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# Proteomic analysis in wild type and Cisplatin resistant yeast strain of Saccharomyces cerevisiae.

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## Introduction

Research carried out in oncology has allowed the identification and characterization of molecular markers of resistance to antineoplastic drugs. It supposes a great advance and allows the administration of customized chemotherapies to patients.

Notable successes in this field of research include the overexpression of the HER2 protein as a molecular marker in patients with breast cancer treated with Trastuzumab, and the deletion of exon 19 or the substitution of exon 21 (L858R) in the EGFR gene, in patients with metastatic non–small-cell lung treated with Erlotinib.

Several studies show that the expression of DNA repair genes such as ERCC1, β-tubulins or Toposiomerases can predict the response to Platinum, Taxanos and other cytotoxic agents. While the use of predictive biomarkers in other types of cancer therapies is something common and used, there is not yet a protocol standardized for chemotherapy.

Thus, this shows the great importance and the need to investigate biomarkers of resistance, with the aim of administering more effective and personalized therapies.

## Objectives

The aim of this work is to carry out a proteomic analysis in the wild type strain and cisplatin resistant strain of *Saccharomyces cerevisiae* in order to look for possible markers of resistance, as well as the mechanisms involved.

## Methodology

Protein extraction, purification and identification were carried out in both the wild type strain and cisplatin resistant strain. The method was an analysis by tandem mass spectrometry using a "nano HPLC-ESI-MS / MS" ion trap system.

The exponentially modified protein abundance index (emPAI) offers a relative quantification of proteins in a mixture.

The increase in emPAI of the resistant strain against the wild strain was calculated by dividing the value of emPAI of the resistant strain between that of the wild type strain.

## Results and conclusions

149 proteins were expressed in both strains. An analysis was carried out in order to evaluate which proteins had a higher overexpression. Proteins with an emPai increase higher than 2.5 were selected. The QCR2 (QCR2 gene), QCR1 (COR1 gene), ALDH4 (ALD4 gene), ATPB (ATP2 gene), ATPA (ATP1 gene), SCW10 (SCW10 gene), HSP26 (HSP26 gene), ATPG (ATP3 gene) and PCKA (PCK1 gene) were analyzed.

The QCR2 gene presented co-expression with the ATP1, ATP2, ATP3, COR1, PCK1, SCW10 and ALD4 genes. The COR1 gene presented co-expression with the ATP1, PCK1 and SCW10 genes. The ALD4 gene presented co-expression with the genes ATP1, COR1, PCK1 and SCW10 genes. The ATP2 gene presented co-expression with the SCW10, ATP1, ATP3, COR1, ALD4 and PCK1 genes. The ATP1 gene presented co-expression with the SCW10 and PCKA1

genes. The SCW10 gene presented co-expression with the QCR2, COR1, ALD4, ATP2, ATP3, PCK1 and ATP1 genes. The HSP26 gene presented co-expression with the ATP1, ATP3, ALD4, PCK1, COR1 and QCR2 genes. The ATP3 gene presented co-expression with the genes ATP1, COR1, SCW10 and ALD4 genes. The PCK1 gene presented co-expression with the SCW10 gene.

These proteins are implicated in hydrogen ion transmembrane transporter activity, cellular respiration, active transmembrane transporter activity, mitochondrial inner membrane and mitochondrial respiratory chain.

The increase of these proteins is logical because the cells have to increase their cellular and molecular mechanisms and produce more proteins to resist to the drug and be able to survive.

In conclusion, the increased of expression of these proteins in the cisplatin-resistant strain suggests that they could be involved in the resistance process. Therefore, they could be good candidates for biomarkers of cisplatin chemoresistance.

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