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Nonlinear Regression with Conditionally Stable
Innovations: A New Definition of Financial
Contagion

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Abstract

We develop a new notion of financial contagion, or the spread of negative characteristics from one market to another, by fitting a conditionally stable model to residuals extracted from a nonlinear regression. More specifically, we estimate the return on a dependent market given the return on an independent market using a spline-based local mean function. Then, instead of assuming that the residuals have a Gaussian distribution, we assume that the residuals are independent stable random variables when conditioned on the covariate market return. In general, the stable distribution depends on four parameters, two of which control skewness and tail heaviness. With our approach, these parameters become functions that are nonparametrically estimated. For various dependent markets, we study the change in the skewness and heaviness functions from the median to the tail of an associated covariate market return distribution (in our case, the U.S. stock market). Using a permutation test, we determine whether, given a value in the tail of the covariate market return distribution, the residuals are more likely to be left-skewed or heavy at the left tail than at the median of the covariate distribution.

1

Introduction

In financial econometrics, the relationship between a covariate market and another dependent market, especially during a time of crisis, is of interest for several reasons. Most importantly, the transmission of crises from one market to another sheds light on the limitations of portfolio diversification theory. In principle, portfolio diversification reduces risk in times of crisis through diversification across international markets [1][5][15][18]. We will refer to this transmission of crises, or more generally the phenomenon that occurs when one market transfers unattractive characteristics to another market, as “contagion.” Primarily, contagion is of concern for financial planners and international investors because if contagion indeed exists, portfolio diversification fails when its benefits are most needed [17]. While several authors have formally characterized multiple notions of contagion, we will develop a notion of contagion more appropriate to a market in which the dependent market is driven by independent, identically distributed innovations from a conditionally stable — rather than a Gaussian — distribution.

In general, we will investigate the skewness and tail heaviness of the residuals derived from a local polynomial fit between dependent and covariate market returns. As a result, we will be able to examine the skewness and tail heaviness of the residuals in the tail of the covariate distribution, and in doing so we will investigate difference

between skewness and tail heaviness at the tail and at the median of the distribution. In short, we will say that contagion exists when skewness or tail heaviness is significantly greater at the tail of the covariate return distribution than at the median. In other words, if the residuals are more likely to be negatively skewed in a day of crisis, we will say that skewness contagion exists. Similarly, we will say that tail heaviness contagion exists when the data has more probability mass in the tail of its distribution, i.e., returns are more likely to be large and negative (or large and positive, although we will not be as concerned with that case) given that a crisis has occurred.

2

Review of Relevant Literature

In order to motivate a new definition of contagion, we will first discuss previously attempts to define contagion and their failings, such as the problems associated with definitions of contagion derived from notions of correlation. Namely, to our knowledge all previous definitions of contagion depend on the assumption that stock return data has a conditionally Gaussian structure, which is widely held to be untrue. Hence, it is for this precise reason that we propose an alternative definition in the next section.

2.1 Contagion

In recent years financial crises, such as those in Mexico in 1995, Thailand in 1997, in Russia in 1999, and most recently the credit crisis in 2008, have provided motivation for concern about international “spillovers” from one market to the next. More specifically, these cross-country comovements of markets in times of crisis have prompted academics and financial practitioners alike to ask whether policies should aim to prevent such comovements in order to protect markets and investors for a variety of reasons, whether it be to preserve the benefits of diversification or to protect countries with economies that cannot afford to feel the impact of an international crisis, a common adverse side effect of international market integration [17].

Problematically, no common definition exists in the literature for “contagion,” the term most commonly associated with the comovements of international equity markets—or the transmission of crisis from a market X to a market Y . Hence, there exists no standard empirical or theoretical tool to identify whether contagion does or does not exist in a given situation. While some authors provide theoretical grounds for identifying contagion, statistical justification does not always exist in order to confirm the existence of contagion during a given crisis. Finally, in some studies on contagion, authors do not distinguish between interdependence and contagion, which causes ambiguity regarding whether two markets are interrelated based on a variety of factors or whether a true transmission of a crisis has occurred. In other words, the comovement of two markets could occur due to a general correlation between the markets rather than a heightened level of interdependence given that a crisis has occurred. Theoretically, contagion is more often associated with the latter case, namely when there exists an *increased* level of dependence during a time of crisis [17].

While no true agreement has become well accepted in the literature regarding a rigorous definition for contagion in the literature, many authors have attempted to produce such a definition. As a result, in perhaps the primary paper on financial contagion, Pericoli and Sbracia (2003) provide a general overview of previous work on contagion and present five definitions along with their statistical counterparts.

First, the authors identify contagion as “a significant increase in the probability of a crisis in one country, conditional on a crisis occurring in another country.” This definition, most commonly used in an attempt to study currency collapses, relies on a crisis indicator i . Specifically, a study following this definition would examine a weighted average of changes in exchange rates, short term interest rates, and international reserves. The study would then take an extreme value of the distribution of the weighted averages, for example two standard deviations from the mean, and call it x . We would then say that a crisis has occurred in country i if the weighted average at time t is greater than x . Finally, the econometrician would then examine

the increase in the probability of crisis in country Y given that a crisis has occurred in country i , using the appropriate control variables.

The second definition which Pericoli and Sbracia present identifies contagion as occurring “when volatility of asset prices spills over from the crisis country to other countries.” This definition “exploits” the empirical evidence of an increase in volatility of asset prices during a time of financial turmoil, and characterizes market uncertainty by an index of market volatility. Intuitively, then, this definition can be easily interpreted as the spread of uncertainty from one market to another. Unfortunately, the coexistence of uncertainty in one market and uncertainty in another market can just as easily be due to market interdependence rather than contagion, which we can think of as a transmission of a negative characteristic of one market to another, rather than simply the coexistence of the same undesirable characteristic in two markets. Statistically, this measure of contagion uses the residual correlation between markets after modeling the returns in a market X as a function of the returns of some market Y . It assumes that the covariance of market X and Y are normally distributed, which results from the assumption that the joint distribution of returns in markets X and Y are normal. Once the model is estimated, one can measure the effects of the country-specific shock on country X at time t on the volatility (or market uncertainty) of market X , the covariance between markets X and Y , and the volatility of market Y .

Third, Pericoli and Sbracia present a more theoretical approach to defining contagion, namely that contagion exists when “cross-country comovements of asset prices cannot be explained by fundamentals,” where by fundamentals the authors mean theoretical beliefs regarding how international markets function and interact, such as, for example, the existence of theoretical market equilibria. Problematically, however, the authors admit that contagion could indeed exist within the theoretic structure, but would not be identified as such by the model.

Fourth, the authors define contagion as “a significant increase in comovements of prices and quantities across markets, conditional on a crisis occurring in one market

or group of markets.” The appeal of this definition is that it seems to capture the most intuitive meaning of contagion, but it is sensitive to the meaning of “significant increase,” which of course can be defined a variety of ways. According to Pericoli and Sbracia, the key to defining contagion in this way is to distinguish between periods of normal comovements, often associated with simple interdependence, and excessive comovements, associated with a structural change in the data. The latter, then, would indicate a period of contagion, while the former would be of less concern. Finally, an examination of the difference in correlation during a period of excessive comovements and a period of normal comovements determines whether contagion exists.

Finally, the authors present a fifth definition of contagion, namely that contagion exists when “the transmission channel intensifies, or, more generally, changes after a shock in one market,” where by transmission channel Pericoli and Sbracia mean, in short, the way in which one market relates to another. For example, if for instance the U.S. market and the French market both typically depend on truffle prices, and yet during a period of crisis cease to depend on truffle prices altogether and instead depend on pork belly prices, we would say that a shift in the structure of transmission channels has occurred, and likely conclude that contagion has occurred as well [17].

Pericoli and Sbracia identify two major drawbacks of the definitions of contagion presented above. First, the notion of contagion often depends heavily on some definition of a crisis or crisis period in a market or group of markets. Admittedly, the identification of crisis and tranquil periods are often arbitrary, and have heavy impacts on whether one can conclude that contagion exists in a give period. Additionally, the presence of a lag might lessen the probability that contagion is correctly identified [17].

The more recent trend in the contagion literature roots itself in econometrics. Specifically, authors have recently exploited regression theory and its relationship to correlation in order to characterize contagion, where markets are treated as random variables. While the most straightforward approach to characterizing the relation-

ship between two random variables is to compute the simple (Pearson’s) correlation coefficient between the two random variables, which is given by

$$\rho_{X,Y} = \text{Cov}(XY)/\text{Var}(X)\text{Var}(Y), \quad (2.1)$$

this measure fails to capture information regarding specifics about the data, such as where the data is located in the distribution. Since contagion is, intuitively, an increase in the probability that a crisis occurs in market Y given that a crisis has occurred in market X , when investigating contagion we are mainly concerned with data located in some set associated with the occurrence of a “crisis.” As is natural, previous literature has concentrated on definitions of correlation dependent on the fact that a crisis has occurred. Specifically, *conditional correlation*, ρ_A , is the correlation between X and Y given that X lies in some set A . This concept is a common starting point when beginning a discussion on contagion. Namely, conditional correlation is given by

$$\rho_A = \rho(X, Y|X \in A). \quad (2.2)$$

Difficulties with this approach have been identified and studied extensively by a variety of authors (see Forbes & Rigobon 2002, Bradley & Taqqu 2004, Boter, Gibson, & Loretan 1999, Hamrick & Taqqu 2009). First, the conditional correlation coefficient requires that one assume that X and Y are jointly Gaussian. In particular, since X and Y are jointly Gaussian, $Y|X = x$ is Gaussian. In financial studies, where X and Y will typically be stock returns or indices, the distribution in question in many cases is not jointly Gaussian. In particular, the Gaussian relationship does not assign sufficient probability to the tails of the distribution. For example, see Figure 2.1. This modeling limitation is particularly problematic when we wish to study large losses and gains [4].

The more subtle drawback to the use of conditional correlation is its dependence on some definition of crisis period. For example, in our context, A serves as some set which contains those returns occurring during a heuristically-defined “crisis period.”

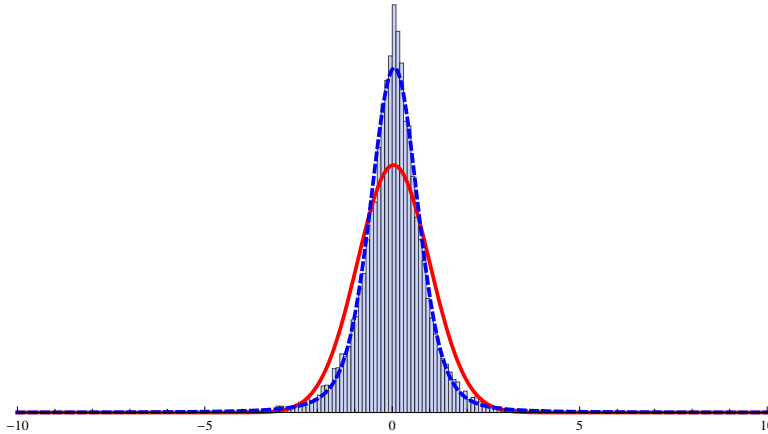


Figure 2.1: In red, the best possible fit of a Gaussian distribution is displayed over a histogram of Standard & Poor's 500 returns since 1991. In blue, the best possible fit of a stable distribution is displayed.

As pointed out by Boyer, Gibson, & Loretan (1999), the way in which the conditional correlation depends on the crisis set A can be problematic. Specifically, in the case where the set A contains returns that are highly variable, estimates of the conditional correlation coefficient are biased upward. For example, during a crisis period, stock returns are, almost by definition, more likely to be highly variable. Consequently, in many cases the use of ρ_A may lead one to conclude too easily that contagion exists. In other words, the upward bias in ρ_A could lead to overzealous claims. Several authors have studied the effects of this bias and provided alternate definitions. For example, Forbes and Rigobon (2002) provide an adjusted ρ_A that accounts for the volatility in A . Unfortunately, as Bradley and Taqqu (2004) point out, the power of Forbes and Rigobon's test for contagion, developed from the adjusted estimator, is very low. In other words, given that contagion is present, a test for contagion with low power identifies the presence of contagion with low probability, which is obviously problematic.

In response to the problems associated with the use of conditional correlation, Bradley and Taqqu study a more general definition of contagion, where the local correlation coefficient is derived from the nonlinear regression model

$$Y_t = m(X_t) + \sigma(X_t)\epsilon_t \quad (2.3)$$

where m is the local mean function and σ is the skedastic function. In the linear case, $m(x) = \alpha + \beta x$ and $\sigma(X_t) = \sigma$. Whereas conditional correlation is developed from the linear case, Bradley and Taqqu use a generalization of the correlation coefficient—the local correlation—to define contagion. The local correlation $\rho(x)$ is given by

$$\rho(x) = \frac{\sigma_X \beta(x)}{\sqrt{\sigma_X^2 \beta^2(x) + \sigma^2(x)}}, \quad (2.4)$$

where $\beta(x)$ is $m'(x)$, the slope of the mean function at x , and $\sigma^2(x) = \text{Var}(Y|X = x)$, the nonparametric residual variance at x . Finally, Bradley and Taqqu define contagion from market X to market Y as the case where $\rho(x_L) > \rho(x_M)$, where x_M is the median of the distribution of X and x_L is a low quantile of X , such as $F_X^{-1}(.025)$. In short, Bradley and Taqqu simultaneously accomplish two goals. First, they escape the unrealistic assumption that a linear relationship exists between X and Y . Second, they account for the correlation at a certain point by employing a more general β and σ that now depend on a particular location within the support of X , avoiding the problems involved with defining a crisis period. Instead, the point at which the presence of contagion is determined is dictated by the structure of the data itself, rather than by a hueristically defined crisis set A .

Using the United States as the independent market in eleven cases, Bradley and Taqqu find evidence for contagion in seven cases, between the United States and Belgium, France, Germany, Italy, the Netherlands, Switzerland, and the United Kingdom. The Hong Kong, Japan, Australia, and Canadian markets did not appear to be contagious with the United States market in Bradley and Taqqu’s investigation [5]. Additionally, these results were reached based on the use of daily return data similar to the data used in this study. Bradley and Taqqu also expanded their analysis to an

investigation of contagion using the United States as the dependent market, in which they were not able to infer that contagion exists. Given that the United States is the dominant international equity market by volume, this result seems intuitive [5].

The advantage of defining contagion in this way, i.e., using local correlation as opposed to conditional correlation, is to escape the problematic consequences of having to define a crisis period. While the move from conditional to local correlation is certainly a viable one, *both definitions* require that the distribution $Y|X = x$ be Gaussian [4]. Unfortunately, it is widely accepted that stock return data do not have a Gaussian distribution [1][15][18]. As evidence of this fact, consider evidence from the US stock market. Over the past century, returns of less than negative twenty percent have occurred twice. Under the assumption of normality, a *single* occurrence of a negative return of that magnitude would occur once every two lifetimes of the earth. While this example is not a definitive reproach on the assumption that stock returns have a normal distribution, it is an easily understood example that evidences the fact that researchers should indeed be wary of this assumption. Indeed, as the literature confirms, the assumption of normality is an undesirable one.

Because implications of contagion for the purposes of portfolio diversification are significant, and because it is easily observed that stock returns are in general not Gaussian, the goal of this project is to provide and investigate a more robust definition of contagion that is free of the assumption that $(X, Y) \sim \mathcal{N}(\mu_X, \mu_Y, \sigma_X^2, \sigma_Y^2, \rho)$. Hence, our estimation will evolve from the assumption that the random variables in question have a conditionally stable distribution. This assumption is a much more reasonable one for two reasons. First, the generalized Central Limit Theorem guarantees that, in the limit, the normalized sums of independent identically distributed random variables is stable—even those with infinite variance [16]. Secondly, stable distributions generally admit heavy tails and skewness, of which we see evidence in return data. Since we will provide our alternate definition of contagion as a model dependent on the stable distribution, a discussion of the class of stable distributions

and their attractiveness with regard to return data is necessary.

2.2 The Stable Distribution

In many models of financial returns, returns are assumed to be jointly Gaussian, or normally distributed. For example, even the fundamental Black and Scholes option pricing theory relies on the normality of returns [3]. However, as early as the 1960s, it has been observed that return data exhibits characteristics of distributions with considerable more probability mass in the tails of the data and higher peaks at the mean [12]. As such, the fields of finance and economics have embraced the use of stable distributions (see Mandelbrot 1963, Fama 1965, Samuelson 1967, Roll 1970, Embrechts et al. 1997, Rachev and Mittnik 2000, McCulloch 1996). Stable distributions are “a rich class of probability distributions that allow skewness and heavy tails and have many intriguing mathematical properties” [16]. In particular, a random variable X is stable if, when X_1 and X_2 are independent copies of X , and for every choice of positive real numbers a and b ,

$$aX_1 + bX_2 \stackrel{D}{=} cX + d \tag{2.5}$$

for some positive c and $d \in \mathbb{R}$, where by $\stackrel{D}{=}$ we mean equal in distribution. More generally, for $X_1, X_2, X_3, \dots, X_n$ independent, identical copies of a random variable X , X is stable if and only if for all $n > 1$, there exists constants $c_n > 0$ and $d_n \in \mathbb{R}$ such that

$$X_1 + X_2 + X_3 + \dots + X_n \stackrel{D}{=} c_n X + d_n. \tag{2.6}$$

While these definitions are intuitive ones in as much as X remains “stable,” or unchanged, under addition, they provide no concrete way of parameterizing the stable distributions with a closed form probability density function. In fact, while the class of all stable distributions is large, only three specific stable distributions contained within the class of all stable distributions have known closed form density functions:

Table 2.1: Comparison of tail probabilities for standard normal, Cauchy, and Levy distributions.

$\mathbb{P}(X > c)$			
c	Normal	Cauchy	Levy
0	.5000	.5000	1.000
1	.1587	.2500	.6827
2	.0228	.1476	.5205
3	.001347	.1024	.4363
4	.00003167	.0780	.3829
5	.0000002866	.0628	.3453

the Gaussian (normal) distribution, the Cauchy (Lorentz) distribution, and the Levy distribution. As an expository example of the wide range of densities included in the class of all stably distributed random variables, consider the differences between these three cases. Specifically, consider a comparison of tail probabilities for the three distributions in Table 2.2 as an example of the varying possibilities of skewness and tail heaviness of different stable distributions.

While the lack of closed form density functions for the majority of stable random variables might seem at first to prevent the use of the stable distribution in practice, recall that even the standard normal cumulative distribution function has no closed form, yet it remains one of the most widely used distributions in all of applied statistics. In particular, recent advances in technology and the computational ability of computers, we can estimate the values of functions without closed forms with ease and accuracy. Hence, while it might seem that the stable distribution is limited to the above four cases in practice, it is in fact the case that we can easily estimate values and quantiles of the stable random variable. Specifically, recall that the characteristic function of a random variable X completely determines the distribution of X . In the

case of the stable distribution, we *do* know the characteristic function $\phi(u)$,

$$\phi(u) = \begin{cases} \exp(-\gamma^\alpha |u|^\alpha [1 + i\beta(\tan \frac{\pi\alpha}{2})(\text{sign} u)(|\gamma u|^{1-\alpha} - 1)] + i\delta u) & \text{if } \alpha \neq 1 \\ \exp(-\gamma |u|^\alpha [1 + i\beta(\frac{2}{\pi})(\text{sign } u)(\log |u|)] + i\delta u) & \text{if } \alpha = 1 \end{cases}$$

so we have the knowledge necessary to characterize the density. In general, the Fourier transform of a random variable is the characteristic function of the random variable, so we can indirectly access the probability density function of a stable random variable, which in turn depends on α , β , γ , and δ .

Equipped with the characteristic function of a stable random variable, we say that a stable random variable X is parameterized by four measures:

1. α , the index of stability or characteristic exponent, where $\alpha \in (0, 2]$,
2. β , a skewness parameter, where $\beta \in [-1, 1]$,
3. γ , a scale parameter, where $\gamma \geq 0$, and
4. δ , a location parameter, where $\delta \in \mathbb{R}$.

More intuitively, α controls the tail heaviness of the data, β controls how much the distribution is left or right skewed, and γ and δ control the rather uninteresting aspects of the distribution—scale and location. Hence, we will remain from this point forward almost exclusively interested in the parameters α and β , which we will relate to contagion shortly. Finally, for a stable random variable X , we say $X \sim \text{Sta}(\alpha, \beta, \gamma, \delta)$. Moreover, since the stable distribution is fully characterized by α, β, γ , and δ , once we are equipped with values for the parameters governing the population of a sample of stock returns, we can exploit the probability density function of that population and develop a notion of contagion that evolves from the skewness and tail heaviness of that population. Namely, if we can access the joint PDF of the returns of a market X and a market Y , since the parameters α and β govern characteristics of the distribution that intuitively relate to contagion, we move towards a formal definition of contagion between two markets as dependent on α and β .

3

Methods

In the process of our estimation, we in general execute the following steps:

1. Using an Epanechnikov kernel weighting function, we estimate functions that govern the stock returns on a foreign market given the stock returns in the United States by fitting a local mean function to the data.
2. We extract the residuals from the results of the local mean function. After doing so, we fit the residuals to a stable model using conditional Maximum Likelihood Estimation (MLE).
3. Modeling the residuals using conditional MLE produces functions for the stable parameters dependent on a point x in the support of the independent variable. With these estimates in hand, we use a technique called *bootstrapping* to create confidence intervals around our estimates at the points of interest within the support of the independent data.
4. Finally, with the bootstrapped estimates in hand, we have the tools necessary to create a measure of contagion independent of any assumption of normality, which is the ultimate goal of this project. Specifically, we will construct and execute a hypothesis test based on these bootstraps, under which the null hypothesis will be that contagion does not exist between the U.S. and some other

market, and conversely where the alternate hypothesis is that contagion does exist between the U.S. market and some other market. These hypotheses will depend on changes in $\alpha(x)$ and $\beta(x)$ from the tail to the mean of the distribution of the U.S. market returns.

3.1 Local Mean Estimation

As opposed most earlier studies on contagion, we will work with a non-linear regression model similar to that of Bradley and Taqqu (2004). Our model, like Bradley and Taqqu's, is also non-parametric and assumes that X affects Y in two ways: through a predictable component m and through an unpredictable component ϵ . Both m and ϵ depend on the the level of the covariate X . In other words, we will work with the model

$$Y_t = m(X_t) + \epsilon(X_t), \tag{3.1}$$

in order to move toward a more robust definition of contagion. The ultimate goal is to model the returns in a market Y given the returns in a market X and to examine the *residuals* from this fit. In doing so, we will concentrate on movements in Y unexplained by X , or more generally *atypical* association between a market X and a market Y rather than the returns on Y given X , which is modeled by the local mean function $m(X_t)$. When attempting to characterize contagion, this atypical association is precisely what we wish to study, which means we wish to extract the residual structure $\epsilon(X_t)$.

In order to fit the local mean function with the end goal of modeling the residual structure, we estimate $m(X_t)$ using *Mathematica* code originally developed by Jeff Hamrick and modified by the author [10]. Specifically, an estimate of the local mean function is obtained by solving a weighted least squares problem using the Epanechnikov kernel, and then fitting a cubic spline to the data. The weighted least squares

problem can be stated as

$$\sum_{i=1}^n \left\{ Y_i - \sum_{j=0}^p \beta (X_i - x_0)^j \right\}^2 K \left(\frac{X_i - x_0}{h} \right), \quad (3.2)$$

where in our case we fit a global polynomial, where $p = 3$, and where K is the weighting function, which controls the amount of weight allocated to each point x_0 in the independent data and hence the degree to which x_0 affects $\hat{m}(x_0)$. The result of solving this problem in *Mathematica* produces a cubic spline, which is the value of the function \hat{m} and its first two derivatives at a given point x in the independent data. Ultimately, the spline is used to fit an interpolation function in *Mathematica*, which takes in a discrete number of values for $\hat{m}(x_t)$ (and its derivatives) at each corresponding x_t and fits a cubic between the triple produced by taking $\{(x_{t-1}, \hat{m}(x_{t-1})), (x_t, \hat{m}(x_t)), (x_{t+1}, \hat{m}(x_{t+1}))\}$ for each t . The result is a smooth function \hat{m} that is both continuous and differentiable for all x in the support of X_t . In Figure 3.1, we see an example fit of an interpolation function to a cubic spline produced by the local mean function. In red, the actual interpolation function is displayed. The data modeled by the spline is shown in blue.

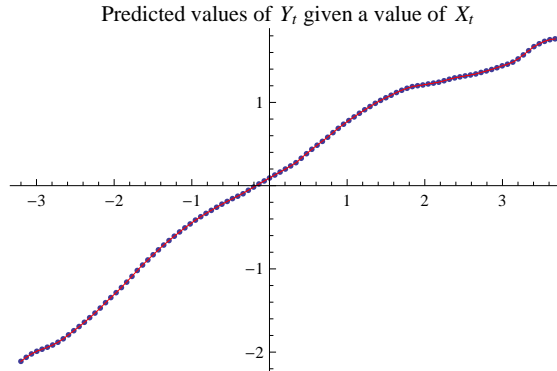


Figure 3.1: In red, an interpolation function generated in *Mathematica* using the spline from the local mean predictions for y_t given x_t , in blue.

The term *local* alludes to the fact that \hat{m} is produced using a weighted least squares model. In particular, at a given x , the estimation technique produces \hat{y} by

taking into account only data that are relatively close in the distribution to x . This estimation technique is preferable because it seems intuitively to be the case that when predicting y at a given x , data closest to x in the distribution should have the greatest affect on the prediction of y . In other words, outlying data is taken to be negligible, so that a “local mean” relationship is modeled. The fit is local in so far as the the least squares problem incorporates the weighting function K to assign less weight to data farther from the point x . The width of the interval which is allowed to affect the estimation of y at a given x is controlled by the parameter h , which we refer to as the *bandwidth* of the kernel.

In the case of the local mean function, it has been shown that the best weighting scheme is implemented using the Epanechnikov kernel [21], and as such we use the Epanechnikov kernel in our estimation. In fact, we not only use the Epanechnikov kernel to estimate m , but due to its widespread use in the literature, we also employ the Epanenchnikov kernel in our “ad hoc” process to fit the a stable distribution to the residual estimates of m . This process and the importance of the Epanechnikov kernel to its implementation will be explored shortly.

3.2 Kernel Smoothing

Kernel Smoothing is a widely-used method in local polynomial fitting for allowing only data in a certain neighborhood of a design point x to affect the prediction of \hat{y} given x [7][20][22]. In particular, the Epanechnikov kernel has been shown to be the best weighting scheme in the context of local polynomial fitting, or local mean estimation, and has the form

$$K_h(x) = \begin{cases} \frac{1}{h} \frac{3}{4} (1 - (\frac{x}{h})^2) & \text{if } -1 < \frac{x}{h} < 1 \\ 0 & \text{otherwise.} \end{cases}$$

Hence, for a given x_0 , values in a neighborhood determined by the choice of h ,

namely values $x \in (x_0 - h, x_0 + h)$ receive weight, with x closer to the boundary of the neighborhood receiving less weight than values of x closer to the center of the neighborhood. Whereas values of x within the neighborhood about x_0 receive weight, values of x excluded from the neighborhood receive zero weight. Hence, as can be seen in the weighted least squares problem (3.2), values of x outside of the neighborhood determined by the choice of h have absolutely no influence on predicting y_0 given x_0 . For an example of the weights associated to x near x_0 , see Figure 3.2, where $x_0 = 0$ and $h = 2$.

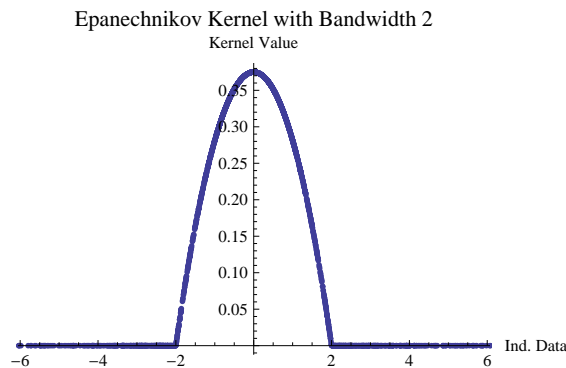


Figure 3.2: An illustrative example for the Epanechnikov Kernel with h set at 2.

3.3 Bandwidth Choice

The Epanechnikov kernel has one as of yet unexplained feature: the preferred choice of h in the context of our estimation problem. More specifically, Fan and Gibjels point out that local mean estimation depends crucially on two parameters, one of which is the degree of the polynomial. In our case, we have pointed out that we fit a global cubic polynomial. However, the bandwidth choice also crucially influences our estimation in as much as it controls the sensitivity of the local mean function to outliers. In the case of stock returns, the data are generally relatively spread out; markets experience returns deep in both tails. As such, we must remain attentive

the appropriate choice of h . Specifically, Fan and Gibjels provide a widely accepted “rule of thumb” bandwidth selection tool that provides the “best” asymptotic choice of h . This particular choice, the rule of thumb (ROT) bandwidth, was in the case of our estimation computed using *Mathematica* with code converted from the MATLAB code used to compute the rule of thumb bandwidths in the Bradley and Taqqu (2005) study [4].

Problematically, the ROT bandwidth is susceptible to the same criticisms as much of the earlier work on contagion in as much as the optimal bandwidth derived by Fan and Gibjels assumes that the structure of $Y|X = x$ is Gaussian. In our study, we assume that the joint return distribution is instead stable, i.e., exhibits heavy tails and skewness. Intuitively, heavy tailed data can be understood to be more spread out than normally distributed data. As such, we believe that the optimal h computed under the Fan and Gibjels model *undersmooths* the estimate, i.e., includes too little data in the estimation of y given x . In response to this concern, an investigation of our results was undergone with a variety of bandwidths, and we indeed found evidence that the data remained undersmoothed given the ROT bandwidth provided by Fan and Gibjels. In response, we reason that in order to include more data surrounding a given x_0 , it seems plausible that we should use the ROT bandwidth as a minimum requirement and scale it upwards by a some positive real number in order to achieve appropriate smoothness. After comparing results under multiple bandwidths, we chose to multiply h_{opt} by the scalar 3.5.

3.4 Fitting the Residual Structure to a Stable Model

As previously mentioned, in this study we wish to investigate the atypical association between the U.S. market and a given foreign market. As such, we remove the “mean” association between the markets by fitting the local mean function and extracting the residuals from that fit. Ultimately, we assume that the residual structure

is stable, i.e. $\epsilon(X_t) \sim \text{Sta}(\alpha, \beta, \gamma, \delta)$, and we derive our new definition of contagion by way of fitting a stable model to the set of residuals associated with the relationship between the US market and some foreign market using Conditional Maximum Likelihood Estimation.

3.5 Conditional Maximum Likelihood Estimation

In our study, as we have said, we will assume that

$$\epsilon(x) \sim \text{Sta}(\alpha(x), \beta(x), \gamma(x), \delta(x)), \quad (3.3)$$

where $\alpha(x), \beta(x), \gamma(x)$, and $\delta(x)$ will be estimated by a conditional maximum likelihood estimation method rather than the traditionally-used maximum likelihood estimation (MLE) method.

In ordinary MLE, the “likelihood” function is globally maximized to produce the “most likely” estimates for parameters which govern the distribution of the data. In particular, let $f(x; \vec{\Theta})$ represent the probability density function governing the data which is characterized by some vector of parameters $\vec{\Theta}$, and call the vector of observed data $\vec{X} = \{x_t\}_{t=1}^n$ such that each x_t is drawn at random from X , where X is governed by the density function f . Ultimately, we want to discover what values $\vec{\Theta} = \Theta_1, \Theta_2, \dots, \Theta_n$ are *most likely* to produce the data which we have drawn from the population. Hence, in the case of MLE, we want to maximize

$$L(\vec{X}; \vec{\Theta}) := f(x_1, x_1, \dots, x_n; \vec{\Theta}) = f(x_1; \vec{\Theta})f(x_2; \vec{\Theta})\dots f(x_n; \vec{\Theta}) \quad (3.4)$$

with respect to $\vec{\Theta}$. This maximization strategy will produce the desired estimates of $\vec{\Theta}$, assuming that the data are drawn at random from a population governed by f . For a full treatment of MLE, see Freund [9].

In our case, we want to investigate the values of α and β at a given point in the distribution of US stock returns, namely at a point deep in the left tail of the distribution and at a point at the median of the distribution. Using this strategy, we will be

able to compare tail heaviness and skewness between “loss” days and “average” days and hence develop a notion of contagion from one market to another by investigating the change in these parameters from the median (associated with “average” days) to the tail (associated with “loss” days) of the distribution. Unfortunately, the likelihood function (3.4) does not provide us with the tools necessary to produce estimates for α and β that are functions of x . If we have estimates $\hat{\alpha}$ and $\hat{\beta}$ that depend on x , where x is a design point located in the support of the independent data, we can then investigate the values of the parameters *conditioned* on a point x_L in the left tail and a point x_M at the median. Luckily, we have already been using a tool which, when incorporated in the likelihood function, can condition the estimates produced on a point x located in the distribution of the U.S. stock returns. So, we define the *conditional* maximum likelihood function as

$$\text{cl}(x, h; \vec{\epsilon}, \vec{X}; \vec{\Theta}) := \prod_{i=1}^N f(\epsilon_i; \vec{\Theta}) K_h(x - X_i), \quad (3.5)$$

where \vec{X} is the independent data (U.S. market returns), $\vec{\epsilon}$ is the vector of residuals extracted from the original regression, f is the density of ϵ , i.e. the stable density, $\vec{\Theta}$ is the parameters which govern the population of ϵ , i.e. $(\alpha, \beta, \gamma, \delta)$, and where K is the Epanechnikov kernel. Similarly to the case of the local mean function, the kernel gives weight in the likelihood function only to data within an h -neighborhood about a given x . In particular, this choice of x is left to be defined by the user of the functions $\hat{\alpha}$ and $\hat{\beta}$. Ultimately, we will be concerned with only two points in the support of the U.S. returns— $x_L = F^{-1}(.025)$ and $x_M = F^{-1}(.5)$, where F is the cumulative distribution function associated with the stable density.

3.6 Bootstrapping

The result of executing conditional MLE will produce functions for $\hat{\alpha}$ and $\hat{\beta}$ which depend on a given point x in the independent data set, the US market returns. For

the given set of data, we will evaluate $\hat{\alpha}$ and $\hat{\beta}$ at x_L and x_M , the loss tail and median of the data, respectively, and consequently this technique will produce *estimates* of the skewness and tail heaviness at the median x_M and loss tail x_L of the covariate market X . Finally, we will compute $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$. In as much as these are *estimates*, confidence intervals must be produced around these estimates if we desire to determine if these differences are significantly different from zero. In order to accomplish this goal, we will bootstrap the results using *Mathematica* using a permutation method. Specifically, we will abolish the relationship between the data and reconstructed at random, and the results of the estimation (the production of $\hat{\alpha}(x)$ and $\hat{\beta}(x)$ at x_M and x_L) will be re-executed many times. In other words, if we randomly permute the X and Y data 5000 times and recompute $\hat{\alpha}(x_M)$, $\hat{\alpha}(x_L)$, $\hat{\beta}(x_M)$, and $\hat{\beta}(x_L)$ each time, we will have a fairly certain idea regarding the intervals in which $\alpha(x_M)$, $\alpha(x_L)$, $\beta(x_M)$, and $\beta(x_L)$ lie, respectively. In other words, bootstrapping is a method whereby we can attempt to reproduce the cumulative density function F governing the distributions of $\hat{\alpha}(x)$ and $\hat{\beta}(x)$ if H_0 is true. Since we do not have any *a priori* knowledge of what the true cumulative densities might be, this non-parametric method is necessary if we wish to compute to test for the significance of our test statistic. In other words, as a result of this bootstrapping method, we can compute $\mathbb{P}(\hat{\theta}(x_M) - \hat{\theta}(x_L) < t)$ by

$$\frac{\text{Number of bootstraps less than } \hat{\theta}(x_M) - \hat{\theta}(x_L)}{\text{Total number of bootstraps}}$$

for $\hat{\beta}(x)$ and similarly for $\hat{\alpha}(x)$. Put differently, we can compute the probability that (assuming the null hypothesis is true) the statistics $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\beta(x_M) - \beta(x_L)$ would actually occur. If this probability is small, then we conclude that the null hypothesis is indeed not true and instead reject the null hypothesis in favor of some appropriately defined alternate hypothesis, in our case the hypothesis that contagion exists.

4

A New Definition of Contagion

Equipped with the tools of conditional MLE and the power of bootstrapping, we are ready to formally define contagion in terms of the stable model. Recall that in our case, the stable model is attractive because we can estimate functions for $\alpha(x)$ and $\beta(x)$, which allow us to generate two new notions of contagion: the contagion associated with the tail heaviness of $Y|X = x$ and the contagion associated with the skewness of $Y|X = x$. Explicitly stated, we will say that tail heaviness contagion exists when $\alpha(x_M) > \alpha(x_L)$, where $\alpha(x_M)$ is the conditional tail heaviness at the median of X , and $\alpha(x_L)$ is the conditional tail heaviness in the loss tail of X . In other words, when the tail of $\epsilon(x)$ is fatter given that x is large and negative, we know that the structure of Y has changed for those “loss” values of x . In short, a crisis in X leads to a greater probability that Y experiences an abnormally large loss. Additionally, we will say that skewness contagion from X to Y exists when $\beta(x_M) > \beta(x_L)$. Intuitively, this definition corresponds to the phenomenon large negative values in X will skew the data in Y leftward, introducing a greater probability that Y has a left-skewed return, which again fits our general notion of contagion. Thus, formally, we state our null and alternate hypotheses as

$$H_{\alpha,0}: \alpha(x_M) \leq \alpha(x_L).$$

$$H_{\alpha,1}: \alpha(x_M) > \alpha(x_L).$$

and

$$H_{\beta,0}: \beta(x_M) \leq \beta(x_L).$$

$$H_{\beta,1}: \beta(x_M) > \beta(x_L).$$

We will test these hypothesis hypotheses using the permutation based bootstrapping method detailed above.

4.1 Data

In order to remain congruent with measures of stock returns used in the literature, we will use data similar to that used in earlier studies [5]. Specifically, we will use the Morgan Stanley Capital International (MSCI) indices for the period from January 1980 to May 2002 to identify the presence (or lack thereof) of tail heaviness and/or skewness contagion between the United States and the following international equity markets:

- | | | |
|--------------|------------|------------------------|
| 1. Hong Kong | 5. Canada | 9. The Netherlands |
| 2. Japan | 6. France | 10. Switzerland |
| 3. Australia | 7. Germany | |
| 4. Belgium | 8. Italy | 11. The United Kingdom |

These data have been taken from Datastream, a financial services database, and they adjust for discrepancies in time of operation and currency accross markets. Additionally, we work with logarithmic return data.

4.2 Results

The remaining sections outline the results of our study on a country-by-country basis. First however, we present in Table 4.1 and Table 4.2 summaries of the following

sub-sections. As the tables convey, we identify the presence of tail heaviness contagion in *zero* cases out of the eleven countries tested. However, we *do* claim that skewness contagion exists in five out of eleven cases, most of which are located in Europe. We arrived at the following conclusions by executing 5000 bootstraps to construct the empirical cumulative density functions of $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ respectively by the process outlined above. If the p-value is less than .1, we reject the null hypothesis (that contagion does not exist) and in favor of the null hypothesis, and we claim that contagion exists between the U.S. and the respective dependent market.

Country	tail heaviness Contagion?	P-value
Hong Kong	No	.634
Japan	No	.659
Australia	No	.175
Belgium	No	.617
Canada	No	.160
France	No	.710
Germany	No	.562
Italy	No	.468
The Netherlands	No	.503
Switzerland	No	.108
The United Kingdom	No	.708

Table 4.1: A table of results for testing for tail heaviness contagion characterized by changes in $\hat{\alpha}(x)$ from the tail of the independent data to the mean.

It must be noted that the lack of existence of tail heaviness contagion between the U.S. market and *any* covariate market seems suspicious. This could be due to the

Country	Skewness Contagion?	P-value
Hong Kong	No	.152
Japan	No	.756
Australia	No	.248
Belgium	No	.473
Canada	Yes	.037
France	Yes	.094
Germany	Yes	.064
Italy	Yes	.062
The Netherlands	Yes	.024
Switzerland	No	.204
The United Kingdom	No	.288

Table 4.2: A table of results for testing for skewness contagion characterized by changes in $\hat{\beta}(x)$ from the tail of the independent data to the mean.

fact that in the sense in which we define contagion, tail heaviness contagion simply does not exist. In fact, we have observed evidence that the function $\hat{\alpha}(x)$ in most cases remains constant across nearly the entire range of the U.S. return data, such as in the Figure 4.1 below. However, the functions associated with the skewness in the data seem more intuitively reasonable, as we can see in Appendix A. Some concluding remarks about these issues are made after the presentation of the results specific to each market below. Additionally, while the functional estimates for δ and γ are uninteresting for the purposes of our study, they are also listed in Appendix A in a similar format to that of Figure 4.1.

We will now discuss in more detail the results for each country.

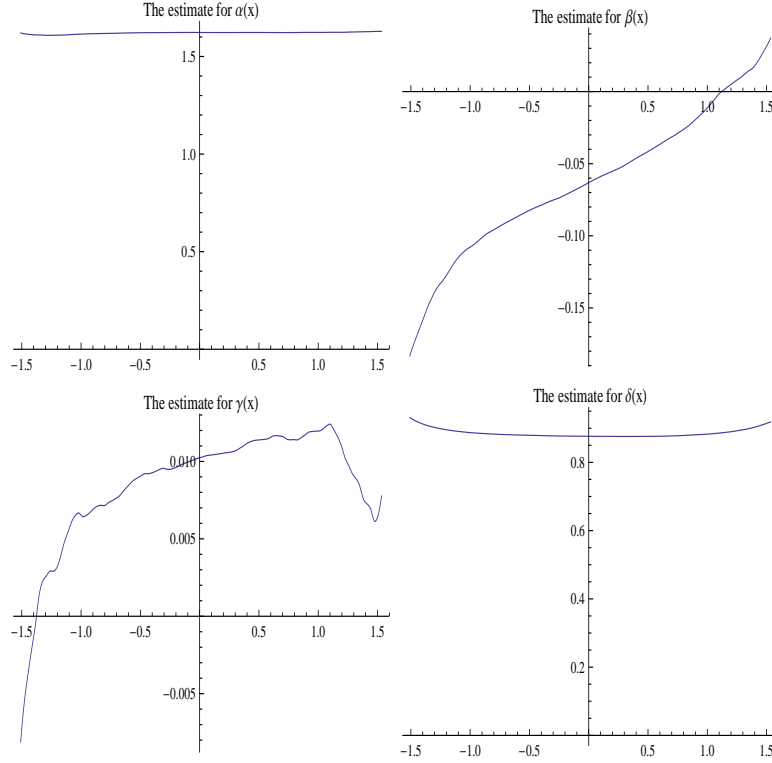


Figure 4.1: Graphs of $\hat{\alpha}$, $\hat{\beta}$, $\hat{\gamma}$, and $\hat{\delta}$ for the Hong Kong market.

4.2.1 Hong Kong

Country	$\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$	$\hat{\beta}(x_M) - \hat{\beta}(x_L)$
Hong Kong	-.02224778	.137369

Table 4.3: The observed values of $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ for Hong Kong.

In the case of the Hong Kong market, we concluded that contagion between it and the US market does not exist under our statistical measure in neither the case of the tail heaviness contagion associated with the parameter α nor in the case of the skewness contagion associated with the parameter β . In Table 4.3 are values for $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$. In Figure 4.2, it is easily observed that in

both cases, the test statistic listed in Table 4.3 is not in the tail of the empirical distribution constructed by our bootstrapping method. Hence, we calculate the high p-values listed in Tables 4.1 and 4.2. As is customary in statistics when faced with a high p-value, we fail to reject the null hypotheses, and we conclude that neither tail heaviness nor skewness contagion exists.

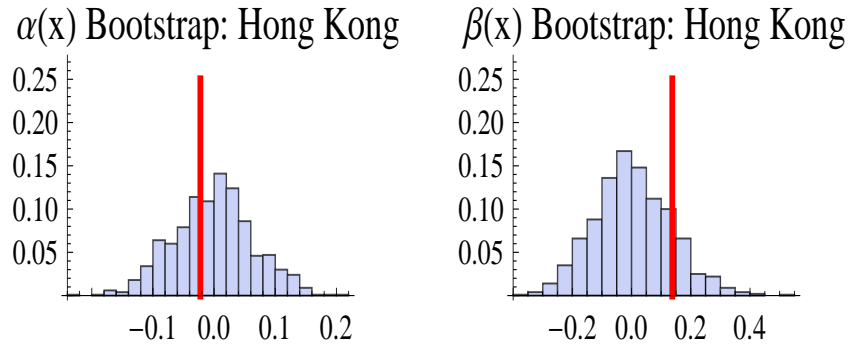


Figure 4.2: The empirical density when H_0 is true along with the test statistics for $\hat{\alpha}$ and $\hat{\beta}$ for Hong Kong.

4.2.2 Japan

Country	$\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$	$\hat{\beta}(x_M) - \hat{\beta}(x_L)$
Japan	-.0507058	-.17544

Table 4.4: The observed values of $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ for Japan.

In the case of the Japanese market, we again concluded that contagion does not exist between it and the US market under either statistic we have defined, which produced the test statistics in Table 4.4 as values for $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$. In Figure 4.3, it is easily observed that in both cases, the test statistic listed in Table 4.4 is not in the tail of the empirical distribution constructed by our bootstrapping method. Hence, we calculate the high p-values listed in Tables 4.1 and 4.2. As is customary in statistics when faced with a high p-value, we fail to reject the null hypotheses, and we conclude that neither tail heaviness nor skewness contagion exists.

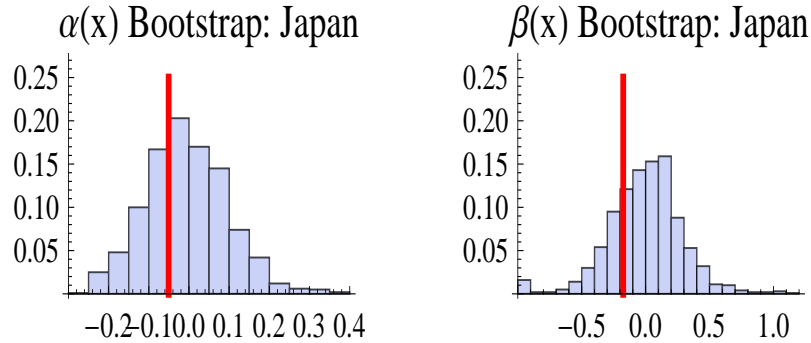


Figure 4.3: The empirical density when H_0 is true along with the test statistics for $\hat{\alpha}$ and $\hat{\beta}$ for Japan.

4.2.3 Australia

Country	$\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$	$\hat{\beta}(x_M) - \hat{\beta}(x_L)$
Australia	.0734785	.264237

Table 4.5: The observed values of $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ for Australia.

In the case of the Australian market, we again conclude that contagion does not exist between it and the US market under either statistic we have defined, which produced the test statistics in Table 4.5 as values for $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$. In Figure 4.4, it is easily observed that in both cases, the test statistic listed in Table 4.5 is not in the tail of the empirical distribution constructed by our bootstrapping method. Hence, we calculate the high p-values listed in Tables 4.1 and 4.2. Thus, we fail to reject the null hypotheses, and we conclude that neither tail heaviness nor skewness contagion exists.

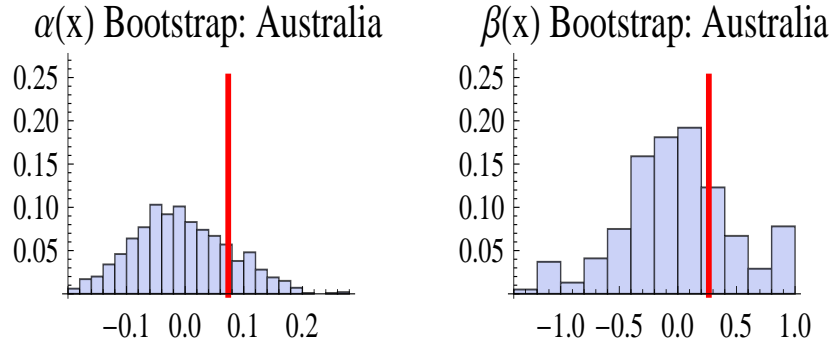


Figure 4.4: The empirical density when H_0 is true along with the test statistics for $\hat{\alpha}$ and $\hat{\beta}$ for Australia.

4.2.4 Belgium

Country	$\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$	$\hat{\beta}(x_M) - \hat{\beta}(x_L)$
Belgium	-.0222803	.0142234

Table 4.6: The observed values of $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ for Belgium.

In the case of the Belgian market, we again conclude that contagion does not exist between it and the US market under either statistic we have defined, which produced the test statistics in Table 4.6 as values for $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$. In Figure 4.5, it is easily observed that in both cases, the test statistic listed in Table 4.6 is not in the tail of the empirical distribution constructed by our bootstrapping method. Hence, we calculate the high p-values listed in Tables 4.1 and 4.2. Thus, as is customary in statistics when faced with a high p-value, we fail to reject the null hypotheses, and we conclude that neither tail heaviness nor skewness contagion exists.

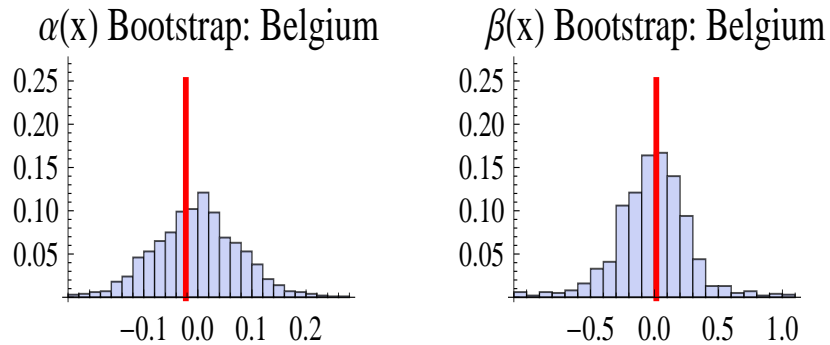


Figure 4.5: The empirical density when H_0 is true along with the test statistics for $\hat{\alpha}$ and $\hat{\beta}$ for Belgium.

4.2.5 Canada

Country	$\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$	$\hat{\beta}(x_M) - \hat{\beta}(x_L)$
Canada	.0754104	.60473

Table 4.7: The observed values of $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ for Canada.

In the case of the Canadian market, we again concluded that contagion between it and the US market does not exist in the case of the tail heaviness definition of contagion associated with the parameter α . However, we do see evidence that skewness contagion exists, which means that given that a crisis has occurred in the U.S. market, the probability of a crisis occurring in the Australian market increases, though not outsizedly. We come to this conclusion based on the relatively low p-value in Table 4.2 and the relatively large statistic for $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ seen in Table 4.7. As we can see in Figure 4.6, the p-value for the hypothesis test is far in the right tail, so we reject the null hypothesis in the case of skewness contagion, and we conclude that skewness contagion does indeed exist.

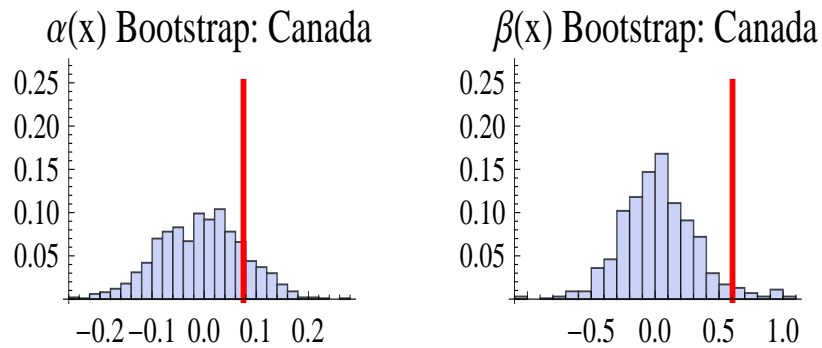


Figure 4.6: The empirical density when H_0 is true along with the test statistics for $\hat{\alpha}$ and $\hat{\beta}$ for Canada.

4.2.6 France

Country	$\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$	$\hat{\beta}(x_M) - \hat{\beta}(x_L)$
France	-.0400363	.406679

Table 4.8: The observed values of $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ for France.

In the case of the French market, we again concluded that contagion between it and the US market does not exist in the case of the tail heaviness definition of contagion associated with the parameter α . However, we do see evidence that skewness contagion exists, which means that given that a crisis has occurred in the U.S. market, the probability of a crisis occurring in the Australian market increases, though not outsizedly. We come to this conclusion based on the relatively low p-value in Table 4.2 and the relatively large statistic for $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ seen in Table 4.8. As we can see in Figure 4.7, the p-value for the hypothesis test is far in the right tail, so we reject the null hypothesis in the case of skewness contagion, and we conclude that skewness contagion does indeed exist.

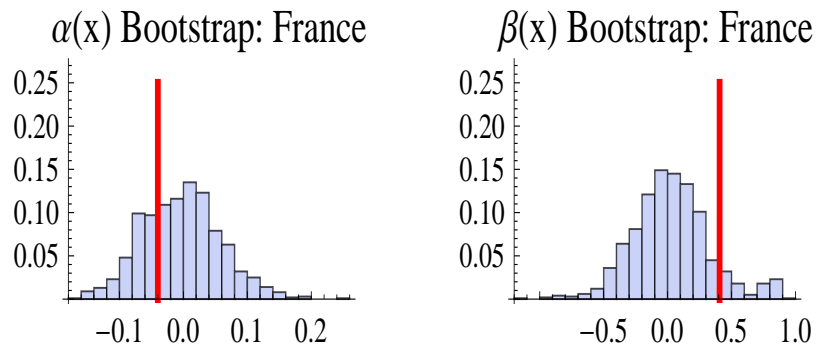


Figure 4.7: The empirical density when H_0 is true along with the test statistics for $\hat{\alpha}$ and $\hat{\beta}$ for France.

4.2.7 Germany

Country	$\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$	$\hat{\beta}(x_M) - \hat{\beta}(x_L)$
Germany	-.0305202	.907952

Table 4.9: The observed values of $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ for Germany.

In the case of the German market, we again concluded that contagion between it and the US market does not exist in the case of the tail heaviness definition of contagion associated with the parameter α . However, we do see evidence that skewness contagion exists, which means that given that a crisis has occurred in the U.S. market, the probability of a crisis occurring in the Australian market increases, though not outsizedly. We come to this conclusion based on the relatively low p-value in Table 4.2 and the relatively large statistic for $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ seen in Table 4.9. As we can see in Figure 4.8, the p-value for the hypothesis test is far in the right tail, so we reject the null hypothesis in the case of skewness contagion, and we conclude that skewness contagion does indeed exist.

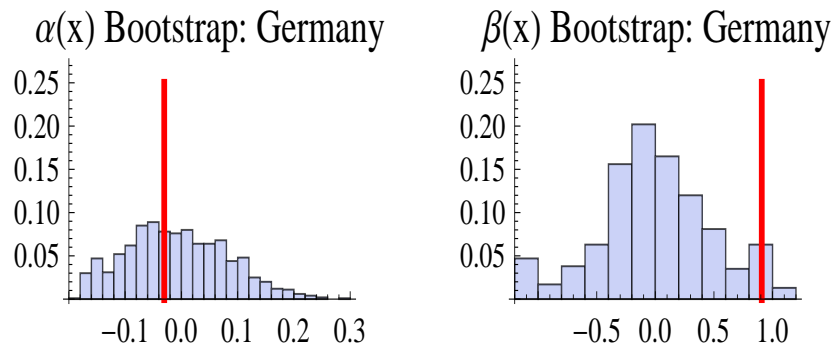


Figure 4.8: The empirical density when H_0 is true along with the test statistics for $\hat{\alpha}$ and $\hat{\beta}$ for Germany.

4.2.8 Italy

Country	$\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$	$\hat{\beta}(x_M) - \hat{\beta}(x_L)$
Italy	-.00105876	.446727

Table 4.10: The observed values of $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ for Italy.

In the case of the Italian market, we again concluded that contagion between it and the US market does not exist in the case of the tail heaviness definition of contagion associated with the parameter α . However, we do see evidence that skewness contagion exists, which means that given that a crisis has occurred in the U.S. market, the probability of a crisis occurring in the Australian market increases, though not outsizedly. We come to this conclusion based on the relatively low p-value in Table 4.2 and the relatively large statistic for $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ seen in Table 4.10. As we can see in Figure 4.9, the p-value for the hypothesis test is far in the right tail, so we reject the null hypothesis in the case of skewness contagion, and we conclude that skewness contagion does indeed exist.

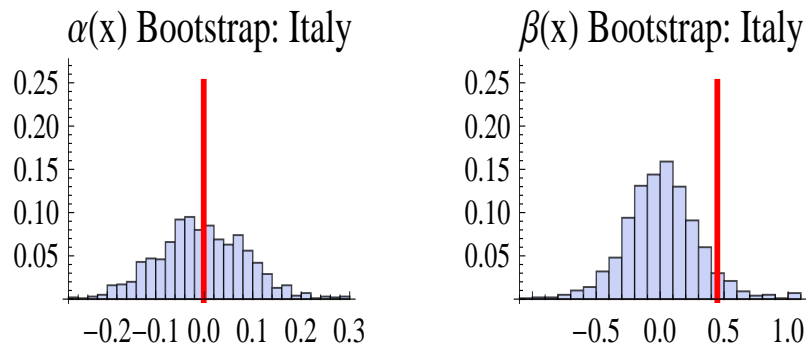


Figure 4.9: The empirical density when H_0 is true along with the test statistics for $\hat{\alpha}$ and $\hat{\beta}$ for Italy.

4.2.9 The Netherlands

Country	$\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$	$\hat{\beta}(x_M) - \hat{\beta}(x_L)$
The Netherlands	-.00888951	.711299

Table 4.11: The observed values of $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ for the Netherlands.

In the case of the Dutch market, we again concluded that contagion between it and the US market does not exist in the case of the tail heaviness definition of contagion associated with the parameter α . However, we do see evidence that skewness contagion exists, which means that given that a crisis has occurred in the U.S. market, the probability of a crisis occurring in the Australian market increases, though not outsizedly. We come to this conclusion based on the relatively low p-value in Table 4.2 and the relatively large statistic for $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ seen in Table 4.11. As we can see in Figure 4.10, the p-value for the hypothesis test is far in the right tail, so we reject the null hypothesis in the case of skewness contagion, and we conclude that skewness contagion does indeed exist.

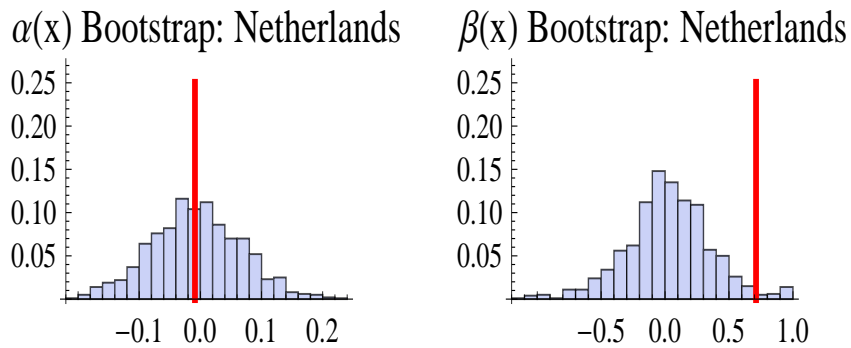


Figure 4.10: The empirical density when H_0 is true along with the test statistics for $\hat{\alpha}$ and $\hat{\beta}$ for the Netherlands.

4.2.10 Switzerland

Country	$\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$	$\hat{\beta}(x_M) - \hat{\beta}(x_L)$
Switzerland	.108323	.36502

Table 4.12: The observed values of $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ for Switzerland.

In the case of the Swiss market, we again concluded that contagion does not exist between it and the U.S. market under either statistic we have defined, which produced the test statistics in Table 4.12 as values for $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$. In Figure 4.11, it is easily observed that in both cases, the test statistic listed in Table 4.12 is not in the tail of the empirical distribution constructed by our bootstrapping method. Hence, we calculate the high p-values listed in Tables 4.1 and 4.2. As is customary in statistics when faced with a high p-value, we fail to reject the null hypotheses, and we conclude that neither tail heaviness nor skewness contagion exist.

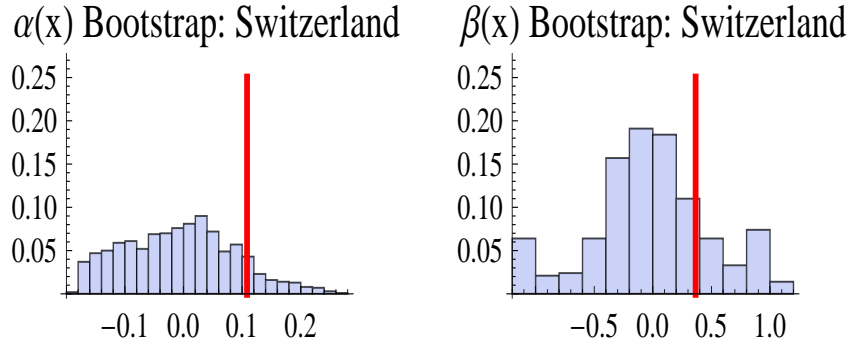


Figure 4.11: The empirical density when H_0 is true along with the test statistics for $\hat{\alpha}$ and $\hat{\beta}$ for Switzerland.

4.2.11 The United Kingdom

Country	$\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$	$\hat{\beta}(x_M) - \hat{\beta}(x_L)$
The United Kingdom	-.0267801	.133274

Table 4.13: The observed values of $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$ for the United Kingdom.

In the case of the British market, we again concluded that contagion does not exist between it and the U.S. market under either statistic we have defined, which produced the test statistics in Table 4.2.11 as values for $\hat{\alpha}(x_M) - \hat{\alpha}(x_L)$ and $\hat{\beta}(x_M) - \hat{\beta}(x_L)$. In Figure 4.12, it is easily observed that in both cases, the test statistic listed in Table 4.2.11 is not in the tail of the empirical distribution constructed by our bootstrapping method. Hence, we calculate the high p-values listed in Tables 4.1 and 4.2. As is customary in statistics when faced with a high p-value, we fail to reject the null hypotheses, and we conclude that neither tail heaviness nor skewness contagion exists.

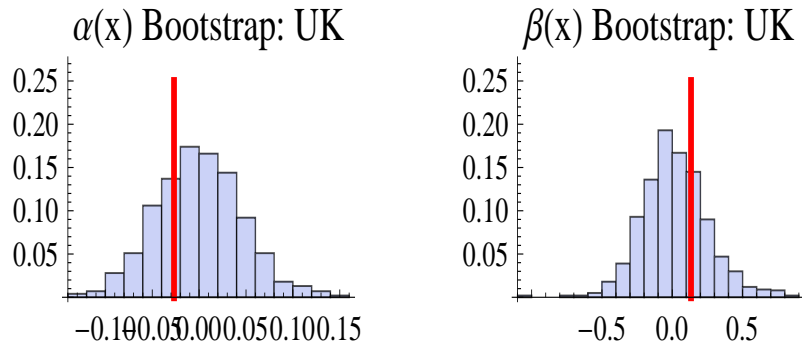


Figure 4.12: The empirical density when H_0 is true along with the test statistics for $\hat{\alpha}$ and $\hat{\beta}$ for the United Kingdom.

4.3 Conclusions

A natural question to ask of the results of a study such as this one concerns how they apply to “the real world.” For financial mathematicians, this study lays a foundation for using a stable model to quantify contagion between international equity markets. More problematically, it also supports the notion that, in some sense, that contagion, although theoretically intuitive, is difficult to define in practice. For example, our results in the case of tail heaviness contagion largely disagreed with those of Bradley and Taqqu. In the case of skewness contagion, our results and the result of Bradley and Taqqu were slightly more congruent, but certainly not identical. However, Bradley and Taqqu’s definition of contagion is a completely different notion, and relies on fundamentally different assumptions. In any case, while both attempts at defining contagion make sense intuitively, the fact that neither agree could indicate that contagion is simply quite difficult to define in practice.

It must be mentioned that additional research in the area of fitting a conditionally stable models to return data in order to identify contagion would be extremely complimentary to this study. In particular, a re-execution of this study with newer data would better our understanding of contagion in current markets, given that fundamental changes to world markets have occurred since 2002. In as much as the relationships between markets could have changed since the end of our data, it is dangerous to claim that contagion exists between the U.S. market and a covariate market based on a study that does not include the latest data. However, the fact that this study has been completed would make such a re-execution fairly easy given the appropriate return data.

Another area for potentially improved understanding of this methodology exists in the opportunity to discover the asymptotic behavior of $\hat{\alpha}$ and $\hat{\beta}$, either pointwise or in some other sense. In particular, if we could discover the distribution of the two statistics as the sample size goes to infinity, we could forgo the bootstrapping method used in this study and remove the fog of war from our estimation strategy. Because

bootstrapping is attractive by virtue of its non parametric nature, we do have some right to claim that it is the best method. However, knowing the asymptotics of $\hat{\alpha}$ and $\hat{\beta}$ would be both informative and ease the computationally expensive method that repeatedly fitting a conditionally stable model entails.

Finally, expanding this study to consider a wider range of markets might be attractive to some financial practitioners. For example, the relationship between the Chinese and U.S. markets is arguably a complicated one, and is hard to model because of the high level of regulation and market manipulation that occurs in the Chinese market. However, in as much as practitioners invest in the Chinese market, they would presumably like to have a better knowledge of how it is related to other markets so as to minimize their accidental exposure to risk. With data that properly quantifies the movement of the Chinese market in hand, a study on contagion between the U.S. and Chinese market, or any of the asian markets for that matter, could be an informative.

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Appendix A

A.1 Hong Kong

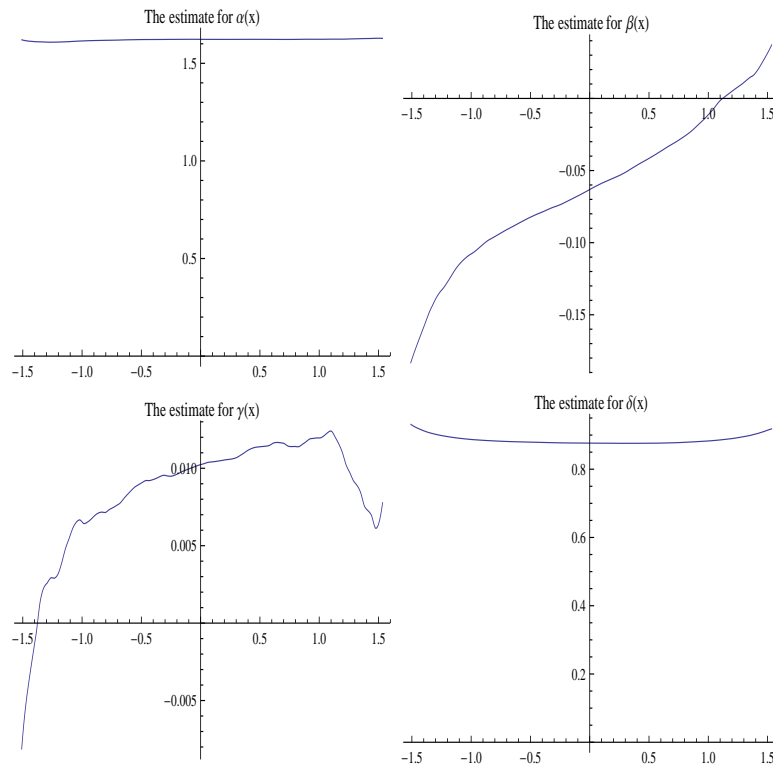


Figure A.1: Graphs of $\hat{\alpha}$, $\hat{\beta}$, $\hat{\gamma}$, and $\hat{\delta}$

A.2 Japan

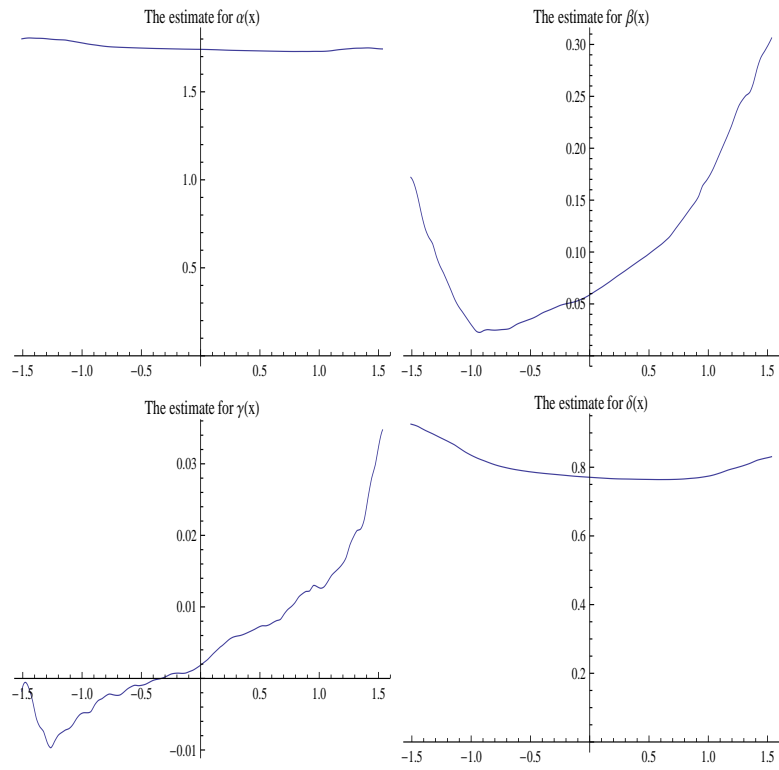


Figure A.2: Graphs of $\hat{\alpha}$, $\hat{\beta}$, $\hat{\gamma}$, and $\hat{\delta}$

A.3 Australia

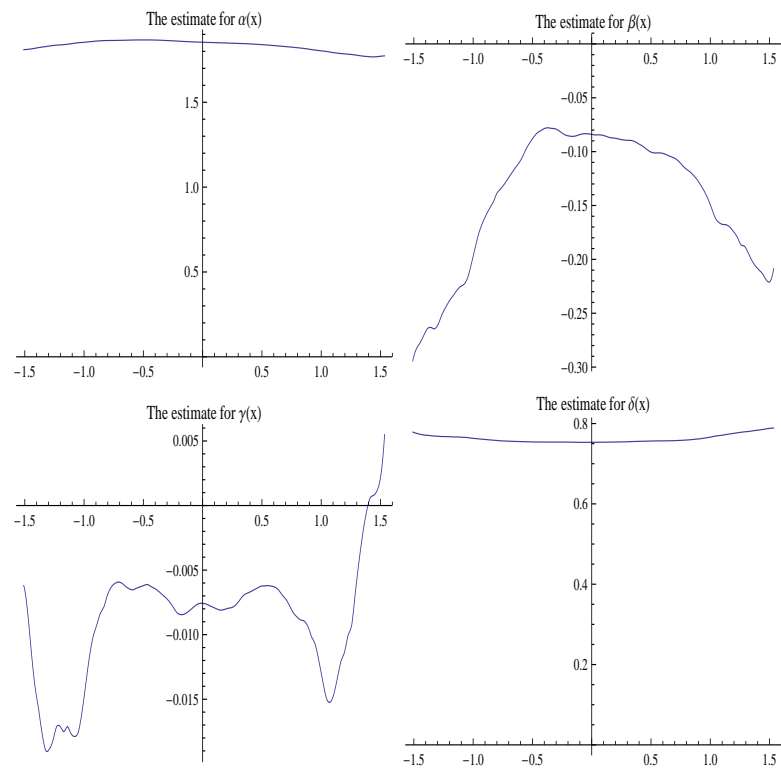


Figure A.3: Graphs of $\hat{\alpha}$, $\hat{\beta}$, $\hat{\gamma}$, and $\hat{\delta}$

A.4 Belgium

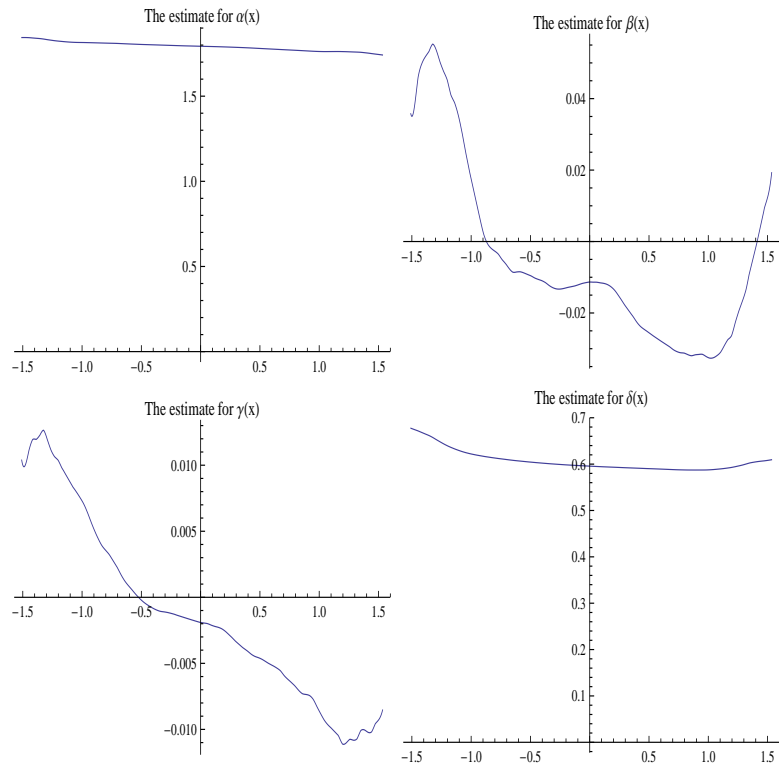


Figure A.4: Graphs of $\hat{\alpha}$, $\hat{\beta}$, $\hat{\gamma}$, and $\hat{\delta}$

A.5 Canada

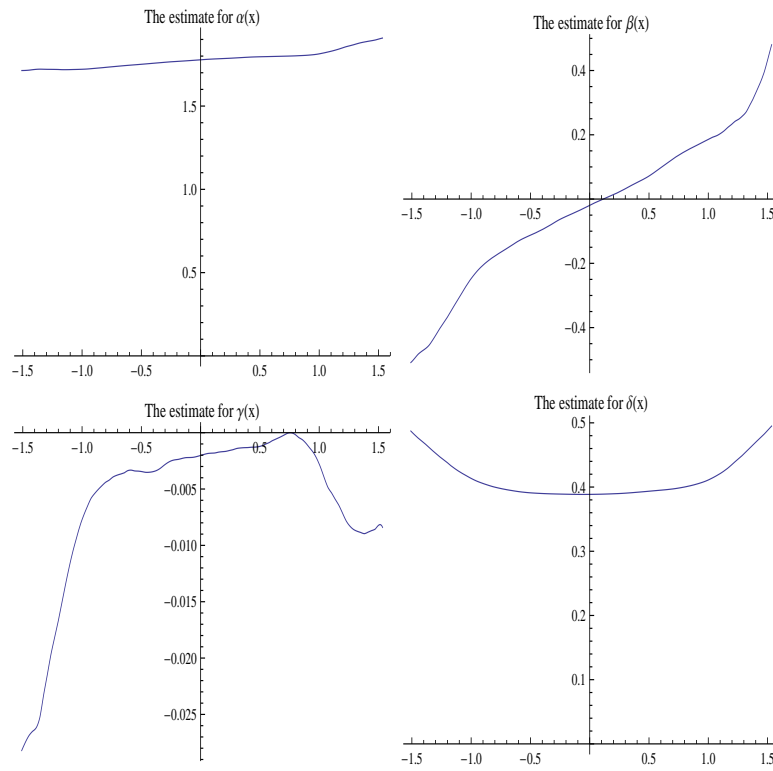


Figure A.5: Graphs of $\hat{\alpha}$, $\hat{\beta}$, $\hat{\gamma}$, and $\hat{\delta}$

A.6 France

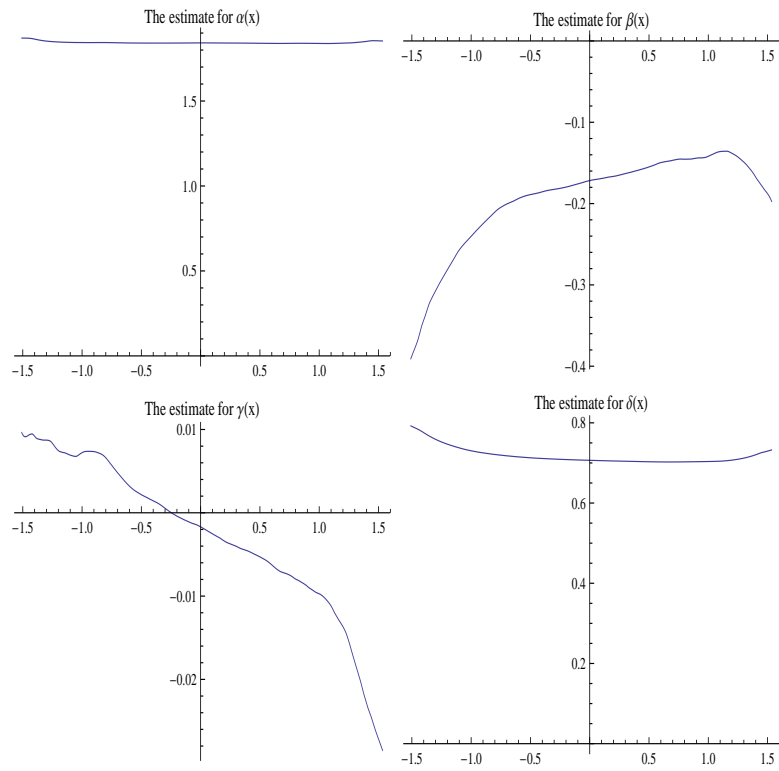


Figure A.6: Graphs of $\hat{\alpha}$, $\hat{\beta}$, $\hat{\gamma}$, and $\hat{\delta}$

A.7 Germany

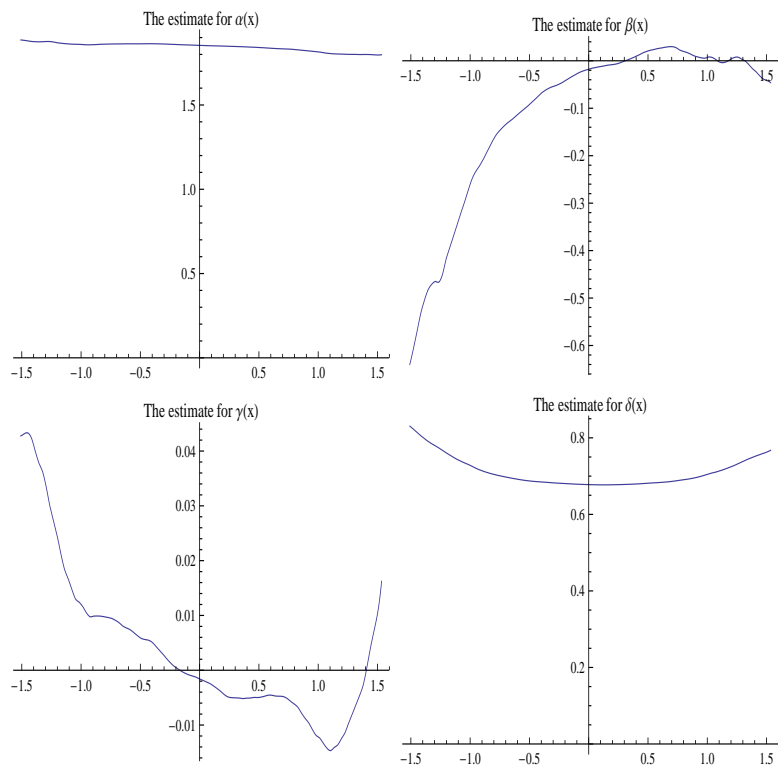


Figure A.7: Graphs of $\hat{\alpha}$, $\hat{\beta}$, $\hat{\gamma}$, and $\hat{\delta}$

A.8 Italy

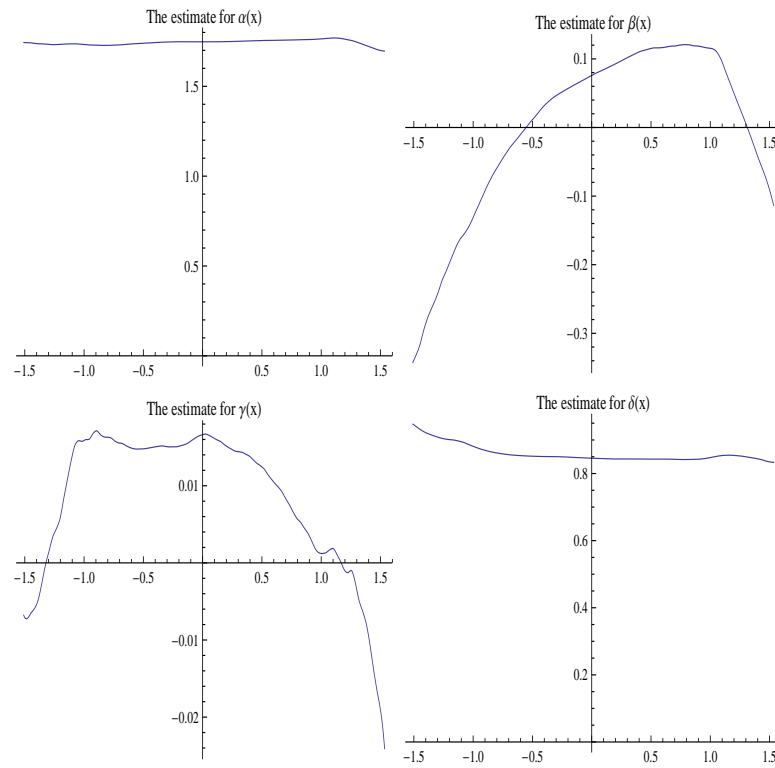


Figure A.8: Graphs of $\hat{\alpha}$, $\hat{\beta}$, $\hat{\gamma}$, and $\hat{\delta}$

A.9 The Netherlands

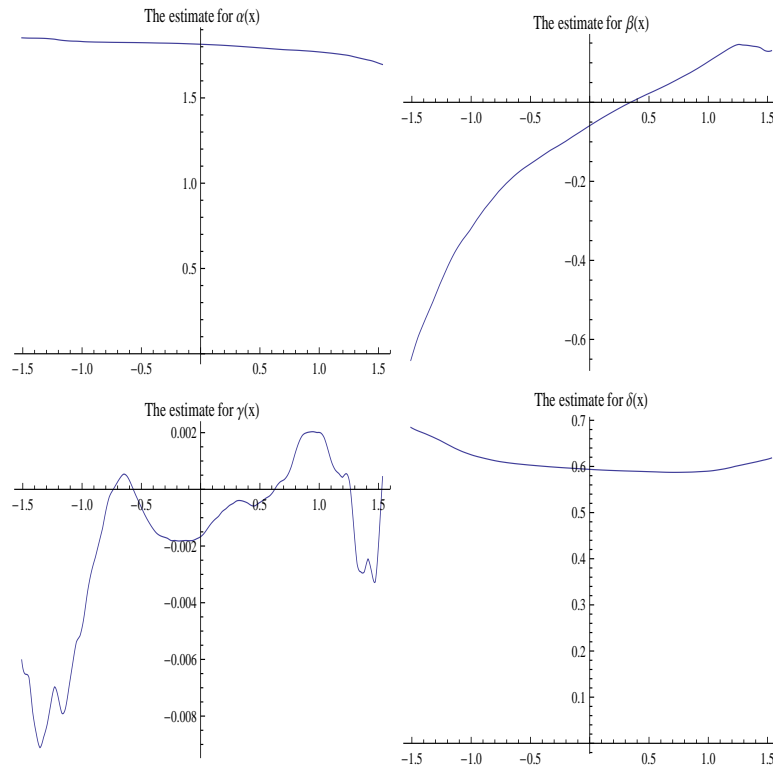


Figure A.9: Graphs of $\hat{\alpha}$, $\hat{\beta}$, $\hat{\gamma}$, and $\hat{\delta}$

A.10 Switzerland

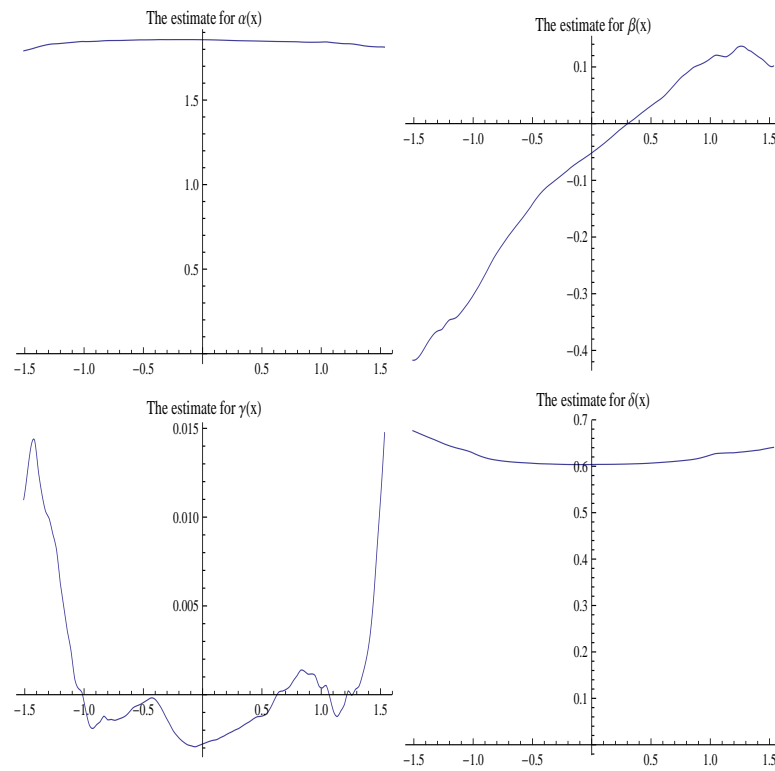


Figure A.10: Graphs of $\hat{\alpha}$, $\hat{\beta}$, $\hat{\gamma}$, and $\hat{\delta}$

A.11 The United Kingdom

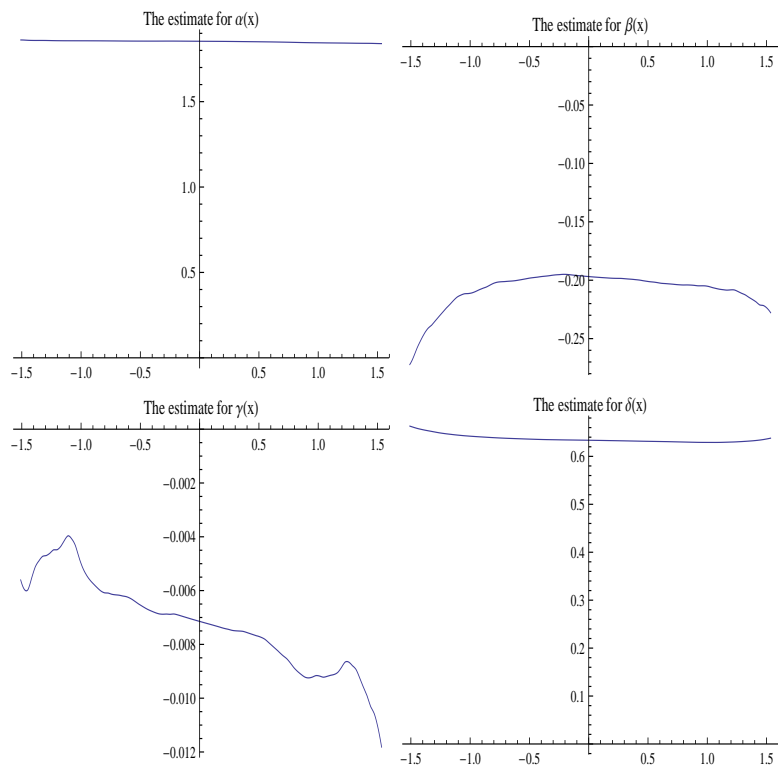


Figure A.11: Graphs of $\hat{\alpha}$, $\hat{\beta}$, $\hat{\gamma}$, and $\hat{\delta}$