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# How smoking encourages infection

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Now new research published in the open access journal *BMC Cell Biology* shows that nicotine affects neutrophils, the short-lived white blood cells that defend against infection, by reducing their ability to seek and destroy bacteria.

Neutrophils are generated by our bone marrow, which they leave as terminally differentiated cells. Although nicotine is known to affect neutrophils, there has been no study until now of the mechanisms at work when nicotine is present during neutrophil differentiation. David Scott from the Oral Health and Systemic Disease Research Group at the University of Louisville School of Dentistry, Kentucky, USA, along with a team of international colleagues decided to investigate how nicotine influenced the differentiation process.

The authors suggest the processes they observed as contributing to impaired neutrophil function partially explain chronic tobacco users' increased susceptibility to bacterial infection and inflammatory diseases. A better understanding of this relationship could pave the way for specific therapeutic strategies to treat a number of important tobacco-associated inflammatory diseases and conditions.
The team modeled the neutrophil differentiation process beginning with promyelocytic HL-60 cells, which differentiated into neutrophils following dimethylsulfoxide (DMSO) treatment both with and without nicotine. The researchers found that nicotine increased the percentage of cells in late differentiation phases (metamyelocytes, banded neutrophils and segmented neutrophils) compared to DMSO alone, but did not affect other neutrophil differentiation markers that they examined.

However, the nicotine treated neutrophils were less able to seek and destroy bacteria than nicotine-free neutrophils. The nicotine suppressed the oxidative burst in HL-60 cells, a function that helps kill invading bacteria. Nicotine also increased MMP-9 release, a factor involved in tissue degradation.

“It must be acknowledged that our study model, DMSO-differentiated HL-60 cells, are not entirely similar to normal neutrophils,” says Scott. “However, this leukemic human cell line does permit the reproducible study of differentiation while retaining many of the key effector functions of primary neutrophils.”

Source: BioMed Central

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