GDE ASE Project 2020

***The price of investing in gold***

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Gold is widely used as a hedge against volatility in investment portfolios and a financial safe haven in times of market turmoil. This belief suggests that gold is negatively related to stock market returns. This projects uses time series data to construct a multilinear regression model to study the relationship between gold and S&P 500 returns during the COVID-19 pandemic and evaluate the relationship between gold and bear markets in the most recent episode of financial market instability.

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**1. Introduction** 500 words

Gold is widely believed to be negatively correlated with stock market performance. This belief has fueled its status as a hedge against volatility and a financial safe haven in times of market turmoil. Baur and Lucey (2010) define a safe haven “as an asset that is uncorrelated or negatively correlated with another asset or portfolio in times of market stress or turmoil”. As such, if gold is to be considered a safe haven, we would expect its price to rise when markets are performing poorly and fall when markets are performing well. Economic theory suggests that an increased demand for gold in times of market instability, such as during a financial crisis, would drive up its price. In other words, the theory suggests that gold is negatively correlated with market performance.

This correlation has been previously observed in practice, at least during short periods, at the beginning of bear markets. Baur and Lucey (2010) found gold and stock prices to be negatively correlated in periods of falling markets in the US, but uncorrelated when returns on the stock market were rising; that is, during bull markets. Their findings suggest that gold acts as a safe haven in bear markets only, and as an uncorrelated commodity in times of increasing market returns.

The COVID-19 crisis represents the most recent period of financial instability since the 2008 financial crisis, almost 12 years ago. The pandemic, which was declared by the WHO on March 11, 2020, has been a decisive factor in ending the bull market that resulted after the 2008 crisis, which had seen stock markets like the S&P 500 rally since the end of 2009.

In this project, I will use time series data on gold and S&P 500 returns during the COVID-19 pandemic to construct a multilinear regression model that shows the relationship between the price of gold and US market prices during the most recent episode of financial market instability. I compare my findings to a period of similar market instability, namely the 2008 financial crisis to

In section 2, I present the data and provide an initial analysis of the descriptive statistics of gold and stock returns in the US from late 2019 to mid-2020. In section 3, I provide a regression model with which to study the relationship between the price of gold and stock market performance throughout the COVID-19 pandemic. This project concludes with section 4, where I present my findings and draw conclusions from the data. Bibliography and Appendices are provided at the end of this paper.

**2. Data** 500 words

*2.1 Data and sources*

2.1.1 Gold

Any values missing from the FRED data were supplemented with data obtained from

2.1.2 S&P 500

Yahoo daily.

*2.2 Descriptive stats*

In this section, refer to Figure 1. Although markets appear to have recovered from this drop… However, in the recent COVID-19 crisis, we have seen a change in the relationship between the stock market and gold price: as the market index fell, so did the price of gold:

Figure 1 presents the prices for the entire sample period for stocks (upper graph) and gold (bottom graph). Stock prices peaked around March 2000 followed by a bear market that ended around March 2003. Bond prices show a different pattern. In general, prices have been rising for the entire sample period with relatively short periods of falling markets compared to stock prices. The bond prices of all three markets are clearly higher at the end of the sample than in the beginning of the sample period. Gold prices are also higher at the end of the sample compared to the beginning but there was no obvious trend of the price for gold. Two gold price regimes are easily discerned: the gold price fell until 2000 and increased afterward.





**Figure 1** Price of gold and S&P 500: (i) historically, (ii) 20-year period since 2000, and (iii) 6-month period during COVID-19 pandemic

*2.3 Correlation analysis*

A correlation analysis of the prices alone shows a significant negative correlation between the prices of gold and S&P 500. Although this is what was expected from the theory, a regression of gold prices on market prices alone does not provide an accurate description of the relationship due to it’s reliance on time.

To remove reliance on time, I chose to used data on the price returns. The raw data was transformed into returns using first difference logs.

2.3.1 Transformation

2.3.2 Correlation matrix

Sample 1: 12/02/2013 12/15/2017 - negative

Sample 2: 12/15/2017 12/16/2019 - no correlation

Sample 3: 7/16/2019 12/16/2019 - negative  
Sample 4: 12/16/2019 5/01/2020 - positive

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | COVID-19 |  |  | VIX |
| S&P 500 | 3386 (Feb 19, 2020) – 2237 (March 23, 2020) | -33.9% | 33 days |  |
| Gold | Same dates:  (Feb 19, 2020) – 2237 (March 23, 2020)  Falling: 1683 (Mar 6, 2020) – 1474 (March 19, 2020) |  |  |  |
|  | 2008 Financial Crisis |  |  |  |
| S&P 500 | 1298 (Aug 11, 2008) – 683 (March 2, 2009) | -47.3% | 203 days |  |
| Gold | 1683 (Mar 6, 2020) – 1474 (March 19, 2020) |  |  |  |

S&P 500

3386 (Feb 19, 2020) – 2237 (March 23, 2020)

- 1683.650 (March 6, 2020) - 1474.250 (March 19, 2020)

-12.5% in gold

- 2972.37 (March 6, 2020) - 2409.39 (March 19, 2020)

-18.9% in S&P 500

- 1683.650 (March 6, 2020) - 1474.250 (March 19, 2020)

-12.5% in gold

1978-2020 and 2000-2020 No significant long-run correlation

Negative correlation

Positive correlation

**3. Regression** 500 words

*3.1 Model*

* Present model equation and reason for this model.
* Present results in an equation and explain what each term means.

*3.2 Results*

3.3.1 Analysis

DW < 1.5 Positive autocorrelation

DW > 1.5 No autocorrelation

DW < 2.5 Negative auto correlation

3.3.2 Sub-sample analysis

**4. Conclusion** 500 words

*4.1 Long-run vs. short-run*

*4.2 Volatility*

**Bibliography**

**Appendix**

**Introduction**

Precious metals, including gold, are widely used to diversify investment portfolios. Gold has because its price increases in response to events that cause the value of paper investments, such as stocks and bonds, to decline. It has served as a hedge against inflation and the erosion of major currencies over the years. This project used a multilinear regression model to analyze recent changes in market returns during the COVID-19 pandemic to evaluate the relationship of gold and market volatility.

On average, although the price of gold can be volatile in the short term, it has always maintained its value over the long term. Gold is an unusual commodity for a number of reasons: it widely used as a safe haven in times of market turmoil and used as a hedge for inflation in portfolios. For this reason, gold is widely believe to be negatively correlated with market performance. In my project, I look at the changes in the return rate of gold since the beginning of the COVID-19 pandemic to try and evaluate its relationship with

Liver diseases are highly prevalent and account for nearly two million deaths each year around the world. One million of these deaths are the results of liver cirrhosis and the other million are due hepatitis and hepatocellular carcinoma (HCC)1. As the sixth most common cancer, HCC incidence has risen faster than that of other cancers in recent years2, 3. Other liver diseases, such as ALD and nonalcoholic fatty liver disease (NAFLD), also represent a heavy health and economic burden to the families of affected individuals and society. The prevention of these diseases is becoming an urgent issue, with the identification of potential factors as the first step.

Previous studies have confirmed several environmental risk factors that contribute to liver diseases, including viral infection, alcohol, and obesity4, 5. Increasing evidence has further revealed that genetic factors also play a crucial role in liver diseases6, 7. In particular, GWAS have identified several contributing loci, such as rs738409 (p.I148M) within *patatin like phospholipase domain containing 3* (*PNPLA3*) and rs58542926 (p.E167K) within *transmembrane 6 superfamily member 2* (*TM6SF2*)8, 9. In 2018, a new variant, rs72613567, within *hydroxysteroid 17-beta dehydrogenase 13* (*HSD17B13*) was first reported to likely provide substantial protection against liver diseases10. *HSD17B13* rs72613567 is a protein-truncating variant with an insertion of A allele adjacent to the splice site of exon 6. Subsequent studies have investigated its role in different types or pathological process of liver diseases, but the results are conflicting10-16.

**Materials and Methods**

This study was carried out in accordance to the statement of PRISMA17. PubMed, Embase, and Web of Science were searched for relevant records without any restrictions (up to March 2020). The search items included “*HSD17B13*”, “polymorphism”, “variant”, and “rs72613567”. The quality of the studies included was assessed using the Newcastle-Ottawa scale (NOS) 18. Three authors participated in the aforementioned work (WP, CW, and YL). Between-study heterogeneity was assessed using Cochran’s Q test and *I2* statistics. *P* < 0.10 or *I2* > 50% indicated significant heterogeneity19. A random-effects model was used to pool the results. Begg’s and Egger’s tests were applied to examine the publication bias20, 21.

**Results**

A total of 23 relevant articles were selected for full-text evaluation. We excluded 16 articles as follows: (1) articles that were not relevant to the association between *HSD17B13* rs72613567 and liver diseases risk or histological features (n = 9) 22-30; (2) articles whose data overlapped with other studies (n = 3)31-33; (3) articles that did not provide allelic data of *HSD17B13* rs72613567 (n = 2) 34, 35; (4) articles that focused on specific populations, such as individuals with Wilson’s disease36 or obese children37 (n = 2). Ultimately, seven eligible studies met all the inclusion criteria10-16. Among these, five studies were included for the meta-analysis of the association of *HSD17B13* rs72613567 with the risk of liver diseases10-14.

Due to the insufficient number of studies for meta-analysis, we summarized relevant studies on the role of *HSD17B13* rs72613567 in NAFLD-histological features10, 11, 15, 16. Features included steatosis, inflammation, hepatocyte ballooning, NAFLD activity score (NAS), fibrosis, and disease severity. Multivariate-adjusted ORs and 95%CIs, genetic models, and adjustment were also demonstrated in Table 2.

We applied a random-effects model based on allelic data to pool the results, as summarized in Figure 2.

***Any liver disease.*** The estimation of any liver disease was analyzed in five studies including 564702 individuals10-14. The results suggested that the mutant TA allele of *HSD17B13* rs72613567 conferred a protective effect on any liver disease, with no significant publication bias. Sensitivity analysis suggested that the results were stable. We further assessed the role of *HSD17B13* rs72613567 in alcoholic liver disease. Compared with the wild allele, the mutant TA allele demonstrated a significant protection against alcoholic liver disease, with low heterogeneity and no publication bias. Despite an insufficient number of studies for the meta-analysis of nonalcoholic liver disease, existing publications supported the protective effect of *HSD17B13* rs7261356710, 13, 15.

***Liver cirrhosis.*** The estimation of liver cirrhosis was performed in four studies including 559834 individuals10-12, 14. *HSD17B13* rs72613567 was significantly associated with a lower risk of liver cirrhosis, with robustness suggested by sensitivity analysis and no publication bias. A stable association was also observed in alcoholic cirrhosis, with no publication bias.

***HCC.*** A total of 183179 individuals from four studies were included10, 12-14. A stable result revealed that *HSD17B13* rs72613567 could reduce the susceptibility of HCC, with no publication bias.

***NAFLD-histological features.*** Four eligible studies are summarized in Table 2 according to histological features10, 11, 15, 16. The sample size of these studies ranged from 356 subjects to 1735 subjects. Overall, *HSD17B13* rs72613567 was associated with decreased inflammation and lower NAS scores, and tended towards reduced ballooning and fibrosis. For disease severity, *HSD17B13* rs72613567 was not associated with steatosis or nonalcoholic steatohepatitis (NASH), but was associated with a decreased risk of progression.

**Discussion**

The major findings are presented as follows: (1) *HSD17B13* rs72613567 could provide a significant protection against several liver diseases, including alcohol liver disease, cirrhosis and HCC; and (2) *HSD17B13* rs72613567 might be associated with milder histological progression of NAFLD, such as reduced inflammation, lower NAS scores, less ballooning and fibrosis.

*HSD17B13* encodes a pivotal lipid-droplet enzyme, which is predominantly expressed in the cytoplasm of hepatocytes, and its aberrant expression has been reported in fatty liver, cirrhosis and HCC38-40. More recently, Abul-Husn integrated exome-sequence data and population study in a large collaborative cohort, firstly revealed a loss-of-function variant, *HSD17B13* rs72613567 that conferred a protective effect on chronic liver disease and mitigation against progressive NASH10. This variant could disturb mRNA splicing and lead to a prematurely truncated protein, and the TA allele is associated with decreased or absence of HSD17B13 expression in an allele-dose-dependent manner10, 15.

Several studies suggested that the protection of *HSD17B13* rs72613567 was observed in patients with ALD, NAFLD, cirrhosis and even HCC10, 12-14, 32. Specially, each TA allele might reduce 15% and 28% of the risk of cirrhosis and HCC, respectively. Also, the TA allele was associated with a lower liver-related mortality rate of 33% in a general population14. By pooling available data from eligible studies, our meta-analysis further confirmed the protective role of *HSD17B13* rs72613567 in liver diseases, ranging from ALD to HCC.

For NAFLD-histological progression, because there are not enough studies for meta-analysis, we instead summarize existing date according to progression features. All these included studies conducted adjustment of confounding factors. Overall, *HSD17B13* rs72613567 demonstrated a trend to mitigate progression of NAFLD. HSD17B13 is a liver-specific enzyme to regulate liver lipid homeostasis, and its aberrant expression and high enzyme activity have been confirmed to promote the development of NAFLD40. So the probable explanation is that *HSD17B13* rs72613567 results in a loss-of-function truncated protein, thus attenuating the progression of NAFLD.

Several limitations should be noted. First, only four eligible studies were included for meta-analyzing the association between *HSD17B13* rs72613567 and liver diseases risk. The limited study number hampered further subgroup analysis, and also probably led to the unstable pooled results of any liver diseased vs. normal and alcohol cirrhosis vs. normal. Second, the effect of *HSD17B13* rs72613567 on histological progression was failed for meta-analysis due to the few eligible studies. Instead, we summarized existing studies based on histological features. At last but not the least, our results did not cover Asian data because of no such studies reported in Asians. The minor allele frequency of *HSD17B13* rs72613567 varies from 5% in the African population to 34% in the East Asian population. The dramatic allele differences require more studies in different ethnic populations to further validate our results.

Our study highlights that *HSD17B13* rs72613567 exhibits a significant protection against multiple categories of liver diseases, and consistently, it tends towards milder progression of NAFLD. In view of disease treatment, this work also supports that people at high risk of liver diseases may be benefit from HSD17B13 inhibition. More studies, especially on Asians, are required to further validate our results.

**Acknowledgements**

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**Conflicts of interest**

Insert here.

**References**

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