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Autore	Firpo, Massimo
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Altri autori (Persone)	Budny, Szymon Paleologo, Iacopo Paruta, Niccolo
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Altri autori (Persone)	CallaghanAlison
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Nota di contenuto	Contents; 8.6.2 Iron status in OA and RA patients; Acknowledgements; Abbreviations; 1 Introduction; 1.1 The range of rheumatic diseases; 1.2 Rheumatoid arthritis (RA):description; 1.3 Osteoarthritis (OA): description; 1.4 Incidence and prevalence; 1.5 Mortality; 1.6 Morbidity; 1.7 Economic cost of arthritis; 1.8 The aim of this book; 2 Classification,pathology and measures of disease assessment; 2.1 Classification of OA; 2.2 Classification of RA; 2.3 Pathology of OA; 2.3.1 General features of OA; 2.3.2 Structure of cartilage; 2.3.3 Pathogenesis of OA; 2.3.3.1 Cartilage degradation 2.3.3.2 Nitric oxide synthesis damages chondrocytes2.3.3.3 Sulphation pattern of GAGs in articular cartilage; 2.3.3.4 Bone changes; 2.3.3.5 Inflammation; 2.3.3.6 Angiogenesis; 2.3.3.7 Oxidative stress; 2.4 Pathology of RA; 2.4.1 General features of RA; 2.4.2 Immunopathogenesis and production of inflammatory mediators; 2.4.3

Autoantibodies:rheumatoid factor; 2.4.4 Glycosylation patterns of immunoglobulins and complement activation; 2.4.5 Dietary lectins,gut translocation and the shared epitope; 2.4.6 Abnormal gut microflora 2.4.7 Reactive oxygen and nitrogen species involved in damage to the rheumatoid joint2.4.7.1 Phagocytosis; 2.4.7.2 Hypoxia reperfusion injury and joint pH; 2.4.7.3 Involvement of nitric oxide and peroxy nitrite; 2.4.7.4 Consequences of the production of reactive oxygen and nitrogen species in the RA joint; 2.4.8 Lipid abnormalities and cardiovascular risk in RA; 2.4.8.1 C-Reactive Protein (CRP); 2.4.8.2 Dyslipidaemia; 2.4.8.3 Endothelial dysfunction; 2.4.8.4 Oxidised LDL in the joint and the formation of fatty streaks; 2.4.8.5 Adhesion molecules; 2.4.8.6 Haemostatic changes 2.4.8.7 Elevated homocysteine and low vitamin B6 status2.4.8.8 Elevated homocysteine and impaired sulphur metabolism; 2.4.8.9 Insulin resistance; 2.4.9 Angiogenesis; 2.4.10 Osteoporosis; 2.5 Assessment of severity of RA and OA; 2.5.1 Outcome measures for RA; 2.5.1.1 Patient 's global assessment; 2.5.1.2 Pain; 2.5.1.3 Disability; 2.5.1.4 Swollen and tender joint counts; 2.5.1.5 Acute phase reactants; 2.5.1.6 RA quality of life index; 2.5.1.7 Radiological assessment; 2.5.2 Some outcome measures for OA; 2.5.2.1 Patient global assessment; 2.5.2.2 Pain score; 2.5.2.3 New joint score 2.5.2.4 Severity score2.5.2.5 Disability; 2.5.2.6 Radiological assessment; 3 Aetiology and risk factors for osteoarthritis and rheumatoid arthritis; 3.1 Introduction; 3.2 Genetic risk factors; 3.3 Age; 3.4 Gender; 3.5 Biomechanical factors as risk factors for OA; 3.5.1 Occupation,sport and physical activity; 3.5.2 Joint trauma and surgery; 3.5.3 Load distribution and malalignment; 3.5.4 Muscle weakness; 3.6 Obesity; 3.7 Smoking; 3.8 Dietary factors; 3.8.1 Olive oil; 3.8.2 Fish and n-3 polyunsaturated fatty acid (PUFA); 3.8.3 Meat; 3.8.4 Fruit and vegetables; 3.8.5 Antioxidants 3.8.6 Vitamin C

Sommario/riassunto

Arthritis affects millions of people throughout the world and while its treatment is usually medical or surgical, there exists an increasingly large body of evidence concerning the positive effects of nutrition on the condition. There are over two hundred forms of rheumatoid disease, with conditions varying in prevalence. In this important title the authors have focussed on osteoarthritis (OA) and rheumatoid arthritis (RA), the most common arthritic diseases with the largest body of dietary data. Including coverage of disease incidence and prevalence, pathology, aetiology and meas
