the brain works in a modular fashion—that is, cognitive processes are specific and independent. Implicit in this perspective is a bottom-up reductionist view of genetics, in which individual modules are the targets of gene action. The findings from multivariate genetic analyses suggest a top-down view, in which genetic effects operate primarily on *g*, rather than suggest a bottom-up view, in which genetic effects are specific to modules. Given that the brain has evolved to learn from a variety of experiences and to solve a variety of problems, perhaps it makes sense that it would function holistically. However, finding genetic correlations near 1.0 does not prove that genetic effects are limited to a single general cognitive process that works in a top-down way. Another alternative is that specific cognitive abilities, as they are currently assessed, might tap many of the same modular processes that are each affected by different sets of genes. This alternative hypothesis could be tested by means of multivariate genetic research on measures of modular processes, such as neuroimaging measures of brain function (Watkins et al. 1999 [in this issue]; Kosslyn and Plomin, in press).

Another direction for genetic research, one that is too new to be mentioned in Mackintosh's book, is the attempt to identify specific genes responsible for the heritability of *g*. DNA associations with *g* have begun to be reported (Chorney et al. 1998), including initial results from a systematic genome scan for association, by means of DNA pooling (Fisher et al. 1999). Neuroscience research with knockout animal models of learning and memory is likely to accelerate research on the molecular genetics of *g*, especially as neuroscientists come to appreciate the broad relevance of *g*. Finding specific genes associated with *g* will facilitate more-precise answers to questions such as modularity. For example, to what extent are genes that are associated with modular processes, such as long-term potentiation, also associated with *g*? Finding genes for *g* will have implications for society as well as for science (Plomin, in press). If, as I predict, *g* will soon take center stage in genetic research on the neuroscience of learning and memory, Mackintosh's excellent overview of research on *g* will be of great help to geneticists and others with an interest in the workings of learning, memory, and intelligence.

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A Means to an End: The Biological Basis of Aging and Death. By William C. Clark. New York: Oxford University Press, 1999. Pp. 234. $27.50 (cloth).

The questions of how and why we age have great intrinsic intellectual appeal and major societal implications. William Clark, an Emeritus Professor of Immunology at UCLA, has written a popular book in an attempt to introduce the subject to nonspecialists. That he himself is a nonspecialist is probably a good thing, since there is the potential to bring a fresh new perspective. He has succeeded in producing a very readable review that does indeed outline the major ideas. Unfortunately, although he quite properly emphasizes the evolutionary theory of *why* we age, his language in many sections of the book indicates a belief that a genetic program has evolved to *produce* senescence.

All serious students of the evolutionary biology of aging would agree that the senescent phenotypes that emerge in age-structured populations are the result of a decline in the force of natural selection with respect to the age of gene effects (Rose 1991). Two classes of gene action are envisaged. The first class, originally outlined by Haldane and Medawar (1952), includes

rare, idiosyncratic constitutional mutations with essentially neutral effects on fitness but with deleterious effects late in life. I refer to these genetic variations as leading to "private" modulations of senescence (Martin 1997). The rare presenilin and beta-amyloid precursor–protein mutations are reasonable examples, although the author in fact discusses these in terms of the second class of gene action, a class that was best elucidated by George C. Williams (1957) and that often is referred to as "antagonistic pleiotropy"; such actions are likely to involve polymorphic alleles that have been selected for enhanced fitness but that result in negative physiological effects that escape the force of natural selection. I now refer to these genetic variants as leading to "public" modulations of aging, since they are relatively prevalent and may be expected to have emerged independently in multiple populations that share certain environmental conditions (Martin 1997). Some such loci could affect the process of aging in a consistent fashion, even among different species. The *APOE* polymorphism discussed by Clark is a good candidate for such a trade-off effect, at least in human populations. My guess is that the "bad" allele, *APOEϵ4*, emerged, in certain populations, as a response to selection for resistance to some endemic infectious agent.

Clark gives star billing to damage from reactive-oxygen species as the mechanism for *how* we age. Most gerontologists would agree, although they would also add peroxynitrate as one of the culprits. Clark emphasizes DNA damage, but others might give relatively more emphasis to proteins, especially to long-lived proteins such as the collagens (the most abundant of our proteins) and the crystallins of the ocular lens. Most senile cataracts probably develop because of posttranslational protein modifications—glycations, methione sulfoxidations, deamidations, isomerizations, and racemizations, among others. The cataracts of the Werner syndrome can be presumed to have a different pathogenesis, given that the altered replication, DNA repair, transcription, and/or recombination that may be ascribed to defects in the Werner helicase can affect only the lens epithelial cells and not the acellular layer of crystallin proteins that constitute the lens itself. The author gives special attention to the Werner syndrome, for which I am grateful, since I have spent more than 30 years researching that disorder. The jury is out, however, as to the degree to which it may eventually inform us about *usual* mechanisms of senescence.

There are a number of errors in the text; I cite a sampling, in the hope the author will make suitable corrections for another edition: (1) The author states (p. 84) that the Hutchinson-Gilford syndrome ("progeria," or "progeria of childhood") is likely to be autosomal recessive. Most experts, notably W. Ted Brown (1992), would argue that it is most likely a sporadic autosomal dominant condition, given the lack of evidence for parental consanguinity and the evidence of advanced paternal age. (2) The author states (p. 86) that approximately 1 in 5,000 persons carries the mutant Werner helicase gene; the figure is closer to 1 in 200, at least in the Japanese population (Satoh et al. 1999). (3) The term "idiopathic" is used incorrectly (p. 137); it is not confined to disorders that are endogenous. (4) mtDNA is not single stranded (p. 151); perhaps the author wanted to indicate that transcription occurs from only a single strand. (5) UV radiation, not "cosmic radiations [*sic*]" (p. 153) is a major cause of degenerative and neoplastic changes in sun-exposed skin. By the standard of the usual error rates in popular books, however (which are orders of magnitude greater than the fidelity of DNA-dependent DNA polymerases!), the author has done well. It is also to his credit that he appears to have often consulted original key papers. Nevertheless, as a general introduction to the biology of aging, I would steer a curious newcomer first to "Why We Age" by Steven N. Austad, because of the latter's clarity in outlining the evolutionary themes of aging.

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