Cellular oxidative stress, derived from imbalance between the production of reactive oxygen species (ROS) and the efficacy of the antioxidant defense, can be a consequence of using the nicotine products.[1-3] Prooxidant properties of the tobacco smoke are accounted for by the abundance of the smoke oxidants. The antioxidant potential of the smoke is scantily addressed in the literature. However, one should take into account that any reactant in oxidation process may exhibit both oxidant and antioxidant propensities depending on the reaction conditions. And we have shown that smoke constituents indeed exhibit at the same time both prooxidant and antioxidant activities.[3] Such smoke-borne antioxidants may be assessed through both the direct chemiluminescence derived from the smoke samples as a function of the smoke tar content and using the probe chemiluminescent preparations of hydrocarbon substrates being oxidized.[3]

Conversely, we have not observed any significant antioxidant activity of aerosols derived from electronic cigarettes (ECs). For ECs, the following feature is noteworthy. We have found for the first time that all ECs, regardless of their technical complexity, generate in their emissions hydroperoxides (ROOH) of propylene glycol (the main component of e-liquids served as solvent for nicotine), which are potential prooxidants (ROS sources), whose physiological significance still requires elucidation.[4] The content of these products depend on the type of EC and on the mode of its use, which makes possible to minimize the ROOH generation.

The activity of neutrophils manifested by the ROS generation is of prime importance for the human immune system, whose functioning may be modified by diverse physiologically active substances entering the human body throughout its lifetime.[4] In particular, using nicotine products by various means (respiratory or oral) may cause oxidative stress developments, depletion of a bioantioxidant defense and other consequences at the cellular level and in the whole organism, which alter the state of immune system.[4] Neutrophils exist in the three states: quiescent, primed or active. Priming and activation of neutrophils exhibit a difference. In the primed state, there is no increase in oxidase activity, however, subsequent stimulation is able to provoke a response that is 10-fold larger than in non-primed activated cells. For assaying the ROS-generating propensity of neutrophils in monitoring the immunomodulating properties of nicotine products, we used non-opsonized zymosan-induced chemiluminescence in a contingently whole blood (erythrocytes were removed from blood samples by spontaneous sedimentation). The chemiluminescence emission was triggered by phagocytosis of the particle matter (zymosan-endotoxin complexes). Studying the correlation between the maximum rate of the ROS generation (manifested by the maximum light-emission intensity) by the cells of the contingently whole blood and the concentration of leukocytes in the peripheral blood has shown that the variability of the chemiluminescence peak value is determined only up to 12 % by the variability of the total concentration of leukocytes. Such an observation implies that only primed neutrophils contribute to the observed light emission. Comparative study of the blood taken from different groups of volunteers has given the following results. During the first year after quitting consumption of any nicotine products, no significant changes in the activity of neutrophils were observed. However, after a longer period of abstinence (from 2.5 years) changes occurred, and for the better. In smokers, neutrophils expectedly are predominantly in a pre-activated state.

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