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## Introduction

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This symposium reviews several important aspects of the pathophysiology and treatment of the disease. The major emphasis of the symposium is on the role of bone and subchondral bone in the pathogenesis of osteoarthritis (OA) and the potential role of bone-modifying agents as disease-modifying agents in the treatment of this disease.

A number of new concepts about the pathogenesis of OA have been described in the last few decades. This has been made possible through a large body of research undertaken by several investigator and research groups. The major role, played by a number of dedicated people who have created the proper environment to support this research, have made it possible for the international community to understand and appreciate such efforts, and this should be recognized. This meeting is quite unique, as it brings together expertise from two major fields of musculoskeletal research; bone and osteoarthritis. Hopefully this is not the last time.

One may question the rationale behind such a venture. Well, we simply believe that it represents a logical approach to understanding the pathophysiology and future treatment of the disease. Recent advances in the field of osteoarthritis research have clearly shown that osteoarthritis is a disease with the global involvement of the three major tissues of the joints, namely, cartilage, synovium, and subchondral bone. The participation, and the role of synovial inflammation in the progression of the structural changes of OA is now a widely accepted concept.

The morphological changes that take place at the subchondral bone level have been studied for many years, and an association between these changes and the initiation and/or progression of the disease have been suggested. Moreover, a number of studies have now provided strong evidence that even in the early stage of OA, a large number of factors such as cytokine and growth factors, are present in excessive amounts in this tissue. These are likely capable not only of locally modifying the bone metabolism, but also of inducing cartilage remodeling and structural changes. Experimental studies indicate that compounds or drugs that can influence bone metabolism may also modify the progression of OA. This has allowed for a better understanding and appreciation of the role of a number of risk factors in the development of OA. Similarly, the work done on clinical trial outcomes has brought to light the necessity of improving the precision of a number of methods used to evaluate the effectiveness of treatment.

This symposium also aims to bring forward the necessity of discussing and evaluating the technology used to objectively evaluate and quantify the structural changes of osteoarthritis. Additionally, we have time for discussion of pertinent topics in OA research including how to evaluate the disease in the clinic, and identify patients at risk of progression.

We hope you will enjoy and appreciate the exciting exchange about the very challenging disease that is osteoarthritis. We would like to thank Procter & Gamble, and in particular, Dr Joan Meyer, for their support in organizing this symposium. We would also like to thank all of the executives at Procter & Gamble, as well as all of the support staff, that have been worked very hard at putting this symposium together. It would not have been possible without the help of all of these people.

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