Pro-Arrhythmic Effects of Gaseous Pollutants under Healthy Conditions: An In-Silico Study

L.C. Palacio[1], J. Saiz[2], C. Tobón[1]*

[1]MATBIOM, Universidad de Medellín, Medellín, Colombia
[2]CI[2]B, Universitat Politècnica de València, Valencia, Spain

Air pollution is the main component of environmental contamination and is responsible for over 6.5 million deaths worldwide each year. Some of the most dangerous gaseous pollutants are sulfur dioxide (SO_2), carbon monoxide (CO), and nitrogen oxides (NO_x), which have been linked to an increased risk of cardiac arrhythmias. However, the underlying pathophysiological mechanisms have not been fully established in humans due to the epidemiological nature of most studies. It has been found that SO_2, CO, and NO affect different ionic currents, which can impact the action potential and potentially contribute to the initiation and maintenance of atrial arrhythmias.

In this study, we use multiscale atrial models to assess the effects of individual gaseous pollutants at low, medium, and high concentrations. For this purpose, mathematical equations describing the effects of these gaseous pollutants under normal physiological conditions were included in the atrial myocyte model developed by Courtemanche to evaluate single-cell electrophysiological characteristics. The modified model was incorporated into a 3D model of human atria to evaluate the dynamics of propagation.

The results indicate that individual gaseous pollutants exhibit pro-arrhythmic effects in a concentration-dependent manner by altering the action potential, which results in a reduction of action potential duration (APD). In the 3D simulations, the main findings showed the existence of reentries, which serve as a trigger for initiating atrial arrhythmias. SO_2 was the pollutant with the highest pro-arrhythmic effects, achieving a 39% decrease in APD at high concentrations and triggering the most chaotic and disordered propagation characterized by a greater number of reentries (7).

In conclusion, our findings suggest that, under healthy conditions, gaseous air pollutants exhibit pro-arrhythmic effects in human atrial models in a concentration-dependent manner.