



Review

Corrosion fatigue of biomedical metallic alloys: Mechanisms and mitigation

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ABSTRACT

Cyclic stresses are often related to the premature mechanical failure of metallic biomaterials. The complex interaction between fatigue and corrosion in the physiological environment has been subject of many investigations. In this context, microstructure, heat treatments, plastic deformation, surface finishing and coatings have decisive influence on the mechanisms of fatigue crack nucleation and growth. Furthermore, wear is frequently present and contributes to the process. However, despite all the effort at elucidating the mechanisms that govern corrosion fatigue of biomedical alloys, failures continue to occur. This work reviews the literature on corrosion-fatigue-related phenomena of Ti alloys, surgical stainless steels, Co–Cr–Mo and Mg alloys. The aim was to discuss the correlation between structural and surface aspects of these materials and the onset of fatigue in the highly saline environment of the human body. By understanding such correlation, mitigation of corrosion fatigue failure may be achieved in a reliable scientific-based manner. Different mitigation methods are also reviewed and discussed throughout the text. It is intended that the information condensed in this article should be a valuable tool in the development of increasingly successful designs against the corrosion fatigue of metallic implants.

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1. Introduction

The selection of a metallic biomaterial to be employed as a load-bearing orthopedic device should be based on a reliable analysis of relevant materials properties. Fatigue resistance is perhaps the most important issue to be addressed in this case. Several reports indicate that fatigue-related mechanisms are responsible for the most part of mechanical failures of implantable medical metallic components [1–4]. Chao and López [5] reported that nearly 90% of the surface fracture of cementless hip prosthesis manufactured with Ti–6Al–4V alloy was due to fatigue mechanisms. In addition to oscillating mechanical loads, implants are exposed to the physiological fluid that consists of a saline solution [6] including Na^+ , Mg^{2+} , Cl^- , SO_4^{2-} and HCO_3^{2-} . Metallic implants owe their corrosion resistance to the formation of a stable, compact and continuous oxide surface film called passive film that prevents the underlying bare metal surface from coming into contact with these aggressive ions [7]. However, the passive film may be locally dissolved, especially by chloride ions, generating pits that rapidly propagate, leading to pitting corrosion. Nucleation of fatigue cracks has been related to the presence of pits on the surface of metallic materials [8]. Under fatigue conditions, the aqueous environment can accelerate the initiation of a surface flaw and propagate it to a critical

size, leading to fracture. This process is known as corrosion fatigue, denoting the failure of a material under the simultaneous action of cyclic loads and chemical attack [9]. The reduction in fatigue life of metallic implants under corrosion fatigue has been well documented [10–12].

Teoh [13] emphasizes the importance of knowing the surface substructure of biomaterials in order to understand the mechanisms of fatigue failure. The substructure is composed of three distinct layers – a molecular absorbed layer, the passive oxide film and the deformed layer – as shown in Fig. 1. Cyclic loadings lead to the generation of wear debris (contact body). The molecular absorbed layer consists of growing tissue (cells) in contact with the physiological environment and the passive layer on the surface of the metallic implant. The deformed layer arises from the cyclic loadings that cause localized plastic deformation, forming the damage zone in the microstructure of the metallic implant. Many questions arise from this picture: how does the intrinsic microstructure of the alloy influence the ability of the material to withstand cyclic loadings without fatigue failure? How does the stability of the passive film in contact with the physiological environment affect corrosion fatigue mechanisms? Does wear participate in the overall fatigue resistance of the material? How does one evaluate these phenomena? How does one maximize the performance of the medical device against corrosion fatigue? Answering these questions correctly may be the difference between a successful implant life and a premature catastrophic failure. If one thinks that all these phenomena are self-related and thus have

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