quite common among the general public, but it is incorrect. According to modern evolution theory, natural selection moulds gene action to optimize reproductive success, not longevity. Natural selection can favour genes that increase reproductive success in early adulthood even though they might promote pathology in later ages—so-called antagonistic pleiotropy (AP).[13] This means that although organismal senescence is not an adaptation, it is genetically programmed: late-life action of genes that bring us into existence eventually cause our death.[17]

Evolutionary theory predicts that senescent pathologies arise from late-life action of many genes. Animal model research has shown that some genes specifying central regulators of growth and development (*i.e.* common to most cell types) are AP determinants of multiple age-related pathologies, including some that contribute to late-life mortality. Inhibiting late-life effects of these genes can lead to amelioration of a wide spectrum of late-life pathologies—typically delaying their onset (decelerated aging). Thus, within the broader AP genetic predisposition one can define discrete genetic etiologies leading to syndromes of age-related pathology. For example, late-life accumulation of senescent cells (*sensu* Hayflick) and, particularly, mTor-activated gene expression in these cells appears to contribute to development of multiple age-related pathologies; research in mice suggests that these pathologies include atherosclerosis, the three major classes of cancer (carcinomas, sarcomas, lymphomas), glomerulosclerosis, cardiomyocyte hypertrophy, cardiac dysfunction, lipodystrophy and cataractogenesis.[18,19]

In conclusion, organismal senescence is a disease multi-syndrome, a set of syndromes and unitary diseases. The main underlying cause is wild-type genetic pre-disposition, in which respect it is etiologically distinct from most other diseases. However, standard etiologies (*e.g.* microbial pathogens, injury, gene mutations) do play a major role in organismal senescence, particularly when programmed aspects of aging increase predisposition to their pathogenic effects.

2.1.3   What is an Anti-Aging Intervention?

If one rejects the traditional dichotomy between normal aging and aging-associated diseases, then the meaning of *anti-aging* becomes problematic. If the aging disease super-syndrome is understood as the sum of senescent pathologies and their causes, this suggests that any treatment of any senescent pathology could be understood to be an anti-aging treatment. This is problematic because treatments for existing senescent pathologies (*e.g.* chemotherapy for late-life cancer or hip replacement operations) do not conform with the objective of improving late-life health by intervening in aging itself. This critical, central aim of biogerontology seems diluted or lost within such a redefinition of anti-aging.

In response to this, a new definition of anti-aging has been proposed[9] to retain the utility of the term. Here, anti-aging refers specifically to the preventative approach to improving late-life health. By this view, anti-aging treatments are interventions that counteract any etiology of organismal senescence. Based on the above account, two basic types of anti-aging