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Pharmacological network study of the effects of Quercetin on gastric cancer using **computerized databases**

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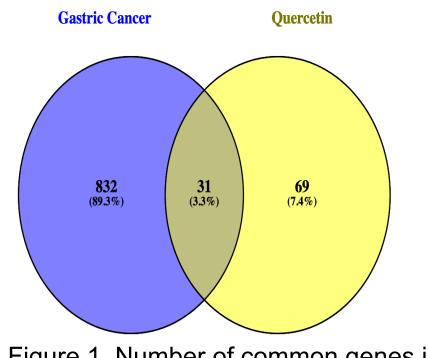
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INTRODUCTION & AIM

The cancer disease can be defined as a part of the body that grows uncontrollably and then migrates to other parts of the body. Normally, cancer can be detected in almost any section of the body (1), including the gastric system. Gastric Cancer is one of the most common types of cancer in the world with a survival rate as low as 10% (2), especially in developing countries (1). Nowadays there are many treatments for GC, including better hygiene, more robust and complete nutrition, and the eradication of pathogens such as Helicobacter pylori. Nevertheless, even with all of those, GC incidence can still be high (3). Therefore the need for more treatments especially those of low cost is more important than ever. For example, those coming from natural sources like onions, grapes, broccoli, citrus fruits, or plants. One product like this is Quercetin (QRC) (4) QRC is a natural phenolic compound that has multiple therapeutic effects including (but not limited to) combating different types of cancer. However, the information regarding the molecular mechanisms of QRC in GC is still unclear. Therefore, this study aims to identify the targets that QRC has, like anti-cancer treatment for GC using different bioinformatic tools and databases



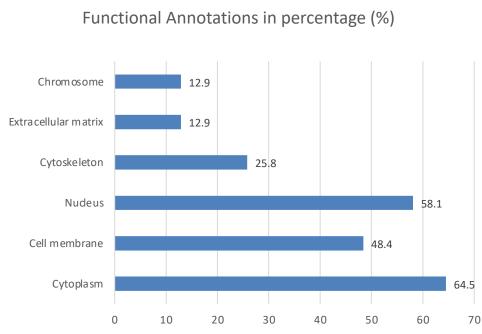
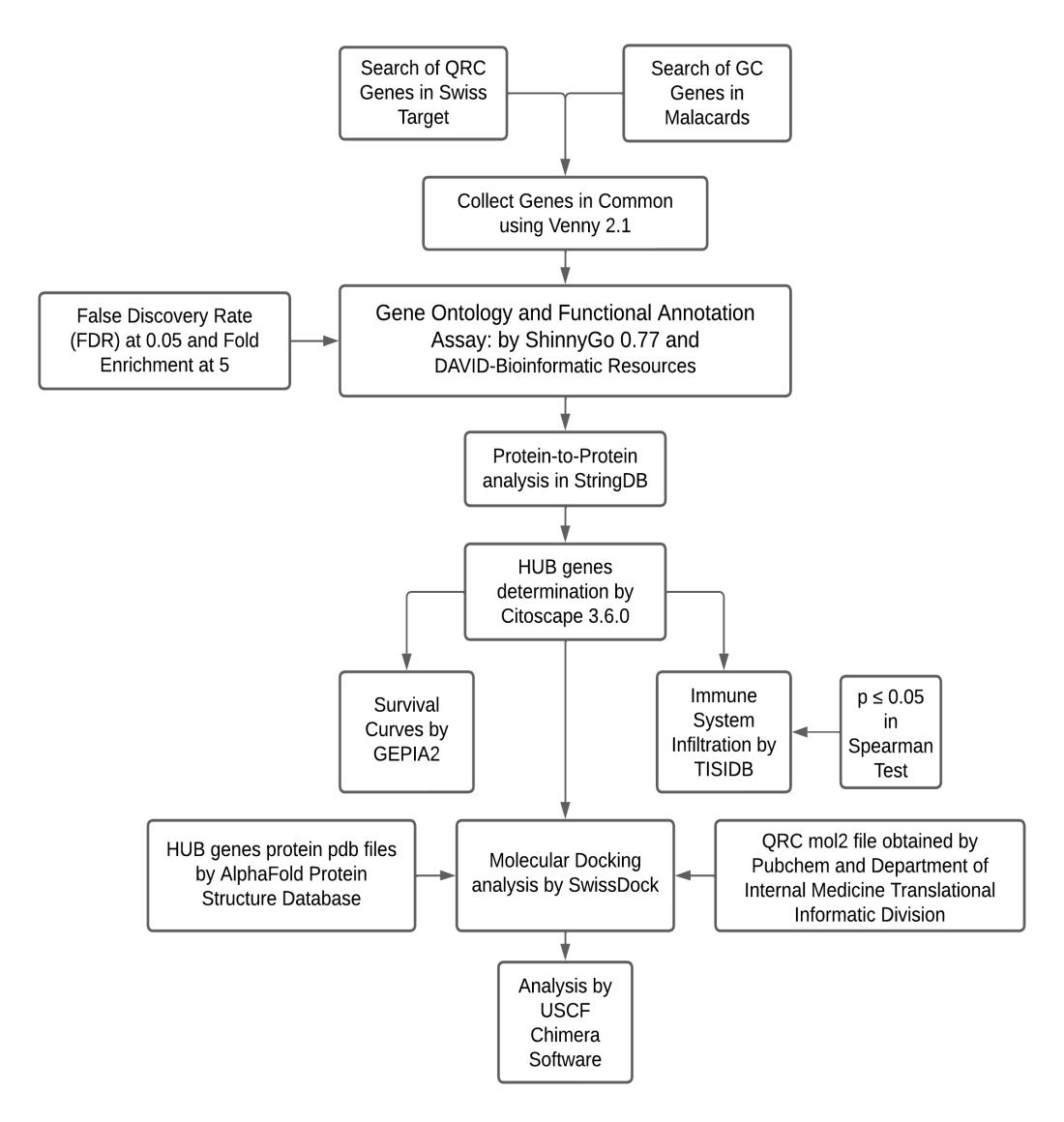


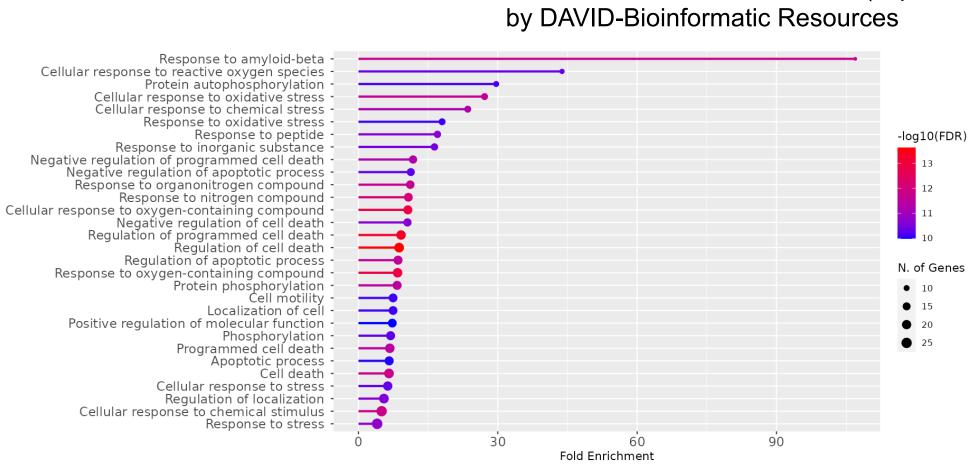
Figure 1. Number of common genes in total between QRC and GE

Figure 2. Functional Annotations of genes in common between QRC and GC (in percentage)

METHOD

The methodology for the following section of the study was based on an article by Martinez-Esquivias et al (5) The full methodology is shown in figure 1.





RESULTS

Figure 3.- Gene analysis onthology Assay by the ShinnyGo 0.77

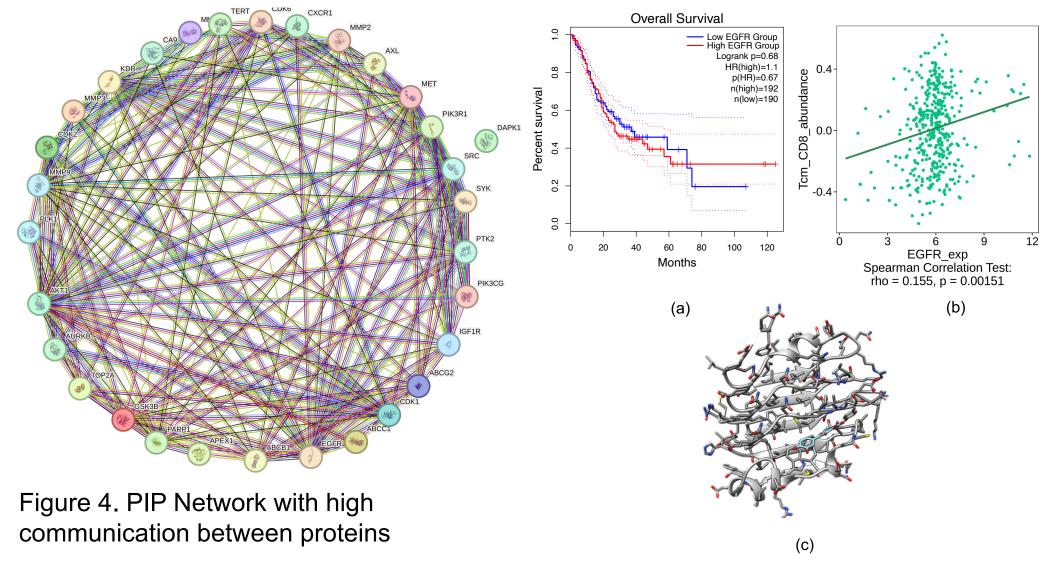


Figure 5. a) Survival rate of EGFR b)Correlation of CD8 Infiltration in Gastric Cancer (Stomach) c) Molecular docking of EGFR and QRC (QRC is light blue, EGFR in mostly white)

HUB genes that were obtained and tested were: AKT1, SYK, PTK2, PIK3R1, PI3K, IGF1R,

Figure 1. Study methodology based on the published by Martinez-Esquivias et al.

GSK3B, CDK6, CDK2, AND EGFR. None of them showed significance in the survival curves analysis or the infiltration analysis (with the exception of EGFR, shown in figure 5)

DISCUSSION & CONCLUSION

While previous research has touched on QRC's molecular mechanisms in cancer, this study represents the first attempt to gauge its efficacy specifically in treating GC. Our bioinformatic predictions diverge notably from prior findings; for instance, Azizi et al. (6) observed QRC's predominant targeting of Cyclines and M2 kidnapping in breast cancer. Such disparities likely stem from cancer-type-specific responses to QRC, evidenced other studies in (7). as While our study identifies numerous potential QRC targets in GC, the precise mechanisms remain elusive, necessitating further investigation.

FUTURE WORK / REFERENCES

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