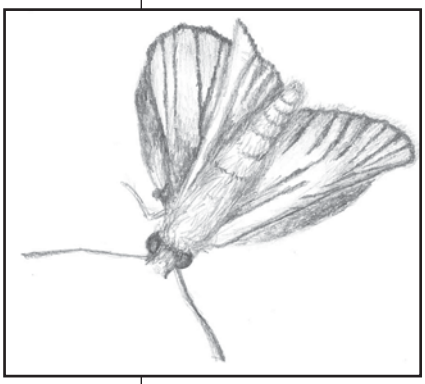
We usually think of climate change in a geophysical sense: we speak of storm patterns, heat waves, melted icecaps, and flooded coasts. These phenomena are immediately pertinent to human life. Yet it is inevitable that global warming will also affect the environment for the estimated 30 million other species that share this planet with us (1). The distribution of biodiversity is being changed in ways that ecologists are just beginning to understand. Some species are in tragic decline, while others are poised to invade areas that were previously outside their inhabitable range. Two articles from the British Ecological Society's Journal of Animal Ecology examine the impact of climate change on animal populations.

The silver-spotted skipper butterfly (*Hesperia comma*) is native to southern England where its preferred food, sheep's fescue, grows in small tufts on open grasslands (2). The conditions required for *H. comma*'s egg deposition are extremely temperature-sensitive: even within their living ranges, egg-laying is limited to microhabitats such as warmer, south-facing hillsides. In the 1980s, populations dropped to endangered levels; however, a study by Davies *et al.* shows that *H. comma* is now staging a comeback (3). Davies *et al.* combined literature with field experimentation to track egg-laying behavior. They identified a positive linear relationship between egg-laying rates and ambient temperature, and they further realized that larger portions of the local environment were becoming suitable for egg-laying. Between 1982 and 2001, the optimum percentage requirement of bare ground for egg-laying shifted from 41% to 21%, indicating sufficient warming in grassy, vegetated microhabitats (3).

As temperatures continue to rise, the authors predict a boom in the population of silver-spotted skipper butterflies; in particular, the northern geographical margins of their current home range are expected to become more hospitable. The authors warn that changes in global climate patterns are likely to shift the butterfly's range out of currently protected areas and into regions that are less inviting and insufficient for sustaining populations (3).

On another continent, data on the North American yellow-billed cuckoo (*Coccyzus americanus*) tell a different tale. Most existing studies of species have narrow applicability across geographical regions due to their focus on a few limited subpopulations. A study by Angela Anders and Eric Post of Pennsylvania State University circumvents the issue of spatial heterogeneity by observing trends across the entire species distribution of *C. americanus* (4). Anders and Post compared long-term climate data with information on all populations of *C. americanus*, which had been collected by the US Geological Survey's Breeding Bird Survey from 1966 to 2002. Cuckoo population densities at breeding sites were found to decrease after warmer years, and populations with breeding grounds located in areas more susceptible to changing climatic patterns declined the most. The authors posit food scarcity as a mechanism for decline, and suggest that *C. americanus*, which feeds on butterfly and moth larvae, is doubly defeated in warmer temperatures: certain caterpillars are less abundant after warm winters, and others peak earlier in the spring. As a late migration landbird, *C. americanus* may miss the feast (4).

The analyses presented in these papers are rooted in empirical fact—climate change is affecting the distribution of biodiversity. For some species, global warming is favorable; the silver-spotted skipper butterfly hovers at the brink of a population explosion. For others, such as the yellow-billed cuckoo, the future is perilous. These papers document evolution in



▲The silver-spotted skipper butterfly (*Hesperia comma*) is poised to expand north of its current habitat in southern England.

▼The decline of the North American yellow-billed cuckoo (*Coccyzus americanus*) correlates strongly with patterns of climactic warming over the past forty years.

credit: Chelsea Gordon, HSR

action; they record the effects of environmental change on population dynamics, and offer predictions for the survival of the fittest species as the tropical zones expand. Climate change is not restricted to the geophysical realm; its consequences on biodiversity and the balance of life could leave a haunting legacy. H

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Fragile-X: A Frontier in Neurology

By Cristina Fernandez

Whenever learning disorders are mentioned, autism, attention deficit disorder (ADD), and attention deficit and hyperactivity disorder (ADHD) are among the first to come to mind. A less well-known learning disorder is Fragile-X Syndrome, despite the fact that it is the most commonly inherited cause of mental impairment. While the disease itself was discovered in 1942, a test was not developed until 1969. Even then, the disease was not readily diagnosed until molecular tests became available in 1991 (1).

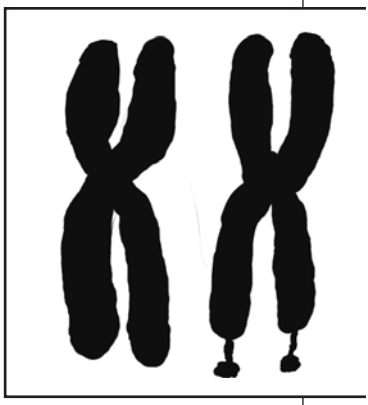
Fragile-X arises due to a mutation in the FMR-1 gene, which encodes a protein that is essential for normal brain function. The mutation involves an expansion of the normal gene sequence by multiple repeats of a particular three-nucleotide sequence. Repetition of this three-nucleotide sequence between 50 and 200 times increases the risk of transmission of the disease to the next generation. The full mutation occurs when there are more than two hundred repeats. In such a case, the patient is fully affected by Fragile-X syndrome (2).

The Fragile-X mutation occurs on the X chromosome and therefore follows its pattern of inheritance. Fathers who are premutation carriers – namely, who possess between 50 and 200 three-nucleotide repeats – can only pass the premutation on to their daughters because for reasons that are still unclear, repeats are not added when the father contributes the X chromosome. Mothers, on the other hand, can pass the full mutation on to their sons or daughters because repeats are added (2). The likelihood that a premutation will develop into a full mutation increases with each generation, and the children of female carriers have a 50% chance of acquiring the full mutation. In many cases, premutations can increase in severity from generation to generation without any sign that the family is carrying a genetic disorder (2). This can occur because carriers do not exhibit any signs of abnormality.

From a biochemical perspective, Fragile-X is caused by the inability of affected individuals to produce the FMR-1 protein, which is involved in shutting down extraneous neural connections during development. Affected individuals show impaired cognition and communication, and often have difficulties with social skills and attention. Moreover, they often exhibit hyperarousal, a condition characterized by easy over-stimulation and by a tendency to overreact to changes in one's environment and routine (2). Nevertheless, it is important to note that the manifestations of the disease vary from patient to patient and that the severity of mental retardation is highly variable.

Interestingly, Fragile-X also appears to affect carriers, or individuals who can transmit the disease but who do not exhibit any symptoms. In the last three years, researchers have discovered that older adult male carriers, usually grandfathers or great-uncles of children with Fragile-X, are prone to developing a neurodegenerative disorder similar to Parkinson's disease (3). This disorder, Fragile X-Associated Tremor Ataxia Syndrome (FXTAS), is characterized by tremors, problems with balance, and memory loss. The disease is usually progressive, resulting in the loss of white matter in the brain, and is almost impossible to treat (3).

Recently, the laboratory of Randi Hager-



▲Normal X-chromosome (at left) is compared to a fragile X chromosome (at right).

credit: Chelsea Gordon, HSR

man, a leading researcher of Fragile-X and related disorders, performed a study on carriers of the FMR-1 mutation. According to their findings, elevated levels of FMR-1 messenger RNA (mRNA) were directly related to such symptoms as obsessive-compulsive behavior, psychosis and FXTAS in male subjects (4). These results support the hypothesis that overproduction of FMR-1 mRNA is responsible for causing FXTAS.

In an effort to identify the proteins that are key in mediating and controlling the abnormal response to the expansion mutation held by Fragile-X carriers, the Hagerman laboratory has identified about twenty proteins believed to be involved in mediating the toxic effects of the FMR1 mRNA (5). In trying to determine the cause of neural degeneration associated with the expansion mutation of the FMR1 gene, the Hagerman laboratory has observed that an elevated level of FMR1 mRNA results in reduced cell viability (6). The reason for this reduced cell viability is the presence of inclusions, or small pockets of foreign matter within the brain. These inclusions greatly reduce brain function and increase in size over time (6). The elucidation of the mechanistic aspects of FXTAS is important for understanding FMR1's role in neural development, as well as for developing a method of treating Fragile-X and FXTAS.

The implications of FXTAS for Fragile-X families are highly significant; the implications for the larger population are unknown, as many cases of FXTAS are probably misdiagnosed. When considering the wealth of knowledge that can be gained by studying the FMR-1 gene and its associated diseases, there is indeed a frontier of neurology that has yet to be explored.

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