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This Week

Research Notes

Articles

Cover: As fatty deposits accumulate, they narrow the coronary arteries supplying blood to the heart — a process that can lead to a heart attack. Some scientists believe a disorder called insulin resistance lies at the core of this process in certain people. (Illustration by Thomas Sims/© American Heart Association)

Departments

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Letters

Only the beginning

Dregs? What a way to describe a youthful galaxy ("The Dregs of the Universe," SN: 7/22/89, p.60) that hasn't even arrived at its maturity! Remember, we're seeing it as it was nearly three-quarters of a billion years ago. It takes time to draw in all that tenuous hydrogen, but time is what Malin 1 and its sibling have plenty of.

Arthur J. Morgan
New York, N.Y.

Psychiatric zeitgeist

A statement in "An early start for panic" (SN: 7/22/89, p.61) carries the seeds of an early start for high blood pressure in some psychiatrists.

"The children significantly improved when treated with drugs that ease the panic and agoraphobia of adults," you write. "Each child also attended psychotherapy sessions."

First, if psychotherapy and drugs were used concurrently, one can say very little about the contribution of either; yet the report implies efficacy for medication and mentions psychotherapy as if by afterthought.

Second, while the present zeitgeist in psychiatry favors chemistry, the extent to which anxiolytics "ease the panic" is still a matter of lively debate.

Malcolm Stewart-Morris
Psychiatrist
Oakland, Calif.

Not just natural selection

I understand J. Eric Triau's comments (Letters, SN: 7/15/89, p.35) concerning Ingrid Wickelgren's misleading use of the verb "evolve," but Triau employs an equally confusing use of the noun "evolution."

Triau uses this word as synonymous with Darwin's theory of evolution, or evolution by natural selection. His interpretation negates other important processes of evolution such as mutation, recombination, genetic drift, migration and inbreeding. Clearly, natural selection interacts with these other processes during evolution, but these are nonetheless considered other valid processes of evolution. If we are looking at subtle misinterpretations, as Triau states he is, then he incorrectly describes evolution as "the nonrandom trend of species toward adaptation based on successful transmission of random mutations conferring survival."

April Ann Fong
Davis, Calif.

Proximate vs. ultimate

William Check (Letters, SN: 7/15/89, p.44) suggests that behavioral scientists are placing too much emphasis on genetic explanations of social behavior. He cites our work on African bee eaters ("Avian Altruism," SN: 6/10/89, p.364) in which we found that: (1) nonbreeding helpers at the nest greatly increased the production of young fledging from such nests; (2) helpers were closely related to the young being reared; and (3) helpers chose to aid close genetic relatives rather than distantly related or unrelated

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"We conclude that healthy persons with [high blood insulin levels] have an increase in risk factors for coronary artery disease, as compared with a well-matched group of healthy subjects with normal insulin levels," the researchers write in the March 16 NEW ENGLAND JOURNAL OF MEDICINE.

Last June, at the annual meeting of the American Diabetes Association in Detroit, French researchers reported that long-standing elevations in blood insulin levels do seem to predict a greater threat of fatal heart attack. Annick Fontbonne and her colleagues at the French National Institute of Health and Medical Research in Paris found a link between blood insulin values and heart disease mortality in a 15-year study of 7,038 Paris policemen, whose ages ranged from 43 to 54 at the study's outset.

"Our studies indicate that the earliest marker of a higher risk of coronary heart disease mortality is an elevation of plasma insulin level," Fontbonne says. When the researchers looked back at their records, they found that the 169 men who died of heart disease during the project had entered the study with fasting insulin values averaging about 20 percent higher than those of survivors. Insulin values measured two hours after a glucose test were 13 percent higher for the 169 than for the survivors, Fontbonne says.

U.S. research also implicates insulin resistance in the development of some forms of coronary artery disease. At the University of Pittsburgh School of Medicine, a new study of 489 healthy, white, premenopausal women aged 42 to 52 shows an increasing risk of heart disease as blood levels of insulin rise. Women with the highest blood insulin levels proved the most likely to have high blood pressure, elevated triglycerides and decreased HDL cholesterol values.

"The finding that insulin, independent of obesity, is related to coronary heart disease risk factors is an important development," study director Rena R. Wing told SCIENCE NEWS. She stresses that many of these women appeared quite healthy. The average study participant weighed 145 pounds and had a total blood cholesterol count of 185 mg/dl, a level considered desirable by the federal National Cholesterol Education Program. Wing and her colleagues, who describe their work in the July/August ARTERIOSCLEROSIS, plan to study the same women as they go through menopause to see whether early signs of coronary disease will lead to higher heart disease mortality rates.

Despite these intriguing findings, the mechanism by which insulin resistance might cause heart disease remains a mystery. Some scientists speculate that high insulin levels may directly damage the artery wall, leading to a lipid buildup that narrows the vessel. Alternatively, insulin's regulation of lipid metabolism may alter the triglycerides or HDL cholesterol in the blood, a process that could lead to artery disease, Reaven notes.

Scientists have yet to unravel the complicated relationship between the genetic and environmental factors underlying insulin resistance and, by inference, syndrome X. A number of scientific reports suggest insulin resistance is inherited, but researchers don't know whether one gene or several control its development. And while scientists do know that obesity often impairs insulin response, they don't know whether other factors might also trigger insulin resistance.

"The most likely possibility is that syndrome X is genetically determined, but the genetic defect remains hidden without some environmental precipitant — probably one involving diet," writes Foster, of the University of Texas, in an editorial accompanying Reaven and Zavaroni's March 16 research report. "Thus, if insulin resistance is pathogenetic, it is more likely an accomplice than the sole killer."

If more extensive research verifies syndrome X, clinicians of the future might screen patients for insulin resistance by monitoring blood insulin levels or by looking for potbellies, Reaven and McKeigue say. Once identified, people with insulin resistance might lower their coronary risk by losing weight or boosting their physical activity, since both measures can help the body use insulin more efficiently, Reaven says.

For Type II diabetics, the theory's implications seem doubly dangerous. "If mild elevations of insulin are harmful, that's a fairly worrisome thing for people who treat diabetes, because diabetics have excess insulin around all the time," comments Foster. Most clinicians focus on controlling diabetics' blood sugar levels and haven't worried much about the heart dangers caused by excess insulin, he says.

Reaven, Fontbonne and other proponents of the theory believe high blood insulin indicates insulin resistance and triggers the process leading to heart disease. But skeptics say high blood insulin could simply flag some as-yet-unknown process that damages heart arteries. They contend insulin resistance itself might play no role, or only a minor role, in the development of heart disease.

Reaven agrees the theory requires further study, but he remains convinced that the experimental evidence strongly suggests that insulin resistance causes heart disease in some people.

"People don't like to have the dogma questioned," he says, "but I've been doing that for 20 years." □

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birds. From these findings we concluded that kin selection is an important force maintaining helping behavior among bee eaters.

Check is correct in suggesting that a proximate mechanism underlying both kin recognition and nepotistic favoritism in helping could be based on early social learning of nestmates. In fact, our cross-fostering experiments, involving exchanges of young between nests, suggest the mechanism of kin recognition *is* based on such social learning.

However, Check confuses proximate and ultimate explanations of behavior. Studies of the *proximate* mechanisms responsible for the choice of a recipient of helping behavior do not address the question of the *ultimate* reason that helping behavior is found in white-fronted bee eaters but not, for example, in red-winged blackbirds. Nor do such studies address the question of how particular behaviors are maintained in populations — i.e., how behaviors expressed by certain individuals ultimately affect the survival and reproduction of additional individuals.

Questions at this level involve studying the functional *consequences* of helping. Behavioral ecologists usually address these questions by measuring the costs and benefits associated with the particular behavior. If helping involved a fitness cost to the performer (for example, if the extra energetic stress resulting from provisioning nestlings led to a greater probability of mortality), then one would predict that, through time, the expression of helping would decrease in frequency and be eliminated. The reason is straightforward Darwinian selection. Individuals that helped less would survive better. This line of reasoning does not imply that there is some single gene for helping; it merely requires that the flexible expression of helping have some heritable component.

Our study demonstrated that helpers among bee eaters *do* incur a personal fitness cost by helping. But through their actions, they also increase the production of younger genetic relatives. When the personal cost and the kin benefit are summed (with appropriate devaluations for differing degrees of relatedness), helping leads to an increase in what William D. Hamilton termed the inclusive fitness of the helper. To state it somewhat differently: If bee eater helpers selected the recipients of their aid randomly, rather than showing kin discrimination, there would be no kin benefit to helping and the personal cost of the behavior would cause it to disappear from the population. It was this reasoning that led us to conclude that kin selection is responsible for maintaining the current expression of helping among bee eaters.

Note that knowledge of the costs and benefits of helping does not aid us in answering the proximate question of how bee eaters recognize their kin. Both proximate and ultimate questions are worthy of study, but they require different types of answers. Social behaviorists will continue to ask both sorts of questions, but they must remember that proximate explanations should not be taken as answers to ultimate questions, nor vice versa.

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