

# Neurotransmitters and Pathogenicity of *Vibrio* sp. – Is there a Connection!

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## Introduction

*Vibrio vulnificus* is a gram negative, motile human pathogen usually found in the estuarine waters and oysters are natural inhabitants in these waters where the salinity is approximately 3.5%. Eating raw or undercooked oysters can cause severe gastroenteritis and also generalized septicemia. Necrotizing wound infections are known to occur when the wound is exposed in the estuarine waters harboring this pathogen. Immune compromised persons are more prone to severity of these symptoms [1].

Many human pathogens respond to stress conditions that may include, e.g. starvation or the unfavorable temperature of the surrounding environment by altering their physiological state. This includes VBNC (viable but non-culturable) state where the organisms are alive but they cannot be cultivated on usual selective laboratory media. They can be resuscitated when the surrounding environment is conducive for their growth and they acquire the cultivable state [2].

Interestingly, when the water temperature drops below 10°C, *V. vulnificus* enters (VBNC) state and this can be confirmed by live dead staining. They then enter a cultivable state when the temperature of this water rises on laboratory selective media. Interestingly, upon environmental stress oysters accumulate noradrenalin and become susceptible to another oyster pathogen *V. splendidus*. In the presence of adrenocorticotrophic hormone *V. splendidus* proliferates [3].

Stress conditions and norepinephrine (NE) are known to accentuate pathogenicity of *Campylobacter jejuni*, *Escherichia coli* and many other gram-negative pathogens [4]. While researching on *V. vulnificus*, my

unpublished research demonstrated that this pathogen proliferates *in vitro* in the presence of NE and formed biofilms in presence of NE. Confocal microscopy revealed clumping of the organism by live dead staining.

Microbial Endocrinology, coined by Freestone is at forefront of research where the role of catecholamines in bacterial pathogenesis cannot be ignored [4]. The idea that begs the question is whether or not these neurotransmitters also play a role in assisting some of human pathogens including *V. vulnificus* to attain a VBNC state *in vivo*, only to emerge when the right conditions for proliferation arise, for example, such as low immunity of the host. Second possibility could be that they play a part in aiding quorum sensing as signaling molecules [5]. Such a scenario may apply to *V. vulnificus* or other pathogens in the near vicinity to indirectly contribute to their survival and proliferation.

## References

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