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Perspective

Catecholamine Stress Hormones and their Impact on Microbial Infections

John Tresguerres*

Department of Genetics, Physiology and Microbiology, Complutense University of Madrid, Madrid, Spain

DESCRIPTION

Although local environmental factors like temperature, pH, and the availability of nutrients are known to affect bacterial growth and pathogenicity, the impact of host signals on bacterial behaviour has only recently come to light. The theory that bacteria have developed systems for sensing host-associated substances like hormones is the cornerstone of the recently recognized microbiology research field known as microbial endocrinology. These hormone sensors give the bacterium the ability to recognize the presence of a suitable host nearby and the right moment to start the production of commensal genes for host colonization or pathogen genes for virulence determinants.

The majority of microbial endocrinology studies to date have concentrated on how bacteria interact with stress-related biochemicals like the catecholamine hormones dopamine, noradrenaline, and adrenaline. This resulted from long-held beliefs that immune function is suppressed by stress hormones in people and animals, increasing their risk of infection. But, by additionally taking into account the effects of the stress event from the perspective of the microbe, seeing host stress *via* the lens of microbial endocrinology provides a fuller awareness of what may be happening with our billions of microbial inhabitants.

In the context of animal welfare, microbial endocrinology has proven to be an effective foundation for building a comprehensive understanding of the elements that influence the interactions between microorganisms and their host during health and disease. A class of extensively active effector substances called catecholamine hormones is produced from tyrosine and other dietary sources. They have a benzene ring with two adjacent hydroxyl groups and an opposing amine side chain as part of their molecular makeup. L-dopa, which is primarily sourced from dietary sources, is the first step in the synthesis pathway for catecholamine stress hormones. From there, dopamine, noradrenaline, and adrenaline are produced through enzymatic conversion. Dopamine, noradrenaline, and adrenaline have both endocrinological and neuronal functions. The Enteric Nervous System (ENS), which is a component of the mammalian body's noradrenergic and dopaminergic nerve terminals, is widely spread throughout the digestive tract.

It has been demonstrated that up to 50% of the noradrenaline produced by mammals is synthesized and used inside the ENS. Noradrenaline is released from storage within sympathetic nerve fibres within the prevertebral ganglia that innervate the gut mucosa in the stomach. Nonsympathetic enteric neurons found in the gut wall are where dopamine is made. The ENS contains a large number of nerve terminals that contain noradrenaline and dopamine, which makes the stomach a noradrenaline and dopamine-rich environment. It is unlikely that adrenaline would typically be found in significant amounts in the gut, save perhaps when the mucosal barrier has been damaged, as the intestinal mucosa lacks neurons that contain phenylethanolamine N-methyltransferase, an enzyme necessary for the synthesis of adrenaline from noradrenaline.

In terms of their signalling capabilities, catecholamines are physiologically ubiquitous and are used by organs and tissues throughout the mammalian body. So, it is reasonable to assume that bacteria living in a range of *in vivo* environments will eventually come into contact with catecholamines and will thereafter have a need to develop sensory systems for keeping track of their host's stress hormone levels. the ability of microorganisms to respond to catecholamines in areas of the body other than the skin or lungs. Several noncatecholamine mammalian hormones are also known to be recognized by pathogenic bacteria, according to the available evidence. The coagulase-negative staphylococci, in particular, are very sensitive to the stress hormone. Compared to controls, the effects of noradrenaline, adrenaline, dopamine, and the synthetic catecholamine inotropes dobutamine and isoprenaline on staphylococcal growth in a medium based on blood or serum were all up to 100,000 times greater. The ability of the bacterium to evade the attack of therapeutic antimicrobials and the host's immune defences is one of the most crucial aspects of bacterial pathogenesis. Although coagulase-negative staphylococci are thought to be of low pathogenicity, their capacity to colonize and

Correspondence to: John Tresguerres, Department of Genetics, Physiology and Microbiology, Complutense University of Madrid, Madrid, Spain, E-mail: jtresguerres@bio.ucm.es

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build biofilms within Intravenous (IV) lines can present a serious infection challenge for critically ill patients. On the same plastic polymers used in line manufacturing, catecholamines at the amounts administered down IV lines were found to promote staphylococcal biofilm formation. Moreover, *P. aeruginosa* biofilm growth on endotracheal tubes was promoted by clinical catecholamine concentrations (used to maintain an open airway in ventilated patients). Three main areas of research on catecholamine stress hormones' effects on bacterial infectiousness have been identified: bacterial growth, pathogenicity, and gene expression. The majority of stress hormone-bacteria interaction research has mainly focused on bacterial interactions with noradrenaline and adrenaline and employed catecholamine levels.