and Johnsen 1986:97). In this view, knowledge generation and knowledge utilization are inseparable.

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On Infection and Sex Ratio of Offspring

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Sieff's article on explaining biased sex ratios in human populations (CA 31:25–48) overlooks the effect of hepatitis B infection on the sex ratio of offspring at birth. There are two kinds of response to infection with hepatitis B. In the first there is no clear manifestation of illness but the infected person will be positive for the surface antigen of the virus afterwards (HBs AG[+]). Apparently there has been no significant response of the immune system and the virus persists in its host, probably without doing much harm. In the second there is severe illness and sometimes significant lasting damage to the liver, probably both due to the immune system's response to infected liver cells rather than to the plasmolytic actions of the virus itself. A person with this type of response will be negative for the surface antigen of the virus afterwards (HBs AG[−]) but positive for anti-HBs immunoglobulin (anti-HBs[+]).

A very striking effect of these responses on the secondary sex ratio of the offspring of infected people has been reported by Drew et al. (1978). Investigating a population of 390 families in Plati, Greece, they found a very high sex ratio in families in which either parent was HBs Ag(+) and an especially high one (3, 4) in families in which the mother was HBs Ag(+) and had not suffered fetal loss. In contrast, families in which the mother was anti-HBs(+) showed a sex ratio below normal. The same researchers report similar findings from Kar Kar Island, Papua New Guinea (Drew et al. 1982).

As hepatitis B infection is prevalent in many populations, especially in the tropics, one would expect a dramatic demographic effect. That this is not actually seen might be explained in terms of the two responses' keeping each other more or less in balance, but even then hepatitis B infection could be a major confounder in studies of other factors affecting sex ratios at birth.

Reply

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Pelt's comment and James's (CA 31:419–20) focus on factors affecting the sex ratio at birth, and given the web of variables that seem to be involved and the confusion over the proximate causes of this variation their contributions are a valuable addition to the discussion. I am grateful to James also for pointing out that the magnitude of the bias he reported is greater than I suggested. The original data were given by the various researchers as ratios (number of males per 100 females); James presented them as proportions, and I converted those incorrectly.

James suggests that because a correlation is not a causal link investigation of mechanisms must precede any adaptive explanation. This view is becoming a commonplace in evolutionary studies of human behavior (see Symons 1987, 1989; Cosmides and Tooby 1987, 1989). It has, however, been argued that adaptive explanations for specific phenomena can be tested without reference to proximate mechanisms (Borgerhoff Mulder 1987, Borgerhoff Mulder and Caro 1987, Betzig 1989). Evolutionary anthropologists might be able to devise an adaptive hypothesis linking high gonadotrophin levels in ill men and a female-biased sex ratio—for example, that men in poor condition will not be able to provide much for their children and therefore, according to Trivers and Willard, "should" produce daughters rather than sons. Whether the result of this strategy is a greater number of surviving offspring and grandoffspring is an empirical question that can, at least in theory, be tested with demographic data from traditional and historical populations. In other words, while almost any sex ratio can post hoc be given an apparently sensible adaptive explanation (as both I and James have recognized), adaptive explanations can be tested against data on the reproductive success of individuals following different strategies (see, e.g., Clutton-Brock, Albon, and Guinness 1987). Of course, even if we find that the data on reproductive success are in agreement with the adaptive hypothesis, this does not necessarily tell us what has been selected during the course of evolution (in this example, illness per se or high gonadotrophin whatever its cause). Questions such as these can be answered only through research of the kind that James advocates. What I was suggesting was the collection of more empirical data on differential reproductive success.

The sex ratio at birth, though biologically important, is only one of the many ratios that may be affected by biased parental investment. Most of the studies I reviewed had to do with biased parental care after birth. Here again, though the researchers have not examined the proximate psychological mechanisms responsible for this biased care, it is possible to investigate its effects on reproductive success and therefore adaptive hypotheses can be empirically tested. Clearly, detailed research on mechanisms is not incompatible with empirical examination of reproductive consequences.[1]

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On Milk-drinking San and the "Myth of the Primitive Isolate"

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The debate between the "Kalahari revisionists" and those who are accused of having constructed a "myth of the primitive isolate" (CA 31:109–46) calls for a better understanding of the history of the various societies of the Kalahari. The revisionists argue that these populations have been in constant contact with pastoralists (e.g., Denbow 1984) or have themselves been animal husbanders for a long time. According to this view the basic strategy has been not foraging but economic flexibility, which has allowed rapid alteration of strategy with change in ecological and/or social conditions. This has resulted in a general pattern of oscillation between herding, foraging, trading, and farming forms of production—a pattern now well documented for many East African communities (Bollig 1987).

1. I thank Amy Parish for helpful comments on this reply.