Cigarette smoking activates platelets, induces endothelial inflammation, and promotes vascular dysfunction leading to accelerated atherosclerosis, plaque rupture, acute coronary syndromes, and death. Over several decades, notable advances in public policy in the United States have reduced initiation and increased cessation of cigarette smoking, thus lowering the prevalence of tobacco product consumption and related cardiovascular disease (CVD). Indeed, most people in developed countries understand the harm of cigarette smoking and the health and economic benefits of abstinence and cessation. However, these public health successes have slowed recently, reflecting perhaps a higher degree of addiction in remaining smokers and also a degree of fatigue in further advancing preventive and treatment strategies, as well as scientific knowledge more generally.

Several realities underscore the current challenges to further reducing tobacco-related CVD. First, rates of tobacco product use continue to increase in developing nations and, in combination with dietary and additional lifestyle changes, will drive a surge of CVD in the coming decades in these societies. Second, even in developed countries, tobacco use continues to be stubbornly prevalent particularly in minorities, in patients with psychiatric disorders, and in economically disadvantaged populations and represents a major cause of preventable morbidity and mortality. Last, and in some respects of most direct concern to our research community, mechanistic insights into how tobacco consumption causes CVDs and how this affects individual risk have not kept pace with epidemiological or public health advances. Although we have gained some knowledge of the genetic basis for cigarette smoking behavior and nicotine addiction, we have an incomplete knowledge at the molecular level of the processes that promote CVD, have learned little regarding individualized and genetic risk of CVD complications in those exposed, and have surprisingly limited knowledge of the specific constituents of cigarette smoke that lead to CVD. Thus, we are poorly positioned to apply mechanism-based strategies to treat smoking-related CVD or to execute preventive strategies in those at greatest risk.

In this miniseries, we focus on mechanistic aspects of tobacco-related CVD as a focus for galvanizing scientific efforts for the 21st century global epidemic challenge. Norman and Curci highlight how cigarette smoking dominates as a risk factor for abdominal aortic aneurysm and emphasize recent experimental insights but acknowledge our incomplete mechanistic understanding of abdominal aortic aneurysm in smokers. Barua and Ambrose provide an update on the role and potential mechanisms of cigarette smoking in platelet activation, thrombosis, acute cardiovascular events, and modulation of therapeutic interventions in coronary thrombosis. Breitling emphasizes the paucity of contemporary genetic studies in understanding the interindividual risk of developing CVD in cigarette smokers, highlights promising epigenetic discoveries, and reviews the priorities for large-scale and rigorous genomic approaches to smoking-related CVD. In a future issue of the journal, additional reviews in this series will focus on tobacco-related endothelial dysfunction and atherosclerosis, as well as present an integrated view of CVD in the context of nicotine addiction, public policies, and global health.

We are now at a critical juncture in the battle against tobacco-related CVD. A striking observation in editing this miniseries was the relative dearth of recent mechanistic and genomic studies of smoking-related CVD. This failure to advance mechanistic and genomic understanding of smoking-related CVD at an individual level coupled with unchallenged marketing and an increasing surge in tobacco product consumption in developing countries may severely challenge our ability to mount effective preventive strategies and manage the global epidemic of related
CVD. This series marks current knowledge, highlights deficiencies, and defines the challenge for future mechanistic, therapeutic, and public policy requirement for prevention and treatment of tobacco-related CVDs in the 21st century.

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References

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