



## Viviparity and Testability

Daniel G. Blackburn

*Science*, New Series, Vol. 241, No. 4864. (Jul. 22, 1988), p. 399.

Stable URL:

<http://links.jstor.org/sici?sici=0036-8075%2819880722%293%3A241%3A4864%3C399%3AVAT%3E2.0.CO%3B2-Y>

*Science* is currently published by American Association for the Advancement of Science.

---

Your use of the JSTOR archive indicates your acceptance of JSTOR's Terms and Conditions of Use, available at <http://www.jstor.org/about/terms.html>. JSTOR's Terms and Conditions of Use provides, in part, that unless you have obtained prior permission, you may not download an entire issue of a journal or multiple copies of articles, and you may use content in the JSTOR archive only for your personal, non-commercial use.

Please contact the publisher regarding any further use of this work. Publisher contact information may be obtained at <http://www.jstor.org/journals/aaas.html>.

Each copy of any part of a JSTOR transmission must contain the same copyright notice that appears on the screen or printed page of such transmission.

---

JSTOR is an independent not-for-profit organization dedicated to creating and preserving a digital archive of scholarly journals. For more information regarding JSTOR, please contact [support@jstor.org](mailto:support@jstor.org).

## AIDS Risk

Harry Rubin states (Letters, 10 June, p. 1389) that available information on the prevalence of human immunodeficiency virus (HIV) infection in the United States and on the risk of AIDS after infection with HIV are inconsistent with observed AIDS incidence. He perceives a "three- to fivefold shortfall" in the observed number of AIDS cases in 1986 (compared with the expected number) and attributes this to differences in risk of AIDS after HIV infection resulting from differences in behavior. Rubin goes on to imply that the probability of developing AIDS after HIV infection may be much lower for heterosexuals who do not use IV drugs than for other HIV-infected individuals and that observed latency periods (times between HIV infection and onset of AIDS) of more than 5 years may be "an illusion created by the need for chronic or repeated exposure to an array of microorganisms or immunosuppressive practices." As we demonstrate below, this reasoning is flawed.

The basis of Rubin's argument for an inconsistency between the 16,000 AIDS cases diagnosed in 1986 and the estimated 1 to 1.5 million persons infected with HIV is that approximately 5% of infected people will develop AIDS each year. In fact, available epidemiologic data indicate that the risk of developing AIDS is not constant, but rather increases with time since infection (1). Therefore, people who have been infected for several years have a greater risk of developing AIDS in the coming year than those recently infected. It follows that the proportion of an HIV-infected population that develops AIDS in a 1-year period depends on the distribution of time since infection in this population. This distribution changes over chronologic time because those infected longer develop AIDS at a higher rate and because the rate of new infection also changes over chronologic time.

In order to determine whether the observed number of AIDS cases is consistent with an estimated number of HIV-infected individuals, it is necessary to take into account the dynamics of the epidemic, that is, changes over time in the prevalence of HIV infection and the incidence of AIDS. Modeling these dynamics under the assumption of a common latency distribution (but allowing different mixes of times since infection) demonstrates that a large range of cumulative infection curves are consistent

with the observed annual incidence of AIDS (2, 3). Indeed, this is one of the approaches used to arrive at the estimate of 1 to 1.5 million people infected with HIV (3). There is, therefore, no inconsistency between the estimated number of people infected with HIV and observed AIDS incidence.

While these results contradict Rubin's statement, they do not prove that behavior or mode of infection is unrelated to latency. Although there is no direct epidemiologic evidence for differences in the latency distribution among gay men, transfusion recipients, and hemophiliacs (4), it would be premature to conclude that no difference exists because of limitations of available data both in structure and in sample size. Precise quantification of this relation will require follow-up of cohorts of individuals infected with HIV from different kinds of contact.

V. DE GRUTTOLA  
S. W. LAGAKOS

Department of Biostatistics,  
School of Public Health,  
Harvard University, Boston, MA 02115

### REFERENCES

1. J. Curran *et al.*, *Science* **289**, 610 (1988).
2. V. De Gruttola and S. Lagakos, "The value of AIDS incidence data in assessing the course of the AIDS epidemic" (Technical Report No. 564, Dana-Farber Cancer Center, Boston, MA, 1987).
3. Centers for Disease Control, *Morbidity and Mortality Weekly Report* **36**, S-6 (1987).
4. M. Eyster *et al.*, *Ann. Intern. Med.* **107**, 1 (1987); K. J. Lui, *Proc. Natl. Acad. Sci. U.S.A.* **83**, 2913 (1986); G. F. Medley, R. M. Anderson, D. R. Cox, *Nature* **328**, 719 (1987); S. W. Lagakos, L. M. Barraj, V. De Gruttola, *Biometrika*, in press.

## Viviparity and Testability

Two recent explanations for the absence of viviparity in birds have been described by Roger Lewin (Research News, 17 Oct. 1986, p. 285; 29 Jan. 1988, p. 465), one by Blackburn and Evans (1) and the other by Anderson, Stoyan, and Ricklefs (2). Although the dialogue on this issue is continuing elsewhere, the alternative interpretations thus far expressed ought not obscure a significant area of agreement: the utility of framing the relevant evolutionary hypotheses in testable terms.

Historically, macroevolutionary issues often have been judged on the basis of plausibility and concordance with unquestioned assumptions and accepted paradigms. When dealt with in terms of falsifiable predictions and testable assumptions, evolutionary questions are brought fully within the bound of empirical science, minimizing the roles of individual bias, chance, and disciplinary parochialism. The universality of oviparity among birds is an example of a long-

standing evolutionary issue that remains approachable, if indirectly, through empirical investigation and rigorous theoretical analysis.

DANIEL G. BLACKBURN  
Department of Biology,  
Trinity College,  
Hartford, CT 06106

### REFERENCES

1. D. G. Blackburn, and H. E. Evans, *Am. Nat.* **128**, 165 (1986).
2. D. J. Anderson, N. C. Stoyan, R. E. Ricklefs, *ibid.* **130**, 941 (1987).

## Military Funding of Biological Research

Reviewing a book on the military-physics relationship in postwar America (15 Apr., p. 341), Karl Hufbauer comments that chapter author Paul Forman's perspective "would surely need major modification for those disciplines such as the biological sciences where military patronage is small."

Military patronage in the biological sciences is by no means small, as attested by federal contracts cited in *Commerce Business Daily*. In a single day's issue (1), large biological research contracts (for more than \$100,000) awarded by the Office of Naval Research alone have totaled \$1,850,217.

KARIN WISIOL  
Karin Wisiol & Associates,  
614 Indian Road, Glenview, IL 60025

### REFERENCES

1. *Commerce Business Daily* (7 April 1988), p. 21. "The Physiology of Selective Attention" (\$324,682); "Research on Creation of a Digital Database of Marine Animal Sounds" (\$350,000); "Natural Anti-fouling Strategies in Gorgonian Corals" (\$129,954); "Molecular Regulation of Light Adaptation and Nitrogen Assimilation in Marine Diatoms" (\$210,000); "Continued Research to Develop Procedures for Enzymatic Conversion of A Red Cells to Type O for Transfusion" (\$265,000); "Molecular Basis of RNA Catalysis" (\$240,531); "Optical Neuromorphs" (\$330,050).

## Longevity and Gender

R. Fischer (Letters, 8 Apr., p. 130) suggests that the main reason why women live longer than men is that they burn their "fire of life"—to use Kleiber's phrase (1)—more efficiently. He falls into the trap, however, of expressing energy expenditure in terms of surface area. For reasons that are not entirely clear this appears to be satisfactory for interspecies comparisons over a wide range of body mass, from mouse to elephant (1). It does not, however, hold for comparisons between the sexes in humans, where body fat as a percentage of total body weight in