Journal of
Dairy & Veterinary Sciences
ISSN: 2573-2196

Juniper Publishers
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Mini Review
Volume 13 Issue 4 - September 2019
DOI: 10.19080/JDVS.2019.13.555868

Dairy and Vet Sci J

Coagulase-Negative Staphylococci (CNS) as Emerging Mastitis Pathogens

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Submission: August 28, 2019**; Published:** September 18, 2019

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Introduction

Mastitis caused by Coagulase-negative Staphylococci (CNS) usually remains subclinical or mildly clinical [1], however it was reported that CNS mastitis could be aggravated to severe clinical cases [2], but most CNS mastitis cases are chronic [3] based on their intramammary persistence for lactation milk exceeding periods, even extended to the upcoming ones [4]. CNS mastitis is a hidden but serious threat to dairy herd including further horizontal transmission to lactating cows and vertical to suckling calves because of environmental origin of most CNS and hidden subclinical nature [5]. CNS mastitis hazards aren't exclusive to the dairy herds, but also extended to public health due to possible horizontal transmission of resistance genes (Soares et al., 2012) to other human pathogens or direct transmission to humans because of shared zoonotic virulent CNS species between animal and humans [6]. Pathogenicity of CNS is generally amplified by two parameters: invasiveness (capability to permeate the protective barriers and to spread) and toxicity (ability to produce enzymes and toxins). CNS are capable of producing enzymes instead of coagulase enable the invasion of host tissues and spread of the inflammatory process (e.g. lipase, fibrinolysin, urease). Moreover, they were found capable of producing proteolytic enzymes, exotoxins and haemolysins, which facilitate the uptake of iron [7]. Besides other various virulence constituents protecting CNS from local and systematic host immunity actions [8].

Antimicrobial therapy is still an important component in any CNS mastitis control or prophylaxis actions. But, with the indiscriminate use of antimicrobials and emerging of multidrug resistant CNS, desired results are no longer obtained [9]. Antimicrobial resistance in CNS and other mastitis pathogens has been a worldwide concern during the past decades and it has also brought increasing attention to the use of antimicrobials in animal agriculture and its potential impact on public health [10]. The contribution of agricultural antimicrobial use to development and spread of resistance to human pathogens, however, remains under investigation and debate [11]. Mechanism of CNS resistance to antimicrobials including genotypic detection of resistance genes have been investigated for long time to update knowledge that may help in CNS control programs [12]. For example, mecA-encoded alternative penicillin binding protein, PBP2a, causing reduced binding to β-lactams antibiotics [13]. β-lactamases encoded by blaZ gene. Also, antimicrobials inactivating enzymes, efflux pumps and protective methylation of the antibiotic's ribosomal target site help resistance to other common antimicrobials used in dairy medicine as tetracyclines, aminoglycosides and macrolides [14].

Ability to form biofilm is a very important virulence constituent, enabling CNS to be organized in multilayered cell clusters embedded in a matrix of extracellular polysaccharide (slime) permitting persistence of CNS in udder tissue unaffected by antimicrobials and protected from host immunity [7,15]. Biofilms improve the ability of microorganisms to resist adverse factors and colonize the environment besides being mainly accused for repeated therapeutic failures as CNS isolates growing within biofilms are less susceptible to antimicrobials commonly used on dairy farms, including β-lactam members [15]. Therefore, biofilm-formation by CNS species could possibly impede antimicrobial therapy [16]. Biofilm formation in CNS also contributes to distinguish them as a main cause of persistent inta-mammary infection (IMI) which enables CNS to survive in the udder tissue from season to season as a constant source of infection [16,17]. Although biofilms do not appear to affect disease severity [18].

Increased antimicrobials resistance of bacteria causing mastitis including CNS is globally and hazardously increasingly. This what recently guided scientific attention to the plant kingdom members, extracts and essential oils (EOs) as cinnamon [19] and carvacrol [20] which might be a substitute cure once the synthetic chemical compounds are unable to perform their role [21].

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DOI: 10.19080/JDVS.2019.13.555868

How to cite this article: Ismail Rahil. Coagulase-Negative Staphylococci (CNS) as Emerging Mastitis Pathogens. Dairy and Vet Sci J. 2019; 13(3): 555867. DOI: 10.19080/JDVS.2019.13.555868