SPL

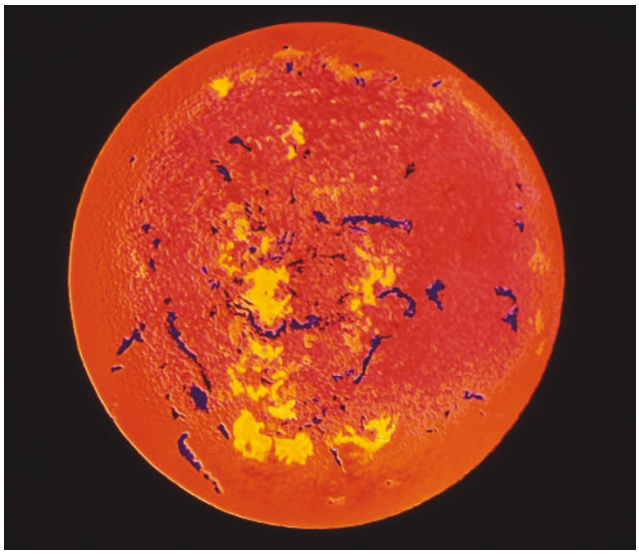

Figure 1 **Solar blemishes. This is a false-colour image of the face of the Sun, with sunspots as black patches. In the work discussed here, Solanki *et al.*[1] have produced a reconstruction of sunspot number going back 11,000 years.**

high sunspot number), the more intense solar wind — the ions streaming out from the Sun — deflects charged particles so that fewer of them enter Earth's atmosphere.

Solanki and colleagues' first step was to determine the rate of ^{14}C production using the tree-ring record of atmospheric ^{14}C concentration after removing the long-term trend in Earth's magnetic field, which modulates the cosmic-ray flux. The concentrations of ^{14}C in the atmosphere may also be affected by variations in ocean circulation, because carbon is partitioned between the atmosphere, the ocean and the biosphere. But there is no evidence of major oceanic variability over the past 11,000 years, and carbon fluxes in the biosphere are not sufficient to cause large changes in atmospheric ^{14}C.

The second step was to calculate the cosmic-ray flux from the data for ^{14}C production, by 'inverting' a model of the transport and modulation of galactic cosmic rays within the envelope of the solar wind; model inversion means working backwards from the answer to find the necessary input to produce that answer. Solanki *et al.* then reconstructed the Sun's open magnetic flux — the magnetic field that extends into the interplanetary medium — from a model of the effect of the open magnetic flux on the transport of galactic cosmic rays. Finally, a model describing the evolution of the open magnetic flux for a given sunspot number was inverted to produce estimates of sunspot number. Within well-defined limits of uncertainty, the series of models reproduce the observed record of sunspots extremely well, from almost no sunspots during the seventeenth century to the current high levels.

Climate variability on centennial to millennial timescales is documented in many palaeoclimate records going back at least as far as the end of the last glaciation, some 12,000 years ago. Whether solar activity is a dominant influence in these changes is a subject of intense debate[3–6]. The exact relationship of solar irradiance to sunspot number is still uncertain[7,8], but the reconstructed sunspot number will nonetheless provide a much-needed record of solar activity. This can then be compared with palaeoclimate data sets to test theories of possible solar–climate connections, as well as enabling physicists to model long-term solar variability. A better understanding of the mechanisms responsible for past climate variability will also help those using global circulation models to predict future climate change.

So does the current episode of high sunspot number imply that the Sun has had a significant role in the global warming of the late twentieth century? The answer is no. Although climate models differ in their estimation of the Sun's contribution to recent warming, even those that include spectrally varying changes in solar irradiance conclude that anthropogenic causes are the prime factor[9–12]. The high probability that this episode will end soon is not likely to cut us much slack in controlling global warming unless we reduce greenhouse-gas emissions. But because the solar influence may be more regionally variable than the effects of greenhouse gases[11], model-based predictions of regional climate change may be improved by this study. It is at the regional level that climate change will have the greatest impact on society. ■

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Evolutionary biology

Mortality and lifespan

Peter A. Abrams

How does natural selection affect lifespan? The question has exercised biologists for some years. The latest twist comes from ingenious experiments on tropical fish from different ecological backgrounds.

On page 1095 of this issue, Reznick *et al.*[1] describe how they have investigated one of the main factors that influence the evolution of an organism's lifespan. That factor is the risk of dying that a population faces as a result of environmental conditions (such as, in this case, predation). The study subjects are guppies, small tropical fish that are widely used in evolutionary studies, and the authors provide the first experimental support for the prediction that a higher environmental risk of mortality can select for inherently longer-lived organisms.

Guppies from the lower reaches of several rivers in Trinidad are subject to much higher rates of predation than those in the upper parts of the same rivers, where waterfalls block access by larger fish. In predator-free lab experiments, Reznick *et al.* found that guppies from the high-predation segments of two of the rivers lived up to 35% longer than those from low-predation segments of the same watercourse. In addition, the guppies from high-predation sites had a 40% longer reproductive span and reproduced at a higher rate. So a background of higher mortality under natural conditions has apparently led to the evolution of both a longer lifespan and a longer reproductive span. Their longest-lived fish, a female, is pictured in Figure 1.

A bit of history is required to see why this observation is surprising. Environmentally caused ('extrinsic') mortality has long been recognized as a key factor determining how natural selection moulds 'intrinsic' mortality — the death rate that a population would have under some standardized, generally benign, set of environmental conditions. Although evolution should favour lower

intrinsic mortality (and a longer intrinsic lifespan) when all else is equal, many organisms face a trade-off between higher levels of reproduction or lower levels of intrinsic mortality. One of the main reasons that senescence occurs is because repair is costly: resources that are devoted to maintaining an organism are not available for reproduction. In the 1950s, Peter Medawar[2] and George Williams[3] pointed out that high extrinsic mortality could favour shorter intrinsic lifespan. Why, they reasoned, should an organism invest in costly repair that will probably only ensure that it is in prime physical condition when its life ends? Higher extrinsic mortality should favour low investment in repair, and thus a high intrinsic mortality and a short intrinsic lifespan.

But this reasoning didn't take account of two further factors. One is that higher extrinsic mortality also slows the rate of population growth, and more slowly growing populations are expected to evolve to have lower rates of intrinsic mortality and a longer lifespan[4,5]. The other is the interaction between extrinsic mortality factors and physiological repair or maintenance[5,6]. If predators can be evaded by fast, but not by slow prey, greater predation risk should select for greater maintenance of the body systems essential for fast movement. This higher level of repair would then prolong intrinsic lifespan.

Higher extrinsic mortality (more predators) could also have indirect effects that Medawar and Williams did not consider. For example, it reduces population size, which in turn increases the abundance of food or other resources. These changes may have their own effects on both population growth and the level of intrinsic mortality favoured by selection. Other complications arise if the mortality factor has a greater effect on some ages than on others[5,6] — if, for example, predators prefer to capture larger, older prey. As a result of these complicating features, many types of mortality are expected to reduce intrinsic death rates at some ages while increasing them at others[6]. In any event, theory suggests that higher extrinsic mortality will produce evolutionary conditions that can either extend or shorten the intrinsic lifespan.

Given these complexities, the curious feature of previous observational[7] and experimental work[8] has been its support for the Medawar–Williams prediction. There have been exceptions[9], if only suggestively so. But the almost unanimous evidence that high extrinsic mortality is associated with shorter lifespan is puzzling because there is no reason to believe that the conditions that produce the opposite outcome are rare in nature.

So it is reassuring that Reznick *et al.*[1] found longer intrinsic lifespans in guppies from populations characterized by higher predation rates. The authors also looked at whether these evolutionary changes might be an indirect consequence of predator-caused deaths, such as the availability, in natural settings, of more food for the remaining guppies. Reznick and colleagues' study is unique in examining this effect. They found that food alone could not account for the difference in intrinsic mortalities seen in their experiments, but that having more food enhanced the lifespan-lengthening effect of a high-predation background.

There is no doubt that the guppies from high-predation sites have both longer intrinsic lifespans and longer reproductive spans. But is it valid to conclude that they have slower senescence? This is a more difficult question. A hypothetical population with no senescence (that is, no age-related decline in survival or reproduction) could still have a short lifespan if it had a high mortality rate that was independent of age. If guppies from high-predation sites begin their adult life with a lower rate of intrinsic mortality than those from low-predation environments, they could have the same rate of increase with age in their mortality rate, but would still have a longer lifespan. One could then argue that the two populations had identical rates of senescence. Some measures of the rate of change of intrinsic mortality with age suggest that senescence is delayed in guppies from high-predation sites.

However, senescence encompasses relationships between many different components of fitness and age, none of which can be adequately summarized by a single number: there are many potential measures of the rate of senescence, and conclusions about this rate depend on the measure chosen. It might be possible to make a case that guppies from high-predation environments are more robust, but age at a rate equal to or higher than that of low-predation guppies. Regardless of the mathematical measure used to quantify the rate of senescence, the work of Reznick *et al.* clearly shows that rates of senescence differ among the different components of fitness examined: survival, reproduction or swimming performance. The reasons for these differences are not yet understood.

It would be surprising if guppies were the only species for which an added risk of mortality lengthens intrinsic lifespan. Similar studies on other species will help us understand the underlying reasons why Medawar and Williams' predictions hold for some species and not for others. Such studies should follow Reznick and colleagues' lead in quantifying declines in several fitness components and studying the indirect ecological consequences of higher extrinsic mortality. ■

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Figure 1 **Star survivor — the longest lived of the guppies studied by Reznick *et al.*[1]. The photo was taken shortly before her death at the age of 1,464 days.**

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Correction

In Yi Zhang's News and Views article "Molecular biology: No exception to reversibility" (*Nature* **431,** 637–639; 2004), there were errors in Fig. 1b. In the side chain of citrulline, a double bond should have been shown between NH and O, rather than between NH and NH_2. Several of the connecting atoms are erroneously shown as H rather than N. And the leaving methylamine should have been represented as $+NH_3CH_3$ rather than $+NH_2CH_3$.