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Microbe-Endocrine Hormone Interactions

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Introduction

The influence of hormones on human cells is very well characterized, yet much less understood is the response to those chemical signals of the 1013-1014 bacteria and fungi that are co-resident within the human frame [1]. Microbial Endocrinology is a research area which seeks to understand the role of microbial interactions with mammalian hormones in conditions of health and disease [2-4]. It takes the view that through their long evolutionary relationship with animals microorganisms have evolved systems for sensing hormones which they use as an indicator that they are within the proximity of a potential host. This article considers what happens when the human microbiota come into contact with the chemical signals of their host, and the health significance of this inter-kingdom-encounter.

Hormones can be classified on the basis of their chemical structures: amino acid, peptide and protein and cholesterol based, and the receptor location by which the hormonal signal is transduced. The function of each hormonal type will be described, and the health implications to the host when the hormone is encountered by potentially infectious bacteria and fungi. Structures of the hormones covered and the microbes which recognize them can be found in Table 1, respectively [5-39].

Table 1: Hormone responsive microorganisms.

Species	Hormone/Metabolite	Growth	Virulence	Reference
Aeromonas hydrophila	NE	+	+	Kinney et al. [5]
Acinetobacter lwoffii	NE	+		Freestone et al. [6]
Bordetella bronchiseptica,B. pertussis	NE, Adr, Dop	+	+	Anderson & Armstrong [7]
Borrelia burgdorferi	NE		+	Scheckelhoff et al. [8]
Brachyspira pilosicoli	NE	+	+	Naresh & Hampson [9]
Campylobacter jejuni	NE	+	+	Cogan et al. [10]
Citrobacterfreundii, C. rodentium	NE	+		Freestone et al. [6], Bailey et al. [11]
Enterobacter agglomerans, E. sakazaki	NE	+		Freestone et al. [6]
Enterococcus faecalis, E. cloacae	NE	+		Freestone et al. [6]
Escherichia coli (commensal and pathogenic)	NE, Adr, Dop, Iso, Dob, DHPG, DHMA	+	+	Lyte & Ernst [12], Freestone et al. [6,13-15], Green et al. [16], Vlisidou et al. [17], Sandrini et al. [18]
Hafnia alvei	NE	+		Freestone et al. [6]
Helicobacter pylori	NE	+		Doherty et al. [19]
Klebsiella oxytoca, K. pneumoniae	NE	+		Freestone et al. [6]
Listeria monocytogenes	NE, Adr, Dop	+		Coulanges et al. [20], Freestone et al. [6]
Morganella morgana	NE	+		Freestone et al. [6]
Mycoplasma hyponeumoniae	NE		+	O'Neal et al. [21]
Proteus mirabilis	NE	+		Freestone et al. [6]

Pseudomonas aeruginosa	NE, Adr, Dop	+	+	Alverdy et al.[22], Freestone et al. [6,23]
Salmonella enterica, Salmonella Typhimurium	NE, Adr, Dop	+	+	Freestone et al. [6,24] Methner et al. [25], Pullinger et al. [26]
Shigella sonnei, S. flexneri	NE	+		Freestone et al. [6,27]
Staphylococcus aureus	NE, Dop	+		Freestone et al. [27]
Staphylococcus epidermidis, S. capitis,				
S. saprophyticus, S. haemolyticus, S. hominis	NE, Adr, Dop, Iso, Dob	+	+	Freestone et al. [6,27]
Streptococcus dysgalactica	NE	+		Freestone et al. [6]
Vibrio parahaemolyticus, V. mimicus, V. vulnificus	NE, Adr, Dop	+	+	Nakano et al. [28]
Xanthomonas maltophila	NE	+		Freestone et al. [6]
Yersinia enterocolitica	NE, Adr, Dop,	+		Freestone et al. [6,29]
Periodontal pathogens	NE, Adr	+		Roberts et al. [30]
Actinomyces gerenscseriae, A. naeslundii, A. odontolyticus, Campylobacter gracilis, Capnocytophaga sputigena, C. gingivalis, Eikenella corrodens, Eubacterium saburreum, Fusobacterium periodonticum, F. nucleatum subsp. Vincentii, Leptotrichia buccalis, Neisseria mucosa, Peptostreptococcus anaerobius, P. micros, Prevotella denticola, P. Melaninogenica, S. intermedius, S. gordonii, S. constellatus, S. mitis, S. mutans, S. sanguis				
Burkholderia pseudomallei	Insulin		+	Wood et al. [31]
Candia albicans	Oestrogen			
Progesterone				
Luteinising hormone	+			
+	+			
+				
+	Kinsman et al. [32], White & Larsen [33], Tarry et al. [34]			
Banerjee et al. [35]				
Bramley et al. [36,37]				
Chlamydia trachomatis	Oestrogen			
Progesterone		+		
+	Sonnex [38]			
Sonnex [38]				
E. coli	ACTH		+	Schreiber & Brown [39]

The '+' indicates that the hormone shown, or their metabolites have induced enhancement of growth or virulence of the bacterial species shown. Key: NE, noradrenaline; Adr, adrenaline; Dop, dopamine; Iso, isoprenaline; Dob, dobutamine; DHPG, dihydroxy phenylglycol; DHMA, dihydroxy mandelic acid, ACTH, adrenocorticotrophic hormone.

This table was adapted with permission from Freestone [3].

Amino Acid-Derived Hormones

These are commonly derived from dietary tyrosine and tryptophan, and comprise two main types: thyroid hormone such as thyroxine and the catecholamines dopamine, noradrenaline and adrenaline [5]. Catecholamines are well studied as they possess a diversity of signaling functions and are widely distributed throughout the tissues and organs of the human body [5]. Noradrenaline and adrenaline are neurotransmitters but also play an integral role in the flight or fight response. In terms of the infection significance of catecholamine release, the field of psychoneuroimmunology has long reported that stress hormone elevations in humans and animals increases their risk of developing an infection. This is in part due to

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stress-released catecholamine and glucocorticoid hormones reducing the functionality of the immune system [6,7]. More recently, Microbial Endocrinology studies have shown that like immune cells many bacteria involved in human infections recognize catecholamines which they appear to use as an indicator that their host is stressed, and possibly less able to mount a defense to the invading microbe [3,4]. Table 1 shows the catecholamine-responsive microbes that have been identified so far. Most analyses of bacterial stress hormone interactions have looked at growth effects using serum- or blood-based culture media, chosen to more closely reflect the host environment in which the hormone will be encountered [40]. Blood or serum containing media is iron limited due to the presence of ferric iron sequestering proteins such as transferrin or lactoferrin which inhibits the growth of most bacterial pathogens [41]. Because iron is so essential for the *in-vivo* growth of bacteria [42], its limitation by transferrin and lactoferrin represents a key immune defense against infection. However, bacteria can directly use catecholamines as a kind of siderophore to steal transferrin and lactoferrin Fe which enables up to 100,000-fold increases in bacterial cell numbers in what normally should be highly bacteriostatic host tissue fluids [14,15,18,23].

Dopamine, noradrenaline and adrenaline exposure can also induce pathogenic bacteria to become even more virulent by inducing expression of genes in toxin release [43], increasing biofilm formation [18] and enhancing attachment to host epithelial tissues [16,17]. Catecholamines can even catalyze recovery of bacteria severely damaged by antibiotic treatment [18,27], and rapidly promote exchange of genetic material between different bacterial species [44]. In terms of the infection significance of catecholamine-microbe interactions, catecholamines are used therapeutically in acutely ill patients to maintain heart and kidney function [5]. Catecholamines at the levels infused down intravenous catheter lines were found to massively increase staphylococcal biofilm formation on the same plastic, while clinically attainable levels of catecholamines also increased *P. aeruginosa* biofilm formation on endotracheal tubing (used to maintain an open airway in ventilated patients) as well as enabling the pathogen to resist antibiotic treatment [18].

Peptide and Protein Hormones

There are reports of peptide-like hormones affecting the infectious potential of pathogenic bacteria. Melioidosis is an infectious disease caused by the Gram-negative bacterium Burkholderia pseudomallei, which tends to be found in soil and water of tropical climates such as Vietnam and parts of Australia. It has been observed that type I diabetes mellitus is an apparent risk factor for the development of the septicemic form of melioidosis [20]. Woods and co-workers found that *B. pseudomallei* can directly bind human insulin and that each bacterial cell expressed around 5000 surface-associated insulin receptors. Woods et al. [31] showed that insulin inhibited the growth of B. pseudomallei and suggested that the deficiency of the hormone at least in part explained the higher risk of melioidosis in insulin-dependent diabetics [31].

Adrenocorticotropic hormone (ACTH) is a peptide hormone that induces the adrenal cortex to produce corticosteroid hormones such as cortisol which contribute to regulation of systemic glucose levels. It is therefore interesting that Schreiber and Brown found that exposure to ACTH increased attachment of *E. coli* O157:H7 to gut epithelia, though the underlying mechanism for this response is not clear [39]. Thyrotropin is a pituitary hormone that induces the thyroid gland to produce thyroxine followed by triiodothyronine which stimulates oxidative respiration and organ development. Interestingly, use of radiolabelled thyrotropin has showed the presence of receptor for thyrotropin in *Yersinia enterocolitica* [45,46]. The thyrotropin specificity of the *Y.enterocolitica* binding activity was similar to that of the thyrotropin receptor in human thyroid tissue. This binding activity is thought to have implications for Graves' disease, which is an autoimmune disease in which thyroid-stimulating antibodies to the thyroid-stimulating hormone receptor mimic thyroid-stimulating hormone, which activates the receptor leading to hyperthyroidism. Thyrotropin binding sites on have been shown to be recognized by antibodies from humans with Graves' disease, and prior infection by *Y. enterocolitica* has been implicated in the pathogenesis of Graves' disease [46]. The outer membrane porins Omp A,C and F have been identified as the *Y. enterocolitica* targets recognized by Graves' patient antibodies, though their role in contributing to development of Graves' disease remains to be shown [47].

Candida albicans is a dimorphic opportunistic fungal pathogen of females and the immunocompromised which has been shown to interact with several human peptide hormones. Luteinizing hormone is required for ovulation and the formation of a corpus luteum in the female menstrual cycle. *C. albicans* has been shown to bind human luteinizing hormone and chorionic gonadotropin [36]. Bramley et al. [36] used (125I)-labeled luteinizing hormone and chorionic gonadotropin to demonstrate the presence of specific binding sites for both hormones in *C. albicans*, and *C. tropicalis* [36]. The binding activity was found to be highly specific and was not surface associated instead being at greatest levels in microsomes and cytoplasmic fractions. Also, of considerable relevance to *C. albicans* infectivity, interaction with the luteinizing hormone was found to stimulate germination of Candida spores and germ tube formation [32].

Cholesterol-Derived Hormones

Cholesterol is the chemical basis of steroid hormones such as oestrogen, progesterone and testosterone which regulate aspects of the metabolism, tissue differentiation and reproductive cycles of females and males. Investigations from a variety of researchers have shown that exposure of some bacteria and fungi to steroid hormones can elevate infection risk in certain patient groups. For instance oestrogen have been shown to increase the likelihood of urogenital infections, particularly during

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pregnancy, or in women taking high oestrogen contraceptives or hormone replacement therapy [38]. *Chlamydia trachomatis* is an important sexually transmitted pathogen, especially in young women; Sonnex [38] reported that treatment of *C. trachomatis* with physiological levels of oestrogen increased infection of human endometrial cells, and enhanced Chlamydia colonisation of female mice. *C. trachomatis* infection of female mice was also increased following pre-treatment with progesterone. *C. albicans* is a major source of fungal infections in women of reproductive age [38] which has been shown to possess an oestrogen binding protein of high affinity and specificity [32,33,34]. Contact with oestrogen has been reported to increase *C. albicans* growth as well as its infectivity, causing the yeast to shift into to a more invasive hyphal morphology [33]. Tarry et al. [34] showed that *C. albicans* vaginal colonization in a rat model of infection was increased over 8-fold when a physiological level of oestrogen was present [38]. Banerjee et al. [35] investigated the effects of progesterone on *C. albicans* gene expression and found that expression of 99 genes was differentially affected by the hormone. Most changes were metabolism associated such as protein synthesis and cellular transport. Of relevance to infection risk was the finding that expression of virulence associated genes such as those involved in hyphal induction, pathogenesis and multi-drug resistance genes were significantly increased in progesterone-treated Candida [48-50].

Conclusion

The effects of endogenous hormones on mammalian cell are well understood, yet although microbes within the human body will repeatedly encounter their host hormones the biological significance to the host of these interactions is only now becoming apparent. This review examined only a few of the many hormones within the human body, but still revealed that there are considerable health implication for some of the microbe-hormone encounters. Table 1 revealed that the most extensively studied area of microbial endocrinology is catecholamine-related, largely because of the long held view of stress increasing infection risk. However it is clear that other types of contact the human microbiota may have with mammalian hormones has health implications. It will be interesting to discover if additional signals within our hormonal milieu are being sensed by the thousands of other species of microbes we host.

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References

1. Sekirov I, Russell SL, Antunes LC, Finlay BB (2010) Gut microbiota in health and disease. Physiol Rev 90(3): 859-904.
2. Lyte M (2004) Microbial endocrinology and infectious disease in the 21st century. Trends Microbiol 12(1): 14-20.
3. Freestone P (2013) Communication between bacteria and their hosts. Scientifica 2013: 361073.
4. Sandrini S, Aldriwesh M, Alruways M, Freestone P (2015) Microbial endocrinology: host–bacteria communication within the gut microbiome. J Endocrinol 225(2): R21-R34.
5. Kinney KS (1999) Catecholamine enhancement of Aeromonas hydrophila growth. Microbial pathogenesis 26(2): 85-91.
6. Freestone PP, Haigh RD, Williams PH, Lyte M (1999) Stimulation of bacterial growth by heat-stable, norepinephrine-induced autoinducers. FEMS Microbiol Lett 172(1): 53-60.
7. Anderson MT, Armstrong SK (2006) The Bordetella bfe system: growth and transcriptional response to siderophores, catechols, and neuroendocrine catecholamines. J Bacteriol 188(16): 5731-5740.
8. Scheckelhoff MR, Telford SR, Wesley M, Hu LT (2007) *Borrelia burgdorferi* intercepts host hormonal signals to regulate expression of outer surface protein A. Proc Natl Acad Sci U S A 104(17): 7247-7252.
9. Naresh R, Hampson DJ (2011) Exposure to norepinephrine enhances *Brachyspira pilosicoli* growth, attraction to mucin and attachment to Caco-2 cells. Microbiology 157(2): 543-547.
10. Cogan TA, Thomas AO, Rees LE, Taylor AH, Jepson MA, et al. (2007) Norepinephrine increases the pathogenic potential of *Campylobacter jejuni*. Gut 56(8): 1060-1065.
11. Bailey MT, Dowd SE, Parry NM, Galley JD, Schauer DB, et al. (2010) Stressor exposure disrupts commensal microbial populations in the intestines and leads to increased colonization by *Citrobacter rodentium*. Infect Immun 78(4): 1509-1519.
12. Lyte M, Ernst S (1993) Alpha and beta adrenergic receptor involvement in catecholamine-induced growth of gram-negative bacteria. Biochem Biophys Res Commun 190(2): 447-452.
13. Freestone PP, Williams PH, Haigh RD, Maggs AF, Neal CP, et al. (2002) Growth stimulation of intestinal commensal *Escherichia coli* by catecholamines: a possible contributory factor in trauma-induced sepsis. Shock 18(5): 465-470.
14. Freestone PP, Lyte M, Neal CP, Maggs AF, Haigh RD, et al. (2000) The mammalian neuroendocrine hormone norepinephrine supplies iron for bacterial growth in the presence of transferrin or lactoferrin. J Bacteriol 182(21): 6091-6098.
15. Freestone PP, Haigh RD, Williams PH, Lyte M (2003) Involvement of enterobactin in norepinephrine-mediated iron supply from transferrin to enterohaemorrhagic *Escherichia coli*. FEMS microbiology letters 222(1): 39-43.
16. Green BT, Lyte M, Chen C, Xie Y, Casey MA, et al. (2004) Adrenergic modulation of *Escherichia coli* O157: H7 adherence to the colonic mucosa. Am J Physiol Gastrointest Liver Physiol 287(6): G1238-G1246.
17. Vlisidou I, Lyte M, van Diemen PM, Hawes P, Monaghan P, et al. (2004) The neuroendocrine stress hormone norepinephrine augments Escherichia coli O157: H7-induced enteritis and adherence in a bovine ligated ileal loop model of infection. Infect Immun 72(9): 5446-5451.
18. Sandrini SM, Raminder S, Jonathan W, Remya M, Richard DH, et al. (2010) Elucidation of the mechanism by which catecholamine stress hormones liberate iron from the innate immune defense proteins transferrin and lactoferrin. Journal of bacteriology 192(2): 587-594.
19. Doherty NC, Tobias A, Watson S, Atherton JC (2009) The Effect of the Human Gut-Signalling Hormone, Norepinephrine, on the Growth of the Gastric Pathogen *Helicobacter pylori*. Helicobacter 14(3): 223-230.
20. Coulanges V, Andre P, Vidon DJM (1998) Effect of Siderophores, Catecholamines, and Catechol Compounds on *Listeria spp.* Growth in Iron-Complexed Medium. Biochemical and biophysical research communications 249(2): 526-530.

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21. Oneal MJ, Schafer ER, Madsen ML, Minion FC (2008) Global transcriptional analysis of Mycoplasma hyopneumoniae following exposure to norepinephrine. Microbiology 154(9): 2581-2588.

22. Alverdy J, Holbrook C, Rocha F, Seiden L, Wu RL, et al. (2000) Gut-derived sepsis occurs when the right pathogen with the right virulence genes meets the right host: evidence for *in vivo* virulence expression in *Pseudomonas aeruginosa*. Ann Surg 232(4): 480-489.

23. Freestone PP, Hirst RA, Sandrini SM, Sharaff F, Fry H et al. (2012) Pseudomonas aeruginosa-catecholamine inotrope interactions: a contributory factor in the development of ventilator-associated pneumonia? Chest 142(5): 1200-1210.

24. Freestone PP, Haigh RD, Lyte M (2007) Blockade of catecholamine-induced growth by adrenergic and dopaminergic receptor antagonists in *Escherichia coli* O157: H7, *Salmonella enterica* and *Yersinia enterocolitica*. BMC Microbiol 7(1): 8.

25. Methner U, Rabsch W, Reissbrodt R, Williams PH (2008) Effect of norepinephrine on colonisation and systemic spread of Salmonella enterica in infected animals: role of catecholate siderophore precursors and degradation products. Int J Med Microbiol 298(5): 429-439.

26. Pullinger GD, Sonya CC, Fathima FS, Pauline MD, Francis D, et al. (2010) Norepinephrine augments Salmonella enterica-induced enteritis in a manner associated with increased net replication but independent of the putative adrenergic sensor kinases QseC and QseE. Infection and immunity 78(1): 372-380.

27. Freestone PP, Haigh RD, Lyte M (2008) Catecholamine inotrope resuscitation of antibiotic-damaged staphylococci and its blockade by specific receptor antagonists. J Infect Dis 197(7): 1044-1052.

28. Nakano M, Takahashi A, Sakai Y, Nakaya Y (2007) Modulation of pathogenicity with norepinephrine related to the type III secretion system of *Vibrio parahaemolyticus*. J Infect Dis 195(9): 1353-1360.

29. Freestone PP, Haigh RD, Lyte M (2007) Specificity of catecholamine-induced growth in *Escherichia coli* O157: H7, *Salmonella enterica* and *Yersinia enterocolitica*. FEMS microbiology letters 269(2): 221-228.

30. Roberts A, Matthews JB, Socransky SS, Freestone PP, Williams PH, et al. (2002) Stress and the periodontal diseases: effects of catecholamines on the growth of periodontal bacteria *in vitro*. Oral Microbiol Immunol 17(5): 296-303.

31. Woods D, Jones A, Hill PJ (1993) Interaction of insulin with *Pseudomonas pseudomallei*. Infect Immun 61(10): 4045-4050.

32. Kinsman O, Pitblado K, Coulson CJ (1988) Effect of Mammalian Steroid Hormones and Luteinizing Hormone on the Germination of *Candida albicans* and Implications for Vaginal Candidosis./; Die Wirkung von Säugersteroidhormonen und Gelbkörperreifungshormon auf die Keimschlauchbildung von *Candida albicans* und ihre Bedeutung für die Vaginalcandidose. Mycoses 31(12): 617-626.

33. White S, Larsen B, (1997) *Candida albicans* morphogenesis is influenced by estrogen. Cell Mol Life Sci 53(9): 744-749.

34. Tarry W, Fisher M, Shen S, Mawhinney M (2005) *Candida albicans*: the estrogen target for vaginal colonization. J Surg Res 129(2): 278-282.

35. Banerjee D, Martin N, Nandi S, Shukla S, Dominguez A, et al. (2007) A genome-wide steroid response study of the major human fungal 8 pathogen Candida albicans. Mycopathologia 164(1): 1-17.

36. Bramley TA, Menzies GS, Williams RJ, Adams DJ, Kinsman OS (1990) Specific, high-affinity binding sites for human luteinizing hormone (hLH) and human chorionic gonadotrophin (hCG) in Candida species. Biochem Biophys Res Commun 167(3): 1050-1056.

37. Bramley T, Menzies GS, Williams RJ, Kinsman OS, Adams DJ (1991) Binding sites for LH in *Candida albicans*: comparison with the mammalian corpus luteum LH receptor. J Endocrinol 130(2): 177-190.

38. Sonnex C (1998) Influence of ovarian hormones on urogenital infection. Sex Transm Infect 74(1): 11-19.

39. Schreiber KL, Brown DR (2005) Adrenocorticotrophic hormone modulates *Escherichia coli* O157: H7 adherence to porcine colonic mucosa. Stress 8(3): 185-190.

40. Li W, Lyte M, Freestone PP, Ajmal A, Colmer HJA, et al. (2009) Norepinephrine represses the expression of tox A and the siderophore genes in Pseudomonas aeruginosa. FEMS Microbiol Lett 299(1): 100-109.

41. Lambert LA, Perri H, Halbrooks PJ, Mason AB (2005) Evolution of the transferrin family: conservation of residues associated with iron and anion binding. Comp Biochem Physiol B Biochem Mol Biol 142(2): 129-141.

42. Ratledge C, Dover LG (2000) Iron metabolism in pathogenic bacteria. Annu Rev Microbiol 54: 881-941.

43. Lyte M, Erickson AK, Arulanandam BP, Frank CD, Crawford MA, et al. (1997) Norepinephrine-induced expression of the K99 pilus adhesin of enterotoxigenic *Escherichia coli*. Biochem Biophys Res Commun 232(3): 682-686.

44. Peterson G, Kumar A, Gart E, Narayanan S (2011) Catecholamines increase conjugative gene transfer between enteric bacteria. Microb Pathog 51(1-2): 1-8.

45. Weiss M, Ingbar SH, Winblad S, Kasper DL (1983) Demonstration of a saturable binding site for thyrotropin in *Yersinia enterocolitica*. Science 219(4590): 1331-1333.

46. Heyma P, Harrison L, Robins BR (1986) Thyrotrophin (TSH) binding sites on *Yersinia enterocolitica* recognized by immunoglobulins from humans with Graves' disease. Clin Exp Immunol 64(2): 249-254.

47. Hargreaves CE, Grasso M, Hampe CS, Stenkova A, Atkinson S, et al. (2013) *Yersinia enterocolitica* provides the link between thyroid-stimulating antibodies and their germline counterparts in Graves' disease. J Immunol 190(11): 5373-5381.

48. Goldstein DS, Eisenhofer G, Kopin IJ (2003) Sources and significance of plasma levels of catechols and their metabolites in humans. J Pharmacol Exp Ther 305(3): 800-811.

49. Reiche EMV, Nunes SOV, Morimoto HK (2004) Stress, depression, the immune system, and cancer. Lancet Oncol 5(10): 617-625.

50. Glaser R, Kiecolt GJK (2005) Stress-induced immune dysfunction: implications for health. Nat Rev Immunol 5(3): 243-251.

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