How long do individuals in different species live? How fecund are they? How big do they grow. How much effort is expended by older individuals (e.g., parents) to help younger individuals (e.g., children)? Why, in particular, do humans have the life histories we have? Such questions about the age-trajectories of mortality, fertility, growth and intergenerational transfers are of fundamental interest to demographers and biologists. Much fundamental work needs to be done to develop theory--and demographers can contribute to this work, as evidenced by contributions by Tuljapurkar {1990, 1997}, Wachter {1999}, Lee {2003} and Vaupel and co-workers {2004}. Lotka pioneered research in evolutionary demography, but following his seminal contributions demographers turned to other topics. The recent resurgence of interest in the theory of evolutionary demography suggests that this area may become one of the most interesting branches of demography. This paper reviews recent theoretical developments and suggests future directions for research.

In particular, the paper discusses the two main types of models used in evolutionary demographic theory. Evolutionary-demographic models of life-history characteristics in general and of senescence in particular fall into two types {Partridge, 1993}, optimization models and non-adaptive age-specific mutation models. In optimization models the forces of evolution are assumed to yield the best-possible design of a species' life history, the design that maximizes Darwinian fitness. Williams {Williams, 1957} proposed an optimization model of senescence, the so-called antagonistic-pleiotropy model. The basic idea is that some genes have a favorable or unfavorable effect on fertility or survival at younger ages but the opposite effect on mortality at older ages. A small positive (or negative) effect at younger ages may be more important than a large opposite effect at older ages if few individuals survive to these ages and if their reproduction is low. Williams' model is often formulated in terms of mutations that have a positive effect at some particular age and a negative effect at some other age {Charlesworth, 1994}. Williams' idea, however, is more general. It is simply an example of the kind of thinking about trade-offs that underlies all optimization modeling. Williams thought that his model implied senescence and he did not consider the logical possibility that such an optimization model might lead to negative senescence, i.e., to the decline in mortality with age {Vaupel, 2004}. The "disposable soma" model {Kirkwood, 1991 and 1992} is a related example of this kind of thinking applied to senescence.

In the second class of models evolutionary forces act in a non-adaptive way, as follows. Evolution acts on randomly occurring mutations. Some of these mutations may have age-specific effects. In particular, some mutations may only be harmful at older ages. There is little selective pressure to remove such mutations from the population because the

individuals who have them have produced most of their offspring before they have reached old age. Hence, such mutations tend to accumulate, resulting in senescence. Charlesworth {1994} provides a general discussion of mutation-selection balance, i.e., of models of the opposing forces of deleterious mutation and subsequent Darwinian selection. Hamilton {1966} developed an influential mutation-accumulation model of senescence.

The potential for evolutionary-demographic theory is enhanced by the fundamental importance of demography in evolution, as briefly explained below. Nothing in biology, Dobzhansky asserted, makes sense except in the light of evolution. An equally valid overstatement is that nothing in evolution can be understood except in the light of demography. Evolution is driven by population dynamics governed by age-schedules of fertility and survival. Lotka emphasized this. Since his pathbreaking research, models of the evolution of fertility, mortality and other life-history patterns have been based on stable population theory. Lotka's equation

$$\int_0^{\omega} e^{-ra} l(a) m(a) da = 1 \qquad (1)$$

specifies the intrinsic growth rate, r, of a closed population, typically of females, as a function of the proportion, $l(a)$, of newborns surviving to age a and age-specific maternity (or fertility), $m(a)$. If a new subspecies emerges as a result of mutation, then the subspecies is assumed to have an evolutionary advantage if its intrinsic growth rate is greater than that of other subspecies.