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Oxidative stress interaction with inflammatory response and its effect on the development of bacterial resistance in chronic infections

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Abstract

Chronic infections are an ongoing health issue on a worldwide level, which has been compounded by the development of bacterial resistance to antimicrobial therapy. To contribute to bacterial adaptation and resistance, two key host-derived conditions interact with each other, specifically, oxidative stress and inflammation. It is a synthesis review of available evidence relating to contributions of long-term oxidative conditions and inflammatory signaling to bacterial persistence, mutation, horizontal gene transfer, and phenotypic resistance. These mechanisms are important to comprehend as they present new perspectives of therapeutic interventions and mitigation of resistance.

Keywords: Oxidative Stress; Effect; Bacterial; Infection

1. Introduction

Chronic infections are a major and increasing health issue in the world that has caused high morbidity, extended hospitalization, and high health expenditures [1]. The persistence of the medical device infection despite vigorous antimicrobial treatment is typical of cystic fibrosis-related lung infections, diabetic foot ulcers, chronic osteomyelitis, periodontitis, and biofilm-associated medical devices infections. Unlike in acute infections, in which pathogens are generally eliminated after a strong yet self-limiting immune response, chronic infections are associated with extended interactions between the host and pathogen, which alters the physiology of these microorganisms, as well as the dynamics between the host cellular immunity and the pathogen, over time. [2].

One of the common characteristics associated with chronic infection is chronic activation of the host immune system. The sustained inflammation of neutrophils, macrophages, and other immunological cells causes sustained synthesis of pro-inflammatory cytokines and reactive oxygen species (ROS) [3]. Though these processes are an indispensable part of the natural protection, an extended activation causes the destruction of tissues, modification of the microenvironmental patterns, and the creation of inflammation niches conditioning the existence of microorganisms [4]. It is caused by repeated waves of oxidative burst that are mediated, at least in part, by NADPH oxidase activity and the action and effects of myeloperoxidase, which exposes bacterial populations to cyclic but persistent oxidative stress [5]. Paradoxically, immune mechanisms will also be able to induce evolution of microbes that are considered adaptive, and they are supposed to kill the pathogens. The damaged DNA, caused by the chronic exposure to ROS, leads to the acceleration of the process of bacteria mutation through the bases and strand breaks [6]. Paradoxically, immune mechanisms will also be able to induce evolution of microbes that are considered adaptive, and they are supposed to kill the pathogens. The damaged DNA, caused by the chronic exposure to ROS, leads to the acceleration of the process of bacteria mutation through the bases and strand breaks [7].

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In addition, chronic inflammation changes the local microenvironment in such a way that indirectly favors bacterial survival. Hypoxia with infiltration of immune cells, nutrient availability with host tissue breakdown and altered pH conditions are a combination of factors that influence bacterial metabolic pathways [8]. These shifts are often conducive to the forming of biofilms (structured microbial community that has extracellular polymeric substances that provide physical protection and metabolic heterogeneity). Biofilms not only limit the penetration of antibiotics, they allow for the easier horizontal transfer of genes and thus spread of resistance determinants [9].

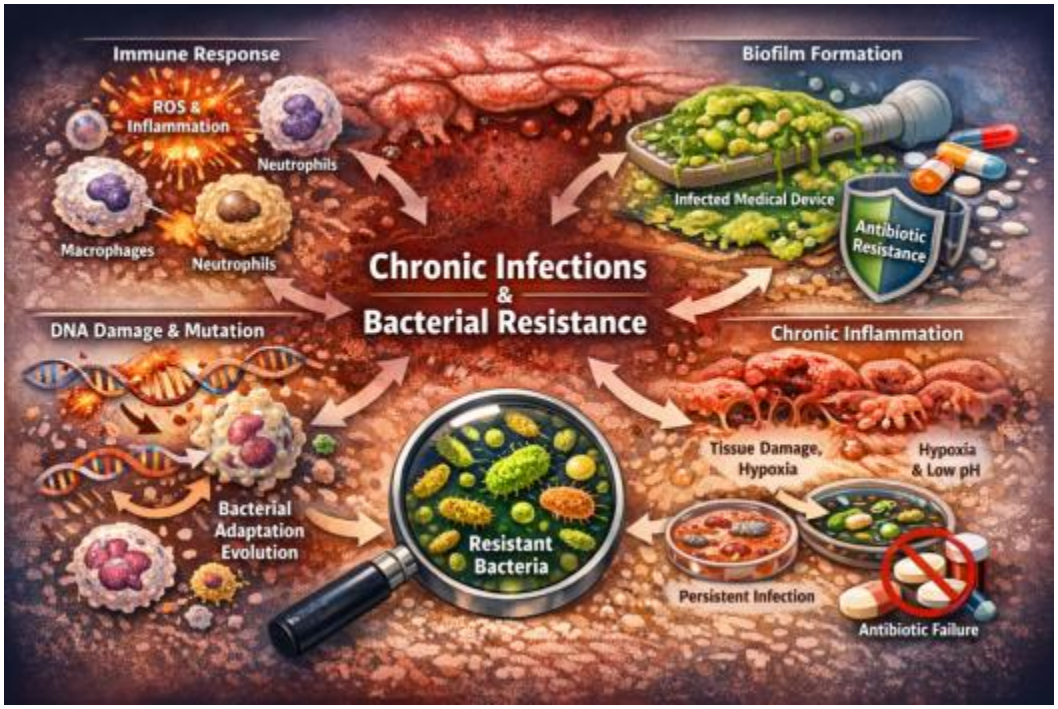

Figure 1 Mechanistic Link Between Chronic Infection and Antimicrobial Resistance

Rather than being simple pressure of bactericide, these host-derived pressures are selective environments in which resilient and genetically adaptable populations of bacteria are selected for. The understanding of this dualistic relationship is critical to the growing crisis of antimicrobial resistance, specifically where treatment failure and relapse are a common problem in chronic clinical settings [10].

This review discusses the mechanistic relationships between chronic oxidative stress and chronic inflammation and the evolution of bacterial resistance [11]. By combining knowledge from immunology, microbiology and molecular pathogenesis, we hope to offer a coherent picture of the role of host defence mechanism in the persistence and adaptive evolutionary developement of bacterial pathogens [12].

2. Oxidative Stress and Its Dual Role

Oxidative stress is a cellular phenomenon that arises when oxidative stress occurs, i.e., the formation of reactive oxygen species (ROS) such as superoxide (O2-), hydrogen peroxide (H2O2), and hydroxyl radicals (OH) becomes greater than the capacity of the antioxidant defense mechanism to detoxify them. In the case of chronic infections, ROS are not passing antimicrobial mediators, but long-term environmental stressors [13]. Long-term oxidative stress alters the genomic stability, bacterial metabolism, and physiology. Although ROS are necessary to clear pathogens, intense oxidative stress forms strong forces of evolution. This two-sidedness, which is antimicrobial and mutagenic, places the oxidative stress in the center of resistance development during chronic infection [14].

Figure 2 The Dual Role of Oxidative Stress in Chronic Infections

2.1. Host ROS Generation

The production of ROS by neutrophils, macrophages, and other phagocytic cells is mainly caused by the activation of the NADPH oxidase complex in the oxidative burst. It is a process that leads to the fast generation of superoxide which is then changed into hydrogen peroxide and other reactive intermediates such as hypochlorous acid through the action of myeloperoxidase. This burst in acute infections is highly controlled and very efficient in killing bacteria. But in chronic infections the activation of the immune system does not stop and there is repeated and prolonged exposure of ROS [15]. Oxidative stress not only destroys the structure of microbes, but also raises the rate of bacterial mutation, triggers global responses to stress, like the SOS system, and induces metabolic transitions to slow-growing or dormant phenotypes that are less susceptible to antibiotics [16].

2.2. Bacterial Responses to Oxidative Stress

A complex antioxidant defense system has been developed by bacteria to endure oxidative attacks, such as catalases, superoxide dismutases, glutathione systems, and peroxiredoxins, as well as enzymes. These systems counteract ROS and uphold a redox homeostasis state in changing environmental conditions. The chronic infection environment has been known to select strains with increased oxidative tolerance and adaptive stress management. [17]. Transcriptional regulators can be activated to repair DNA more actively and frequently in an error-prone manner, to increase genomic variability e.g. through activation of transcriptional regulators like OxyR and the LexA/RecA-mediated SOS response [18]. Moreover, oxidative stress supports the development of biofilm and communal defenses, which additionally support phenotypic tolerance and selective development of antibiotic resistant subpopulations [19].

3. Inflammation as a Selection Pressure

Inflammation is a well-coordinated mechanism of host defense which is coordinated by interaction of cytokines (e.g. IL-1 1 and TNF-), chemokines, adhesion molecules, and recruiting of leukocytes [20]. Nevertheless, during the chronic infection, the inflammation process becomes dysregulated and persistent, leading to the perpetual activation of the immune system and the sequential destruction of the tissue. The chronic inflammatory signaling changes the physicochemical characteristics of the surrounding microenvironment such as oxygen concentration, pH, nutrients, and redox potential [21]. It is these dynamic changes that produce selective pressures that cause microbial adaptation and not elimination, thus, leading to long-term bacterial persistence and development of resistance [22].

3.1. Impact on Bacterial Survival

The long-term effects of the inflammatory conditions of the niche of infection are simply transformative and result in the indirect survival of bacteria. Due to tissue remodelling, increased vascular permeability determines microanatomical niches through which bacteria can avoid effective immune clearance in order to define immune-privileged or poorly perfused niches [23]. At the same time, in the event of the destruction of the host tissues, they release amino acids, lipids, iron and other micronutrients that could be used by the resistant pathogens as metabolic substrates. Hypoxic gradients or microaerophilic bacterial metabolism prone to the action of infiltrating immune cells is also implicated in chronic inflammation [24]. Such environmental changes favour the growth of biofilm facilitating the adoption of organised community lifestyles that are marked by increased resistance to antibiotics and immune effectors by bacteria. All these together turn the areas of inflammation into evolutionary laboratories of resistant populations. [25].

3.2. Cytokine-Modulated Bacterial Behavior

The latest research has shown that bacteria are not anymore mere innocent victims of the action of the inflammatory mediators but may actively react to the action of the host-derived cytokines. Other pathogens detect molecules (catecholamines or pro-inflammatory cytokines) through surface receptors or two-component regulatory systems and overall control the expression of virulence genes [26] . Cytokine-inducing environments are capable of modifying quorum sensing routes, metabolic processes, and stress-response pathways, and increasing adaptive fitness. The upregulation of multidrug efflux pumps and membrane transport systems, along with the decrease in accumulation of intracellular antibiotics, have also been associated with the action of inflammatory mediators, which may also enhance phenotypic heterogeneity in bacterial populations, and thereby enhance the probability of selecting resistant or tolerant subpopulations [27].

4. Interplay Between Oxidative Stress and Inflammation

The biological relationship between oxidative stress and inflammation is that they increase one another in infection. Immune cells, including neutrophils and macrophages, produce reactive oxygen species (ROS), which stimulates intracellular signal transduction, in particular, the NF-kappaB transcription factor, which increases the expression of pro-inflammatory cytokines [28]. This stimulation causes the mediators including TNF-a and IL-6 to be released more, which enhances inflammatory response. Continuous inflammation in its turn brings in other immune cells that generate more ROS which secondary strengthens oxidative stress. The resultant effect is a self-reinforcing cycle of feedback because of the destruction of tissues and selectivity of microbes [29].

This hostile microenvironment will not be so specific in killing all bacteria, but will be biased towards those strains that have better stress-response systems. Bacteria capable of causing antioxidant defense (e.g., catalase, superoxide dismutase) are in a better position to survive as a target of oxidative damage facilitated by the immune system [30]. The evolutionary pressure is brought about in the long-term through continued exposure to oxidative and inflammatory stress. This pressure favors the variants that are tolerant to host immunity and also to antimicrobial therapy. This, therefore, leaves chronic inflammatory niches with harbors of resilient and possibly multidrug-resistant populations [31].

5. Mechanisms of Resistance Evolution

The synergist effect of oxidative stress and inflammation enhances or speeds up the adaptation of bacteria by enhancing genetic variability and selection pressure. Damage via ROS modulates nucleotides, proteins and membranes, which forms an environment and promotes the rate of mutation [32]. Inflammatory mediators in the meantime alter the local oxygen tension, pH and nutrient availability and control the behavior of microbes. It is these environmental pressures that cause rapid selection of resistant phenotypes. They combined, they constitute inflamed tissues evolutionary hotspots of the evolution of antimicrobial resistance [33].

5.1. Mutagenesis

Reactive oxygen species directly cause damage to bacterial DNA, resulting in a change in base, strand breakage, and error in replication. Such changes add to the rate of point mutation of which some can result in antibiotic target site or efflux pump regulation [34]. DNA polymerases that are error-prone are also activated by this response, further raising the rates of mutation. Consequently, oxidative stress leads to mutagenesis that is highly accelerated to generate antibiotic-resistant strains [35].

Additionally, this effect is exacerbated by sublethal exposure to antibiotics in the course of inflammation because mutants with survival advantages are selected. Stress-induced DNA repair mechanisms tend to be less accurate which leads to genomic instability [36]. Membrane permeability or enzyme affinity can be changed even by a small change of nucleotides. With time, resistance profiles get improved due to accumulation of adaptive mutations [37].

5.2. Biofilm-Driven Resistance

Biofilms are structured communities of microorganisms that have been implanted in an autolytically generated extra-cellular matrix that shields bacteria against environmental risks [38]. In biofilms, there are heterogeneous micro environments formed by oxygen and nutrient gradients. Such gradients enhance phenotypic diversity such as the slow-growing or dormant persister cell that is able to withstand antibiotics. As a result, biofilms have great impact on the survival of bacteria in the presence of oxidative and inflammatory stress [39].

Moreover, the presence of oxidative gradients within biofilms can cause local stress responses that cause selection of highly adaptive subpopulations. The proximity of cells makes communication through quorum sensing and coordinated defense strategies easy [40]. Bacteria that are part of a biofilm tend to have more efflux pumps and genes of stress response expressed. Such group strength renders infections hard to eliminate. As such, biofilm formation is a highly important process that connects inflammation with long-lasting antimicrobial resistance [41].

5.3. Horizontal Gene Transfer (HGT)

Both horizontal gene transfer, conjugation, transformation, and bacteriophage-mediated transduction are stimulated by stressful inflammatory conditions. Oxidative stress is capable of enhancing the permeability of the membrane and freeing of DNA, which enables exchange of genes. Antibiotic resistance determinants are often found in mobile genetic components like plasmids, transposon and integron. With selective pressure, bacteria that carry these elements have an advantage to survive. As a result, microbial communities become resistant to genicides at faster rates [42].

Stress responses caused by inflammation can also increase genes that deal with the uptake of DNA and recombination. Biofilm-to-Biofilm close cell-to-cell contact also encourages conjugative plasmid transfer. Transduction by bacteriophages that have been carried to inflamed tissues can also occur, increasing the spread of resistance mechanisms. These processes over time enlarge the resistome of the pathogenic populations. Hence, horizontal gene transfer is an effective evolutionary motor in the oxidative and inflammatory conditions [43].

6. Clinical Implications

The realization that the host-derived oxidative stress plays a role in resistance development changes the treatment emphasis towards the pathogen approach to treatment to host and pathogen interaction management. Several inflammation results in increased selective pressure, which puts stress-resistant and resistant strains at an advantage. Consequently, a more sustained use of antibiotics could be made by balancing instead of escalating the activation of the immune system [44].

Anti-inflammatory interventions applied to the adjoining area may contribute to decreasing the over-stimulation of pathways (NF-kappaB) containing excessive production of cytokines and secondary generation of ROS. Regulated changes in inflammation can potentially lessen tissue damage and cause the decrease in the evolutionary pressure which promotes mutagenesis. This strategy should be strictly moderated not to hamper the surveillance immunity [45].

In like manner, specific antioxidant measures might inhibit the action of ROS-mingled DNA fractures without completely inhibiting antimicrobial oxidative balance. Tuning oxidative processes would help reduce mutation rates and genomic instability in bacterial populations. Simultaneously, individualized treatment plans, according to the immune details of specific patients, can maximize the antimicrobial choice and dosing [46].

Future Directions

Future investigation is required into finding host biomarkers that are reliable when to indicate the potential to drive resistance evolution under inflammatory environments. These biomarkers might inform the early intervention measures and specific treatment plans. This accuracy method can be useful to avoid the establishment of chronic infections [47].

The other attempt is to establish therapeutics capable of disrupting the vicious circle of oxidative stress and inflammation. The stabilisation of the local microenvironment may be attained by targeting some of the signaling cascades or redox- sensitive pathways. [48].

Lastly, improved immune modulation would be worth investigating as a means of prevention of chronic infections. Controlling the level of immune response instead of completely silencing it can help with increased pathogen clearance and reduced collateral stress. One such direction of future research in translational research is to combine immunology and antimicrobial stewardship [49] [50].

7. Conclusion

Chronic infection of the microenvironment leads to the synergic effect of oxidative stress and inflammation to impact the bacterial evolution. Chronic host-associated stress, rather than their mere killing, leads to genetic diversification, phenotypic selection and resistance-selection-of bacteria. To develop new strategies to fight the development of antimicrobial resistance, one should understand the mechanisms thoroughly.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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