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Section

Disease Biomarker

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Section Editor-in-Chief

Prof. Dr. Lorenza Rimassa 1. Department of Biomedical Sciences, Humanitas University, 20072 Pieve Emanuele, Milan, Italy
2. Medical Oncology & Hematology Unit, Humanitas Cancer Center, IRCCS Humanitas Research Hospital, 200289 Rozzano, Milan, Italy

lorenza.rimassa@hunimed.eu

Message from the Section Editor-in-Chief

The Section "Disease Biomarker" aims to publish the latest and most relevant research in the field of biomarkers for a broad spectrum of diseases. Important works on solid tumors, blood neoplasms or non-oncological diseases, such as cardiovascular and metabolic diseases, infectious diseases, and neurodegenerative diseases, are welcome. This section supports the submission of manuscripts based on a multidisciplinary and multifaceted approach to promote the development of innovative and personalized therapies for patients with cancer or other severe diseases. Immunotherapy, targeted agents, and new combinations have made significant breakthroughs in cancer treatment and have broadened treatment options. However, the identification of predictive biomarkers still represents an unmet need for most diseases. We encourage the submission of clinical and translational studies that focus on the identification of diagnostic, prognostic, and predictive biomarkers that can help to select and stratify groups of subjects at risk for a given disease, groups of patients with different prognoses, and groups of patients who may benefit more from specific treatment approaches.

This Section aims to provide knowledge of the most significant advances and novel therapeutic options and approaches in the field of precision medicine. Original research papers, systematic reviews, meta-analyses, and reviews are welcome.

Invitation to Submit

Biomarkers for Cholangiocarcinoma/Biliary Tract Cancer
Guest Editor: Prof. Dr. Lorenza Rimassa
Deadline: 10 December 2023

Biomarkers in Diagnosis and Treatment of Pulmonary Disease
Guest Editors: Dr. Kelly Domvri and Dr. Konstantinos Porpodis
Deadline: 5 January 2024

The Gut Microbiome as a Target for the Treatment of Inflammatory Bowel Diseases
Guest Editor: Dr. Limin Shi
Deadline: 15 February 2024

Cancer Biomarkers: Promises and Challenges
Guest Editor: Dr. Vincenzo Quagliariello
Deadline: 29 February 2024

Multiple Myeloma: Biomarkers and Target Therapy
Guest Editor: Dr. Aneta Szudy-Szczyrek
Deadline: 15 April 2024

Gut Microbiome and Its Impact on Human Health
Guest Editors: Dr. Joanna B. Bierła and Dr. Qing Ai
Deadline: 20 April 2024

Biomarkers for Inflammatory and Metabolic Disorders
Guest Editors: Dr. Aldona Wierzbicka and Dr. Joanna B. Bierła
Deadline: 1 June 2024

Biomarkers and Personalized Therapies in Non-hodgkin Lymphomas
Guest Editor: Dr. Joanna Zawitkowska
Deadline: 5 June 2024

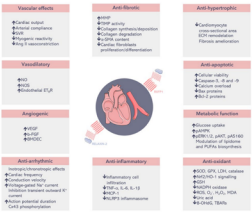
Tailoring Treatment with Biomarkers: Advancements in Heart Failure Management
Guest Editors: Prof. Dr. John S. Skoularigis and Dr. Andrew Xanthopoulos
Deadline: 31 August 2024

Featured Papers

DOI:0.3390/jpm12071021

Relaxin-2 as a Potential Biomarker in Cardiovascular Diseases

Authors: Alana Aragón-Herrera, Sandra Feijóo-Bandín, Laura Anido-Varela, Sandra Moraña-Fernández, Esther Roselló-Lletí, Manuel Portolés, Estefanía Tarazón, Oreste Gualillo, José Ramón González-Juanatey and Francisca Lago

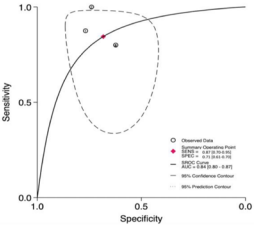


Abstract: The pleiotropic hormone relaxin-2 plays a pivotal role in the physiology and pathology of the cardiovascular system. Relaxin-2 exerts relevant regulatory functions in cardiovascular tissues through the specific receptor relaxin family peptide receptor 1 (RXFP1) in the regulation of cardiac metabolism; the induction of vasodilatation; the reversion of fibrosis and hypertrophy; the reduction of inflammation, oxidative stress, and apoptosis; and the stimulation of angiogenesis, with inotropic and chronotropic effects as well. Recent preclinical and clinical outcomes have encouraged the potential use of relaxin-2 (or its recombinant form, known as serelaxin) as a therapeutic strategy during cardiac injury and/or in patients suffering from different cardiovascular disarrangements, especially heart failure. Furthermore, relaxin-2 has been proposed as a promising biomarker of cardiovascular health and disease. In this review, we emphasize the relevance of the endogenous hormone relaxin-2 as a useful diagnostic biomarker in different backgrounds of cardiovascular pathology, such as heart failure, atrial fibrillation, myocardial infarction, ischemic heart disease, aortic valve disease, hypertension, and atherosclerosis, which could be relevant in daily clinical practice and could contribute to comprehending the specific role of relaxin-2 in cardiovascular diseases.

DOI:0.3390/jpm13091283

The Value of Fournier's Gangrene Scoring Systems on Admission to Predict Mortality: A Systematic Review and Meta-Analysis

Authors: Antonio Tufano, Piervito Dipinto, Francesco Passaro, Umberto Anceschi, Giorgio Franco, Rocco Simone Flammia, Flavia Proietti, Luca Antonelli, Giovanni Battista Di Pierro, Francesco Prata, Roberta Rullo, Sisto Perdonà and Costantino Leonardo

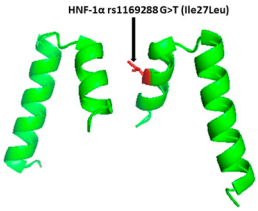


Abstract: Objective: To systematically review and meta-analyze the predictive value of the Fournier gangrene severity index (FGSI), the simplified FGSI (SFGSI), and the Uludag FGSI (UFGSI) on mortality in patients affected by Fournier's Gangrene (FG). Methods: A search was performed in PubMed, Web of Science, Embase, and the Cochrane Library, from January 2000 to May 2023, to identify original cohorts comparing data between surviving and non-surviving FG patients. The statistical analysis consisted of two parts. First, the mean and standard deviation (SD) of the FGSI, SFGSI, and UFGSI at admission were extrapolated from each study, and the pooled mean difference (MD) with 95% confidence interval (95% CI) was obtained using the Der Simonian–Laird random-effect model. Second, to evaluate the accuracy of the FGSI, SFGSI, and UFSGI in predicting mortality, true positive (TP), false positive (FP), true negative (TN), and false negative (FN) values were extracted where possible and reported in 2 × 2 contingency tables. The sensitivity, specificity, and AUC values were pooled, and summary receiver operating characteristic (SROC) curves were constructed. Results: Overall, forty studies comprising 2257 patients were included. The pooled analysis revealed that the FGSI, SFGSI, and UFGSI values at admission were higher in non-survivors than survivors (MD: 5.53 (95% CI: 4.68–6.37); MD: 2.41 (95% CI: 1.06–3.77); and MD: 5.47 (95% CI: 3.68–7.26), respectively). Moreover, the AUC values of the FGSI, SFGSI, and UFGSI were 0.90 (95% CI: 0.87–0.92), 0.84 (95% CI: 0.80–0.87), and 0.94 (95% CI: 0.92–0.96), respectively. Conclusions: The higher scores of the FGSI, SFGSI, and UFGSI on admission were associated with mortality. Moreover, when comparing accuracy rates, the UFGSI exhibited the highest AUC value.

DOI:0.3390/jpm13081270

Comprehensive Molecular Evaluation of HNF-1 Alpha, miR-27a, and miR-146 Gene Variants and Their Link with Predisposition and Progression in Type 2 Diabetes Patients

Authors: Rashid Mir, Imadeldin Elfaki, M. E. Elangeeb, Mamdoh S. Moawadh, Faris Jamal Tayeb, Jameel Barnawi, Ibrahim Altedlawi Albalawi, Amnah A. Alharbi, Marwan H. Alhelali and Basim S. O. Alsaedi

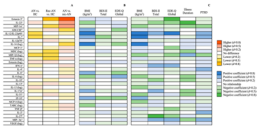


Abstract: Background: Type 2 diabetes (T2D) is a metabolic condition induced by insulin resistance and pancreatic beta cell dysfunction. MicroRNAs (miRNAs) have biological significance because they regulate processes such as the molecular signaling pathways involved in the pathophysiology of diabetes mellitus. The hepatocyte nuclear factor-1 alpha (HNF-1 alpha) is a transcription factor found hepatocytes and the pancreas. Mutations in the HNF-1 alpha gene were reportedly associated with maturity-onset diabetes of the young (MODY). The objective of the present study was to examine the associations between MiR-27a, MiR-146, and HNF-1 alpha single-nucleotide variations (SNVs) with T2D risk in the Saudi population. Methodology: We evaluated the association of SNVs of miR-27a rs895819 A>G, 146a-rs2910164 C>G, and HNF-1 alpha rs1169288 G>T (I27L) with the risk of T2D in Saudi patients with the Amplification Refractory Mutation System PCR (ARMS-PCR). For the miR-27a SNVs, we used 115 cases (82 males, 33 females) and 117 matched healthy controls (HCs); for the Mir-146 SNVs, we used 103 cases (70 males, 33 females) and 108 matched HCs; and for the HNF-1 alpha, we employed 110 patients (80 males, 30 females) and 110 HCs. The blood biochemistry of the participants was essayed using commercial kits, and the methods of statistical analysis used were the Chi-square test, the Fisher exact test, and a multivariate analysis based on logistic regression, like the odds ratio (OD) and risk ratio (RR), with 95% confidence intervals (CIs). Results: The MiR-27a rs895819 AG genotype was linked to increased T2D susceptibility, with OR = 2.01 and p-value = 0.011, and the miR-146 rs2910164 CG genotype and C allele were linked to an elevated risk of T2D, with OR = 2.75, *p*-value < 0.0016, OR = 1.77, and *p*-value = 0.004...

DOI:0.3390/jpm11080814

Reduced MIP-1β as a Trait Marker and Reduced IL-7 and IL-12 as State Markers of Anorexia Nervosa

Authors: Johanna Louise Keeler, Olivia Patsalos, Raymond Chung, Ulrike Schmidt, Gerome Breen, Janet Treasure, Hubertus Himmerich and Bethan Dalton



Abstract: Alterations in certain inflammatory markers have been found in individuals with anorexia nervosa (AN). However, their relation to clinical characteristics has not been extensively explored, nor is it clear whether they are trait or state features of the disorder. This cross-sectional study measured serum concentrations of 36 inflammatory markers in people with acute AN (*n* = 56), recovered AN (rec-AN; *n* = 24) and healthy controls (HC; *n* = 51). The relationship between body mass index (BMI), eating disorder psychopathology, depression symptoms and inflammatory markers was assessed. Statistical models controlled for variables known to influence cytokine concentrations (i.e., age, ethnicity, smoking status and medication usage). Overall, most inflammatory markers including pro-inflammatory cytokines were unchanged in AN and rec-AN. However, in AN and rec-AN, concentrations of macrophage inflammatory protein (MIP)-1β were lower than HCs. Interleukin (IL)-7 and IL-12/IL-23p40 were reduced in AN, and concentrations of macrophage-derived chemokine, MIP-1α and tumor necrosis factor-α were reduced in rec-AN compared to HC. In conclusion, a reduction in MIP-1β may be a trait marker of the illness, whereas reductions in IL-7 and IL-12/IL-23p40 may be state markers. The absence of increased pro-inflammatory cytokines in AN is contradictory to the wider literature, although the inclusion of covariates may explain our differing findings.

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