Cigarette smoking is considered as one of the most important preventable risk factors for the development of early atherogenesis, which precludes most forms of cardiovascular disease. Endothelial dysfunction in particular can be induced by smoking through increased expression of adhesion molecules and reduced nitric oxide bioavailability. Additionally, smoking-related increased adherence of platelets and macrophages has been detected as being causal for pro-coagulant and inflammatory processes along the vessel wall (1).

The two most substantially (dys)-regulated miRNAs in microvesicles from smokers in the present study by Badrnya et al. (4), miR-29b and miR-223, have previously been identified as crucial modulators in various cardiovascular disease pathologies. Interestingly, the only miRNA, which is decreased in circulation of smokers, miR-223, has recently been detected as a plasma biomarker in prospective studies, predicting patients with greater risk for future myocardial infarction and type 2 diabetes mellitus (2). The second miRNA, which was expressed substantially higher in microvesicles derived from smokers, miR-29b, has been linked in previous studies to modulate metalloproteinase-2 and -9 levels, as well as targeting several collagen isoforms, thus contributing to vascular and cardiac fibrosis, and furthermore innate immune responses (3). All of these effectors are relevant attributes and important contributors to different forms of cardiovascular disease.

There is still a great gap regarding our knowledge about the effects of smoking in cardiovascular disease development. We do know that smoking cessation is a very effective measure to augment cardiovascular damage and to prevent fatal outcome. In this regard, the study by Badrnya et al. (4) opens up a new chapter on how smoking influences the micro-milieu of our body, elucidating microvesicles that can carry microRNAs, which are potentially an essential component of intercellular communication and could probably be utilised for future therapeutic and biomarker approaches.

Conflicts of interest
None declared.

References