People's tissues accumulate an increasing number of somatic mutations as they age. Although the majority of these mutations have little or no functional impact, a mutation that confers a fitness advantage on a cell may occur. When this occurs in the hematopoietic system, a significant proportion of circulating blood cells may be derived from a single mutated stem cell. This type of outgrowth, known as "clonal hematopoiesis," is very common in the elderly. We will discuss recent advances in our understanding of clonal hematopoiesis, its relationship to cancer, its link to nonmalignant aging diseases, and its potential impact on immune function. Clonal hematopoiesis offers insight into the process of mutation and selection that occurs in all somatic tissues. Traditional risk factors are only partially predictive of the development of cardiovascular disease, which is the leading cause of death in the elderly. Recent epidemiological studies have found that human ageing is associated with an increased frequency of somatic mutations in the hematopoietic system, which give a mutant cell a competitive advantage, allowing for clonal expansion, a phenomenon known as clonal hematopoiesis. Surprisingly, these mutations have been linked to an increased risk of cardiovascular disease, implying a previously unknown link between somatic mutations in hematopoietic cells and cardiovascular disease. We present an up-to-date review of clonal hematopoiesis and its relationship to ageing and cardiovascular disease in this article. We also provide a detailed report on the experimental studies that have helped us understand the relationship between clonal hematopoiesis and cardiovascular disease, as well as the mechanisms by which hematopoietic somatic mutations contribute to disease pathology. Clonal expansions of mutated hematopoietic cells, known as clonal hematopoiesis, are common in the elderly. One expected consequence of mutation-associated clonal hematopoiesis is an increased risk of hematologic cancers, as demonstrated by several studies. The hematopoietic stem cells that acquire these somatic mutations, however, also give rise to mutated immune effector cells such as monocytes, granulocytes, and lymphocytes. These effector cells have the potential to influence a wide range of disease states, particularly those with a chronic inflammatory component. Indeed, several studies have now found a link between clonal hematopoiesis and an increased risk of atherosclerotic cardiovascular disease. Emerging evidence links clonal hematopoiesis to other nonhematologic diseases. In this section, we will look at recent research that has linked clonal hematopoiesis to altered immune function, inflammation, and nonmalignant aging diseases. Clonal hematopoiesis is a common premalignant condition characterised by the abnormal expansion of clonally derived hematopoietic stem cells harbouring somatic mutations in leukemia-related genes. Aside from increasing age, this phenomenon is more common in people with lymphoid or solid tumours and is linked to genotoxic stress exposure. In this context, clonal hematopoiesis increases the risk of developing therapy-related myeloid neoplasms and appears to contribute to poor cancer-related survival via a variety of potential mechanisms.

References