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CAUSAL EFFECT ESTIMATION OF POLICY RATES ON INFLATION RATES USING NONPARAMETRIC METHODS

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ABSTRACT

This study applies a newly developed nonparametric test based on a cross-fitted nonparametric estimator of an integral of the causal dose-response curve, to determine if there is an average causal effect for different lags of the policy rates, which are the interest rates set by central banks as part of monetary policy, on inflation. Furthermore, nonparametric regression is used to estimate the density between policy rates and inflation to provide insight into the potential shape of the causal-dose-response curve. In this study, both Swedish and US data are analyzed. The results indicate a causal effect of policy rates after 2-4 months for the Swedish data and 2-5 months for the US data. However, the nonparametric regressions are not able to capture any kind of relationship as the estimated density curves are flat.

Keywords: Inflation, Policy rates, Interest rates, Causal inference, Continuous exposure, Nonparametric regression, Time series.

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Contents

1 Introduction

Inflation is the increase of the general price levels, resulting in a decrease in the purchasing power of goods and services given the same amount of money. It can arise from various factors such as general increasing economic demand, increased firm expenses because of higher wages, production costs, or supply shocks. Additionally, inflation can result from expectations of future price increases, prompting firms and consumers to raise prices and wages accordingly (Sveriges Riksbank 2022).

The most important factor alongside fiscal policy is monetary policy, which policymakers, typically the central banks, can use to control the price levels (Tran 2018). The modern monetary policy framework in most developed countries stems from the idea of inflation targeting, where central banks try to maintain the inflation rate at a constant value, typically at or around 2 percent. This was first adopted in the 1990s by several industrial countries in the wake of the difficulties these countries faced in implementing monetary policy using an exchange rate peg or some monetary aggregate as an intermediate target (International Monetary Fund 2006). This also included Sweden, as the Swedish central bank, Sveriges Riksbank, officially introduced inflation targeting in 1993 and began to apply it in 1995. The US central bank, The Federal Reserve, officially adopted inflation targeting in 2012. Although before that, monetary policy in the USA was still being conducted without an explicit target (The Federal Reserve 2023). The reason for having an inflation target is that it helps create favorable conditions for economic development by maintaining a stable inflation rate (Sveriges Riksbank 2024).

The primary tool on which central banks rely to steer inflation is the policy rate, which is the daily interest rate that banks use when lending and borrowing to each other. This affects other interest rates in the economy, such as the banks and mortgage institutions. Thus, by adjusting policy rates, central banks can influence the pace and direction of the overall economic activity affecting aggregate output, employment, and inflation (Friedman 2000). Although not of interest to this study, there are other complementary tools that central banks can use to influence the interest rates such as quantitative easing, which developed after the 2008 financial crisis and involves the large-scale purchase of financial assets such as government bonds (Bernanke 2020).

The mechanism through which monetary policy affects the economy is called the monetary transmission mechanism. It is one of the most studied areas of monetary economics, and there

is a large amount of literature on this subject (Boivin et al. 2010). This is crucial because policymakers must have an adequate assessment of how monetary policy affects the economy and the timing of those effects to make sound economic decisions and effectively determine monetary policy at a specific point in time. (Boivin et al. 2010).

However, for estimating the actual causal effects of the policy rate on inflation, there is very little available literature. Many of the previous studies in the econometrics framework have typically focused on making predictions. They have commonly used methods such as vector autoregression models and Granger causality for time series data, often revolving around analyzing monetary shocks. Although these types of analysis have their usage, they are in a broad sense more oriented toward causal discovery, which is the task of identifying and understanding causal relationships in the data. A problem with such methods is that they lack causal effect estimation, which is the estimation of the causal effect of a policy or treatment on a target variable. Instead, other methods typically use metrics like the average treatment effect (ATE) for binary variables to estimate how a treatment variable, also known as the exposure variable, affects an outcome variable. These methods are based on the causal inference framework, as defined by the Rubin causal model (Moraffah et al. 2021). In contrast to traditional methods using the policy rate to forecast inflation, very little literature is based on the causal inference framework. This may be due to the methods in causal inference being primarily developed for binary or polytomous discrete treatment variables. Thus, within the causal inference framework, there is much less literature on continuous exposure variables. However, research on continuous exposure variables has advanced recently with the introduction of new statistical tests, which create more opportunities to explore causality. This paper aims to address the existing gap in the literature on the causal effect of the policy rate on inflation.

1.1 Research questions

Within the causal inference framework for continuous exposure variables, the estimand of interest is often the causal dose-response curve, which can be interpreted as the average effect of a treatment variable on a target variable for any given level of treatment. This study aims to estimate the causal dose-response curve where the policy rate is seen as the treatment variable and the inflation rate as the outcome variable to determine whether there is a causal effect from policy rates on inflation for different lags of the policy rates. To the best of my knowledge,

no other paper has done so previously. Thus, this paper is the first to analyze the causal dose-response curve on the effects of policy rates on inflation, accounting for different lags of policy rates using the proposed method.

Furthermore, using non-parametric regression, this study will also try to estimate this density to give an idea of what the relationship between policy rates and inflation rates may look like. However, as a limitation, the nonparametric regression method is not based on the causal inference framework. Consequently, findings from the nonparametric regressions should be interpreted with caution with regard to causality.

1.2 Outline of the paper

This paper is structured as follows: Section 2 reviews the literature and presents the theoretical framework based on the macroeconomic theory of the transmission mechanism. Section 3 details the data used in this study, including its sources, collection methods, and minor issues. Section 4 outlines the statistical methods and tests employed in this study, divided into various subsections. Section 5 presents the results and discusses their implications. Finally, Section 6 provides the conclusion.

2 Previous research and theoretical framework

This section begins with a literature review in Section 2.1, examining general interest rates and their effects on inflation, focusing on the methodological framework from a statistical and econometric perspective. It highlights the most commonly used models and their challenges in determining causality. Note that this review provides a general overview as the specific literature on the causal dose-response curve, which this paper investigates, is limited. Section 2.4 outlines the theoretical framework of macroeconomic theory on the transmission mechanism, discussing the key channels through which it exerts its causal effect. Section 2.3 discusses how the Phillips curve relates unemployment to the inflation rate. Lastly, Section 2.4 addresses the debated aspects of the causal effect and the challenges of generalizing the results to all countries.

2.1 Literature review

There is an abundance of literature within the general area on the effects of interest rates on inflation, even before central banks officially adopted the idea of maintaining an inflation target by the policy rate. Although interest rates and policy rates are not strictly the same thing as the policy rate refers to the one officially set by the central banks, they are closely related as they move in the same direction, and the work of using policy rates stems from the early work of interest rates in a more general sense. However, the focus has not typically been so much on trying to estimate any direct causal effect, but rather on using interest rates to predict the inflation rate. Much of the early work on this subject was based on regression analyses, often in some autoregressive setting. Some early notable works are, for example, Fama (1975) or Nelson & Schwert (1977), using short-term interest rates as predictors for inflation, in which there are found to be significant results. Fama derived the relationship between interest rates and inflation as:

$$E\left(Y_t \mid I_{t-1}\right) = E\left(R_t - r_t \mid I_{t-1}\right) = R_t - E(r_t \mid I_{t-1}) \tag{1}$$

where R_t is the nominal return from one month treasury bills and Y_t is the inflation rate and r_t the inflation adjusted real return.

But, as Xu et al. (2022) points out, there are limitations with this type of linear regression-based framework. Most importantly, there is reason to suspect that the relationship between inflation and interest rates may not be constant over a longer period. Furthermore, the conditional mean represents the average return, but it does not provide much insight into the distribution of the return. Two distributions can have the same mean, yet exhibit very different shapes, and overlooking these differences can lead to incorrect conclusions. In their work, Xu et al. (2022) used locally stationary quantile regression instead to model inflation and interest rates and came to two conclusions: the first was that the inflation rate is positively correlated with a one-month lagged inflation rate for the quantiles considered. The second conclusion was that the relation between inflation and interest rates exhibits more complicated quantile-specific and time-varying features.

However, for a long time, the use of VAR models has traditionally dominated the methodological framework of the causality of interest rates on inflation, starting with the seminal works by Sims (1980). In contrast to autoregression, which is a single-equation, single-variable linear model, where a current variable is regressed on its past values, a VAR is an n-equation, n-

variable linear model where each variable is regressed on its past values as well as the current and lagged values of the other n variables (Stock & Watson 2001). VAR models are commonly used with the Granger causality concept attributed to Granger (1969). A variable X is said to cause some other variable Y if past values of X help explain future values of Y, that is, it contains information about Y that is not available in past values of Y (Moraffah et al. 2021). With VAR models this is typically modeled as:

$$Y_t = \sum_{\tau=1}^{\tau_{\max}} \phi_\tau Y_{t-\tau} + \epsilon_t, \tag{2}$$

where $Y_t = (Y_{1t}, \ldots, Y_{nt})$ indicates time series Y at time step t, ϕ_τ is the $N \times N$ coefficient matrix at lag $\tau, \tau_{\max}$ denotes the maximum time lag, and ϵ_t represents an independent noise. If any coefficient in $\phi_{ji}(\tau)$ is non-zero, then $_i$ is said to cause Granger to cause Y_j with the lag τ.

The relationship can be shown by $Y^i_{t-\tau} \rightarrow Y^j_t$, which demonstrates the causal link between Y_i and Y_j at lag τ.

One notable study is Stock & Watson (2001), in which they use VAR and do not find evidence that the federal funds rate (the US policy rate) helps predict inflation, but instead that inflation helps predict the federal funds rate. Boivin et al. (2010) also using VAR models, does find in contrast to Stock & Watson that the Federal Reserve has played a role in reducing the volatility of inflation by its use of the Federal Funds rate. Tran (2018) uses a Vector error corrected model (VECM), which is a restricted VAR model, and finds that there is a short-run causal relationship from the policy rate to inflation, based on data from Vietnam.

Adolfson et al. (2007) makes the argument that although VAR models may have good statistical properties and can be useful as a way to make good forecasts, they come with little economic theory, and a drawback is that they typically are over-parameterized. Having many parameters causes imprecise estimates leading to large intervals for the pulse response functions. Hence, VAR models cannot give more in-depth answers about the monetary transmission mechanism, and the actual effects of monetary policy are not captured. One proposed solution is to instead incorporate Bayesian methods such as Bayesian Retrogressive (BVAR) models. These models can include some macroeconomic theory by the use of priors, for example, such as the concept of the steady state of the system as done by Villani (2006) and in which they found that BVARS can make inflation forecasts as good as more complicated models used at central banks. Another example is Berger & Österholm (2011), where they use BVAR to determine

whether money growth Granger causes inflation based on US data, for which including interest rate consistently gave better results.

Although the literature within this area is well-developed, this paper lacks a close relationship to them from a methodological perspective. They attempt to establish whether policy rates influence inflation as well as determine the magnitude of this effect. However, the results from these models do not allow for a direct causal interpretation, even though they offer an in-depth analysis of varying statistical dependencies within a set of economic variables to better evaluate causality. As mentioned briefly in the introduction, this paper aims to take an approach based on the causal inference framework, which is scarce in comparison.

There are some examples of causal inference methods that are being used in a broader context of analyzing monetary policy. For example, Angrist et al. (2018) analyzes the effect of the policy rate on different macroeconomic variables using inverse probability weighting estimator. However, they limit their analysis to a discretization of the policy rate. Vonnák (2021) extends the work of Angrist et al. by generalizing to the continuous case, but seems to rely on parametric assumptions about the specified propensity score. In addition, even though they estimate the causal dose-response curve, they do not formally test whether the causal dose-response curve is flat. Hence, to the best of my knowledge, this study is the first one with this explicit aim.

2.2 The transmission mechanism

The traditional channels of the transmission mechanism stem from the neoclassical framework of investment, consumption, and international trade models, which operate through different channels. In this paper, the focus is not on the actual transmission mechanism itself. Thus, no emphasis will be placed on the individual channels. Instead, the focus is on a more aggregate scale to answer the question if there can be determined to be any causal effect or not. For this reason, it is nonetheless important to understand the transmission mechanism. See, for example, Gertler & Karadi (2015) for more details and the history of how the transmission mechanism has developed over time.

According to traditional Keynesian models, one of the channels of the transmission mechanism is the interest rate channel; the more traditional way by adjusting the nominal money affecting the interest rate or more directly through the policy rate. However, in practice, they have the same effects in that they affect aggregate production, employment, and overall price levels

(Mishkin 2016). It should also be noted that it is the real interest rates and not the nominal interest rates that affect asset pricing and spending through the transmission channel. Furthermore, it is not only the current interest rates that matter, but also the expected interest rates. However, setting short-term nominal interest rates gives central banks control over the contemporaneous real rates and the expected rates in the future for some horizon ahead (Gertler & Karadi 2015).

Businesses and households tend to look at the long horizon when factoring in variations in interest rates in investment decisions. Although a contractionary monetary policy by increasing the policy rate increases the short-term nominal interest rates, it also increases the longer-term nominal interest rate, for example, because investors seek to eliminate disparities of risk-adjusted expected return. This will also translate into changes in the real interest rates. Hence, the user capital cost increases. At the same time, capital asset demand decreases due to increased borrowing costs for both firms and individuals. This situation leads to lower investment spending and a decrease in aggregate spending and demand, which has a damping effect on inflation (Ireland 2010). Furthermore, monetary policy also has a direct effect on household consumption. Lowering short-term interest rates, for example, increases demand for assets such as common stocks and housing, thus raising their cost. This results in a general increase in total wealth, stimulating household consumption and aggregate demand (Gertler & Karadi 2015).

Another way the transmission mechanism works is through the exchange rate channel for open economies; as a result of falling interest rates, the domestic currency depreciates vis-à-vis other currencies. This is because the return on domestic assets decreases compared to that of foreign assets, causing domestic goods to be cheaper than foreign goods and an increase in foreign demand for domestic goods, leading to expenditure switching, a switch between foreign and domestic goods, and hence a rise in net exports. Thus, the increase in economic activity as a result of higher aggregate demand raises inflation. Increased interest rates have the opposite effect. Hence, the exchange rate channel plays an important role in how monetary policy affects the economy (Taylor 2001).

There is also the asset price channel as described by Tobins q-theory (Tobin 1969), where tobins q is a ratio of the market value of a company's assets divided by the replacement cost of those assets. Low interest rates typically increase the q ratio by raising market values and lowering replacement costs, while high interest rates usually decrease the q ratio by lowering market values and raising replacement costs. For example, when q is lower, each firm has to

issue more new shares of stock to finance any new investment project, making the investment more expensive. Consequently, across all firms, investment projects that were only marginally profitable before monetary tightening are now unfunded due to the decline in q. This results in a decrease in output and employment (Ireland 2010).

In addition to those channels, there are several other mechanisms through which monetary policy affects inflation, not just those discussed here. For example, the credit channel constitutes another mechanism which influences inflation by impacting borrowing costs and credit availability. For further details, see Bernanke & Gertler (1995) or Kashyap & Stein (1994). However, the main point moving forward is that the way monetary policy works is a complex phenomenon, and it is difficult to single out each effect. However, the channels mentioned here are the main ones commonly mentioned in the literature. Furthermore, it is worth noting that there are other factors affecting the interest rates in a country. For example, the general level of global interest rates. Hence, the policy rate is not solely responsible for being the factor that affects the inflation rate, but it does nevertheless play an important role.

2.3 Unemployment rate and inflation

The relationship between inflation and unemployment stems from the concept of the Philips curve, originally developed by Phillips (1958). This curve describes an inverse relationship: When the inflation rate is high, the unemployment rate is low, and vice versa. The inverse relationship was largely attributed to the dynamics of labor demand and wage adjustments. Philips theorized that times of high demand for labor lead to increased wages, whereas during times of low demand, workers were willing to accept lower wages. Philips also noted that the relationship between the unemployment rate and nominal wage change should be highly non-linear due to the rigidity stemming from the reluctance of workers to low rates even during times of high unemployment. Since then, economists have continuously developed the concept of the Phillips curve to incorporate new ideas. For example Friedman (1968) together with Phelps (1967, 1968) extended it to include the idea of the natural rate of unemployment, which revolves around the idea that there is one specific level of unemployment where the inflation rate will continue at an existing pace. When unemployment deviates from that level, inflation will rise or fall accordingly. Their work also contributed to the idea of the augmented Phillips curve, which expanded on previous theories by incorporating the role of inflation expectations.

Unlike the previous Philips curve as developed by Philips, which primarily focused on the direct relationship between unemployment and inflation levels, the augmented Phillips curve suggested that it is not the level of unemployment that influences inflation directly, but rather how unemployment affects the rate at which inflation changes based on expectations about future inflation. However, the amount of literature on this topic is vast, and this paper will not go into more detail on this aspect. See, for example, Gordon (2011) and Mavroeidis et al. (2014) for a more detailed overview and more recent developments. However, because of its importance to the Philips curve, the unemployment rate is commonly used in similar studies, and hence why it is also included in this study as it is an important confounder variable.

2.4 Causality debate

Though the effects of monetary policy on inflation are widely accepted, there is some debate about its actual effectiveness, especially in developing countries (Islam & Ahmed 2023). A key aspect is the different financial structure; for example, the absence of effective and functional markets for instruments like fixed-income securities, equities, and real estate. Furthermore, even though banks are the largest financial institutions, the financial system is typically small relative to the total economy (Mishra & Montiel 2013). Developing countries typically have limited and imperfect connections with private international capital markets, leading their central banks to heavily intervene in foreign exchange markets (Mishra et al. 2012). This institutional context indicates that the mechanism of monetary transmission in low-income countries can differ significantly from that of advanced and emerging economies. However, because this study focuses only on US and Swedish data, it does not further analyze this aspect, as these countries are classified as advanced economies by the IMF (Nielsen 2011; International Monetary Fund 2023). As a result, the findings may not apply to other countries, particularly to developing countries.

3 Data

The data used in this study are monthly time series for the variables inflation rate, unemployment rate, and policy rate for Sweden and the United States. The data consists of 355 months covering the periods from June 1994, when the Riksbank officially started to track the policy rate, to December 2023. Thus, to make the results more comparable, the time series for the

other variables have been restricted to align with the same period as the Swedish policy rate data, even though data is available from earlier points in time for those variables. All the data are collected from various public governmental sources. The Swedish policy rates are obtained from Riksbanken, while the Swedish inflation rate and the unemployment rate are obtained from Statistics Sweden (Statistika Centralbyrån, SCB). The US Federal Funds rate is obtained from the US Federal Reserve Bank, with data on unemployment and inflation retrieved from the U.S. Bureau of Labor Statistics. Although in some literature a larger collection of variables is used, such as in example Leeper et al. (1996) using 13 variables in VAR-based models, much of the literature considers these three variables in particular with a notable being Stock & Watson (2001). In this case, it is also preferable to try to limit the number of variables to keep the models as parsimonious as possible because of the somewhat limited available data.

A price index is used to operationalize the concept of inflation for both the US and Swedish data. Typically, the consumer price index (CPI) is used as a measure of inflation. It measures the change in prices of goods and services acquired for private domestic consumption, based on weighted averages for specific sets or baskets of products. However, this study will use the 12-month percentage difference of the consumer price index with fixed rates (CPIF) for Swedish inflation. For the US data, this study will use the 12-month percentage difference of the personal consumer price index (PCE). These are the measures used by the respective central banks as the inflation target. Both measures closely relate to the CPI; CPIF measure inflation similarly to the CPI, while discarding the effects of mortgages for consumers. The PCE reflects consumer spending and adapts more quickly to changes in spending patterns (Sveriges Riksbank 2023; Federal Reserve 2024).

Regarding the unemployment rate, it should be noted that although they are roughly similarly defined in the Swedish and US cases, some differences exist. Generally, the unemployment rate for both countries refers to people who are part of the labor force, which means that they do not currently have a job but are actively seeking one. Hence, people without jobs but who are not actively seeking jobs are not considered unemployed. For the US data, these data are originally collected from surveys done by the Bureau of Labor Statistics, whereas the Swedish data are obtained from Arbetskraftundersökningarna (AKU), a survey done by Statistics Sweden. Thus, there are some differences in the exact method by which the surveys were conducted, such as the target population for the survey and the definitions used in the surveys. There have also been

some changes over the years. For example, in 2021 AKU made because of a new framework law implemented by the EU, which included changes in the target population of the surveys and some definitions used. Thus, comparisons between the periods before and after this date are not straightforward (Statistics Sweden 2023). However, SCB has worked on making the time series comparable through different means, such as imputations. Nevertheless, this is considered a minor problem for this study, and potential differences are not further analyzed.

4 Method

Section 4.4 describes the nonparametric test for the causal dose-response function used to answer the first research question. Following this, Section 4.2 discusses alternative approaches commonly used in similar contexts and explains the rationale for choosing this specific test. Section 4.3 details the generalized linear models that estimate some nuisance estimators as part of the nonparametric test. Section 4.4 explains the nonparametric regression used to address the second research question. Furthermore, the study uses two time-series tests: the augmented Dickey-Fuller test for stationarity in Section 4.5, and the Ljung-Box test for autocorrelation, presented in Section 4.6.

4.1 Nonparametric test of the causal dose-response function

This study will use a relatively new test as developed by Westling (2022). It is a doubly robust nonparametric test that relies on a cross-fitted estimator of an integral of the dose-response curve. To my knowledge, it is the first such nonparametric test that considers global inference for continuous exposure. The focus of this section will be to explain the test while keeping the most relevant parts based on the results from Westling (2022). More details are provided in the original paper, and the proofs are available in the supplementary material to that paper. The test is also available in R with the package ctsCausal. Thus, the notations and explanations are largely based on the paper from Westling (2022).

First, the exposure variable, or the treatment variable, which in this case represents the policy rate, is denoted as A with support $\mathcal{A} \subset \mathbb{R}$. Using the potential outcomes of the causal inference framework, for each exposure value, $a \subset \mathcal{A}$, the potential outcomes are: $Y(A = a) \in B \subset \mathbb{R}$ under the intervention of setting the exposure to $A = a$, where B is bounded within the reals. This outcome reflects what would be observed if it were possible to assign a unit to that specific

exposure value, thereby allowing an assessment of how that level of exposure would influence the outcome. For the remainder of the paper, $Y(A = a)$ will be denoted as $Y(a)$.

The causal parameter of interest is the counterfactual means defined as $m(a) := E[Y(a)]$, which is the average outcome under the assignment of the entire population to the exposure level $A = a$. The function $m : \mathcal{A} \to \mathbb{R}$ is the causal dose-response curve, also called the average dose-response function. The fundamental problem of causal inference is that it is impossible to observe all potential outcomes. Instead, for each unit, only one of the possible outcomes can be observed. The observed data are assumed to be generated from some unknown distribution P_0 such that: $O_1, \ldots, O_n \sim P_0$ for the data unit $O = (Y, A, W)$, and where $Y := Y(A)$ is the observed outcome and $W \subset \mathbb{R}^d$ are some pre-exposure covariates. For the remainder of this paper, the functions or parameters indexed by 0 indicate dependence on the unknown distribution P_0.

The null and alternative hypotheses to be tested are as follows:

$$H_0 : E\left[Y(a)\right] = c, \text{ for all } a \in \mathcal{A}$$
$$H_1 : E\left[Y(a_1)\right] \neq E\left[Y(a_2)\right] \text{ for some } a_1, a_2 \in \mathcal{A}.$$

Under the null hypothesis, the causal dose-response curve m is flat on $\mathcal{A}$. An alternative way to view it is that the average outcome under assigning all units to $A = a$ is the same for all $a \in \mathcal{A}$. However, since $Y(a)$ is not observable for all units and only the observed outcome is available, m does not map the joint distribution of the pair (A, Y). As a result, the observed data cannot directly test this null hypothesis. To make it testable, certain assumptions are required:

1. The potential outcomes of each unit are independent of the exposure of all other units (the stable unit treatment assumption).

2. The observed outcome Y almost surely equals $Y(A)$.

Furthermore, if the following assumption were to hold:

3. $Y(a) \perp\!\!\!\perp A$ for all $a \in \mathcal{A}_0$,

Then m becomes identifiable through univariate regression, and the null hypothesis can be tested using parametric approaches. However, when confounder variables influence both A and $Y(a)$, nonparametric regression produces invalid type 1 error rates. Therefore, the following assumptions are required:

4. $Y(a) \perp\!\!\!\perp A \quad W$ for all $a \in \mathcal{A}$

5. all $a \in \mathcal{A}$ are in the support of the conditional distribution of A given $W = w$ for almost every w. Or alternatively : $p(a \mid w) > 0$

Assumption 4 ensures that all confounding variables are captured by W, while Assumption 5 allows every unit to receive any treatment a within its support. Given that all the assumptions 1-2 and 4-5 hold, then:

$$E[Y(a) \mid W = w] = E_0(Y \mid A = a, W = w) := \mu_0(a, w), \text{ so}$$
$$E[Y(a)] = E_0\left[\mu_0(a, W)\right] := \theta_0(a),$$

which is known as the *backdoor formula*, or G-*computation* (Robins 1986). This implies that when the conditions are satisfied, the G-formula can estimate the counterfactual mean parameter. $\mu_0(a, W)$ represents the conditional mean of the observed Y for treatment a, calculated as the expectation over the marginal distribution of the covariates W.

Under these conditions, the null and alternative hypotheses can be equivalently expressed in terms of the observed data distribution as follows:

$$H_0 : \theta_0(a) = \gamma_0 \text{ for all } a \subset \mathcal{A} \text{ and } \gamma_0 := E_0\left[\theta_0(A)\right]$$
$$H_1 : \theta_0(a) \neq \gamma_0, \text{ for some } a \subset \mathcal{A}$$

In practical terms, the null hypothesis holds if the g-computed regression function $\theta_0(a)$ remains constant across the support of a, which is the case only when it equals its average value. Here, γ_0 represents the average value of the causal dose-response function over the marginal distribution of the exposure A.

The next step is to derive the test. Since direct estimation of θ_0 is difficult, the method proposed by Westling (2022) is to estimate a primitive parameter of θ_0. The logic behind this approach can be illustrated as follows: Consider a density function g_0 that is flat over the interval $[0, 1]$. The flatness of g_0 implies that the cumulative distribution function $G_0(X)$ is the identity function on $[0, 1]$, which is equivalent to $G_0(x) - x = 0$ for all $x \subset [0, 1]$.

Then the null hypothesis can be expressed in terms of a primitive transformation of θ_0 using

the following definitions:

$$
\begin{aligned}
F_0(a) &= P_0(A \leq a) \\
\gamma_0 &= \int_{-\infty}^{\infty} \theta_0(a) dF_0(a), \\
\Gamma_0(a) &= \int_{-\infty}^{a} \theta_0(u) dF_0(u), \\
\Omega_0(a) &= \Gamma_0(a) - \gamma_0 F_0(a).
\end{aligned}
$$

$F_0(a)$ is the marginal cumulative distribution of the observed exposure a, while γ_0 denotes the average value of the dose-response function over the marginal distribution of A. The function $\Gamma_0(a)$ is the partial average of θ_0 up to the point a. The following proposition can then be derived:

Proposition 1 *if θ_0 is continuous on $\mathcal{A}$, then θ_0 is constant on $\mathcal{A}$ if and only if $\Omega_0(a) = 0$ for all $a \subset \mathcal{A}$*

Unlike θ_0, $\Omega_0(a)$ is pathwise differentiable relative to the nonparametric model at each a with an estimable influence function. This can be done regardless of the form of the form of F_0. Thus, it is possible to construct a uniformly asymptotically linear estimator of Ω_0. In the supplementary materials to Westling (2022), it is proven that the efficient influence function is:

$$
\begin{aligned}
D^*_{a_0,0}(y,a,w) :=& \left[I_{(-\infty,a_0]}(a) - F_0(a_0)\right] \\
&\times \left[\frac{y - \mu_0(a,w)}{g_0(a,w)} + \theta_0(a) - \gamma_0\right] \\
&+ \int \left[I_{(-\infty,a_0]}(u) - F_0(a_0)\right] \mu_0(u,w) F_0(du) \\
&- 2\Omega_0(a_0).
\end{aligned} \tag{3}
$$

Letting V denote the number of folds, and where $F_{n,v}$ and Qn_v denote the empirical marginal distributions of A and W, then a cross-fitted estimator $\Omega_n(a_0)$ of $\Omega_0(a_0)$ is given by:

$$
\begin{aligned}
\frac{1}{V}\sum_{v=1}^{V} \Bigg\{ & \frac{1}{N_v} \sum_{i \subset \mathcal{V}_{n,v}} \left[I_{(-\infty,a_0]}(A_i) - F_{n,v}(a_0)\right] \\
&\times \frac{Y_i - \mu_{n,v}(A_i, W_i)}{g_{n,v}(A_i, W_i)} \\
&+ \frac{1}{N_v^2} \sum_{i,j \subset \mathcal{V}_{n,v}} \left[I_{(-\infty,a_0]}(A_i) - F_{n,v}(a_0)\right] \mu_{n,v}\left(A_i, W_j\right) \Bigg\}.
\end{aligned} \tag{4}
$$

This also requires the estimators for each fold $u_{n,v}$ of the outcome regression u_0 as well as $g_{n,v}$,

which is the hybrid standardized propensity score defined as:

$$g_0(a,w) := \begin{cases} \frac{P_0(A=a|W=w)}{P_0(A=a)}, & \text{for } a \text{ such that } P_0(A=a) > 0 \\ \frac{p_0(a|w)}{f_0(a)}, & \text{for } a \text{ where } F_0 \text{ is absolutely continuous.} \end{cases}$$

Both u_0 and g_0 are estimated using generalized linear models and generalized additive models, as detailed in Section 4.3. This is done using the Superlearner package consisting of flexible libraries. SuperLearner applies cross-validation to assess the performance of the selected models and constructs an ensemble by taking a weighted average of the models' performances.

Additionally, Westling (2022, p. 6) shows that under certain standard doubly robust conditions, $n^{1/2}(\Omega_n - \Omega_0(a))$ converges weakly to a Gaussian process Z_0. Thus, by using the cross-fitted estimator Ω_n of Ω_0, it is possible to construct an approximation $T_{n,p,a}$ of the $1-\alpha$ quantile of the limiting distribution of $n^{1/2} ||\Omega_n - \Omega_0||_{F_0,p}$. Westling further shows that this test is doubly-robust consistent, meaning that this test remains consistent even if either the outcome regression μ_0 or the generalized propensity score g_0 is inconsistent. The test procedure is outlined in the following algorithm of five steps:

1. Split the sample into V equal size sets $\mathcal{V}_{n,1}, \ldots, \mathcal{V}_{n,V}$
2. Use the training set $\mathcal{T}_{n,v}$ for each fold v to construct the estimates $\mu_{n,v}$ and g_{n_v} of μ_0 and g_0.
3. For each value a within the observed set of $\mathcal{A}_n := [A_1, \ldots, A_n]$, use the estimates $\mu_{n,v}$ and $g_{n,v}$ to construct $\Omega_m^\circ(a)$ as defined by equation 4
4. Let p denote the norm, and $\mathbb{P}$ denote the empirical distributions for each of the validation folds $\mathcal{V}_{n,v}$. Furthermore, let $T_{n,\alpha,p}$ represent the $1-\alpha$ quantile of $\left(\frac{1}{n}\sum_{i=1}^n |Z_n(A_i)|^p\right)^{1/p}$ for $p < \infty$ or $\max_{a \subset \mathcal{A}_n} |Z_n(A_i)|$ for $p = \infty$, where, conditional on $O_1, \ldots, O_n$, $(Z_n(A_1), \ldots, Z_n(A_n))$ is mean-zero multivariate normal distributed with covariance $\Sigma_n\left(A_i, A_j\right) := E\left[Z_n(A_i) Z_n\left(A_j\right) \mid O_1, \ldots, O_n\right] = \frac{1}{V}\sum_{v=1}^V \mathbb{P}_{n,v} D^*_{A_i,n,v} D^*_{A_j,n,v}$ for $D^*_{a_0,nv}(y,a,w)$ equal to:

$$\begin{aligned} &[I_{(-\infty,a_0]}(a) - F_{n,v}(a_0)]\left[\frac{y-\mu_{n,v}(a,w)}{g_{n,v}(a,w)} + \theta_{n,v}(a) - \gamma_{n,v}\right] \\ &\quad + \int [I_{(-\infty,a_0]}(u) - F_{n,v}(a_0)]\,\mu_{n,v}(u,w) F_{n,v}(du) \\ &\quad - 2\Omega_{\mu_{n,v},F_{n,v},Q_{n,v}}(a_0) \end{aligned}$$

where $\theta_{n,v}(a) := \int \mu_{n,v}(a,w) dQ_{n,v}(w)$ and $\gamma_{n,v} := \iint \mu_{n,v}(a,w) dF_{n,v}(a) dQ_{n,v}(w)$

5. At the final step, reject H_0 for a given level of α if $n^{1/2} \left\|\Omega_n^\circ\right\|_{F_n,p} > T_{n,\alpha,p}$

For this study, the significance level α is chosen to be 0.05 for this test, as well as for all other tests used.

4.2 Alternative considerations for testing the causal effect

In traditional classical tests of association, causality can often be asserted due to randomization. In the case of observational data, establishing causality is more difficult due to potential confounding between the exposure and outcome variables. Much of the literature in the causal inference framework focuses on cases involving binary exposure variables. Various methods have been developed for such cases, including matching estimators (Rubin 1973), inverse probability weighted (IPW) estimators (Horvitz & Thompson 1952) or augmented inverse propensity weighted estimators (Robins et al. 1994). These methods allow for the estimation of counterfactual means under the different exposure levels and for testing whether these means are equal. In addition, these techniques can be extended to address polytomous discrete exposures.

However, estimating the causal dose-response function with continuous variables is more difficult, and the literature in this area is less developed compared to discrete cases. A common approach to address this is to discretize the exposure variable, thereby enabling the application of methods suited for discrete variables, such as those mentioned previously. However, discretization is not ideal for several reasons. The process of converting a continuous exposure variable to a discrete one can lead to information loss, which typically reduces the power of the test compared to the use of the original continuous variable (Westling 2022). Furthermore, discretization complicates the interpretation of the causal parameter (Young et al. 2019), and the choice of discretization method can affect the results, as there is no universally optimal approach.

As Westling argues, one approach to estimating the dose-response curve is to assume that the regression for the outcome on the exposure variable and potential confounders follows a linear model. Given that the model is correctly specified, the coefficient for the exposure corresponds to the slope of the dose-response curve. However, if the model is misspecified, the results will be inconsistent. While IPW may be used, it will cause inconsistent results if the model for the propensity score is wrong. Hence, for this reason, the nonparametric test as developed by Westling seems preferable. It makes fewer assumptions about the data-generating mechanism

than other parametric methods, making it more robust.

4.3 Generalized linear models

Generalized linear models (GLMS) extend traditional linear regression to be able to model outcomes with non-normal response distributions as well as nonlinear functions of the mean. For this case, they are used specifically to estimate the nuisance estimators g_0 and μ_0 using the identity link function and the logit link function. A GLM consists of three components: a random component, a linear predictor, and a link function.

The random component is the response variable $y_1, \cdots y_n$ with a PDF or PMF from the exponential family in which case a general expression can be written as:

$$f\left(y_i; \psi_i, \phi_i, \right) = \exp\left\{\frac{y_i\psi_i - b\left(\psi_i\right)}{\kappa_i(\phi)} + c\left(y_i, \phi\right)\right\}, \tag{5}$$

where ψ_i is the natural parameter, ϕ_i is the dispersion parameter, and $\kappa(\cdot), b(\cdot)$ and $c(\cdot)$ denotes some known functions.

For each observation i, a linear predictor relates parameters η_i concerning $E(y_i)$ to the independent variables $x_1, \cdots, x_p$ by a linear combination, expressed as:

$$\eta_i = \sum_{J=1}^{P} x_{ij}\beta_j, \quad i = 1, \ldots, n \tag{6}$$

which can also be written in matrix notation as: $\eta_i = \boldsymbol{x}_i' \boldsymbol{\beta}$

The link function $f(\cdot)$ connects the random components with the linear predictor to model the expected outcome. The link function transforms $u_i = E(Y_i)$ to the linear predictor such that $f(u_i) = \eta_i = \boldsymbol{x}' \boldsymbol{\beta}$. The link function must be monotonic and differentiable.

The natural parameter for a normal distribution is the mean, and the link function $f(u_i) = u_i$ is the identity link function, which has $\eta_i = u_i$. This is used by the GLM known as the linear model corresponding to the linear regression model: $u_i = \boldsymbol{x}_i' \boldsymbol{\beta}$

One advantage of expressing it in terms of a generalized linear model is that the logit model as well as the GAM model can easily be expressed in terms of a GLM.

For example, consider a Bernoulli trial for some observation i and with the binomial parameter π_i, so that $p(y_i = 1) = \pi_i$ and $p(y_i = 0) = 1 - \pi_i$. The natural parameter is the log odds

$\log [u_i/1 - u_i)]$. Then, the logistic regression can be expressed in terms of a GLM by using the canonical logit link function:

$$\text{logit}(\pi_i) = \log(\frac{\pi_i}{1 - \pi_i}) = \boldsymbol{x}_i' \boldsymbol{\beta} \tag{7}$$

The generalized additive model can be considered as a general extension of the generalized linear model by replacing the linear predictor with additive smooth functions of the covariates. For example, instead of the previous GLM structure with the logit link as described by equation 7, it is instead generalized to $f(\mu_i) = \sum_{j=1}^{p} s_j(x_{ij})$ where $s_j(\cdot)$ is an unspecified smooth function of the predictor j. Using the same logit link, the GAM model would then be expressed as:

$$\text{logit}(\pi_i) = \log(\frac{\pi_i}{1 - \pi_i}) = \sum_{j=1}^{p} s_j(x_{ij}) \tag{8}$$

GAMs provide greater flexibility compared to regular GLMs, as they do not require that the relationship between Y, or the logit transformation of Y in the logistic regression case, and $\boldsymbol{X}$ to be linear. This flexibility allows GAMs to discover patterns that are otherwise missed using GLM, leading to better predictions. One drawback is the reduced interpretability of the effects of covariates when smooth terms are included. Furthermore, statistical inference with GAMs is more difficult as it is unclear how to apply confidence intervals. GAMs also typically require a larger sample, as smoothing methods can have a large number of parameters. Nevertheless, in this study, the GAMs and GLMs are not used directly to make inferences, but rather to estimate the nuisance estimators for the nonparametric test. Thus, this aspect is not considered to be a problem.

4.4 Nonparametric regression

Nonparametric smoothing regression is a type of regression analysis used to estimate the relationship between a dependent variable and one or more independent variables without assuming a predefined form (like a linear or polynomial form) for the relationship. This flexibility allows the model to adapt more closely to the actual data, making it particularly useful in situations where the relationship between variables is complex or unknown. Hence, it can model relationships between variables while also being robust against functional form of misspecification and can detect structures that may otherwise be missed (Racine & Li 2004). As has been mentioned previously, the relationship between interest rates and inflation, and the relationship between

inflation and unemployment, are assumed to be nonlinear, justifying the reason for using nonparametric regression in this case. This method aims to capture the relationship between policy rates and inflation and approximate the form of how the causal dose-response curve may be shaped. However, this method is not based on the causal inference framework and no result can be attributed to a direct causal interpretation.

In nonparametric smoothing regression, smoothing refers to the process by which predictions are computed as weighted averages of observed responses in the training data. In kernel regression, the degree of smoothing is controlled by the bandwidth denoted h. Smoothing very little ($h \to 0$), means that the model can have very small details of any potential "true" regression function. However, it also causes each of the predictions to be an average of fewer observations, making the predictions more noisy. It is important to consider the relationship between bias and variance through the total error composed as: total error = noise + bias^2 + variance. Changing the amount of smoothing affects both bias and variance. To determine the optimal amount of smoothing, cross-validation is typically used. The bandwidth with the lowest error under cross-validation is then used to fit the regression curve for all data.

This study uses a nonparametric regression method adapting a cross-validated local linear estimator with the method developed by Racine & Li (2004) and Li & Racine (2004). This approach can use both continuous and categorical data through kernels, with the bandwidth selected by a data-driven approach. This is computed in R using the npreg package. Although the method naturally handles both types of data, this study only considers continuous data. Consequently, the methodology applied follows the specific approach described by Li & Racine (2004).

First, the nonparametric regression model can be expressed as:

$$y_j = \xi\left(x_j\right) + \epsilon_j, \quad j = 1, \ldots, n, \tag{9}$$

and where $x_j \in \mathbb{R}^q$. The derivative of $\xi(x)$ is defined as $\beta(\boldsymbol{x}) := \nabla\xi(x) \equiv \partial\xi(x)/\partial x$ and the gradient $\nabla\xi(\cdot)$ is a $q \times 1$ vector. Then $\delta(x) = (\xi(x), \beta(x)')'$ so that $\delta(x)$ is a $(q+1) \times 1$ vector-valued function. The first component of $\delta(x)$ is $\xi(x)$ and the other q components are the first derivatives of $\xi(x)$.

By Taylor expanding $\xi(x_j)$ at x_i, it results in:

$$\xi\left(x_j\right) = \xi\left(x_i\right) + \left(x_j - x_i\right)' \beta\left(x_i\right) + R_{ij} \tag{10}$$

where $R_{ij} = \xi\left(x_j\right) - \xi\left(x_i\right) - \left(x_j - x_i\right)' \beta\left(x_i\right)$.

Then, equation 9 can be rewritten as:

$$\begin{aligned} y_j &= \xi\left(x_i\right) + \left(x_j - x_i\right)' \nabla\xi\left(x_i\right) + R_{ij} + u_j \\ &= \left(1, \left(x_j - x_i\right)'\right) \delta\left(x_i\right) + R_{ij} + u_j. \end{aligned} \tag{11}$$

By using kernel weighted regression of y_j on $\left(1, \left(x_j - x_i\right)'\right)$, a leave-one-out local linear kernel estimator of $\delta\left(x_i\right)$ can be expressed as:

$$\begin{aligned} \hat{\delta}_{-i}\left(x_i\right) &= \begin{pmatrix} \hat{\xi}_{-i}\left(x_i\right) \\ \hat{\beta}_{-i}\left(x_i\right) \end{pmatrix} \\ &= \left[\sum_{j\neq i} K_{h,ij} \begin{pmatrix} 1, & \left(x_j - x_i\right)' \\ x_j - x_i, & \left(x_j - x_i\right)\left(x_j - x_i\right)' \end{pmatrix}\right]^{-1} \sum_{j\neq i} K_{h,ij} \begin{pmatrix} 1 \\ x_j - x_i \end{pmatrix} y_j, \end{aligned} \tag{12}$$

where $K_{h,ij} = \prod_{s=1}^{q} h_s^{-1} k\left(\left(x_{js} - x_{is}\right)/h_s\right)$ is the product kernel function and $h_s = h_s(n)$ is the smoothing parameter associated with the s th component of x.

Next, ι_1 is defined as a $q + 1 \times 1$ vector, with the first element being 1 and all the rest being 0. The leave-one-out kernel estimator of $\xi\left(x_i\right)$ is given by $\hat{\xi}_{-i}\left(x_i\right) = \iota_1' \hat{\delta}_{-i}\left(x_i\right)$, and $h_1, \ldots, h_q$ are chosen to minimize the least-squares cross-validation function given by:

$$CV\left(h_1, \ldots, h_q\right) = \sum_{i=1}^{n} \left[y_i - \hat{\xi}_{-i}\left(x_i\right)\right]^2. \tag{13}$$

$\hat{h} = \left(\hat{h}_1, \ldots, \hat{h}_q\right)$ denotes the cross-validation choices of $h_1, \ldots, h_q$ which minimize equation 12. After $\hat{h}$ is computed, then $\delta(x)$ is estimated by:

$$\begin{aligned} \hat{\delta}(x) &= \begin{pmatrix} \hat{\xi}(x) \\ \hat{\beta}(x) \end{pmatrix} \\ &= \left[\sum_{i=1}^{n} K_{\hat{h},ix} \begin{pmatrix} 1, & (x_i - x)' \\ x_i - x, (x_i - x)(x_i - x)' & \end{pmatrix}\right]^{-1} \sum_{i=1}^{n} K_{\hat{h},ix} \begin{pmatrix} 1 \\ x_i - x \end{pmatrix} y_i \end{aligned}$$

where $K_{\hat{h},ix} = \prod_{s=1}^{q} \hat{h}_s^{-1} k\left(\left(x_{is} - x_s\right)/\hat{h}_s\right)$, and $\xi(x)$ is estimated by $\hat{\xi}(x) = e_1' \hat{\delta}(x)$.

4.5 Dickey-Fuller test for stationarity

The concept of stationarity is an important property of time series.

Denoting the jth autocovariance as:

$$\begin{aligned} \upsilon_{jt} = \operatorname{Cov}\left(Y_t, Y_{t-j}\right) &= \operatorname{E}\left(Y_t - \mu_t\right)\left(Y_{t-j} - \mu_{t-j}\right) \\ &= \int_{-\infty}^{\infty}\int_{-\infty}^{\infty}(y-\mu_t)\left(x-\mu_{t-j}\right) f_{Y_t,Y_{t-j}}(y,x)\mathrm{d}y\ \mathrm{d}x \end{aligned} \tag{14}$$

Then, if neither the expectation μ_t nor the autocovariances γ_{jt} depend on the time t, the process for Y_t is said to be covariance-stationary or weakly stationary:

$$\begin{aligned} \operatorname{E}\left(Y_t\right) &= \mu, && \text{for all } t; \\ \operatorname{E}\left(Y_t - \mu\right)\left(Y_{t-j} - \mu\right) &= \upsilon_j < \infty, && \text{for all } t \text{ and any } j \end{aligned} \tag{15}$$

Time series typically need to be stationary to ensure the properties remain the same over time and to be able to make valid inferences. Using nonstationary time series in regression settings has potential consequences, such as inefficient estimators, sub-optimal forecasts, and invalid significance tests (Granger & Newbold 1974). This is tested with an augmented dickey fuller test as follows:

Consider, for example, an AR(1) process as: $y_t = \alpha + y_{t-1} + \delta_t + e_t$ where e_t is a martingale difference sequence such that $\operatorname{E}\left(e_t \mid y_{t-1}, y_{t-2}, \ldots y_0\right) = 0$. Then y_t has a unit root if and only if $\rho = 1$

The (non-augmented) Dickey-Fuller test involves fitting the model by OLS and testing the null hypothesis of $\rho = 1$ against the alternative of $\rho < 1$. However, to control for serial correlation the augmented Dickey-Fuller test fits a model on the form:

$$\Delta y_t = \Upsilon + \lambda y_{t-1} + \delta t + \zeta_1 \Delta y_{t-1} + \zeta_2 \Delta y_{t-2} + \cdots + \zeta_k \Delta y_{t-k} + \epsilon_t \tag{16}$$

for some specified number of τ lags. The test of $\mathrm{H}_0 : \lambda = 0$ against $\mathrm{H}_1 : \lambda < 0$ is then equivalent to the test of if $\rho = 1$ in the former model.

The test statistic does not have a standard t-distribution, and there are several versions of the test. See, for example, Hamilton (1994, Chapter 17) where the different test statistics are derived.

4.6 Ljung-Box test for autocorrelation

Since observations in time series data are typically dependent on the previous values, they are often autocorrelated. To test for autocorrelation in the time series, this study will use the Ljung-

Box test (Ljung & Box 1978), which tests for autocorrelation up to any specified length of τ lags. The empirical autocovariance function is given by: $\hat{\upsilon}_j = 1/T \sum_{t=i+1}^{T} \left(Y_t - \bar{Y}\right)\left(Y_{t-j} - \bar{Y}\right)$, from which it follows that the empirical autocorrelation function is given by: $\hat{\rho}_j = \hat{\upsilon}_j/\hat{\upsilon}_0$

The null and alternative hypotheses are

$$H_0 : \rho_1 = \rho_2 = \ldots = \rho_\tau = 0$$

$$H_1 : \rho_j \neq 0 \text{ for some } j \in \{1, 2, \ldots, \tau\}$$

The test statistic is given by the Ljung-Box statistic as:

$$Q_{LB}(\tau) = T(T+2) \sum_{j=1}^{\tau} \frac{\hat{\rho}_j^2}{T-j} \sim \chi^2(\tau - p) \tag{17}$$

where p denotes the number of parameters to be estimated.

5 Results

This section is structured as follows. First, the summary statistics and graphical representations are presented in 5.1. The results addressing the research questions are given in 5.2, which is followed by a discussion of the results in 5.3. Some additional supplementary results are provided in the Appendix.

5.1 Summary statistics

Table 1: Summary statistics for the different variables

	n	mean	sd	median	min	max	skew	kurtosis
us_interest	355	2.45	2.24	1.75	0.05	6.54	0.37	-1.52
swe_interest	355	2.26	2.30	2.00	-0.50	8.91	0.84	0.39
us_unemployment	355	5.62	1.82	5.20	3.40	14.80	1.43	2.27
swe_unemployment	355	7.60	1.16	7.60	4.90	10.50	0.15	-0.66
swe_CPIF	355	1.94	1.63	1.60	-0.40	10.20	2.89	9.80
us_PCE	355	2.76	2.54	2.60	-14.70	25.00	1.06	23.85

Table 1 lists the summary statistics for the variables. Comparing the Swedish and US data, it can be seen that the US inflation rate on average is higher than the Swedish inflation rate at

2.76 percent compared to 1.94 percent as defined by their respective consumer price indices (CPIF and PCE). These values align with the 2 percent inflation target, indicating the central banks' success in maintaining the target. The US inflation rate exhibits more variation, as indicated by the higher standard deviation of 2.54 compared to 1.63, as well as having a large difference between the min and max values. Furthermore, the US inflation rate also has a considerably higher kurtosis at 23.85 compared to 9.80 for the Swedish inflation rate, indicating a distribution with heavier tails. In contrast, interest rates and unemployment rates show more similarity between the two countries. Although there are minor differences in the interest rates, the Swedish unemployment rate is, on average, higher, with somewhat less variation and skewness compared to the US.

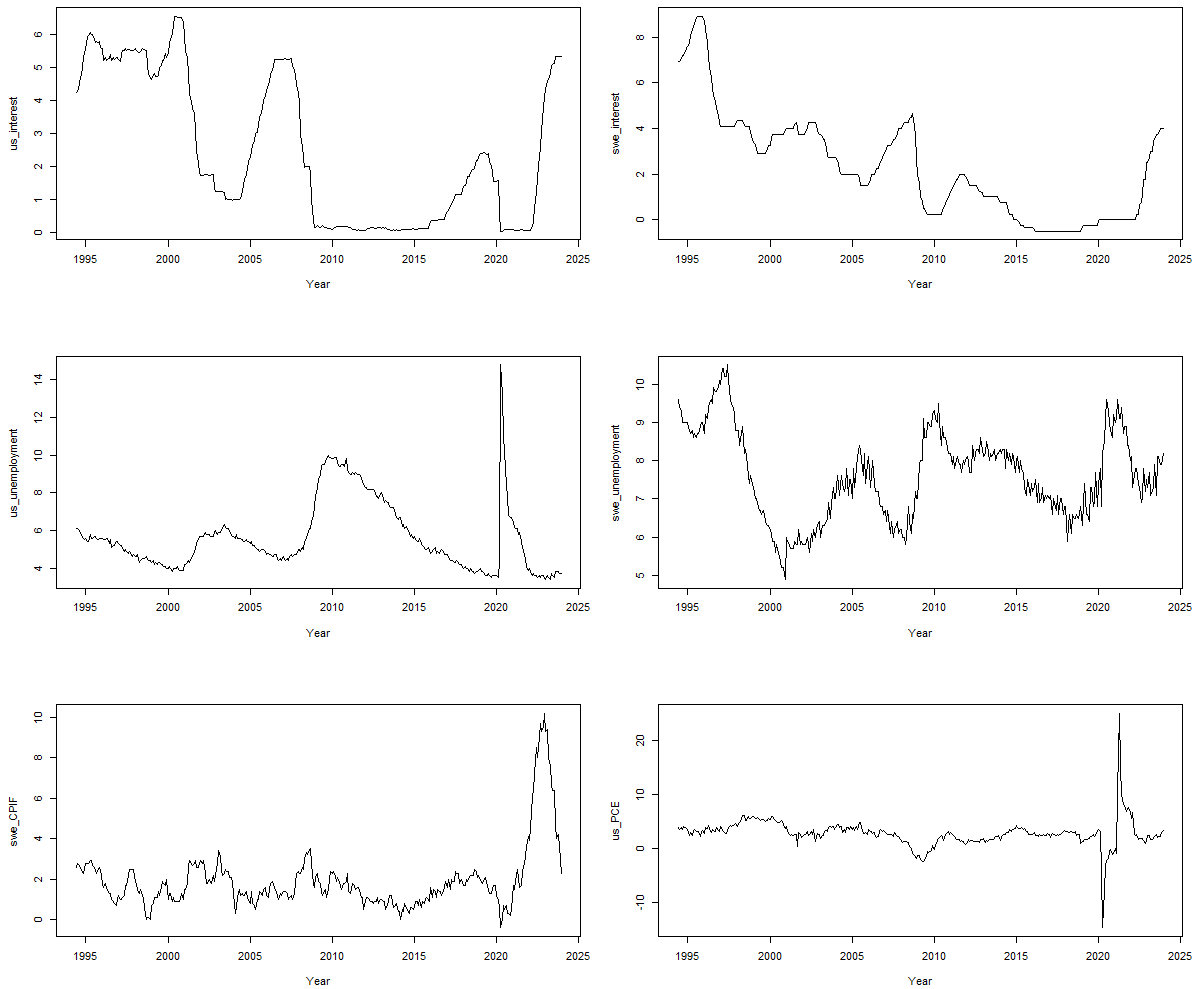

Figure 1: Variables over time

Figure 1 shows the different variables plotted over time. The visualized graphs align with the

results presented in Table 1. The interest and unemployment rates in both countries exhibit similar overall trends and peak patterns, likely due to their shared economic cycles. One noticeable difference is observed in the US policy rates, which remained near zero from approximately 2010 to 2015. Regarding the inflation rates, both countries demonstrated relative stability until 2020, when a significant positive spike was observed in both cases, alongside a substantial negative spike for the US. Similar patterns are present in the other variables; however, the magnitude of the spikes is less notable compared to those in the inflation rates, with previous fluctuations also observed.

5.2 Empirical results

The first test performed is the Dickey-Fuller adjusted stationarity test, with the results shown in Table 2.

Table 2: Adjusted Dickey-Fuller test results

	statistic	p.value
us_interest	-2.31	0.45
swe_interest	-2.51	0.36
us_unemployment	-2.22	0.48
swe_unemployment	-3.40	0.05
swe_CPIF	-2.46	0.38
us_PCE	-4.01	0.01

Since all p-values are not statistically significant except for the US PCE, the time series for the variables are not considered to be stationary, and the null hypothesis of a unit root cannot be rejected. This could raise some issues. However, by examining the time series in Figure 1, it was determined that they are fairly stable over time with respect to both mean and variance, and there is no evident long-term trend. It is important to note that ADF tests are sensitive to large spikes, such as the one apparent around the year 2020 which may impact the test performance. Furthermore, economic theory indicates that the variables should be relatively bounded: interest rates and inflation typically do not remain negative for extended periods and are usually only negative during severe recessions, when significant efforts are made to ensure positive values. Similarly, the unemployment rate cannot be below 0 percent and has not

exceeded 14.80 percent in the US or approximately 14 percent in Sweden. Consequently, these processes are unlikely to exhibit a random walk or other forms of persistent long-term trends. Therefore, even though the time series are deemed nonstationary by the adjusted Dickey-Fuller test, it should still be possible to use them without further adjustments. Although their first-order difference proved to be stationary, it would not make more sense to use it since it is still based on the difference between two consecutive values, in which case the overall pattern is still relatively similar.

Another issue of concern is the autocorrelation of the data. The ljung box-tests indicate present autocorrelation (Table 4 in the appendix). Additionally, a visual inspection of the Autocorrelation functions (ACFs) and partial autocorrelation functions (PACFs) showed the behavior of a typical AR process in a slow decay of the ACF, and with a sharp cutoff for the PACFs. The PACFs show significant values for the initial lags and a notable spike at the 12th lag, which seems reasonable considering it is monthly data. These observations suggest that positive values are frequently followed by other positive values, while negative values tend to follow negative values.

Nevertheless, it is not feasible to incorporate an AR(12) model to account for the 12:th lag, and it is more desirable to keep the model parsimonious. Hence, for the nonparametric test for the causal dose-response curve and the nonparametric regression used, the approach used is to keep only one autoregressive part of the inflation rate. Although it is uncertain whether using more autoregressive parts would change the results in any major way, using a model with one autoregressive part is simpler while also being based on the economic theory that inflation at some period t for one time period can be modeled by the information given by the previous period $t-1$, as done in Fama (1975) mentioned in section 2.1.

The results of the nonparametric test of a flat dose-response function as detailed in Section 4.1 are shown in Table 3. The analysis covers six different models using different lags of the unemployment rate and the policy rates while keeping an autoregressive part of the inflation for the previous month. For example, the results for lag 1 are based on the setting where the policy rate is the exposure variable for the inflation rate as the outcome, while the covariates are the one-month lag of the interest rate, the unemployment rate, and the inflation rate. For the lag 2 results, the lag length of interest rate and unemployment rates is increased to use the two-month lagged data instead, while maintaining the autoregressive term for inflation at $t-1$.

This is then tested up to lag 6.

Table 3: Results for causal dose-response curve test

	Swedish Data	**US Data**
Lag	p.val	p.val
1	0.18	0.26
2	0.02	0.00
3	0.00	0.00
4	0.03	0.00
5	0.09	0.00
6	0.31	0.37

The results show significant p-values for lags 2, 3, and 4 for the Swedish data. Likewise, for the US data, the p-values are significant for lag 2, 3, 4, and 5. Consequently, the null hypothesis that the causal dose-response curve is flat is rejected in these cases. This indicates that the causal effect of the Swedish policy rate on inflation is seen after 2-4 months and after 2-5 months for the federal funds rate.

Several nonparametric models are fitted using the method described in Section 4.4 while using the same lags of the variables for each case as done for the nonparametric significance test. The figures corresponding to the significant models in Table 3 are included in the appendix (Figures 2-9). Examination of these plots reveals no notable relationship between inflation rates and policy rates, while taking into account the unemployment rates and the lagged values of the inflation rate. The curves for both the Swedish data and the US data appear relatively flat. Furthermore, the automatically chosen bandwidths (Tables 5, 6 in the appendix) for the Swedish variables are around 0-3 for all the variables. However, for some of the lags of the US unemployment and policy rates, the bandwidths are excessively high, exceeding several thousand. Hence, these results did not provide any insight into the relationship between the policy rate and inflation. Although not formally analyzed in this study, one potential problem could be the high spikes in inflation rates following the COVID-19 epidemic. Another concern with respect to US data is that the policy rates were close to 0 for a long time during the period 2010-2015. It is not clear whether these two concerns have had any impact on the performance of the tests and regressions. Still, they may explain why bandwidths for the US

data are unreasonably high, as the models may have trouble smoothing the data trying to capture a relationship.

5.3 Discussion

The significant results of the nonparametric test of the causal dose-response curves indicate that there is a lagged causal effect and that the causal dose-response curve is not flat. However, it is also important to consider the underlying assumptions of the test. Perhaps the most important assumption is regarding the absence of unobserved confounder variables. If this is not fulfilled, the results may be biased. This study only considers the unemployment rate and lagged inflation values as potential confounders, based on their theoretical relevance and common use in other studies as potential confounder variables. Future research might benefit from using additional variables.

There is also a possibility that the model specifications may be incorrect, as there are no clear guidelines on how to handle the lagged effects of the variables. In the present case, the same lag is applied to both the unemployment rate and the interest rates. It is possible that the "true" lagged effects if, they can be distinctly identified, are different for interest rates and unemployment rates. Another possibility is that the causal effect may arise from multiple lags simultaneously and hence is not restricted to one lag. These sorts of considerations may also be of interest in future studies.

Furthermore, the models used to specify and estimate the nuisance functions μ_0 and g_0 for the causal null test are based on the GLM framework. Although the test is doubly robust and retains asymptotical validity even if one model is misspecified, the limited amount of available data may not be enough to ensure that the asymptotic properties can be applied in this context. Also, given the time series nature of the data, other types of models may be better suited to capture the dependencies of the autocorrelation in the time series data.

6 Conclusion

The primary objective of this study was to analyze whether the causal dose-response curves of the policy rate on inflation are flat for the US and Swedish data and to use nonparametric regression to try and capture what that relationship may look like. The key finding is that the

nonparametric test for the causal-dose response curve yielded significant results for lags 2-4 for the Swedish data and lags 2-5 for the US data, indicating that the causal-dose response curve is not flat. These findings suggest that the causal effects of policy rates are seen within 2-4 months for Sweden and 2-5 months for the USA. This also aligns with the economic theories that there is a lag between the effects. Thus, these results provide potential new evidence of the causality of policy rates on inflation based on the dose-response function for the causal inference framework.

The nonparametric models are not able to adequately estimate the density, as the estimated curves do not show any special relationship, while some of the models exhibit unreasonably high bandwidths. This issue may arise from limitations in the data or potential misspecifications of the models concerning the nature of the time series data.

To my knowledge, this study is the first to analyze the dose-response curve using nonparametric tests to determine whether there is an average causal effect. Given the limited literature on the dose-response curve of inflation and policy rates, future research is encouraged to build on these findings and explore potential improvements.

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Appendix

Table 4: Ljung-Box test

	statistic	parameter	p.value
us_interest	3444	12	0.00
swe_interest	3410	12	0.00
us_unemployment	2386	12	0.00
swe_unemployment	2904	12	0.00
swe_KPIF	2300	12	0.00
us_PCE	722	12	0.00

	swe_KPIF_{t-1}	$\text{swe\_unemployment}_{t-\tau}$	$\text{swe\_interest}_{t-\tau}$
$\tau = 1$	2.96	1.22	1.34
$\tau = 2$	2.67	0.76	1.17
$\tau = 3$	2.19	0.74	1.38
$\tau = 4$	1.75	0.30	1.91
$\tau = 5$	2.06	0.51	1.81
$\tau = 6$	1.70	0.79	0.26

Table 5: Bandwidths for Swedish data for different lags τ

	us_PCE_{t-1}	us_unemployment	us_interest
$\tau = 1$	5.74	3.86	4795118.89
$\tau = 2$	5.94	1789395	0.31
$\tau = 3$	5.59	1564231	3285292
$\tau = 4$	5.58	6451167	5093350
$\tau = 5$	5.56	2939304	2911466
$\tau = 6$	1.16	0.28	5.67

Table 6: Bandwidths for US data for different lags τ

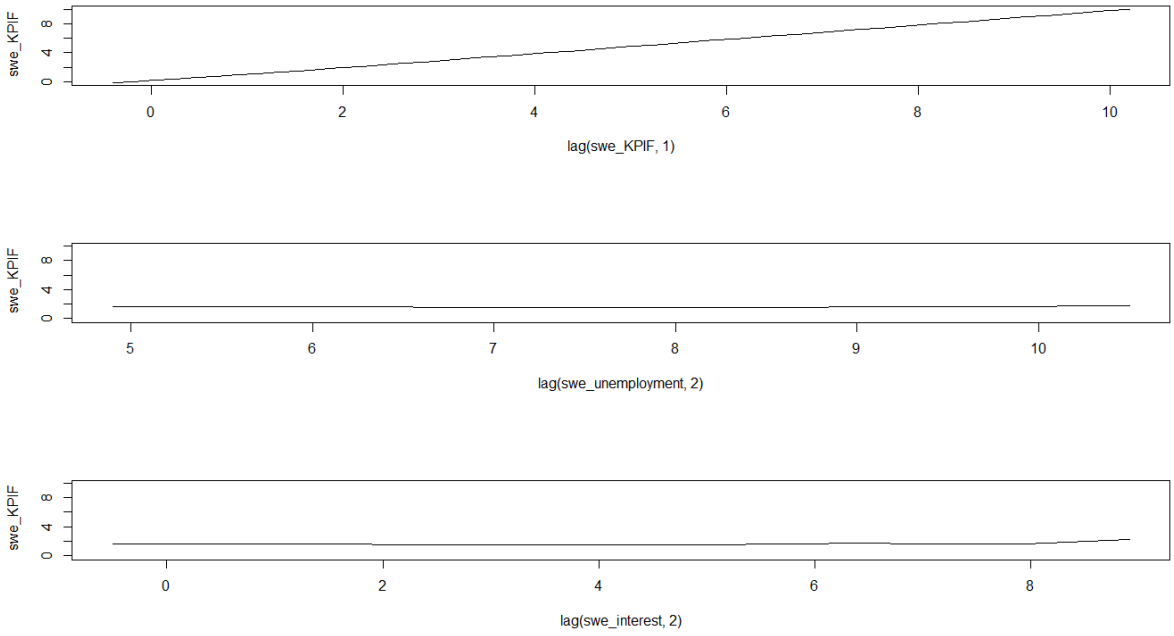

Figure 2: Nonparametric regression curves for Swedish CPIF using 2 lag

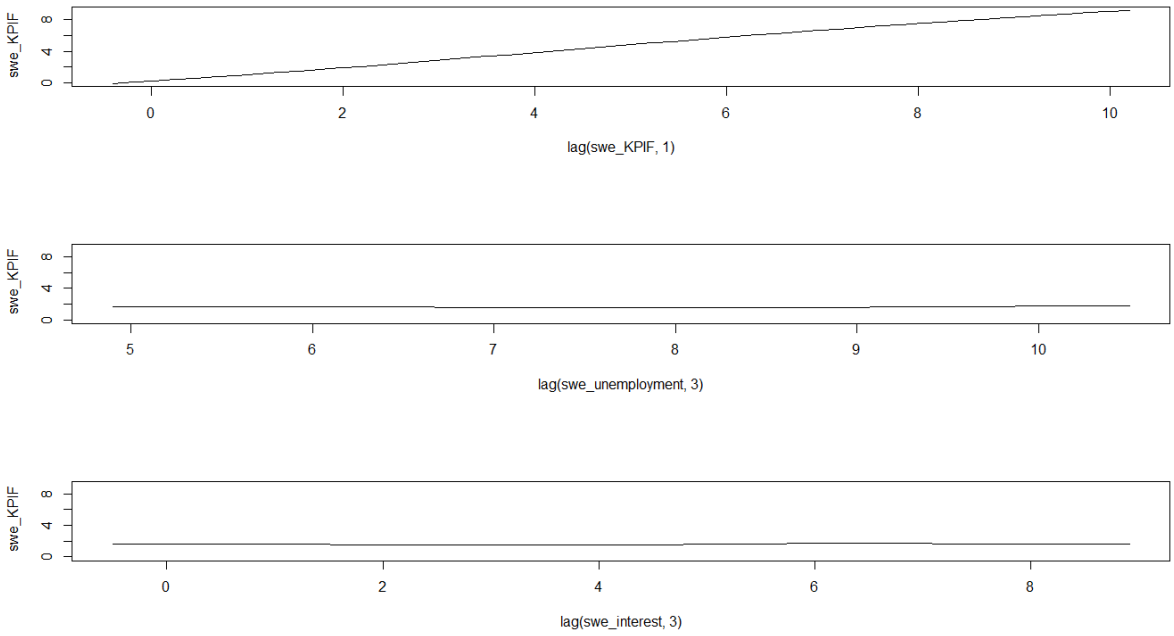

Figure 3: Nonparametric regression curves for Swedish CPIF using 2 lags

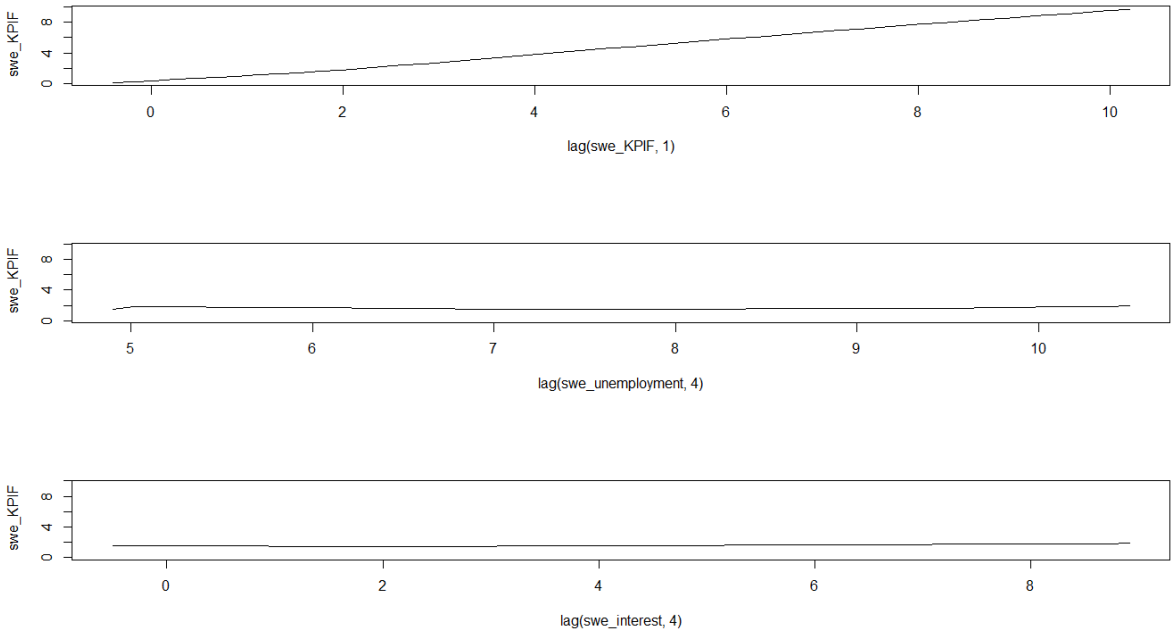

Figure 4: Nonparametric regression curves for Swedish CPIF using 4lags

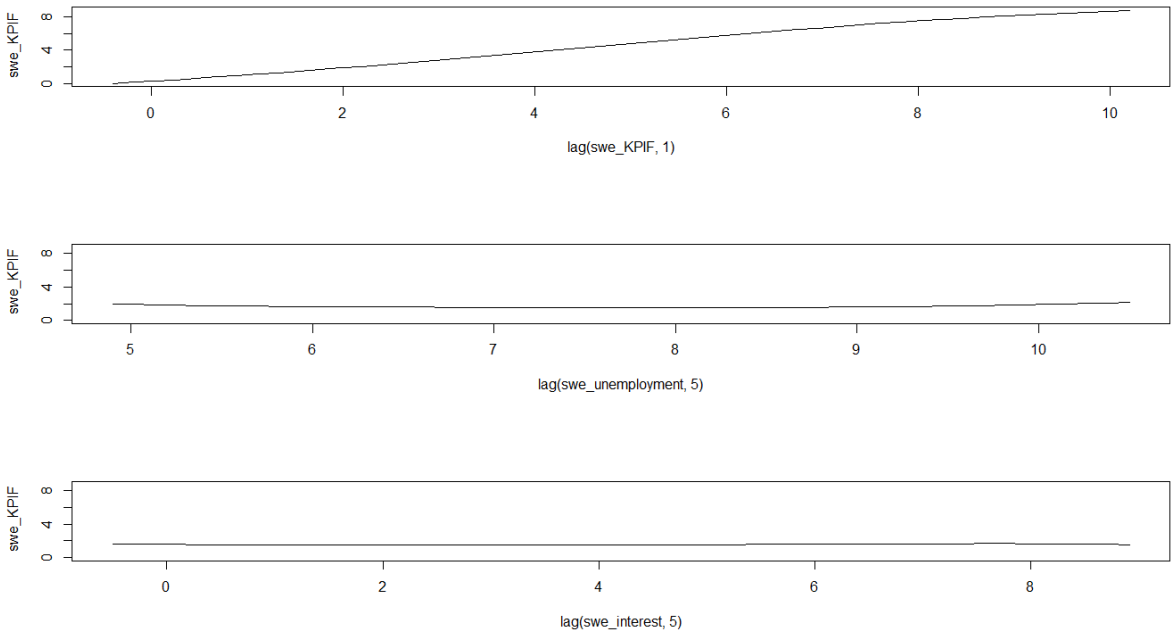

Figure 5: Nonparametric regression curves for Swedish CPIF using 5 lags

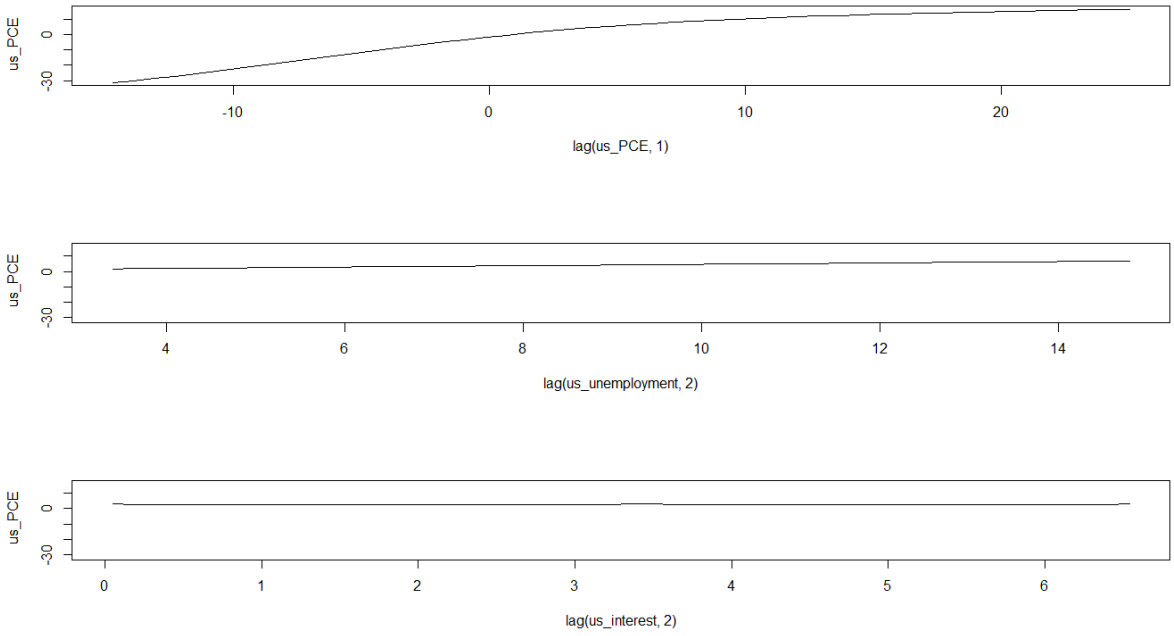

Figure 6: Nonparametric regression curves for US PCE using 2 lags

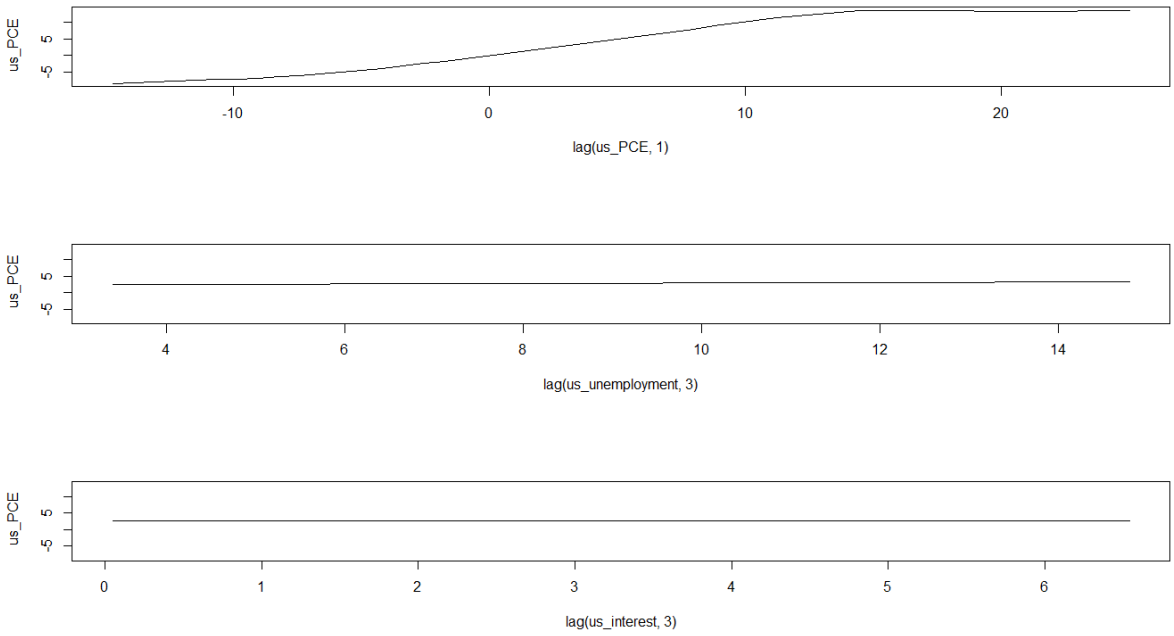

Figure 7: Nonparametric regression curves for US PCE using 3 lags

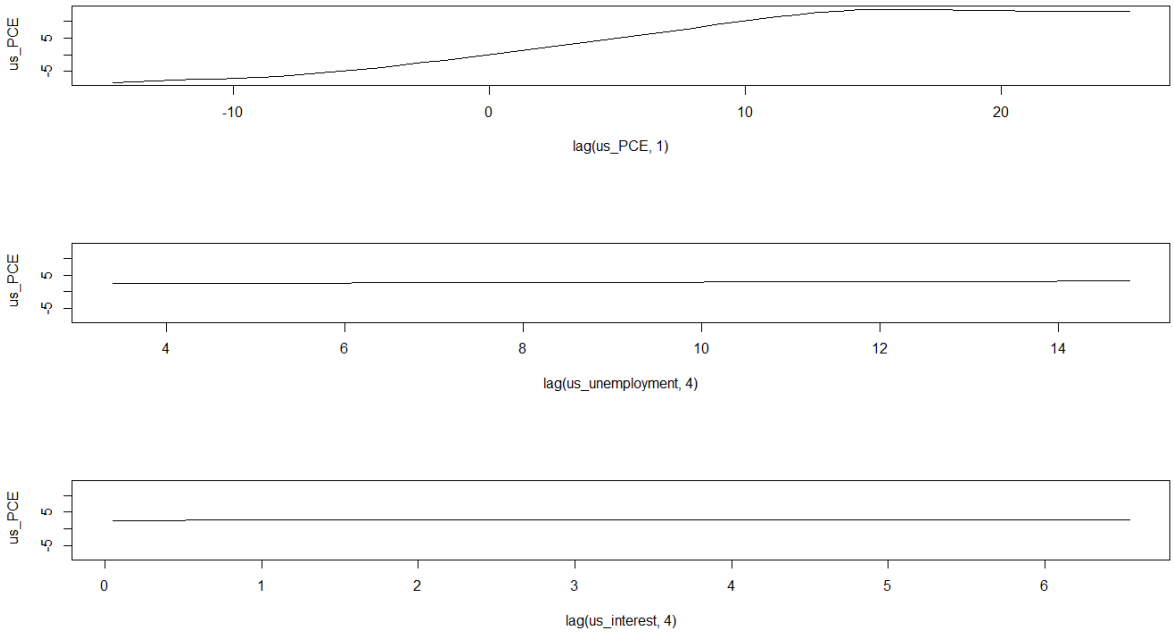

Figure 8: Nonparametric regression curves for US PCE using 4 lags

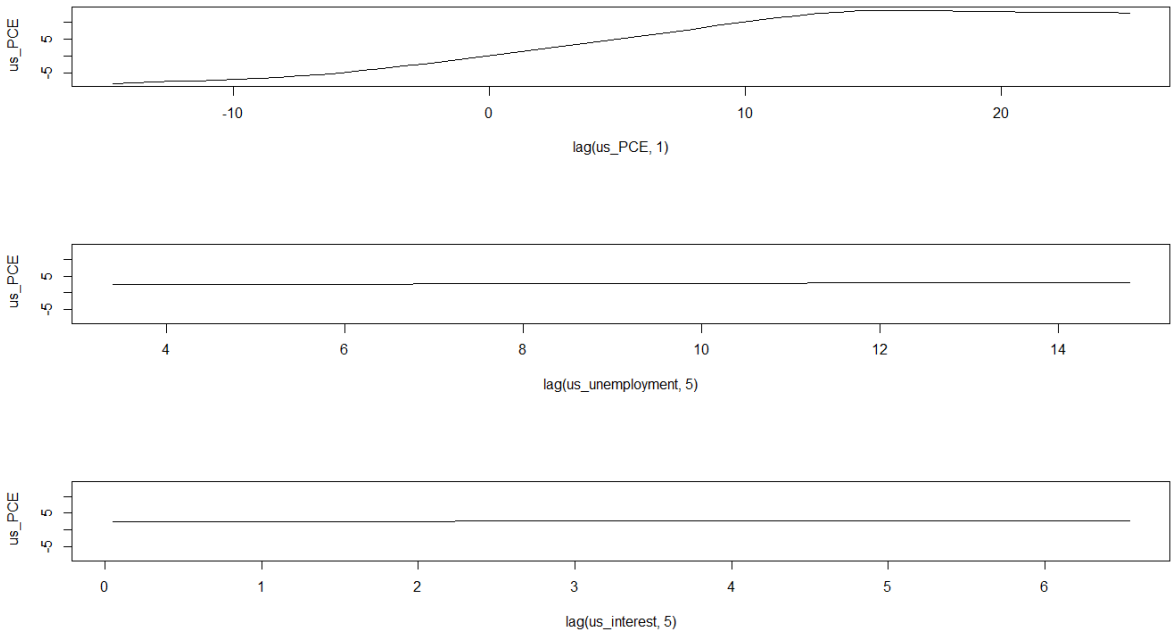

Figure 9: Nonparametric regression curves for US PCE using 5 lag