Oral neutrophils: cells with multiple personalities

Neutrophils are protective immune cells that swarm sites of infection to target and kill invading bacteria. The importance of neutrophils in the maintenance of human health is highlighted by multiple severe and life threatening diseases caused by a lack of functioning neutrophils. However, neutrophils can also cause substantial damage to normal tissues when present in great numbers or for a prolonged duration. Unintended neutrophil-mediated tissue damage is a hallmark of many long-term or chronic inflammatory diseases such as severe gum disease or chronic periodontitis.

While studying neutrophils in gum disease we noticed that neutrophils are also recruited in great numbers to the mouth, even in the absence of gum disease. What is really interesting is that neutrophils recruited to healthy gums co-exist with bacteria found around teeth and gums in a symbiotic manner. Healthy oral neutrophils grade their inflammatory response so as to not allow bacterial overgrowth while at the same time not damaging the normal human tissues. With the onset of gum disease, which is characterized by a change in the bacterial community, oral neutrophils completely change their personality or “phenotype”.

Oral neutrophils found in a gum-diseased mouth are significantly more aggressive, resulting in more bacterial killing but also significantly more neutrophil-mediated tissue damage. If this excess neutrophil activity goes unchecked, it leads to progressive damage to the supporting apparatus of the teeth, ultimately leading to tooth loosening and tooth loss. While oral neutrophils in disease had reached their full inflammatory potential, healthy oral neutrophils could change states and become pro-inflammatory with a strong exogenous stimulus. Until now, it had not been demonstrated that tissue neutrophils could exist in multiple activation states, allowing for restrained oral neutrophil function in health. Using an established CP inflammation model system, we were able to conclusively show how neutrophils change their phenotype from health to disease, which correlates to increase activity and tissue damage with the onset of disease.

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