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Identification of novel biomarker candidates for hypertrophic cardiomyopathy and other cardiovascular diseases leading to heart failure

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In-depth proteome discovery analysis represents new strategy in an effort to identify novel reliable specific protein markers for hypertrophic cardiomyopathy and other life threatening cardiovascular diseases. To systematically identify novel protein biomarkers of cardiovascular diseases with high mortality, we employed an iTRAQ-based quantitative proteome technology to make comparative analysis of plasma samples obtained from patients suffering from non-obstructive hypertrophic cardiomyopathy, stable dilated cardiomyopathy, aortic valve stenosis, chronic stable coronary artery disease and stable arterial hypertension. We found 128 plasma proteins whose abundances were uniquely regulated among the analyzed cardiovascular pathologies. Most of them have not been described yet. Additionally, application of statistical exploratory analyses of the measured protein profiles indicated the relationship in pathophysiology of the examined cardiovascular pathologies.

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Role of post-translational modifications by ubiquitin family of proteins in resistance mechanisms of pancreatic cancer cells

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Pancreatic cancer remains nowadays one of the deadliest forms of cancer. This situation is partly due to the fact that these tumors become rapidly resistant to any kind of therapies. Our aim is to identify new resistance mechanisms which would explain the multi-resistant phenotype of pancreatic cancer cells. Chemotherapies trigger cellular stress responses that help the cell to survive. These responses are based on the post-translational modification (PTMs) of key components of these pathways. Among all possible PTMs, modifications mediated by ubiquitin family members appear to play major roles in these processes but, so far, have not been really studied in this context. Therefore, we intent to identify alterations of the ubiquitin and ubiquitin-like pathways and to determine which ones are involved in resistance mechanisms. To this end, we use cell lines expressing tagged version of each ubiquitin and ubiquitin-like studied, to specifically purify modified proteins and identify them by tandem mass spectrometry. Hence, we have established the PTMs profiles of pancreatic cancer cells, treated and not treated with chemotherapeutic drugs, resistant or not to them. We could observe that chemotherapeutic treatment, as well as acquisition of the resistant phenotype, is associated with an important modulation of PTMs profiles. Among those, we could validate the role of the modification of one candidate regarding the survival of the cell challenge by gemcitabine. Hence, studying alterations of PTMs mediated by the ubiquitin family of proteins associated with cancer resistance has the potential to reveal important new molecular mechanisms involved in the phenomenon.

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