Essays on Time Series Econometrics and Health Economics

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ABSTRACT

Essays on Time Series Econometrics and Health Economics

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This thesis contains two different topics that investigate issues in time series econometrics and applied health economics.

The first chapter (joint work with Nikolay Gospodinov) proposes a bootstrap unit root test in models with GARCH(1,1) errors and establishes its asymptotic validity under mild moment and distributional restrictions. While the proposed bootstrap test for a unit root shares the power enhancing properties of its asymptotic counterpart (Ling and Li, 2003), it corrects the substantial size distortions of the asymptotic test that occur for strongly heteroskedastic processes. The simulation results demonstrate the excellent finite-sample properties of the bootstrap unit root test for a wide range of GARCH specifications.

Both the second and third chapters study the obesity epidemic in Canada in recent years. Chapter 2 focuses on changes in obesity prevalence of Canadian adults, while chapter 3 focuses on their BMI shift pattern among different BMI categories.

By applying the quantile regression to three health surveys conducted from 1978 to 2004, chapter 2 explores the effects of some widely used demographic, geographic, and socioeconomic factors, as well as lifestyle, on the body mass index (BMI). The results of this study show that, changes of BMI that are attributable to these factors differs at different points of the BMI distribution, and the importance of any given factor changes over time. The epidemic of obesity in recent years is more closely

related to lifestyle and socioeconomic factors than demographic factors. By applying the Markov chain analysis to the BMI for individuals from the longitudinal National Population Healthy Survey (NPHS), chapter 3 explores how the pattern of weight of Canadian individuals has shifted among six categories of BMI during the 1994/95-2006/07 period. Two policy implications are suggested by this study: first, the effort to prevent the occurrence of new obese cases seems to be much more effective than the effort to reduce the number of existing obese people, and second, health policies for reducing obesity should focus more on the physically inactive people and provide incentives for them to be more physically active.

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Introduction

This thesis contains two different topics that investigate issues in time series econometrics and health economics. The first chapter focuses on a bootstrap unit root test in models with GARCH(1,1) errors, and both the second and third chapters study the obesity epidemic in Canada in recent years using econometric methods such as quantile regression and Markov Chain analysis.

Autoregressive time series with a unit root is a very important subject in the econometrics literature. Nelson and Plosser (1982) found that most U.S. macroeconomic time series could be characterized as a univariate unit root process. Moreover, financial time series, such as stock price and foreign exchange rates, also exhibit properties of unit root processes. While the Dickey-Fuller test (DF) for unit root is extremely popular in applied work, its low power and size distortion in the presence of conditional heteroskedasticity is widely documented.

A strand of literature that emerged recently (Ling and Li, 1998, 2003; Ling, Li and McAleer, 2003; Seo, 1999) derives the asymptotic distributions of unit root tests with GARCH errors and demonstrates the power gains of incorporating the GARCH structure into the testing procedure. The form of the asymptotic distribution of the unit root test in this case is a mixture of a Dickey-Fuller (DF) and a standard normal distribution with a mixing coefficient that depends on the degree of conditional heteroskedasticity.

Despite the non-trivial power gains of the unit root tests with GARCH errors (see, for example, Seo, 1999), the applied work with these tests has been very limited. There are several possible reasons why empirical researchers may find these tests not to be particularly appealing. First, they require nonlinear (maximum likelihood) estimation as opposed to OLS estimation for the Dickey-Fuller tests. More importantly, the asymptotic distribution depends on nuisance parameters which involves additional computation for obtaining critical values. Finally, as we show later in the paper (see also Seo, 1999), the tests based on asymptotic critical values suffer from substantial size distortions especially for some GARCH parameter configurations that are typically documented in empirical studies with financial time series data.

The first chapter proposes a bootstrap unit root test in models with GARCH(1,1) errors and establishes its asymptotic validity under mild moment and distributional restrictions. While the proposed bootstrap test for a unit root shares the power enhancing properties of its asymptotic counterpart, it offers a number of important advantages. In particular, the bootstrap procedure does not require explicit estimation of nuisance parameters that enter the distribution of the test statistic and corrects the substantial size distortions of the asymptotic test that occur for strongly heteroskedastic processes. The simulation results demonstrate the excellent finite-sample properties of the bootstrap unit root test for a wide range of GARCH specifications.

As in the U.S. and many other developed countries, obesity is becoming a severe social problem in Canada. The prevalence of obesity among adults aged 18 or older in Canada has been increasing significantly since the end of 1970s, from 13.8% in 1978/79 to 23% in 2004 (Tjepkema, 2006). Almost all studies of the impact of obesity reveal a

strong positive relationship between excess weight and diseases such as cardiovascular disease, diabetes and hypertension. Obesity is also believed to be responsible for the increased prevalence of psychological disorders, such as depression (Wadden et al., 2002). As a person's weight jumps to a high level of obesity, the risk of having these diseases increases dramatically (Allison et al., 1999; Engeland et al., 2003; Flegal et al., 2005). Sturm (2002) states that obesity outranks both smoking and drinking in its deleterious effects on health and health costs. He argues that obesity has roughly the same association with chronic health conditions as does twenty years of aging, and this greatly exceeds the associations of smoking or problem drinking. In Canada, the total direct medical cost attributable to the obesity was estimated over $1.8 billion in 1997. It corresponded to 2.4% of the total health care expenditures for all diseases in Canada in 1997 (Birmingham et al., 1999). If the indirect cost of obesity such as production loss due to obesity is included, the above number is even higher. In the U.S., the obesity-related conditions in 2008 account for 9.1 percent of all medical spending, up from 6.5 percent in 1998. During that time, the medical costs of obesity almost doubled and have risen from $ 78.5 billion to $147 billion (Finkelstein et al., 2009).

The second chapter explores the influences of some widely used demographic, geographic, and socioeconomic factors, as well as lifestyle, on the body mass index (BMI). By applying the quantile regression to three health surveys conducted from 1978 to 2004, a period in which the prevalence of obesity in Canada was stable at first and almost doubled thereafter, this study attempts to detect two kinds of changes: changes of BMI that are attributable to factors such as age, resident region, physical

activity and family income at different points of the BMI distribution, and changes in the importance of these factors over time. The result of the study shows that, the influence of factors on BMI differs at different points in the BMI distribution; moreover, the importance of any given factor changes over time. The epidemic of obesity in recent years is more closely related to lifestyle and socioeconomic factors than demographic factors. During this period, the role of education has shifted from the weakest factor to the strongest factor affecting BMI. This in turn, implies that the relationship between the capacity to avoid obesity and education is strong. On the other hand, working status is the least important factor related to the BMI for both men and women. This not only indicates the dominance of sedentary work and the continually decreasing expenditure of energy in the workplace, but also reveals that physical activity is the main way for people to expend energy. The fact that age becomes less important at higher quantiles of the BMI distribution suggests that a change in the age structure of Canadians is not contributing much to the obesity epidemic in Canada.

Movements among different BMI categories could provide very important information about the obesity epidemic and the shift patterns of different groups. This information is useful for health care, health policies and other health related issues. For example, since each category of the BMI reflects a different level of health risk and movements among different BMI categories imply changes in health risk. The information of movements among different BMI categories could be used to forecast the prevalence of some diseases such as cardiovascular disease, hypertension and diabetes II that mainly resulted from obesity, as well as the demand for health care

and other medical resources related to theses diseases. More importantly, if the shift patterns of different groups are obtained, more pertinent and effective policies that prevent obesity could be designed.

Chapter 3 studies how the pattern of weight of Canadian individuals has shifted among six categories of BMI during the 1994/95-2006/07 period, and seeks to determine the relationship between this pattern and the obesity epidemic over the last twelve years. By applying the Markov chain analysis to the BMI for individuals from the longitudinal National Population Healthy Survey (NPHS), the BMI shift patterns of Canadian adults is identified and estimated. The results reveal that: (i) although men and women differ in the BMI shift pattern, their BMI shift patterns throughout the period exhibit a positive trend, thus confirming that the prevalence of obesity in Canada is increasing; (ii) the BMI shifts are not stationary for both men and women; however, the BMI shifts for physically moderately active men and women are stationary. Two policy recommendations emerge naturally from the results of this study. First, the findings suggest that the effort to prevent the occurrence of new obese cases seems to be much more effective than the effort to reduce the number of existing obese people. Second, since people who are inactive in physical activity account for the biggest portion of Canadian population and their obesity prevalence is much higher than that of the physically active and moderate groups, health policies for reducing obesity should focus more on the inactive people and provide incentives for them to be more physically active. Using the estimated BMI shifts for physically

moderately active men and women, it turns out that if all inactive people are encouraged to become moderately active, the obesity prevalence in Canada can be confined below 25% in the long run.

CHAPTER 1

Bootstrap Unit Root Tests in Models with GARCH(1,1) Errors

(joint with Nikolay Gospodinov)

1.1. Introduction

The simultaneous presence of high persistence and conditional heteroskedasticity is a common characteristic of many economic time series. The stark differences between the long-run behavior and implications of nonstationary and stationary processes led to the development of a large class of unit root tests with good size and power properties. While the limiting theory for possibly unit root processes has been established under fairly general conditions, including some types of time-varying volatility, the explicit modeling of the higher-order dynamics is often expected to improve the efficiency of the conditional mean estimates and the power of the tests. For instance, a strand of literature that emerged recently (Ling and Li, 1998, 2003; Ling, Li and McAleer, 2003; Seo, 1999) derives the asymptotic distributions of unit root tests with GARCH errors and demonstrates the power gains of incorporating the GARCH structure into the testing procedure. The form of the asymptotic distribution of the unit root test in this case is a mixture of a Dickey-Fuller (DF) and a standard normal distribution with a mixing coefficient that depends on the degree

of conditional heteroskedasticity. As the degree of conditional heteroskedasticity increases (i.e., the sum of the GARCH coefficients approaches one), the standard normal distribution carries more weight and the corresponding smaller critical values give rise to a more powerful testing procedure. Note that the Dickey-Fuller distribution is still valid in the presence of GARCH errors but it is conservative and provides an upper bound for the critical values.

Despite the non-trivial power gains of the unit root tests with GARCH errors (see, for example, Seo, 1999), the applied work with these tests has been very limited. There are several possible reasons why empirical researchers may find these tests not to be particularly appealing. First, they require nonlinear (maximum likelihood) estimation as opposed to OLS estimation for the Dickey-Fuller tests. More importantly, the asymptotic distribution depends on nuisance parameters which involves additional computation for obtaining critical values. Finally, as we show later in the paper (see also Seo, 1999), the tests based on asymptotic critical values suffer from substantial size distortions especially for some GARCH parameter configurations that are typically documented in empirical studies with financial time series data.

In this chapter we propose a bootstrap method for approximating the finite-sample distributions of unit root tests with GARCH(1,1) errors and establish its asymptotic validity. We extend the results of Basawa *et al.* (1989, 1991), Ferretti and Romo (1996), Heimann and Kreiss (1996) and Park (2003), among others, to unit root models with conditional heteroskedasticity estimated by maximum likelihood (ML). The implementation of the proposed bootstrap procedure is straightforward and is valid under some fairly weak conditions. In particular, we follow Ling and Li (2003) and

derive the consistency of the bootstrap distribution assuming finite second moments and symmetry of the errors. This allows for highly persistent GARCH specifications (with sum of the GARCH parameters arbitrarily close to one) that are commonly estimated in the empirical finance literature. Some related bootstrap results are derived in Gospodinov (2008) in the context of testing for nonlinearity in models with a unit root and GARCH errors.

The finite-sample results demonstrate the excellent size and power properties of the proposed bootstrap test. While the tests based on asymptotic critical values tend to overreject (in some situations, up to 40-50% at 5% significance level), the bootstrap test is always very close to its nominal size regardless of the degree of conditional heteroskedasticity. Furthermore, the power of the bootstrap test that incorporates the GARCH structure of the model exceeds the size-adjusted power of the standard DF test by a substantial margin when the conditional heteroskedasticity is strong.

The properties of the proposed bootstrap test prove to be of great practical importance for identifying the mean reverting behavior in processes with GARCH structure. In our empirical analysis of several U.S. interest rate processes, we show that the DF test does not provide any evidence against the null of a unit root which has important implications about the long-run properties of the data. In contrast, the bootstrap DF-GARCH test tends to reject convincingly the unit root hypothesis due to its superior power properties. This lends support to the mean reverting specification as an underlying process for interest rate dynamics in many economic and finance models.

The rest of the chapter is organized as follows. The main model and notation are introduced in Section 2. Section 3 describes the proposed bootstrap procedure and derives its asymptotic validity. Section 4 presents Monte Carlo simulation experiment that assesses the finite-sample performance of the asymptotic and bootstrap tests. Section 5 concludes. The proofs of all results in the paper are relegated to the Appendix.

1.2. Model and Notation

Consider the first-order AR process with GARCH(1,1) errors

$$
\begin{aligned}
z_t &= d_t + y_t \\
y_t &= \phi y_{t-1} + \varepsilon_t \\
\varepsilon_t &= \sqrt{h_t}\eta_t \\
h_t &= \omega + \alpha\varepsilon_{t-1}^2 + \beta h_{t-1},
\end{aligned}
\tag{1.1}
$$

where $\phi = 1$, d_t is a deterministic component and $\eta_t \sim iid(0,1)$. This model can be generalized to an AR(p) model $A(L)(1-\phi L)y_t$ with a $(p-1)$ lag polynomial $A(L)$ with roots that lie outside the unit circle and higher-order GARCH processes. For simplicity, we present the results for the first-order model (1.1) but the limiting representations and the bootstrap procedure can be extended in a straightforward manner (but with more cumbersome notation) to higher-order processes.

Let $\delta = (\omega, \alpha, \beta)$ denote the vector of the unknown GARCH parameters. The parameter of interest is ϕ and the estimation is performed on the raw, demeaned or detrended data depending on whether $d_t = 0$, $d_t = \mu_0$ or $d_t = \mu_0 + \mu_1 t$, respectively.

The Gaussian quasi-likelihood function of this model is given by

$$L_T(\phi, \delta) = \frac{1}{T}\sum_{t=1}^{T} l_t(\phi, \delta), \tag{1.2}$$

where $l_t(\phi, \delta) = -\frac{1}{2}\ln h_t - \frac{1}{2}\frac{\varepsilon_t^2}{h_t}$. We follow Ling and Li (2003) and assume that the following conditions are satisfied.

ASSUMPTION 1 *Assume that*

(a) $\eta_t \sim iid(0, 1)$, $E(\eta_t^3) = 0$, $E(\eta_t^4) = \kappa < \infty$ *for all* t;

(b) $\Psi = \{(\omega, \alpha, \beta) : 0 < \omega_l \leq \omega \leq \omega_u, 0 < \alpha_l \leq \alpha \leq \alpha_u, 0 < \beta_l \leq \beta \leq \beta_u, \alpha + \beta < 1\}$;

(c) $y_0 = 0$ *and* h_0 *is initialized from its invariant measure.*

Assumption 1 imposes some very weak moment and distributional conditions on the error term. The standardized errors are assumed to be symmetric *iid* random variables with a finite fourth moment. The assumed symmetric distribution of η_t may appear restrictive but this allows us to weaken the moment requirements on the error term ε_t (see Ling and Li, 2003). In particular, the limiting results and the validity of the bootstrap procedure are derived assuming the existence of finite second moment of ε_t which is satisfied under fairly general conditions on the GARCH parameters. More specifically, the conditions in part (b) ensure that $E(\varepsilon_t^2) < \infty$ and the processes $\{h_t\}$ and $\{\varepsilon_t\}$ are strictly stationary, ergodic and β-mixing with exponential decay (Carrasco and Chen, 2002; Francq and Zakoïan, 2006) and allow for strong conditional heteroskedasticity that is typically present in financial data. Part (c) specifies the initialization of the conditional mean and variance functions.

Assuming y_0 to be fixed at a different value than zero or to be $o_p(T^{1/2})$ does not affect the limiting results derived below. Similarly, the asymptotic distributions are invariant to the assumption on the initial condition of h (Lee and Hansen, 1994; Ling and Li, 2003).

By the block diagonality of the Hessian matrix (Bollerslev, 1986; Ling, Li and McAleer, 2003), the conditional mean and variance parameters can be estimated separately without any efficiency loss. Let $\widehat{\phi}_{LS} = (\sum_{t=1}^{T} y_{t-1}^2)^{-1}(\sum_{t=1}^{T} y_t y_{t-1})$ denote the OLS estimator of ϕ and note that $T(\widehat{\phi}_{LS} - 1) = O_p(1)$ under Assumption 1. The parameter vector δ can be estimated from the OLS residuals $\widehat{\varepsilon}_t = y_t - \widehat{\phi}_{LS} y_{t-1}$ and the corresponding estimates $\widehat{\delta}$ are asymptotically equivalent to the estimates obtained from the true ε_t. Then, for some preliminary T-consistent estimator $\widetilde{\phi}$, the one-step QMLE estimator of ϕ is given by

$$\widehat{\phi}_{ML} = \widetilde{\phi} - \left[\sum_{t=1}^{T} \frac{\partial^2 l_t\left(\phi, \widehat{\delta}\right)}{\partial \phi^2}\right]_{\phi=\widetilde{\phi}}^{-1} \left[\sum_{t=1}^{T} \frac{\partial l_t\left(\phi, \widehat{\delta}\right)}{\partial \phi}\right]_{\phi=\widetilde{\phi}}$$

and (Ling and Li, 2003)

$$T(\widehat{\phi}_{ML} - 1) = -\left[\frac{1}{T^2}\sum_{t=1}^{T} \frac{\partial^2 l_t\left(\phi, \delta\right)}{\partial \phi^2}\right]_{\phi=1}^{-1} \left[\frac{1}{T}\sum_{t=1}^{T} \frac{\partial l_t\left(\phi, \delta\right)}{\partial \phi}\right]_{\phi=1} + o_p(1).$$

The OLS estimator $\widehat{\phi}_{LS}$ can be used as an initial preliminary estimator. Then, the iterative estimator that updates the estimates of $\widehat{\delta}$ and $\widehat{\phi}_{ML}$ until convergence is asymptotically equivalent to the full MLE.

Let

$$t_{\phi_{LS}=1} = (\sum_{t=1}^{T} y_{t-1}^2)^{1/2}(T^{-1}\sum_{t=1}^{T} \widehat{\varepsilon}_t^2)^{-1}(\widehat{\phi}_{LS} - 1)$$

and

$$t_{\phi_{ML}=1} = \left[-\sum_{t=1}^{T} \frac{\partial^2 l_t(\phi, \delta)}{\partial \phi^2} \right]^{1/2}_{\phi=\widehat{\phi}_{ML}, \delta=\widehat{\delta}} (\widehat{\phi}_{ML} - 1)$$

be the t-statistics of $H_0 : \phi = 1$ for the OLS and ML estimators, respectively. Let also $\Rightarrow$ signify weak convergence, $D[0,1]$ denote the space of real valued functions defined on the interval [0,1] that are right-continuous at each point in [0,1] and have finite left limits, and $B_1(r)$ be a standard Brownian motion on $D[0,1]$. The following lemma is a restatement of some results in Ling and Li (2003) and Seo (1999).

LEMMA 1 *Suppose that $\phi = 1$ and Assumption 1 holds. Then, as $T \to \infty$*

$$t_{\phi_{LS}=1} \Rightarrow \frac{\int_0^1 B_1(r) dB_1(r)}{\left(\int_0^1 B_1^2(r) dr \right)^{1/2}}, \tag{1.3}$$

$$t_{\phi_{ML}=1} \Rightarrow \sqrt{\frac{K}{F}} \left[\rho \frac{\int_0^1 B_1(r) dB_1(r)}{\left(\int_0^1 B_1^2(r) dr \right)^{1/2}} + \sqrt{1-\rho^2}\xi \right], \tag{1.4}$$

where $\rho = 1/\sigma\sqrt{K}$, $E(h_t) = \sigma^2$, $K = E(1/h_t) + (\kappa - 1)\alpha^2 \sum_{k=1}^{\infty} \beta^{2(k-1)} E(\varepsilon_{t-k}^2 / h_t^2)$, $F = E(1/h_t) + 2\alpha^2 \sum_{k=1}^{\infty} \beta^{2(k-1)} E(\varepsilon_{t-k}^2 / h_t^2)$ and ξ is a standard normal random variable distributed independently of $B_1(r)$.

PROOF See Ling and Li (2003) and Seo (1999).

The results in Lemma 1 are presented for the case with no deterministic terms, i.e. $d_t = 0$. The limiting representations for $d_t = \mu_0$ and $d_t = \mu_0 + \mu_1 t$ can be obtained

by replacing $B_1(r)$ in (1.3) and (1.4) by its demeaned version $B_1(r) - \int_0^1 B_1(s)ds$ and its detrended version $B_1(r) - \int_0^1 (4-6s)B_1(s)ds - r\int_0^1 (12s-6)B_1(s)ds$, respectively.

Several interesting results emerge from the limiting representations in Lemma 1. The asymptotic distribution of $t_{\phi_{ML}=1}$ is a scaled mixture of a Dickey-Fuller and a standard normal distribution with a mixing coefficient that depends on the degree of conditional heteroskedasticity and non-normality of the errors. In the case of normally distributed errors ($K = F$), the Dickey-Fuller distribution provides an upper bound for the critical values of $t_{\phi_{ML}=1}$. As the degree of conditional heteroskedasticity increases,[1] more weight is assigned to the standard normal distribution and the corresponding smaller critical values increase the power of the test.

Another version of the test standardizes $(\widehat{\phi}_{ML} - 1)$ with the robust variance covariance matrix (Bollerslev and Wooldridge, 1992)

$$\left[-\sum_{t=1}^{T}\frac{\partial^2 l_t(\phi,\delta)}{\partial\phi^2}\right]^{-1}\left[-\sum_{t=1}^{T}\left(\frac{\partial l_t(\phi,\delta)}{\partial\phi}\right)^2\right]\left[-\sum_{t=1}^{T}\frac{\partial^2 l_t(\phi,\delta)}{\partial\phi^2}\right]^{-1},$$

evaluated at the ML estimates of ϕ and δ, whose limiting distribution is given by $\rho\frac{\int_0^1 B_1(r)dB_1(r)}{\left(\int_0^1 B_1^2(r)dr\right)^{1/2}} + \sqrt{1-\rho^2}\xi$. This test is expected to have more robust size properties with possibly non-normally distributed errors although at the cost of moderate power losses for Gaussian errors.

Despite its potential for non-trivial power improvements, the test in (1.4) has the unappealing property that its asymptotic distribution is non-pivotal and depends on nuisance parameters. In principle, one could tabulate critical values for the test

[1] Boswijk (2001) derives an approximate expression of ρ in terms of the GARCH parameters as $\rho \approx \sqrt{\frac{(1-\alpha-\beta)(1-\beta^2)}{(1-\alpha-\beta+\alpha^2)(1-\beta^2+2\alpha^2)}}$. Then, it is easy to see that high persistence in the conditional variance ($\alpha+\beta$ near one) is typically associated with low values of ρ.

$t_{\phi_{ML}=1}\sqrt{\widehat{F}/\widehat{K}}$ on a grid of values for ρ (Seo, 1999), where the nuisance parameters are estimated from the data, although this makes the testing procedure somewhat cumbersome. More importantly, the nuisance parameters involve infinite sums and estimates of α, β, κ and h that enter in a highly nonlinear fashion which could impair the precision with which these quantities are computed. As we demonstrate below, this may lead to severe size distortions of the tests even for very large sample sizes. The bootstrap method that we propose in this paper proves to be very useful for approximating the finite-sample distribution of $t_{\phi_{ML}=1}$ as it avoids the explicit calculation of the nuisance parameters. In addition to the substantially improved size properties of the unit root test, the straightforward implementation of the bootstrap offers practical advantages and can be easily extended to processes that accommodate more general seral correlation and conditional heteroskedasticity structure.

1.3. Bootstrap Approximation

In this section, we propose a bootstrap method for approximating the finite-sample distribution of the unit root test $t_{\phi_{ML}=1}$. We start by discussing the bootstrap procedures based on resampling the symmetrized residuals and generating repeated samples under the null of a unit root. In proving the asymptotic validity of this bootstrap approximation, we first verify if the bootstrap samples satisfy the conditions of Assumption 1 and if the effect of the initial conditions is asymptotically negligible. Then, we develop a bootstrap invariance principle with conditionally heteroskedastic errors and establish the weak convergence of the bootstrap statistic to the limiting distribution in Lemma 1.

1.3.1. Description of Bootstrap Procedure

Let $\{z_1, z_2, ..., z_T\}$ be a sequence of T observations generated by model (1.1). Suppose that the deterministic component is removed by an OLS regression of $\{z_t\}$ on a constant or on a constant and a linear trend and let $\{y_1, y_2, ..., y_T\}$ denote the residuals from these regressions. As argued above, the conditional mean and variance parameters of (1.1) can be estimated separately. Let $\widehat{\delta} = \left(\widehat{\omega}, \widehat{\alpha}, \widehat{\beta}\right)'$ denote the ML estimates of the GARCH parameters, $\{\widehat{h}_t\}$ be the conditional variance computed recursively from these estimates for some initial value h_0 and $\widehat{\phi}_{ML}$ denote the one-step or iterated MLE of ϕ introduced in the previous section.

Define the residuals $\widetilde{\varepsilon}_t = y_t - \widehat{\phi}_{ML} y_{t-1}$. While these residuals could also be constructed imposing the null of a unit root ($\phi = 1$), we follow Paparoditis and Politis (2003) and compute the residuals using the MLE of ϕ which helps to retain the important characteristics of the data and improve the power of the unit root test. We then construct the recentered standardized residuals as $\widetilde{\eta}_t = \widetilde{\varepsilon}_t / \sqrt{\widehat{h}_t} - T^{-1} \sum_{i=1}^{T} \widetilde{\varepsilon}_i / \sqrt{\widehat{h}_i}$ for $t = 1, 2, ..., T$ with empirical distribution function denoted by $\widetilde{F}_T(\eta) = T^{-1} \sum_{t=1}^{T} I(\widetilde{\eta}_t \leq \eta)$ that is used for resampling. Since the underlying distribution of η_t is assumed to be symmetric (Assumption 1, part (a)), we need to ensure that the empirical distribution from which the bootstrap samples are drawn is also symmetric. For this reason, we construct the collection $\{\pm\widetilde{\eta}_1, \pm\widetilde{\eta}_2, ..., \pm\widetilde{\eta}_T\}$ and resample with replacement from its (symmetrized) empirical distribution function $\widetilde{F}_T^{sym}(\eta)$ to obtain the sequence $\{\eta_t^* : t = 0, ..., T\}$ (Jing, 1995).

The bootstrap procedure for approximating the distribution of $t_{\phi_{ML}=1}$ takes the following steps. First, draw a random sample $\{\eta_1^*, \eta_2^*, ...\eta_T^*\}$ from $\widetilde{F}_T^{sym}(\eta)$ with replacement and for initial conditions h_0^* and y_0^*, construct a bootstrap sample recursively as

$$h_t^* = \widehat{\omega} + (\widehat{\beta} + \widehat{\alpha}\eta_{t-1}^{*2})h_{t-1}^*$$

$$y_t^* = y_{t-1}^* + \sqrt{h_t^*}\eta_t^*.$$

The (possibly demeaned/detrended) bootstrap sample $\{y_1^*, y_2^*, ...y_T^*\}$ is first used to get the bootstrap QMLE estimates $\delta^* = (\omega^*, \alpha^*, \beta^*)$ from $\varepsilon_t^* = y_t^* - \phi_{LS}^* y_{t-1}^*$, where $\phi_{LS}^* = (\sum_{t=1}^T y_{t-1}^{*2})^{-1} (\sum_{t=1}^T y_t^* y_{t-1}^*)$. Then, the one-step bootstrap QMLE of ϕ is obtained as

$$\phi_{ML}^* = \widetilde{\phi}^* - \left[\sum_{t=1}^T \frac{\partial^2 l_t^*(\phi, \delta^*)}{\partial \phi^2}\right]_{\phi=\widetilde{\phi}^*}^{-1} \left[\sum_{t=1}^T \frac{\partial l_t^*(\phi, \delta^*)}{\partial \phi}\right]_{\phi=\widetilde{\phi}^*},$$

where $\widetilde{\phi}^*$ is a preliminary consistent estimate, typically ϕ_{LS}^*. The iterative bootstrap estimator can be computed by updating the estimates of δ^* and ϕ_{ML}^* until convergence. The estimators δ^* and ϕ_{ML}^* are finally used to calculate the Hessian $\left[-\sum_{t=1}^T \frac{\partial^2 l_t^*(\phi,\delta)}{\partial \phi^2}\right]_{\phi=\phi_{ML}^*, \delta=\delta^*}^{-1}$ and the t-statistic of a unit root

$$t_{\phi_{ML}=1}^* = \left[-\sum_{t=1}^T \frac{\partial^2 l_t^*(\phi, \delta)}{\partial \phi^2}\right]_{\phi=\phi_{ML}^*, \delta=\delta^*}^{1/2} (\phi_{ML}^* - 1).$$

This algorithm is repeated B times[2] and each time the bootstrap unit root statistic $t^*_{\phi_{ML}=1}$ is computed. Let P^* denote the distribution of $(y_1^*, y_2^*, ..., y_T^*)$ conditional on the sample $(y_1, y_2, ..., y_T)$ and $G_T^*(x) = P^*(t^*_{\phi_{ML}=1} \leq x)$ be the bootstrap distribution of $t^*_{\phi_{ML}=1}$. Bootstrap critical values can be obtained by taking the corresponding quantile of $G_T^*(x)$ and bootstrap p-values of the unit root test are constructed as $B^{-1}\sum_{j=1}^{B} I(t^*_{\phi_{ML}=1} \leq t_{\phi_{ML}=1})$.[3]

1.3.2. Asymptotic Validity of the Bootstrap Approximation

This section analyzes the asymptotic properties of the symmetrized-residual bootstrap procedure. We first demonstrate that the bootstrap samples also satisfy the conditions of Assumption 1. We also show that the initial values used for generating bootstrap samples do not affect the asymptotic distribution of the test statistic. We then establish the bootstrap invariance principle for partial sums of processes with GARCH errors and prove the weak convergence of the bootstrap unit root test statistic to the asymptotic distribution (1.4) in Lemma 1.

From the properties of the MLE estimator $\widehat{\delta}$ and the constraints imposed in the estimation of the GARCH parameters, it is easy to verify that part (b) of Assumption 1 still holds for the bootstrap data generating process. As a result, we focus on establishing if the bootstrap samples satisfy the conditions of parts (a) and (c) of Assumption 1.

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[2]See, for example, Davidson and MacKinnon (2000) for guidance in selecting the number of bootstrap replications.

[3]GAUSS and MATLAB codes for implementing the bootstrap procedure are available from the authors upon request.

Let $d_2(.)$ denote the Mallows metric[4] of degree 2, defined as

$$d_2(F_X, F_Z) = \inf \left(E \left| X - Z \right|^2 \right)^{1/2}$$

over all joint distributions for the random variables X and Z with marginal distributions F_X and F_Z. Also, let $\widetilde{F}_T^{sym}(\eta)$ denote the empirical distribution function of the symmetrized recentered residuals $\{\pm\widetilde{\eta}_1, \pm\widetilde{\eta}_2, ..., \pm\widetilde{\eta}_T\}$ and F be the true distribution of the standardized errors η_t. We use the Mallows metric d_2 to show that the symmetrized empirical distribution function of the recentered standardized residuals provides a good approximation to the true distribution function and the bootstrap errors satisfy the conditions for establishing the bootstrap invariance principle.

LEMMA 2. *Let E^* and Var^* refer to the expected value and variance of P^*, $\{\eta_t^*\}_{t=1}^T$ be drawn with replacement from $\widetilde{F}_T^{sym}(\eta)$ and suppose that Assumption 1 holds. Then,*

(a) $d_2\left(\widetilde{F}_T^{sym}, F\right) \to 0$ *as* $T \to \infty$,

(b) $E(\varepsilon_t^*) = 0$.

(c) $Var^*(\varepsilon_t^*) = \sigma^2$ *as* $T \to \infty$,

(d) $E^*(\varepsilon_t^*)^3 = 0$.

PROOF. See Appendix.

The bootstrap sequences $\{h_t^*\}$ and $\{\varepsilon_t^*\}$ are constructed for some initial values h_0^* and η_0^*. Auxiliary Lemma 2 in Appendix A.1 establishes that if η_0^* is drawn from $\widetilde{F}_T^{sym}(\eta)$ and h_t^* is initialized from its invariant measure, the bootstrap sequences

[4]For the properties of the Mallows metric, see Section 8 in Bickel and Friedman (1981).

$\{h_t^*\}$ and $\{\varepsilon_t^*\}$ are strictly stationary and ergodic. Furthermore, Auxiliary Lemma 3 in Appendix A.1 shows that the expected difference (under P^*) of partial sums constructed from sequences that start from infinite past and finite past tend to zero as $T \to \infty$.

The following lemma demonstrates that different initial values of h_t^* have no asymptotic effect on the bootstrap procedure.

LEMMA 3. *Define the processes* $\xi_t^* = \lambda_1 \varepsilon_t^* + \lambda_2 [\frac{\varepsilon_t^*}{h_t^*} - \frac{\hat{\alpha}}{h_t^*}(\frac{\varepsilon_t^{*2}}{h_t^*} - 1) \sum_{k=1}^{t-1} \hat{\beta}^{k-1} \varepsilon_{t-k}^*]$ *and* $S_{[Tr]} = T^{-1/2} \sum_{t=1}^{[Tr]} \xi_t^*$ *for* $\{0 \leq r \leq 1\}$, *where* $\lambda = (\lambda_1, \lambda_2)'$ *is a constant vector with* $\lambda\lambda' \neq 0$. *Let* h_{01}^* *and* h_{02}^* *are two different initial values of* h_t^* *and* (h_{t1}^*, h_{t2}^*), $(\varepsilon_{t1}^*, \varepsilon_{t2}^*)$ *and* (ξ_{t1}^*, ξ_{t2}^*) *are bootstrap sequences corresponding to these initial values, respectively. Then, under Assumption 1 and as* $T \to \infty$,

(a) $E^* |h_{t1}^* - h_{t2}^*| \to 0$,

(b) $E^* |\varepsilon_{t1}^* - \varepsilon_{t2}^*| \to 0$,

(c) $E^* \left| \frac{1}{\sqrt{T}} \sum_{i=1}^{T} \varepsilon_{i1}^* - \frac{1}{\sqrt{T}} \sum_{i=1}^{T} \varepsilon_{i2}^* \right| = O\left(T^{-1/2}\right)$,

(d) $E^* \left| S_{[Tr]}^{(1)} - S_{[Tr]}^{(2)} \right| = O\left(T^{-1/2}\right)$, *where* $S_{[Tr]}^{(1)} = \frac{1}{\sqrt{T}} \sum_{t=1}^{[Tr]} \xi_{t1}^*$ *and* $S_{[Tr]}^{(2)} = \frac{1}{\sqrt{T}} \sum_{t=1}^{[Tr]} \xi_{t2}^*$.

PROOF. See Appendix.

Finally, we show that the bootstrap delivers consistent estimates of the nuisance parameters that enter the limiting distribution of the unit root test.

LEMMA 4. *Under Assumption 1 and as* $T \to \infty$,

(a) $E^*(h_t^*) \to E(h_t)$,

(b) $E^*(1/h_t^*) \to E(1/h_t)$.

(c) $K^* \to K$,

(d) $F^* \to F$.

PROOF. See Appendix.

Now we can establish the bootstrap invariance principle for partial sums of GARCH processes.

LEMMA 5. *Under Assumption 1,*

$$\left(T^{-1/2} \sum_{t=1}^{[Tr]} \varepsilon_t^*, T^{-1/2} \sum_{t=1}^{[Tr]} \left[\frac{\varepsilon_t^*}{h_t^*} + (1 - \eta_t^{*2}) \frac{\widehat{\alpha}}{h_t^*} \sum_{j=1}^{t} \widehat{\beta}^{j-1} \varepsilon_{t-j}^* \right] \right) \Rightarrow [W_1(r), W_2(r)]$$

for all $r \in [0,1]$, *conditionally on the sample* $(y_1, y_2, ... y_T)$, *where* $[W_1(r), W_2(r)]$ *is a bivariate Brownian motion in* $D[0,1] \times D[0,1]$ *with mean zero and covariance matrix* $\Omega = r \begin{pmatrix} E(h_t) & 1 \\ 1 & K \end{pmatrix}$, *where* K *is defined in Lemma 1.*

PROOF. See Appendix.

The results in Lemmas 2 to 5 provide sufficient conditions for the asymptotic validity of the bootstrap procedure. The next theorem shows that the bootstrap approximation to the distribution of the $t_{\phi_{ML}=1}$ test converges weakly to the limiting distribution in Lemma 1 which implies that the bootstrap is first-order asymptotically correct.

THEOREM 1. *Under Assumption 1 and the null hypothesis* $H_0 : \varrho = 1$, *for any* $x \in \mathbb{R}$ *and* $\epsilon > 0$,

$$\lim_{T\to\infty} \Pr\left\{\sup_x \left|P^*(t^*_{\hat{\varrho}_{ML}=1} \leq x) - P(t_{\hat{\varrho}_{ML}=1} \leq x)\right| > \epsilon\right\} = 0$$

where $P(t_{\hat{\varrho}_{ML}=1} \leq x)$ *is the limiting distribution (1.4) of the* $t_{\hat{\varrho}_{ML}=1}$ *test in Lemma 1.*

PROOF. See Appendix.

The result in Theorem 1 implies that the critical and p-values for the unit root test with GARCH errors can be approximated by bootstrap that avoids the explicit estimation of nuisance parameters. An interesting extension that is beyond of the scope of this paper is to investigate the power of the bootstrap test under the alternative and show that it converges to the power function of the asymptotic test as in Swensen (2003). Also, while establishing the higher-order accuracy of the bootstrap might be interesting, the bootstrap is not expected to offer any asymptotic refinements since the test statistic is not pivotal.

The next section shows that the asymptotic distribution (1.4) provides a very poor approximation to the finite-sample distribution of the unit root test when the degree of conditional heteroskedasticity is high. This seems to be due to the imprecise estimation of the nuisance parameters as the conditional heteroskedasticity is close to an integrated GARCH process. In contrast, the size of the bootstrap-based test is near the nominal level across all GARCH parameterizations without any adverse effects on the power.

1.4. Numerical Illustrations

1.4.1. Monte Carlo Simulation

This section reports the results from a Monte Carlo experiment that assesses the size and power properties of the asymptotic and bootstrap unit root tests in models with GARCH errors. Repeated sample paths are generated from the following model

$$
\begin{aligned}
y_t &= \phi y_{t-1} + \varepsilon_t \\
\varepsilon_t &= \sqrt{h_t}\eta_t \\
h_t &= \omega + \alpha\varepsilon_{t-1}^2 + \beta h_{t-1},
\end{aligned} \tag{1.5}
$$

where $\eta_t \sim iidN(0,1)$.[5] The sample size is $T = 200$ and the number of Monte Carlo replications is 2,000.

The autoregressive parameter ϕ takes values of 1 and 0.92 in evaluating the size and the power of the unit root test, respectively. We also normalize the unconditional variance to be one by setting $\omega = 1 - \alpha - \beta$. The performance of the tests is evaluated for different degrees of conditional heteroskedasticity that cover the conditional homoskedastic case ($\alpha + \beta = 0$) and some highly persistent GARCH specifications ($\alpha+\beta = 0.999$). We consider specifications that are typically estimated from financial data (for example, ($\alpha = 0.399, \beta = 0.6$) and ($\alpha = 0.199, \beta = 0.8$)) as well as specifications (large α and small β, for instance) that are not frequently encountered in

[5]Additional results fot t- and χ^2-distributed errors are available from the authors upon request. The asymmetric errors are used to investigate the sensitivity of the tests to the symmetry condition in Assumption 1.

economic applications. It should be noted that while all specifications satisfy the moment condition $E\varepsilon_t^2 < \infty$, most of the considered GARCH parameterizations render $E\varepsilon_t^4$ infinite.

We investigate the empirical size and power performance of the asymptotic test based on the OLS estimator ($ASY - DF$), the DF test with critical values approximated by the wild bootstrap ($BOOT - DF$), the asymptotic test based on the ML estimator of the GARCH model ($ASY - GARCH$) and its bootstrap analog ($BOOT - GARCH$) discussed in Section 3. All tests are constructed using demeaned data which is equivalent to including an intercept in the estimated models. In the ML estimation of the GARCH parameters, we impose the restriction $\alpha + \beta < 1$.

The GARCH bootstrap generates samples under the null of a unit root by resampling the centered, symmetrized standardized residuals. These samples are used to approximate the distribution of the unit root test with 199 bootstrap replications that delivers the corresponding bootstrap critical values. The asymptotic critical values for the test based on the OLS estimator are obtained from the Dickey-Fuller tables. For the asymptotic test based on the ML estimator with GARCH errors, we use the true values of α, β and κ to obtain the values of the nuisance parameters F, K and ρ (by truncating the infinite sums at a large integer value) and then interpolate the appropriate critical values from Table 3 in Seo (1999).

The empirical rejection probabilities under the null of a unit root at 1%, 5% and 10% nominal levels are reported in Table 1.1. The asymptotic DF test is well sized in the conditionally homoskedastic case and slightly overrejects for low to moderate degrees of conditional heteroskedasticity. As the GARCH persistence approaches the

unit boundary, the size distortions of the DF test are substantial (see also Valkanov, 2005) and are bigger when α exceeds β. Several recent papers (Beare, 2008; Cavaliere and Taylor, 2008) have proposed modified unit root test procedures that are robust to the presence of certain types of conditional heteroskedasticity.[6] Here, we consider the wild bootstrap approach of Cavaliere and Taylor (2008) who extend the results of Gonçalves and Kilian (2004, 2007) to nonstationary volatility models with a unit root. The second column of Table 1 presents the results based on the wild bootstrap method. The wild bootstrap reduces the size distortions of the asymptotic DF test but there are still some relative large overrejections when the sum of the GARCH parameters is near unity. This reflects the stronger moment requirements on the errors that are needed for establishing the validity of the wild bootstrap (Cavaliere and Taylor, 2008).

The results for the $ASY-GARCH$ test $t_{\hat{\phi}_{MLE}=1}$ are reported in the third column of Table 1.1. While the size distortions of this test are smaller than those of the DF test, there are still fairly large despite the fact that the $ASY-GARCH$ test is designed to handle explicitly the presence of conditional heteroskedasticity. Substantial overrejections occur when the GARCH specification borders an integrated GARCH process. Our numerical experiments suggest that these overrejections are due to imprecise estimation of some nuisance parameters as $\alpha+\beta$ is close to one.

[6]Some other popular methods for size correction may not be valid or appropriate in our context. For example, using a robust variance covariance matrix tends to reduce the size distortions (Kim and Schmidt, 1993) but the consistency of this procedure for nonstationary processes has not been formally established. Also, while the resampling scheme that incorporates the GARCH structure of the model can certainly be used for the DF test, it is not obvious why one would employ it for this test and not for the more powerful test based on the ML estimator.

In order to illustrate this point, we present in Table 2 the values of the estimated nuisance parameters $\sigma^2 = E(h_t)$, $E(1/h_t)$ and K (which is equal to F for normal errors) for several GARCH parameterizations and truncation value $k = 3,000,000$. The table shows that when $\alpha + \beta = 0.9$, regardless of which combination of α and β is chosen, the computed values of σ^2 are very close to the true value of 1. However, when $\alpha + \beta = 0.99$, the estimates of σ^2 start to deviate significantly from 1 and tend to be biased towards 0. The difference becomes even more extreme for $\alpha + \beta = 0.999$ and large values of α. The accuracy of the approximation of σ^2 is an indicator of the accuracy of the estimates of $E(1/h_t)$ which in turn affects the estimates of K and the mixing parameter ρ.

In contrast to the large size distortions of the asymptotic tests, the bootstrap controls the size of the unit root test with GARCH errors uniformly across all GARCH specifications and nominal levels. This impressive performance of the bootstrap unit root test is achieved despite the small number of bootstrap replications. Overall, our bootstrap procedure proves to be very effective for correcting the overrejections of the $ASY - GARCH$ test.

Table 1.3 reports the empirical power of the unit root tests with simulated data from model (1.5) with $\phi = 0.92$. The rejection probabilities for the asymptotic tests ($ASY - DF$ and $ASY - GARCH$) is size-adjusted power whereas the power of the bootstrap test ($BOOT - GARCH$) is raw power. One interesting observation that emerges from the results is that the DF test is not able to detect any deviations from the null hypothesis when the conditional heteroskedasticity is very strong. For example, if ($\alpha = 0.6, \beta = 0.399$) and ($\alpha = 0.8, \beta = 0.199$), the size-adjusted power of

the DF test is only 6.70% and 7.12% at 10% nominal level. Even for the parameterization ($\alpha = 0.399, \beta = 0.6$) that is more often encountered in financial applications, the power is 9.55% at 10% nominal level.

The tests that incorporate the GARCH structure of the model suffer only a small power loss in the conditionally homoskedastic case and offer power gains at 10% nominal level of 16-34 percentage points (when the conditional heteroskedasticity is not very strong) to more than 85 percentage points (for the highly persistent GARCH specifications). These extreme power improvements, combined with the size correction property of the bootstrap method. illustrate the potential of the ML-based tests to detect the mean reversion in processes with strong conditional heteroskedasticity. The raw power of the bootstrap test is very close, albeit slightly below, the (typically infeasible in practice) size-adjusted power of $ASY - GARCH$. Davidson and MacKinnon (2006) analyze the discrepancy that arises between the rejection probabilities of the bootstrap test and the size-adjusted power of the asymptotic test and suggest possible ways of minimizing it.

1.4.2. Testing for Unit Root in U.S. Interest Rates

The correct specification of the dynamics of interest rates plays an important role in derivative pricing, hedging and term structure modeling. For example, most diffusion models of spot interest rate that are used for bond valuation impose a mean reverting behavior on the underlying process. Yet, unit root tests for post-war U.S. interest rates rarely reject the null of nonstationarity which requires that this nonstationarity is taken into account in modeling and long-run forecasting of interest rates. This empirical finding not only creates some tension between the dynamics of interest

rates in theoretical finance and the specification adopted in practice but it also may cause substantial size distortions in testing the parameters in term structure models (Elliott, 1998).

While the conditional heteroskedasticity is a widely documented characteristic of interest rates, the unit root tests typically do not incorporate explicitly the strong GARCH effect into the testing procedure. We re-examine the possibility of a mean reversion in U.S. interest rates using the bootstrap test proposed in this paper. The data employed in the analysis include the Federal Funds rate, 3-month Treasury bill rate (secondary market), 1-, 5- and 10-year Treasury bond yields (constant maturity) and the default premium constructed as the difference between the Aaa and Baa corporate bond yields. The series are annualized rates at monthly frequency covering the period July 1954 - November 2008 and are downloaded from Table H.15 of the Federal Reserve Statistical Release (`http://www.federalreserve.gov/releases/h15/data.htm`). The dynamics of the five interest rates and the default premium are plotted in Figures 1.1 and 1.2, respectively. The graphs show that all series exhibit high persistence over the sample period. The short-term interest rates appear to be more volatile than the long-term rates and the dynamics become smoother as the time to maturity increases.

The results from the Dickey-Fuller and the GARCH-based unit root tests are reported in Table 1.4. Since the interest rates do not exhibit any trending behavior, we consider a model that includes an intercept but not a linear trend. The values of the DF statistic for all interest rate processes do not exceed the asymptotic critical values at 5% and 10% significance level (-2.86 and -2.57, respectively). The bootstrap p-values of the DF tests (computed with data generated from the bootstrap procedure

described in Section 3) are between 0.36 and 0.64 and provide no evidence against the null of a unit root. The results from our bootstrap test with GARCH errors stand in sharp contrast with this finding. The bootstrap p-values of the $BOOT - GARCH$ test indicate that the null of a unit root can be rejected at 5% significance level for all interest rates except for the 10-year yield whose bootstrap p-value is 0.068. The last two columns in Table 1.4 confirm the high persistence in the conditional heteroskedasticity of interest rates. Incorporating the pronounced GARCH effect into the testing procedure appears to deliver the substantial power gains documented in the previous section. This rejection of the unit root hypothesis also lends empirical support to the mean reverting diffusion specification that is typically used in financial economics to describe the dynamics of short-term interest rates.

1.5. Conclusion

This paper proposes a bootstrap test for a unit root in processes with GARCH errors and shows its asymptotic validity under very weak moment and distributional assumptions. The proposed method offers several important advantages over the existing tests that do not exploit the information in the conditional variance and its asymptotic counterpart. First, the test delivers impressive power gains by explicitly incorporating the GARCH structure of the errors, especially for highly persistent GARCH specifications with power improvements over the DF-type tests. While the asymptotic counterpart of the test requires the computation of nuisance parameters and suffers from relatively large size distortions, the proposed bootstrap procedure is straightforward to implement and appears to control the size uniformly over all possible GARCH specifications that guarantee the existence of second moments of the

errors. Finally, while generalizing the asymptotic theory to more complicated setups would be quite involved, our bootstrap method can be easily adapted to models with a lag length that goes to infinity at certain rate, asymmetric errors and other types of conditional heteroskedasticity (other models from the GARCH class, stochastic volatility model etc).

Table 1.1. Empirical size (in %) of unit root tests.

	$ASY-DF$			$ASY-GARCH$			$BOOT-GARCH$		
	1%	5%	10%	1%	5%	10%	1%	5%	10%
$\alpha=0,\beta=0$	1.00	5.05	9.35	1.35	5.40	10.06	1.05	5.35	9.85
$\alpha=0.5,\beta=0.4$	3.20	10.16	15.46	1.95	8.05	15.71	1.00	5.05	10.51
$\alpha=0.25,\beta=0.7$	2.85	8.70	14.21	2.15	7.75	15.21	1.10	5.10	9.50
$\alpha=0.399,\beta=0.6$	29.61	43.17	50.58	9.35	26.41	39.82	1.40	4.95	10.05
$\alpha=0.199,\beta=0.8$	13.51	25.66	35.07	9.15	29.16	42.72	1.00	4.95	9.95
$\alpha=0.7,\beta=0.25$	4.80	11.71	17.71	2.65	11.41	21.61	1.00	4.70	10.00
$\alpha=0.6,\beta=0.399$	31.83	45.95	53.55	8.91	24.02	37.04	1.50	5.51	10.11
$\alpha=0.8,\beta=0.199$	31.43	40.64	47.05	7.81	22.02	33.83	0.90	4.70	9.91

Notes: The empirical size is computed from 2,000 Monte Carlo replications with data generated from model (1.5) with $\phi=1$ and $T=200$.

Table 1.2. Values of the nuisance parameters $E(h_t)$, $E(h_t)$ and K computed with truncation value $k = 3,000,000$.

ω	α	β	$\alpha+\beta$	$E(h_t)$	$E(1/h_t)$	K
0.1	0.1	0.8	0.9	1.0001	1.0853	1.1333
0.1	0.3	0.6	0.9	0.99138	1.6085	1.8705
0.1	0.5	0.4	0.9	0.99008	2.457	3.1214
0.01	0.1	0.89	0.99	0.96559	1.8322	1.9721
0.01	0.3	0.69	0.99	0.94684	6.8752	8.2567
0.01	0.5	0.49	0.99	0.69926	15.051	19.623
0.001	0.1	0.899	0.999	0.71754	8.9415	9.6827
0.001	0.3	0.699	0.999	0.23821	59.192	71.376
0.001	0.5	0.499	0.999	0.12845	141.01	184.42

Table 1.3. Empirical power (in %) of unit root tests.

	$ASY-DF$			$ASY-GARCH$			$BOOT-GARCH$		
	1%	5%	10%	1%	5%	10%	1%	5%	10%
$\alpha=0,\beta=0$	31.17	68.83	85.89	25.41	66.68	84.44	24.66	63.93	81.19
$\alpha=0.5,\beta=0.4$	15.91	48.72	67.68	56.93	87.54	94.70	52.83	86.34	93.50
$\alpha=0.25,\beta=0.7$	15.61	49.62	72.49	33.02	73.74	88.64	29.36	70.79	87.04
$\alpha=0.399,\beta=0.6$	0.15	2.35	9.55	22.21	63.68	76.19	28.71	61.28	73.39
$\alpha=0.199,\beta=0.8$	0.35	9.60	27.71	11.31	55.43	73.29	17.36	53.18	71.49
$\alpha=0.7,\beta=0.25$	6.50	42.17	63.43	74.54	94.45	97.30	68.43	91.70	96.50
$\alpha=0.6,\beta=0.399$	0.10	1.35	6.70	38.57	76.19	83.44	46.52	73.94	82.64
$\alpha=0.8,\beta=0.199$	0.00	2.20	7.12	70.63	89.00	92.63	63.39	84.61	90.30

Notes: The empirical power is computed from 2,000 Monte Carlo replications with data generated from model (1.5) with $\phi=0.92$ and $T=200$. The power reported for the asymptotic tests ($ASY-DF$ and $ASY-GARCH$) is size-adjusted power and the power for the bootstrap test ($BOOT-GARCH$) is raw power.

Table 1.4. Unit root tests for U.S. interest rates.

	DF		$BOOT-GARCH$		$\widehat{\alpha}$	$\widehat{\beta}$
	test	$p-value$	*test*	$p-value$		
Fed funds rate	-2.052	0.359	-5.382	0.000	0.328	0.671
3-month rate	-1.994	0.370	-3.582	0.008	0.279	0.720
1-year rate	-1.957	0.370	-3.020	0.023	0.273	0.724
5-year rate	-1.629	0.541	-2.979	0.032	0.170	0.829
10-year rate	-1.578	0.552	-2.662	0.068	0.155	0.844
default premium	-1.329	0.641	-4.650	0.014	0.338	0.661

Notes: The p-values of both tests are bootstrap p-values, where the bootstrap samples are generated using the estimated GARCH parameters (last two columns in the table). The number of bootstrap replications is 1,999.

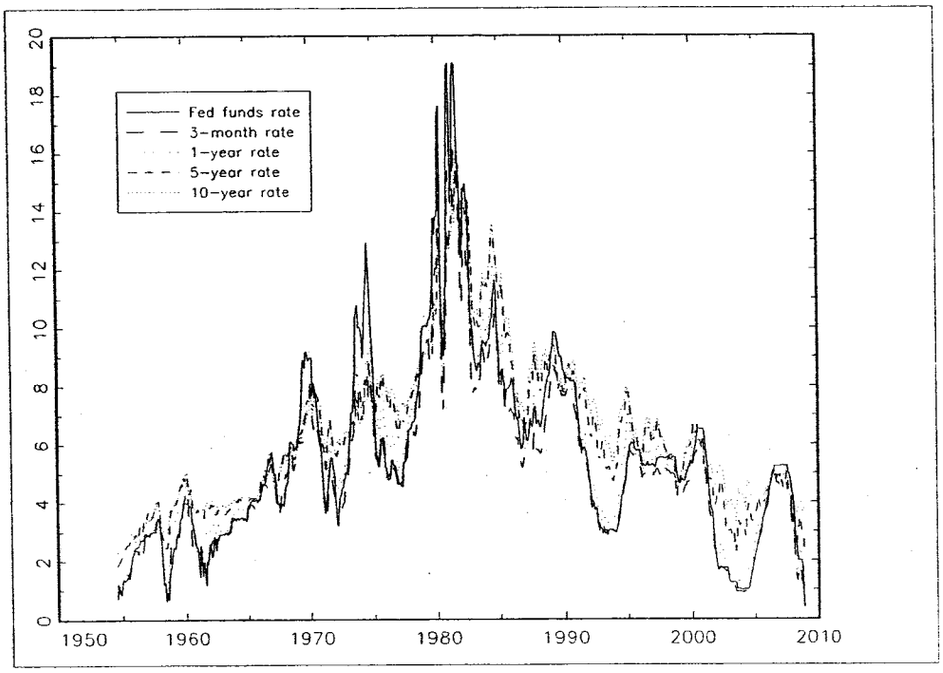


Figure 1. U.S. interest rates.

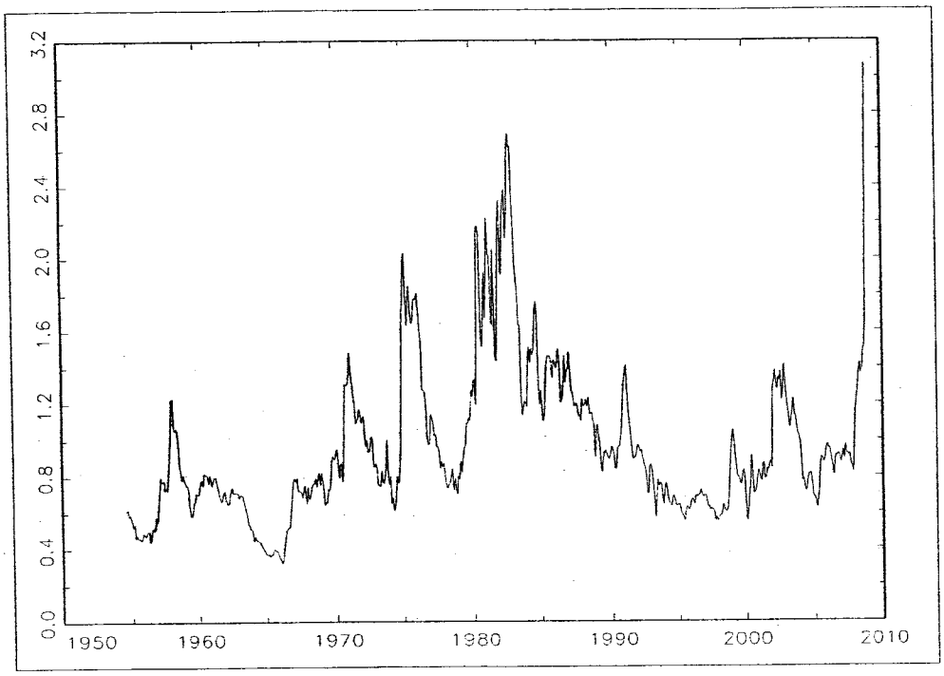


Figure 2. U.S. default premium (Baa corporate bond yield - Aaa corporate bond yield).

CHAPTER 2

Changes in Obesity Prevalence in Canada

2.1. Introduction

As in the U.S. and many other developed countries, obesity is becoming a severe social problem in Canada. The prevalence of obesity among adults aged 18 or older in Canada has been increasing significantly since end of 1970s, from 13.8% in 1978/79 to 23% in 2004 (Tjepkema, 2006). Almost all studies of the impact of obesity reveal a strong positive relationship between excess weight and diseases such as cardiovascular disease, diabetes and hypertension. Obesity is also a reason for the increased prevalence of psychological disorders, such as depression (Wadden et al., 2002). As a person's weight jumps to a high level of obesity, the risk of having these diseases increases dramatically (Allison et al., 1999; Engeland et al., 2003; Flegal et al., 2005). Sturm (2002) states that obesity outranks both smoking and drinking in its deleterious effects on health and health costs. He argues that obesity has roughly the same association with chronic health conditions as does twenty years of aging, and this greatly exceeds the associations of smoking or problem drinking.

Obesity is a condition of excessive body fat which results from a chronic energy imbalance whereby energy intake exceeds energy expenditure (Katzmarzyk, 2002). Bleichet et al. (2007) estimate the relative contribution of caloric intake and physical activity in the developed countries, including Canada, and show that rising obesity is primarily the result of consuming more calories. For example, in 1970, an average

Canadian consumed less than 3000 calories per day; however, average energy intake after 2000 is above 3500 calories.

It is certain that changes in the environment affect human energy intake and physical activity (Hill et al., 2003), and these changes are the direct causes of the obesity epidemic. Bleichet et al. (2007) argue that increase in caloric intake is associated with technological innovations that have resulted in reduced food prices, as well as changing sociodemographic factors, such as increased urbanization and increased female labor force participation. Nonetheless, while changes in the environment increase the possibility of consuming more food, they are just the preconditions for an obesity epidemic. It is individual reactions to changes in the environment that determine who will gain weight, and people with different personal characteristics may respond differently to changes in the environment.

By comparing the average body mass index (BMI) and saving rates among different countries, Komlos et al. (2004) state that the trend in obesity is related to an increase in time preference. By applying a large set of indicators for the individual discount rate from a Dutch survey, Borghans and Golsteyn (2005) suggest that the increase in the average discount rate may be a contributing factor in the rise in BMI in the Netherlands. Although there are many disagreements in the measurement of time preference, these studies imply that responses to changes in the environment lead to the overconsumption of food and the adoption of a sedentary lifestyle. Actually, obesity is the common result of environmental change and personal reactions to it. Given the difficulty and the dispute in the measurement of time preference

and other types of preferences, this study only relates demographic, geographic and socioeconomic characteristics, as well as characteristics in lifestyle, to their BMI.

People's demographic characteristics such as age and race are closely related to their weight. In general, weight increases with age (Baum II and Ruhm, 2007). For both men and women, the prevalence of obesity is significantly different among age groups. It is low among young adults and high among the middle-aged and older people. In Canada, obesity was less than 12% for young adults between the age of 18 to 24, and peaked at around 30% among 45 to 64-year-olds in 2004 (Tjepkema, 2006).

Various studies show that the prevalence of overweight and obesity varies across Canadian provinces. For example, the prevalence of obesity is higher for adults in the Atlantic provinces than the rest of Canada (Heart and Stroke Foundation of Canada, 1999) and the same result was also found among children (Williams et al., 2003). In 2004, the obesity rate for men was significantly above the national level (22.9%) in Newfoundland and Labrador (33.3%) and Manitoba (30.4%). The women's rate surpassed the national figure (23.2%) in Newfoundland and Labrador (34.5%), Nova Scotia (30.3%) and Saskatchewan (32.9%) (Tjepkema, 2006).

Baum II and Ruhm (2007) show that weight is inversely related to socioeconomic status for Americans. In Canada, Tjepkema (2006) argues that " the likelihood of being obese varied by marital status for women, in contrast, the percentages of married, widowed, separated, divorced and never-married men who were obese did not differ significantly. Men aged 25 to 64 with no more than secondary school graduation had significantly higher obesity rates, compared with men who were postsecondary

graduates. Among women, those with less than secondary graduation were more likely than postsecondary graduates to be obese. Men in lower-middle income households were less likely to be obese than those in the highest income households. For women, those in middle and upper-middle income households had a significantly elevated likelihood of being obese, compared with women in the highest income households".

Seven factors are selected to reflect people's body mass index in this study. These are resident region, age, marital status, education, working status, family income and physical activity index which is chosen as a personal characteristic of lifestyle. Among these, six factors other than physical activity are selected with the intention of reflecting their influence on both individuals' energy intake and expenditure, while physical activity is selected as the main way to expend energy.

Studies of the distribution of obesity continually confirm that the BMI has been increasing more over time at the higher than at the lower values, and this implies obesity and severe obesity have increased much faster than median body weight (Ruhm, 2007). In Canada, from 1978/1979 to 2004, the prevalence of overweight people rose from 35.4% to 36.1%, the prevalence of obesity class I rose from 10.5% to 15.2%. However, Class II more than doubled, from 2.3% to 5.1% and Class III tripled, from 0.9% to 2.7%, much higher than the increase in overweight and obesity class I (Tjepkema, 2006).

These differences across the BMI distribution suggest that a linear regression is not a suitable tool for the analysis of obesity, because it only yields a summary for the averages of distributions, conditional on a set of covariates. Thus linear regression supplies a rather incomplete picture for a set of conditional distributions

(Mosteller et at.,1977). A quantile regression, on the other hand, provides a more comprehensive framework for describing changes in the conditional distributions. The major advantage is that the quantile regression can extract the trends in different sections of the set of distributions. Buchinsky (1994) uses this method to study changes in the U.S. wage structure from 1963 to 1987 and provides a range of estimates on the return to schooling and experience at different points of the wage distribution. Ruhm (2007) uses the quantile regression to project the prevalence of obesity in the U.S. and the projected BMI prevalence rate for 2001 is surprisingly close to the actual rate in 1999-2004.

In order to study the obesity epidemic in Canada in recent years, this chapter analyzes the influence of personal factors on BMI along two dimensions: quantiles of the BMI distribution and time. By using quantile regression techniques, this chapter explores the influence of each of the selected seven factors on the body mass index (BMI) at different points of the BMI distribution, and attempts to detect changes in the BMI that are attributable to them. By applying the quantile regression to three health surveys that were conducted from 1978 to 2004, in which the prevalence of obesity was stable at first and almost doubled thereafter, this study attempts to expose changes in the importance of these factors on the BMI over time. Although Ruhm also uses the quantile regression to study obesity, he only relates people's BMI to their race and age and the main aim of his study is predicting the future of obesity prevalence in the United States. Because he applies quantile regression to integrate data from several surveys conducted at different years, the factors' influence on BMI

is averaged over time[1]. This study relates the BMI to more types of personal characteristics and applies quantile regression to data from different surveys respectively. This allows changes in the influence of the factors on the BMI along the BMI distribution *and* over time. Such procedure allows the identification of factors that are closely related to the development of the obesity epidemic in recent years.

By using quantile regression techniques, in conjunction with a backward elimination method, the results indicate that, between 1978 and 2004, age, region and marital status are important determinants of men's and women's BMI, especially for the low quantiles. The influence of marital status on the BMI appears to weaken as the quantile increases. Although the influence of region and age on the BMI also tend to be lower as the quantile increases, in general, their effects are still strong. Since 1994, working status has rarely been an important factor. Over the same period, at higher quantiles, education and physical activity become the main determinants of the BMI, the more education the lower the BMI, the more physical activity the less the BMI; specifically, education plays the most important role in reducing men's BMI and physical activity plays the most important role in reducing women's BMI at the higher quantiles. In this study, men and women are analyzed separately (as in most studies on obesity). Based on the estimation of the quantile regression, the paper also provides a prediction of the future prevalence of obesity in Canada. While the analysis in this paper is based on quantile regression, some linear regression results are also included for comparison.

[1]In his study, Ruhm merges several cross-sectional surveys conducted in different years as one data set.

The rest of this paper is organized as follows. Section 2 describes the available data and defines the variables, and Section 3 introduces the model used. Section 4 reports the estimation results for the distribution of BMI. Section 5 analyzes the importance of factors to the BMI and their evolution. Section 6 suggests a method of prediction and uses it for predicting the BMI in 2007 and 2014, and Section 7 summarizes the conclusions.

2.2. Data

The data used in this research come from three health surveys conducted by Statistics Canada between 1978 to 2004. These are the Canada Health Survey conducted in 1978/1979, the National Population Health Survey (Cycle 1) conducted in 1994/1995 and the Canadian Community Health Survey (Cycle 2.2, Nutrition) conducted in 2004.

The Canada Health Survey (CHS) was proposed as a means of obtaining information required for planning and evaluating health policies and programs. This survey covered the non-institutionalized Canadian population, excluding residents of the Territories, Indian Reserves and remote areas as defined by the Canadian Labour Force Survey. In total, these exclusions comprise less than 3% of the entire Canadian population. The National Population Health Survey (NPHS) is designed to collect information relating to the health of the Canadian population. The target population of the NPHS includes household residents in all provinces, with the principal exclusion of populations on Indian Reserves, Canadian Forces Bases and some remote areas in Quebec and Ontario. The Canadian Community Health Survey (CCHS)

collects information relating to health status, health care utilization and health determinants for the Canadian population. The CCHS (Cycle 2.2) collected responses from persons of all ages, living in privately occupied dwellings in the ten provinces. Excluded from the sampling frame were individuals living in the three Territories, on Indian Reserves and on Crown Lands, institutional residents, full-time members of the Canadian Forces, and residents of certain remote regions.

The BMI is calculated as the weight in kilograms divided by height in meters squared. According to the standard for obesity designed by WHO (World Health Organization, 1997), the BMI for adults is classified into six categories: underweight is defined as $BMI < 18.5$; normal as $18.5 \leq BMI < 25$; overweight as $25 \leq BMI < 30$; obese I as $30 \leq BMI < 35$; obese II as $35 \leq BMI < 40$; and obese III as $BMI \geq 40$.

The BMI from the CHS (1978/1979) and the CCHS (2004, Nutrition) are derived from the interviewer-measured height and weight, while the BMI from the NPHS (1994/1995) is self-reported or proxy-reported. Studies on BMI continually acknowledge that a self-reported BMI is not reliable because a large part of respondents, especially respondents with heavier weight, tend to understate their weight or overstate their height[2]. However, the first cycle of NPHS (1994/1995) was conducted primarily through personal interviews at a selected dwelling, and so understated weight and overstated height were effectively controlled. As a result, the data used in this paper will be considered reliable.

[2]According to the 2004 CCHS, 23.1% of Canadians aged 18 or older were obese, this is significantly higher than estimates derived from self-reported data collected in 2003, which yield an obesity rate of 15.2%.

The data for males and females are analyzed separately as in most studies on obesity. In both linear and quantile regressions, the dependent variable is the BMI, and the covariates are dummy variables corresponding to the following factors: respondents' resident region, age, marital status, education, working status, family income and physical activity index. These dummy variables reflect the characteristics that we are interested in, insofar as they have a relationship with the BMI. The data used include only respondents who were 20 to 64 years old when the survey was conducted. Pregnant women and immigrants who have lived in Canada for less than 10 years prior to the survey are excluded. Observations with missing data for any of the variables described below are dropped from the analysis. This process yielded a sample of 2733 respondents for the CHS (1978/1979), a sample of 11006 respondents for the NPHS (1994/1995) and a sample of 7613 respondents from the CCHS (2004, Nutrition).

The respondents' resident regions are divided into five areas: Atlantic provinces, Quebec, Ontario, Prairie provinces and British Columbia. In the quantile regression, the omitted part (the reference group) is British Columbia so coefficients for respondents' resident regions may be interpreted relative to this province. The respondents' age in years is categorized into three groups: 20-34, 35-49 and 50-64. The omitted category is the 20-34 years group. However, the age groups obtained from the CCHS (2004, Nutrition) are 19-35, 36-50 and 51-65 because of the different method of categorization for age group adopted by this survey. It produces different content in the age-group dummy variables from the same dummy variables based on the other two surveys. However, since there is only a small difference in the categorization of age

group between CCHS and the other two surveys, the errors that result from it are ignored. Marital status is put into three categories, single, married or common law and widowed/separated/divorced. Education is classified by the highest education level that a respondent completed. Three classes are formed: less than secondary graduation, secondary graduation to some post-secondary, and post-secondary graduation, but less than secondary graduation is omitted. Job status describes a respondent's working status during the last 12 months, which include: currently working; not currently working but had a job during the last 12 months; and not working during the last 12 months. The omitted category is: not working during last 12 months. A respondent's family income is divided into 5 categories: lowest; lower middle; middle; upper middle; and, highest level. The omitted category is the lowest category. Although the criterion for income categorization changed over time, it is put into 5 categories in all the three surveys. Hence, the same income categories in different surveys are interpreted as representing the same socioeconomic groups. The physical activity index categorizes respondents as being: active, moderate, or inactive based on total daily energy expenditure values (kcal/kg/day). Active is defined as the averaged energy expenditure above 3.0 kcal/kg/day, moderate as between 1.5 and 3.0 kcal/kg/day and inactive as below 1.5 kcal/kg/day[3]. The omitted category is the inactive group. *Table* 2.1 lists each factor and all its categories. The adopted notation for all dummy variables is listed in the last column of *Table* 2.1.

Besides the categorization of age between the first two surveys and the last survey are slightly different, there are also two other limitations of our data. First, pregnancy

[3]This approach is consistent with other studies, such as the Campbell's Survey and the Ontario Health Survey.

status was not asked in the CHS (1978/79), however, pregnant women in the NPHS (1994) and CCHS (2004) were dropped. Second, in the CHS (1978/79), working status was categorized as employed, unemployed and not in the labor force, whereas it was categorized as currently working, not currently working but had a job during last 12 months, and not working during last 12 months three categories in the other two surveys. Although these differences in the categorization of data may cause some bias in the results, the view taken is that such biases are relatively small and do not significantly affect the general picture that emerges. This view is supported by the fact that many results of this study are consistent with the results of many previous studies.

2.3. Model

The quantile regression model used in this paper is given by

$$(2.1) \qquad bmi_i = X_i'\beta_\theta + \varepsilon_{\theta i} \text{ with } Quant_\theta(bmi_i \mid X_i) = X_i'\beta_\theta \quad (i = 1, ..., n)$$

where β_θ and X_i are $k \times 1$ vectors, and $x_{i1} \equiv 1$. $Quant_\theta(bmi_i \mid X_i)$ denotes the θth conditional quantile of the body mass index given X. Specifically, in this paper $X_i' = (1, reg_i', age_i', mars_i', ed_i', ws_i', incq_i', pai_i')$ where $reg_i' = (reg_i^1, reg_i^2, reg_i^3, reg_i^4)$, $age_i' = (age_i^2, age_i^3)$, $mars_i' = (mars_i^2, mars_i^3)$, $ed_i' = (ed_i^2, ed_i^3)$, $ws_i' = (ws_i^2, ws_i^3)$, $incq_i' = (incq_i^2, incq_i^3, incq_i^4, incq_i^5)$ and $pai_i' = (pai_i^2, pai_i^3)$. The corresponding linear regression model is

$$(2.2) \qquad bmi_i = X_i'\beta + \varepsilon_i \quad (i = 1, ..., n)$$

and we choose the ordinary least squares model as a counterpart of the quantile regression model.

In the above regression models, there are 18 covariates, plus an intercept. For each covariate, the coefficient can be interpreted as the impact of a one-unit change in the covariate on the BMI, holding other covariates fixed. This gives the difference in BMI between the group represented by the covariate and the group represented by the omitted category from the same factor at the θth percentile of the distribution, holding all other factors fixed. For example, the coefficient for pai_i^3 at the $50th$ quantile represents the difference in BMI between the active group and the inactive group at $50th$ percentile of the distribution, holding the other covariates fixed. The intercept of the model could be interpreted as the conditional quantile function (mean for linear regression) of the BMI of a single male or female who lives in British Columbia at age between 20-34 with less than secondary graduation education, who has not worked during the last 12 months, has the lowest family income and is identified as an inactive person. British Columbia as well as the lowest category of each the factor are chosen to represent the base group because first, the prevalence of obesity in British Columbia is relative low and second, we want to observe the relative changes in the BMI as people get older, richer, more educated and active.

2.4. Estimation

OLS estimation and estimation of the quantile regression at each of 6 quantiles, $10th, 25th, 50th, 75th, 90th$ and $99th$ percentiles based on data for both male and female from CHS1978, NPHS1994 and CCHSN2004 are presented in *Table* 2.2 to

Table 2.7. *Table* 2.2. *Table* 2.3 and *Table* 2.4 report estimation results for males and the other three contain estimation results for females.

The OLS estimation is significantly different from the quantile regression estimation for most covariates at most of the quantiles, particularly at the high quantiles. If we use the estimation from OLS to represent the relation between the factors and the BMI for people in this range of the BMI distribution, the conclusion would be totally unreliable. Essentially, OLS treats every observation in the sample equally, it only reflects the relationship between the dependent variable (BMI) and independent variables at the average level and does not reveal specific information at different parts of the distribution. In the rest of the paper, the focus is only on the quantile regression.

As *Table* 2.2 to *Table* 2.7 show, the parameter estimates from the quantile regression vary from one quantile to another. Noticeably, the quantile regression estimates at the median are significantly different from the OLS estimates for most of the covariates and imply some asymmetry or skewness in the conditional density of the BMI. In the next section we analyze the contribution of each covariate for both males and females in the three survey periods, 1978, 1994 and 2004.

2.4.1. Estimation Results

2.4.1.1. Males. *Table* 2.2, *Table* 2.3 and *Table* 2.4 present the results for men for the 1998, 1994 and 2004 surveys, respectively. *Table* 2.2 shows that the coefficients on reg_i^k $(k = 1, 2, 3, 4)$, age_i^l $(l = 2, 3)$, $mars_i^2$ and $incq_i^3$ are positive, and the coefficient on covariate ed_i^3 is negative at each quantile; the estimated coefficients on the other covariates are positive at some quantiles and negative at others. The coefficients of

reg_i^1, reg_i^4, age_i^2, age_i^3 and $mars_i^2$ are significantly different from 0 at most quantiles. We may interpret that these covariates have significant impact on BMI at the different quantiles of the conditional distribution of BMI. In summary, in 1978/79, men's' weight has the following characteristics. (a) On average, men who live in Atlantic and Prairie provinces weigh more than men who live in British Columbia. Compare to British Columbia, it is more likely for men to be obese in those two regions. (b) Age is a major factor that affects the BMI. In general, men in the second and third age groups weigh more than those in the first group. (c) Compared to singles, married men are more likely to be overweight or obese. (d) Looking at the coefficients on ed_i^3, ws_i^2 and $incq_i^2$, their negative values at most quantiles suggest that these covariates generally reduce men's weight [4]. However, the coefficients on these covariates are mostly insignificant. Next, we discuss the results for 1999.

Table 2.3 reveals that the estimated coefficients on reg_i^1, age_i^l $(l = 2, 3)$ are positive at each quantile; the coefficients on $mars_i^m$ $(m = 2, 3)$ and $incq_i^4$ are positive except for a few quantiles, the coefficients on ed_i^2 and ed_i^3 are negative at each quantile and the estimated coefficients on other covariates are positive at some quantiles and negative at others. The variables age_i^l $(l = 2, 3)$, $mars_i^2$ and ed_i^3 appear to be significant at most quantiles except for the high or low quantiles and the other variables tend to have an insignificant effect on BMI. This implies that, in 1994, age is a major factor that affects BMI at quantiles smaller than the $90th$ percentile. Married men weigh more than singles at quantiles less than $90th$ percent. Men who had completed

[4]From the quantile estimation at higher quantile such 75, 90 and 99 quantiles, the estimated coefficients for pai_i^3 are also negative, implying that active physical activity reduces men's weight. However, as for ws_i^2, these coefficients tend to be insignificant.

post secondary education weigh less than those who had not completed secondary education. The estimated coefficients on ed_i^2 suggest that men who had completed secondary education weigh less than those who had not, although these effects are statistically insignificant. We now turn our attention to the results from the most recent survey in 2004.

Table 2.4 shows that the estimated coefficients on reg_i^4 , age_i^l $(l = 2, 3)$, and $incq_i^3$, $incq_i^4$, $incq_i^5$ are positive, and the coefficients on covariates ed_i^n $(n = 2, 3)$, ws_i^3 and pai_i^2 are negative at each quantile; the estimated coefficients are positive for reg_i^1 and $mars_i^m$ $(m = 2, 3)$ and negative for pai_i^3 at most quantiles except one quantiles. Compare to the surveys in 1978 and 1999, the most important changes in 2004 survey are the variables ed_i^n $(n = 2, 3)$ tend to be statistically significant and the coefficients of resident areas, reg_i^k, become insignificant. Specifically, the estimation results imply that, in 2004: (a) the influence of resident area to men's weight becomes weak; (b) age is still a major factor that affects BMI, but its influence is uncertain for men with heavy weight; (c) men who had completed secondary or even some post-secondary education and who had completed all of post-secondary education weigh less than those who had not completed secondary education, but its influence is uncertain for men with big weight; (d) for obese men, physical activity is negatively related to their BMI; those who were active or medium in physical activity are more likely to have a lower BMI than were the inactive ones; and (e) men with family income at the middle, upper middle and highest levels tend to have more weight than men with family income at the lower middle and lowest levels.

2.4.1.2. Females. *Table* 2.5, *Table* 2.6 and *Table* 2.7 report the quantile estimation results for women for 1978, 1994 and 2004 surveys, respectively. *Table* 2.5 shows the estimated coefficients on covariates reg_i^1, reg_i^2, age_i^l $(l = 2, 3)$, $mars_i^2$ and $incq_i^3$ are positive, and the coefficients on covariate ed_i^3, ws_i^2 are negative at each quantile; the coefficients on reg_i^3, reg_i^4 and ws_i^3 are positive, and the coefficients on ed_i^3, $incq_i^2$and $incq_i^5$ are negative except for one quantile; the estimated coefficients on reg_i^1, age_i^2, age_i^3 are significantly different from 0 at most quantiles; the variables ed_i^3 and pai_i^3 appear to be significant at high quantiles. In 1978/79, women's BMI has the following characteristics: (a) on average, women who live in the Atlantic provinces weigh more than women who live in British Columbia; (b) age is a major factor that affects BMI. Women in the second and third age groups weigh more than those in the first group; (c) women who have completed post-secondary education weigh less than those with less than secondary education in the obese level; and (d) for obese women, those who were active in physical activity are more likely to have a lower BMI than the inactive ones. The positive coefficients for reg_i^2 $mars_i^2$ and $incq_i^3$ and the negative coefficients for ws_i^2, $incq_i^2$and $incq_i^5$ at most quantiles mean that these covariates tend to increase and decrease women's weight, respectively. However, their impacts on BMI are not statistically significant.

Table 2.6 shows the estimated coefficients on reg_i^1, reg_i^3, reg_i^4, age_i^l $(l = 2, 3)$ and $mars_i^m$ $(m = 2, 3)$ are positive and the coefficients on ed_i^3, ws_i^2, $incq_i^3$, $incq_i^2$and $incq_i^5$and pai_i^3 are negative at each quantile; the coefficients on covariate ws_i^3, $incq_i^2$ and pai_i^2 are negative except one quantile. The coefficients on reg_i^1, reg_i^4 and age_i^l $(l = 2, 3)$ are significantly different 0 at the most quantiles; the variables $mars_i^m$

$(m = 2, 3)$ and $incq_i^5$ and pai_i^3 appear to be significant at low quantiles and high quantiles, respectively. This means that, in 1994, age was still a major factor affecting the weight of most women, except for the heavist women. For women with normal weight, those who were married weigh more than those who were single. For over weight and obese women, those with family income at the highest level weigh less than those with family income at the lowest level and those who were physically active weigh less than the inactive ones. The variables $incq_i^2$, $incq_i^3$, pai_i^2, $ed_i^3$and ws_i^2 tend to reduce women's weight at almost every quantile. However, their impact on BMI is not statistically significant.

Table 2.7 shows the estimated coefficients on age_i^l $(l = 2, 3)$ are positive and the coefficients on ed_i^n $(n = 2, 3)$ are negative at each quantile; the coefficients on $mars_i^m$ $(m = 2, 3)$ are positive at all quantiles less than $90th$ quantile and the coefficients on pai_i^3 are negative from $25th$ to $99th$ quantile. The coefficients on age_i^l $(l = 2, 3)$ and ed_i^n $(n = 2, 3)$ are significant different from 0 at almost all quantiles. The variables pai_i^3 are significant at high quantiles and the impact of reg_i^k $(k = 1, 2, 3, 4)$ on BMI are not statistically significant at each quantiles. This implies that, in 2004: (a) age is still a major factor that affects BMI, but its influence is uncertain for heavy people; (b) women with high education tend to weigh less than those with low education; (c)-for obese women, physical activity is negatively related to their BMI, those who were active in physical activity are more likely to have a lower BMI than the inactive ones; and (d) the impact of the resident's region on women's BMI become weak and insignificant.

The above results confirm findings of other previous study on obesity, moreover, instead of estimating an average change of BMI which contributes to each factor, this study can detect the contribution of each factor at any point of the BMI distribution. The foregoing is the estimation of BMI distribution for 1978, 1994 and 2004, and the nature of the BMI distribution in these years are well captured by this estimated distribution.

2.4.2. Estimation of the Distribution of BMI

By using quantile regression, the conditional distribution and density function for both males and females were estimated from 1978 to 2004. In order to get a clear refinement of the distribution, 99 quantiles from the $1st$ to the $99th$ quantile were set. First, the coefficients for all covariates at each quantile were estimated. Second, a weighted average for each covariate was calculated. In the last step, the estimation of BMI at θth quantile was produced by the following formula:

(2.3) $$bmi_\theta = \bar{X}'\beta_\theta, \text{ where } \bar{X}' \text{ is weighted average of } X.$$

Figure 2.1 and *Figure* 2.2 plot the distribution functions for both men and women from 1978 to 2004. These figures show that, for both men and women, the difference of BMI distributions is very small between 1978 and 1994, but increases substantially between 1994 and 2004. This confirms what Tjepkema (2006) found in his research. The BMI distribution in 2004 is always below its 1978 and 1994 counterparts and the range of the distribution is extended to the right dramatically. *Figure* 2.3 and *Figure* 2.4 present the density functions for each BMI distribution for both men and

women. From these two figures, it is very clear that density in 2004 is below the 1978 and 1994 densities for values of BMI less than 28 (approximation). However, after that point, the density in 2004 is above the 1978 and 1994 densities almost everywhere and exhibits a very long right tail. This implies that the probability of the union of normal and overweight decreases, while the probability of being obese increases.

Table 2.8 lists the percentage distribution of BMI in 1978, 1994 and 2004 estimated for each year. This table also includes the percentage distribution of BMI in 1978 and 2004 that was reported in Tjepkema (2006). We find that our percentage distribution of BMI is close to that of Tjepkema (2006) between the *Normal* to *Obese I* categories. There are big differences at the extreme ends of both sides. However, in both of our estimation and Tjepkema's (2006) results, the data for the underweight and *Obese III* categories should be interpreted with caution. One factor which might explain part of the difference in the percentage distribution of the BMI between this research and Tjepkema (2006) is the selection of the sample. In this research, the sample is formed by respondents aged from 20 to 64 while it is formed by respondents aged from 18 to 64 in the research of Tjepkema (2006).

2.5. Importance of Factors and Its Evolution

To identify those factors which have the largest impact on the BMI is a major objective of this research. However, because each factor includes at least two covariates, it is hard to compare the influence of factors to the BMI by relying on the estimation of each covariate. In addition, there are two other problems. First, the estimation of some coefficients at some quantiles are not so small, but statistical significances are low. Second, the influence of a covariate is generated not only by its coefficient, but

also by the covariate itself. For example, in the case that a dummy variable has lots of *zeros*, the value of its coefficient hardly affects the dependent variable (BMI), even if it is significantly high. In order to solve these problems and make the analysis more accurate, we need to employ more powerful tool.

A method called backward elimination is now used to solve the problems just described above. Backward elimination is a method which uses the amount of variance a variable adds to the complete model (all remaining variables) as the criterion for exclusion from the model. In this method, the full model (with all variables included) is first computed. Then each variable is removed from the model alternatively, and the variable that causes the least reduction in accounted variance by its removal is the first to be eliminated. This process continues until all remaining variables contribute a significant amount of variance reduction to the final model. Obviously, the variable that is removed first has the weakest influence on the dependent variable and the variables that are kept at the end have the strongest influence on the dependent variable. Instead of applying backward elimination to variables, in this paper, this technique was applied to the factors[5] selected for the model.

Backward elimination was applied at seven quantiles, 10, 25, 50, 75, 90, 95 and 99 percent for both males and females from CHS1978, NPHS1994 and CCHSN2004. The backward elimination used in this paper can be described as follows.

(*a*) By model (1), the estimated BMI for the full model is $bmi_i = X_i'\beta_\theta$.

[5]In this paper we use seven factors, region, age, marital status, education, working status, family income and physical activities which may relate to BMI, and each of them contains at least two covariates. When one factor is eliminated from the model, it means all the covariates that are included in this factor are eliminated.

(*b*) Remove all the covariates corresponding to factor k from the model and reestimate the new simplified model, then the estimated BMI for the new model can be obtained, denoted as $bmi_i^k = X_i^{k'}\beta_\theta$ $(k = 1.2, ..., 7)$.

(*c*) Calculate $\sum_{i=1}^{n}\left(bmi_i - bmi_i^k\right)^2$ for each k, and compare their value. Suppose $\sum_{i=1}^{n}\left(bmi_i - bmi_i^3\right)^2$ is the smallest one, then the third factor or factor marital status is the first factor removed from the full model and new model is formed.

(*d*) Treat the newly formed model as the full model and continue this process until there is only one factor left.

Table 2.9 and *Table* 2.10 report the results from this procedure. *Table* 2.9 is for male and *Table* 2.10 is for female. It is easy to find that some results from these two tables confirm what is found in *Table* 2.2 to *Table* 2.7, however, some important information, especially the order of the importance of the factors to BMI, which was disclosed in *Table* 2.9 and *Table* 2.10 cannot be obtained from *Table* 2.2 to *Table* 2.7. The findings from *Table* 2.9 and *Table* 2.10 can be summarized as follows.

In general, in 1978, age, region and marital status are the most important factors that influence the BMI of men at the low quantiles, but as the quantile increases, family income, working status and physical activities become the most important factors that influence the BMI, and the influence of age and region weakens. Education is the weakest factor to the men's BMI at all quantiles except 99*th* quantile and working status weakly relate to their BMI at the quantiles less than 75 percent at that time. In 1994, age, region and marital status are still the most important factors that influence the BMI of men at the low quantiles. As the quantile increases, education becomes the most important factor that influences men's BMI and marital

status is the weakest factor. Also, the influence of region becomes strong and the influence of age weakens. Physical activity is the weakest factor for men at the low quantiles and remains weak as the quantile increase. In 2004, marital status, family income and age are the most important factors that influence the BMI of men at the low quantiles. As the quantile increase, education and physical activity become the most important factors which influence men's BMI. In the following context, the importance of factors on BMI for women and its evolution are discussed.

In 1978, age is the most important factor that influences the BMI of women, except at the highest quantile. Region and working status are important only at very low quantiles. As the quantile increases, the influence of family income and physical activities on women's BMI also increases, and physical activity even become the most important factor among all the factors at the $99th$ quantile. Education is the weakest factor on women's BMI at all quantiles and marital status is the second weakest factor except at the $99th$ quantile. In 1994, age is still the most important factor for women at all quantiles less than the $90th$ percentile. Although the influence of region decreases after the $90th$ quantile, it is always an important contributor to women's BMI. As the quantile increase, the influence of family income and physical activity also increase and become the two most important factors at high quantiles. Education is always a weak factor for women at all quantiles in 1994. In 2004, as in 1994, age, region and marital status are the most important factors that influence women's BMI at the very low quantiles. As the quantile increases, the influence of physical activity and education on the women's BMI increase and physical activity becomes the most important factor after $75th$ quantile. In 2004, working status is

the weakest contributor to the BMI of women at all quantiles except the $99th$ and marital status is the second weakest factor except at the lowest quantiles.

From 1978 to 2004, age, region and marital status are always important factors for both men and women's BMI. The influence of marital status to the BMI becomes pretty weak as the quantile increases. Although the influence of age to the BMI also becomes weak as the quantile increases, in general, its influence is still strong. Since 1994, working status is rarely an important factor, during the same period, the influence of income on BMI increases for men and decreases for women. For overweight and obese men and women, the importance of education and physical activity increases over the period 1978 to 2004: specifically, education plays the most important role in men's BMI and physical activity plays the most important role in women's BMI at the high quantiles.

2.6. Prediction in BMI

Causes of the difference in the BMI distribution between two adjacent surveys may usefully be analyzed by decomposing each quantile into three distinct components, using estimates from the quantile regressions. Let T and t represent the years when the surveys were conducted, $T > t$, and $\bar{X}$ denotes the weighted average of X in (2.1), then, at the θth quantile ($\theta = 1, 2, ..., 99$).

$$(2.4) \quad bmi_\theta^T - bmi_\theta^t = \left(\bar{X}^{T'} - \bar{X}^{t'}\right)\beta_\theta^t + \bar{X}^{t'}\left(\beta_\theta^T - \beta_\theta^t\right) + \left(\bar{X}^{T'} - \bar{X}^{t'}\right)\left(\beta_\theta^T - \beta_\theta^t\right)$$

In equation (2.4), a change in the BMI distribution, at each quantile θ, has been decomposed into (i) a change in the *structure* of factors $\left(\bar{X}^{T'} - \bar{X}^{t'}\right)\beta_\theta^t$; (ii) a change

in the *function* of factors, $\bar{X}^{t'}\left(\beta_\theta^T - \beta_\theta^t\right)$; (ii) a change in the *interaction* of *function* and *structure*, $\left(\bar{X}^{T'} - \bar{X}^{t'}\right)\left(\beta_\theta^T - \beta_\theta^t\right)$. These three factors are refers to hereinafter as simply *structure*, *function* and *interaction*. By using the decomposition (2.4) at each of the 99 quantiles, a decomposed shift in the BMI distribution may be formed. Because the change in this distribution is much greater between 1994 and 2004 than it was between 1978 and 1994, the main focus will be on decomposition of the former; this may then be compared with decomposition of the letter. In this way, the reasons for the significant shift in the BMI distribution in recent years may be explored.

Actual computation reveals that, between 1994 and 2004, the influence caused by *function*, $\bar{X}^{94'}\left(\beta_\theta^{04} - \beta_\theta^{94}\right)$, is positive and dominant at almost all quantiles, for each of males and females, accounting for most of the changes in the BMI distributions. The exceptions are: two quantiles less than the $7th$ quantile for males and the $2nd$ quantile for females. The influence of *structure*, $\left(\bar{X}^{04'} - \bar{X}^{94'}\right)\beta_\theta^{94}$, is consistently negative for both males and females, save for two low quantiles for females. *Interaction*, $\left(\bar{X}^{04'} - \bar{X}^{94'}\right)\left(\beta_\theta^{04} - \beta_\theta^{94}\right)$, has a positive effect for males but a negative effect for females at most quantiles. The dominance of *function* in determining the change in the BMI distribution may be seen by looking at its percentage contribution to the whole change. For males, from the $16th$ to the $44th$ quantiles, *function* accounts for from 62% to 86%, or on average 74%, of the total change and for most of the rest of the quantiles *function* contributes a little over 90% of the total change. For females, the contribution of function is even more dominant. For all quantiles, except the second to the $13th$, the contribution of *function* represents 110% to 117% of the totals, because the combined influence of *structure* and *interaction* is negative. When

the combined influence is positive, that is, from the second to the $13th$ quantile, the contribution of *function* ranges from 60% to 90%, with an average of 77%, of the total change in the BMI distribution. These results are presented graphically in *Figure* 2.5 and *Figure* 2.6.

Turning now to changes in the BMI distribution between 1978 and 1994, the result are presented graphically in *Figure* 2.7 and *Figure* 2.8. Generally speaking, there is no dominant cause among *structure*, *function* and *interaction* for males and for females. Comparing the decomposition by quantiles between 1978-1994 and 1994-2004, there is no significant difference attributable to *structure* and *interaction*. These two decomposition parts are in the same range during the two periods and, hence, so is their combined influence. But there is a huge difference caused by the change in *function*. For most quantiles, these differences increased tenfold in the second period (see *Figure* 2.9 and *Figure* 2.10).

The decomposition by quantiles from 1978 to 2004, has two important properties which have implications for predicting the BMI distribution beyond 2004: (a) the significant shift in the BMI distribution during 1978 to 2004 was caused principally by change of *function* from 1994 to 2004; (b) the changes due to the combined effects of *structure* and *interaction* have been comparatively small and stable throughout the whole period from 1978 to 2004. The situation described by (a) and (b) above may be cast in symbols based on equation (2.4). Let τ represent the next period ahead of T , i.e. $T < \tau$ in (2.4), then at quantile θ

$$bmi_\theta^\tau - bmi_\theta^T = \bar{X}^{T'}\left(\beta_\theta^\tau - \beta_\theta^T\right) + adjustment, \tag{2.5}$$

whereupon

$$bmi_\theta^\tau = bmi_\theta^T + \bar{X}^{T'}\left(\beta_\theta^\tau - \beta_\theta^T\right) + adjustment. \tag{2.6}$$

Given this background, two predictions will be made for 2007 and 2014, subject to two assumptions: ($A1$) the BMI evolution in the next decade beginning 2005 will be the same as in the decade preceding 2005; ($A2$) changes in the BMI distribution occurs evenly through time. Under the two assumptions, the prediction of the BMI at the θth quantile in year τ becomes, from (2.6)

$$bmi_\theta^\tau = bmi_\theta^{04} + \bar{X}^{04'}\left(\beta_\theta^\tau - \beta_\theta^{04}\right) + adjustment \tag{2.7}$$

in which $\tau = 2007$ or 2014. Under the assumption $A1$ and $A2$, $\beta_\theta^\tau - \beta_\theta^{04}$ may be estimated as $(\beta_\theta^{04} - \beta_\theta^{94}) * (\tau - 2004)/9.5$, 9.5 being the length of time, in years, from the middle of the survey NPHS1994/95 to survey CCHSN2004. The coefficients applied to the prediction for 2007 and 2014 are those obtained for the decomposition analysis, based equation (2.4), for 1994 and 2004. If the combined influence caused by the changes in *structure* and *interaction* is considered, the prediction bias denoted as *adjustment* in (2.7) may be reduced. The bias could be expressed as the product of the effect of *function*, $\bar{X}^{04'}(\beta_\theta^\tau - \beta_\theta^{04})$, and average quotients that combined effects of *structure* and *interaction*, $\left(\bar{X}^{T'} - \bar{X}^{t'}\right)\beta_\theta^t + \left(\bar{X}^{T'} - \bar{X}^{t'}\right)\left(\beta_\theta^T - \beta_\theta^t\right)$, to the effect of *function* in different sections of the BMI distribution. However, the quotients used for the prediction of this paper are those obtained from the decomposition analysis at each quantile for the period from 1994/95 to 2004. The predicted percentage distributions for the BMI in 2007 and 2014 are given in *Table* 2.11 and the predicted

distributions are displayed graphically in *Figure* 2.11 and *Figure* 2.12 for men and women, respectively.

2.7. Conclusion

The analysis of this paper has applied quantile regression to determine the quantitative influence of several observable factors at different points on the Canadian BMI distribution, using data from three distinct Canadian surveys undertaken in 1978, 1994 and 2004. The results obtained strongly suggest that the epidemic of obesity in Canada is mainly developed during the period 1994 to 2004. This epidemic is more closely related to lifestyle and socioeconomic factors than demographic factors[6]. Educational achievement has a negative impact, that is to say, the higher the educational level a person achieved, the lower the likelihood that person will be obese. The role of the education has shifted over the years from the weakest to the strongest factor affecting the BMI. This, in turn, implies that the relationship between the capacity to avoid obesity and education is strong. Among the seven factors in this study, being physically inactive is the most important reason for women to be obese. On the other hand, working status is the least important factor related to the BMI for both men and women. This not only indicates the dominance of sedentary work and continually decreasing expenditure of energy in the workplace, but also reveals that physical activity is the main way for people to expend energy. The fact that age becomes less important in affecting the BMI at higher quantiles indicates that a change in the age structure of Canadians is not a reason for the obesity epidemic in Canada.

[6]Jeuier and Tappy (1999) argue that genetic changes are not the cause of increased obesity over such a short period of time. Rather, changes in the energy balance are key; consuming more calories than are expended leads to weight gain.

A decomposition by quantiles has been introduced, in (2.4), in an effort to explore the development over time of the Canadian BMI distribution, especially in the future. The calculation reveals that, after 1994, the component of most importance in changing the BMI distribution has been the *function* of factors, that is, the changing intensity with which factors impinge on BMI. *Structural* changes, that is, changes in the factors themselves, and the *interaction* of *structure* and *function* are of trivial importance. To put the matter technically: in a quantile regression analysis of the BMI by cross-sections over time, the most significant changes have been those in the coefficients, not changes in the variables or changes in the interaction between the two.

Table 2.1: Definition of the Variables

Factors	Covariates	Notation
Region	Atlantic Provinces	reg^1
	Quebec	reg^2
	Ontario	reg^3
	Prairie Provinces	reg^4
Age	35 to 49	age_g^2
	50 to 64	age_g^3
Marital Status	Married	$mars^2$
	Widowed/Separated/Divorced	$mars^3$
Education	Secondary to Some Post Secondary	ed^2
	Post Secondary	ed^3
Working Status	Currently Working	ws^2
	Not Currently Working	ws^3
Family Income	Lower Middle Level	inc_q^2
	Middle Level	inc_q^3
	Upper Middle Level	inc_q^4
	Highest Level	inc_q^5
Physical Activities	Moderate	pai^2
	Active	pai^3

Table 2.2: Estimate of the influence to BMI at the mean and some quantiles (CHS1978_Male)

Coef.	OLS	Quantiles					
		10th	25th	50th	75th	90th	99th
reg[1]	1.238 (.451)	.719 (.371)	1.759 (.293)	1.327 (.406)	.957 (.417)	1.48 (.845)	3.033 (.901)
reg[2]	.538 (.467)	.653 (.370)	.248 (.308)	.524 (.417)	.132 (.421)	.944 (.941)	2.740 (.342)
reg[3]	.951 (.464)	.283 (.342)	1.321 (.277)	.859 (.394)	.746 (.421)	.818 (.906)	4.54 (.507)
reg[4]	1.058 (.409)	.972 (.344)	1.49 (.273)	1.061 (.383)	.935 (.398)	1.489 (.86)	3.48 (.568)
age_g[2]	1.075 (.383)	1.905 (.228)	1.522 (.216)	1.682 (.324)	.596 (.351)	.179 (.7)	1.232 (.442)
age_g[3]	1.347 (.422)	1.045 (.281)	1.62 (.231)	2.16 (.353)	.794 (.37)	1.387 (.763)	.135 (.334)
mars[2]	1.239 (.378)	.346 (.325)	.638 (.242)	1.036 (.375)	1.786 (.361)	2.111 (.676)	.79 (.321)
mars[3]	.917 (.787)	-.146 .418 ()	.233 (.448)	.013 (.802)	2.565 (.537)	.541 (1.157)	1.525 (1.047)
ed[3]	-.322 (.335)	-.163 (.242)	-.136 (.202)	-.254 (.303)	-.715 (.314)	-.216 (.628)	-.818 (.501)
ws[2]	-.206 (.53)	-.817 (.377)	-.16 (.263)	.094 (.503)	-.0978 (.475)	-2.958 (.845)	-2.005 (.325)
ws[3]	-.361 (.669)	-.894 (.426)	-.211 (.326)	-.175 (.603)	.0720 (.559)	-2.13 (1.084)	1.625 (.339)
inc_q[2]	.001 (.771)	-.113 (.435)	-.417 (.336)	-.078 (.555)	-.0558 (.716)	2.053 (1.286)	-.834 (.261)
inc_q[3]	.55 (.644)	.174 (.454)	.0016 (.32)	.899 (.551)	1.169 (.684)	2.178 (.978)	.432 (.514)
inc_q[4]	-.211 (.613)	-.0513 (.511)	.164 (.32)	-.106 (.556)	-.311 (.658)	1.525 .861 ()	.37 (.591)
inc_q[5]	-.387 (.581)	.535 (.457)	-.141 (.336)	-.107 (.546)	-.808 (.636)	.552 (.725)	-1.359 (.408)
pai[2]	.778 (.460)	1.105 (.288)	.945 (.247)	.809 (.378)	.632 (.404)	-.919 (.755)	5.114 (.382)
pai[3]	.207 (.356)	.687 (.244)	.36 (.202)	.6 (.295)	-.358 (.305)	-1.402 (.632)	-.143 (.454)
cons	23.3 (.774)	19.91 (.609)	20.7 (.450)	22.35 (649)	25.65 (.73)	29.68 (1.351)	32.14 (.599)

Table 2.3: Estimate of the influence to BMI at the mean and some quantiles (NPHS1994_Male)

Coef.	OLS	Quantiles					
		10th	25th	50th	75th	90th	99th
reg^1	.656 (.219)	.55 (.245)	.3 (.267)	.529 (.2359)	.912 (.236)	.8 (.559)	1.092 (1.875)
reg^2	-.377 (.23)	-.733 (.258)	-.8 (.282)	-.358 (.246)	-.1 (.239)	-.3 (.580)	.046 (1.79)
reg^3	.442 (.203)	-.033 (.234)	-.033 (.253)	.386 (.221)	.837 (.216)	.8 (.511)	1.219 (1.606)
reg^4	.303 (.220)	-.15 (.250)	-.1667 (.278)	.357 (.237)	.8 (.230)	.2 (.543)	1.304 (1.790)
age_g^2	.760 (.155)	.7 (.171)	.708 (.198)	.786 (.167)	.725 (.156)	.6 (.355)	1.781 (1.35)
age_g^3	1.3 (.19)	1.166 (.222)	1.408 (.229)	1.471 (.2)	.963 (.193)	.9 (.448)	1.462 (1.351)
$mars^2$	.718 (.174)	.9 (.193)	.95 (.206)	.9 (.176)	.987 (.166)	-.1 (.394)	-.181 (1.392)
$mars^3$	.141 (.239)	.467 (.262)	.567 (.28)	.286 (.246)	.162 (.246)	-.6 (.573)	.392 (2.3)
ed^2	-.265 (.190)	.1 (.208)	-.108 (.208)	-.057 (.184)	-.513 (.18)	-.5 (.429)	-1.3 (1.464)
ed^3	-.877 (.188)	-.283 (.202)	-.592 (.211)	-.886 (.185)	-1.5 (.181)	-1.1 (.422)	-1.55 (1.289)
ws^2	.143 (.251)	.483 (.273)	.37 (.274)	.1 (.231)	-.062 (.228)	-.9 (.521)	-1.573 (2.001)
ws^3	.009 (.320)	-.067 (.345)	-.2 (.358)	.2 (.297)	-.062 (.298)	-.8 (.662)	-.292 (2.561)
inc_q^2	.169 (.369)	-.083 (.351)	.292 (.394)	.557 (.349)	-.237 (.350)	-.3 (.776)	-.454 2.631 ()
inc_q^3	.388 (.319)	.433 (.305)	.767 (.330)	.886 (.292)	-.175 (.295)	-.5 (.674)	-.288 (1.658)
inc_q^4	.596 (.316)	.567 (.3)	.808 (.33)	.929 (.29)	.125 (.294)	.3 (.673)	-.158 (1.918)
inc_q^5	.603 (.340)	.567 (.330)	.883 (.367)	1.36 (.323)	-.075 (.321)	-.36 (.743)	-.812 (2.213)
pai^2	-.0141 (.166)	.3 (.186)	.225 (.197)	-.0857 (.169)	-.275 (.165)	-.2 (.386)	-.431 (1.517)
pai^3	-.173 (.163)	.317 (.189)	.125 (.203)	-.0143 (.175)	-.488 (.17)	-.9 (.405)	-1.246 (1.526)
cons	24.66 (.416)	19.87 (.421)	21.76 (.449)	23.72 (.387)	27.43 (.38)	31.7 (.904)	36.96 (2.921)

Table 2.4: Estimate of the influence to BMI at the mean and some quantiles (CCHSN2004_Male)

Coef.	OLS	Quantiles					
		10th	25th	50th	75th	90th	99th
reg^1	.125 (.538)	.472 (.585)	.458 (.236)	.139 (.429)	-.474 (.559)	.0486 (1.166)	2.828 (1.945)
reg^2	-.832 (.529)	.024 (.509)	-.286 (.232)	-1.045 (.438)	-1.039 (.578)	-.471 (1.151)	-.099 (1.944)
reg^3	-.24 (.518)	-.03 (.512)	-.204 (.213)	-.465 (.412)	-.312 (.538)	.543 (1.163)	1.6 (1.681)
reg^4	.483 (.515)	.132 (.505)	.198 (.226)	.507 (.415)	.07 (.528)	.789 (1.102)	3.718 (2.333)
age_g^2	.973 (.413)	.79 (.351)	.838 (.166)	.616 (.319)	.728 (.429)	.641 (.981)	2.929 (1.31)
age_g^3	1.503 (.435)	1.418 (.376)	1.476 (.17)	.968 (.329)	1.409 (.467)	1.438 (.997)	4.561 (1.259)
$mars^2$	.696 (.419)	1.32 (.309)	1.164 (.148)	1.602 (.299)	.534 (.421)	-.051 (.885)	-3.289 (1.708)
$mars^3$	.322 (.743)	.288 (.599)	.752 (.228)	.549 (.485)	.406 (.63)	.646 (1.453)	-3.552 (2.299)
ed^2	-.989 (.575)	-.422 (.386)	-.616 (.195)	-1.09 (.378)	-1.018 (.489)	-3.02 (1.273)	1.797 (1.616)
ed^3	-1.329 (.526)	-.872 (.366)	-.416 (.174)	-1.085 (.329)	-1.391 (.448)	-4.144 (1.28)	-.918 (1.6)
ws^2	-.767 (.578)	.012 (.371)	-.534 (.199)	-.84 (.464)	-1.932 (.671)	.487 (.987)	-.604 (1.531)
ws^3	-.744 (.594)	-.436 (.421)	-.85 (.210)	-.917 (.482)	-1.183 (.688)	-.05 (.986)	-.149 (1.631)
inc_q^2	-.282 (1.152)	-.168 (.892)	-1.574 (.411)	-1.105 (.919)	.97 (.925)	1.264 (1.655)	3.756 (3.597)
inc_q^3	1.977 (.883)	1.016 (.681)	1.974 (.394)	1.499 (.704)	2.776 (.871)	3.177 (1.602)	3.298 (3.169)
inc_q^4	1.789 (.785)	1.314 (.638)	1.482 (.376)	1.285 (.65)	2.859 (.733)	2.077 (1.352)	.677 3.248 ()
inc_q^5	2.344 (.805)	1.804 (.646)	2.36 (.386)	1.731 (.676)	3.31 (.788)	2.594 (1.446)	.995 (3.047)
pai^2	-1.056 (.361)	-.296 (.349)	-.646 (.167)	-.572 (.294)	-1.393 (.366)	-2.713 (.765)	-3.179 (1.217)
pai^3	-.886 (.411)	.022 (.354)	-.082 (.153)	-.367 (.3)	-1.693 (.389)	-2.15 (.972)	-3.722 (1.257)
cons	26.76 (1.005)	20.16 (.905)	22.17 (.446)	26.23 (.804)	30.05 (1.015)	34.71 (2.047)	39.73 (3.274)

Table 2.5: Estimate of the influence to BMI at the mean and some quantiles (CHS1978_Female)

Coef.	OLS	Quantiles					
		10th	25th	50th	75th	90th	99th
reg[1]	1.625 (.451)	.632 (.357)	1.064 (.413)	1.515 (.561)	2.582 (.510)	2.483 (.981)	4.183 (3.1)
reg[2]	1.341 (.475)	.5 (.35)	.422 (.432)	.725 (.58)	2.354 (.528)	2.853 (1.028)	.926 (1.888)
reg[3]	1.279 (.432)	.427 (.338)	.224 (.407)	1.224 (.561)	2.093 .5 ()	2.613 (1.027)	-.804 (1.593)
reg[4]	.554 (.4)	-.0752 (.362)	-.046 (.394)	.501 (.53)	.936 (.468)	1.092 (.926)	-.329 (1.729)
age_g[2]	2.077 (.393)	.822 (.297)	1.771 (.33)	1.787 .441 ()	1.702 (.396)	3.761 (.825)	1.063 (1.18)
age_g[3]	3.512 (.438)	2.212 (.289)	3.096 (.345)	3.682 (.477)	4.082 (.453)	3.867 (.983)	2.964 (.726)
mars[2]	.615 (.425)	.193 (.376)	.251 (.462)	.347 (.562)	.986 (.523)	1.032 (.912)	1.552 (.971)
mars[3]	.0614 (.641)	.042 (.488)	.238 (.582)	-.268 (.742)	.688 (.684)	.102 (1.388)	-.876 (.984)
ed[3]	-.97 (.359)	.28 (.265)	-.415 (.354)	-.884 (.457)	-1.471 (.396)	-2.259 (.760)	-2.262 (.818)
ws[2]	-.562 (.403)	-.67 (.265)	-.443 (.325)	-.377 (.419)	-1.054 (.391)	-1.13 (.913)	-.193 (1.145)
ws[3]	.429 (.576)	-.133 (.422)	.0297 (.517)	.942 (.633)	.206 (.56)	.344 (1.282)	.89 (.797)
inc_q[2]	-.994 (.585)	.3 (.382)	-.048 (.462)	-.503 (.628)	-1.632 (.577)	-2.12 (1.267)	-.012 (.887)
inc_q[3]	-1.15 (.561)	.397 (.326)	-.01 (.46)	-.484 (.644)	-2.307 (.615)	-2.723 (1.23)	.563 (1.87)
inc_q[4]	-.208 (.619)	.171 (.367)	.118 (.478)	.295 (.652)	-.817 (.615)	1.006 (1.273)	2.222 (.831)
inc_q[5]	-1.225 (.605)	.406 (.334)	-.189 (.513)	-.876 (.663)	-1.549 (.6)	-2.589 (1.26)	-.02 (1.219)
pai[2]	-.472 (.446)	.39 (.294)	.245 (.354)	-.428 (.466)	-.383 (.448)	-1.66 (.987)	-3.391 (.82)
pai[3]	-.406 (.387)	.337 (.259)	.092 (.32)	-.231 (.418)	-.351 (.379)	-2.224 (.827)	-2.96 (1.124)
cons	23.11 (.718)	18.56 (.527)	20.21 (.632)	22.16 (.859)	25.2 (.779)	28.96 (1.613)	36.5 (1.781)

Table 2.6: Estimate of the influence to BMI at the mean and some quantiles (NPHS1994_Female)

Coef.	OLS	Quantiles					
		10th	25th	50th	75th	90th	99th
reg[1]	1.377 (.259)	.5 (.262)	.8 (.265)	1.287 (.303)	2.455 (.412)	1.775 (.594)	1.133 (1.518)
reg[2]	-.121 (.258)	-.344 (.274)	-.367 (.270)	-.226 (.312)	-.025 (.424)	.025 (.613)	1.367 (1.685)
reg[3]	.907 (.243)	.172 (.251)	.25 (.253)	.705 (.288)	1.523 (.393)	1.675 (.568)	1.933 (1.337)
reg[4]	1.146 (.251)	.467 (.255)	.583 (.261)	.976 (.301)	1.959 (.413)	1.7 (.59)	2.167 (1.511)
age_g[2]	.878 (.191)	.367 (.175)	.767 (175)	.889 (.209)	1.32 (.296)	.975 (.444)	.067 (1.153)
age_g[3]	1.789 (.215)	1.172 (.208)	1.817 (.205)	2.268 (.246)	2.259 (.342)	1.35 (.493)	-.067 (1.184)
mars[2]	.687 (.228)	.739 (.231)	.8 (.222)	.679 (.249)	.293 (.342)	.8 (.503)	.4 (1.301)
mars[3]	.67 (.274)	.672 (.273)	.65 (.268)	.713 (.306)	.419 (.415)	1.25 (.586)	1.133 (1.516)
ed[2]	-.163 (.229)	.083 (205)	-.1 (.204)	-.411 (.244)	-.19 (.352)	.15 (.493)	-.367 (1.448)
ed[3]	-.524 (.232)	-.017 (.221)	-.25 (.218)	-.589 (.256)	-.8 (.36)	-.475 (.514)	-.9 (1.394)
ws[2]	-.475 (.199)	-.011 (.181)	-.167 (.182)	-.168 (.22)	-.99 (.313)	-1.3 (.447)	-.433 (1.105)
ws[3]	-.515 (.333)	-.567 (.294)	-.317 (.291)	-.247 (.346)	-.871 (.506)	-.45 (.755)	.2 (1.685)
inc_q[2]	-.302 (.401)	.067 (.324)	-.2 (.328	-.084 (.408)	-.38 (.559)	-.8 (.832)	-.533 (2.08)
inc_q[3]	-.698 (.374)	-.306 (.33)	-.45 (.315)	-.716 (.383)	-.6 (.519)	-1.45 (.769)	-.933 (1.922)
inc_q[4]	-.662 (.373)	-.072 (.324)	-.267 (.314)	-.589 (.385)	-.634 (.520)	-1.425 (.781)	-1.433 (1.882)
inc_q[5]	-1.184 (.412)	-.106 (.367)	-.367 .355 ()	-1.089 (.442)	-1.459 (.601)	-2.55 (.91)	-2.933 (1.894)
pai[2]	-.205 (.192)	.006 (.177)	-.167 (.178)	-.05 (.209)	-.464 (.293)	-.325 (.454)	-1.567 (1.158)
pai[3]	-.917 (.196)	-.044 (.198)	-.267 (.2)	-.747 (.24)	-1.206 (.35)	-1.95 (.497)	-3.367 (1.09)
cons	24.2 (.456)	18.82 (.459)	20.58 (.424)	23.09 (.49)	26.71 (.657)	31.33 (.949)	37.93 (2.596)

Table 2.7: Estimate of the influence to BMI at the mean and some quantiles (CCHSN2004_Female)

Coef.	OLS	Quantiles					
		10th	25th	50th	75th	90th	99th
reg[1]	1.146 (.639)	.794 (.269)	1.483 (.334)	.405 (.364)	1.219 (.702)	2.325 (1.324)	-1.716 (6.61)
reg[2]	-.412 (.711)	-.211 (.259)	.49 (.347)	-.715 (.389)	-1.224 (.788)	-1.59 (1.427)	-.992 (6.573)
reg[3]	-.101 (.601)	-.404 (.255)	.347 (.341)	-.235 (.354)	.052 (.686)	-1.482 (1.21)	-.347 (6.822)
reg[4]	.708 (.602)	.666 (.252)	1.163 (.329)	.265 (.361)	-.0432 (.692)	-.025 (1.228)	2.302 (7.52)
age_g[2]	1.728 (.511)	1.055 (.201)	.877 (.271)	2.025 (.292)	1.705 (.634)	3.020 (.998)	2.336 (3.772)
age_g[3]	1.854 (.509)	1.449 (.195)	1.772 (.278)	2.335 (.295)	1.962 (.645)	3.66 (1.051)	.742 (4.951)
mars[2]	.232 (.464)	.182 (.162)	1.017 (.264)	.4 (.292)	.49 (.634)	-2.367 (.938)	-.461 (3.293)
mars[3]	.682 (.641)	1.269 (.189)	1.57 (.310)	.775 (.390)	1.423 (.925)	-2.316 (1.3)	.569 (5.481)
ed[2]	-1.198 (.706)	.116 (.149)	-.985 (.282)	-2.04 (.352)	-1.434 (.758)	-2.808 (1.41)	-.072 (3.974)
ed[3]	-1.928 (.631)	-.195 (.148)	-1.238 (.27)	-2.72 (.335)	-1.448 (.729)	-3.65 (1.313)	-.717 (3.333)
ws[2]	-.687 (.587)	-.021 (.165)	-.02 (.287)	-.17 (.308)	-.872 (.609)	.33 (1.093)	-2.592 (5.53)
ws[3]	-.786 (.589)	.0764 (.178)	.333 (.304)	.155 (.340)	-.813 (.677)	-.808 (1.126)	-5.195 (4.89)
inc_q[2]	.254 (.876)	-.19 (.315)	-1.705 (.553)	.61 (.716)	-.457 (1.239)	3.313 (1.879)	3.057 (3.986)
inc_q[3]	1.071 (.771)	.474 (.239)	-.575 (.508)	.82 (.687)	1.378 (1.196)	3.661 (1.681)	4.881 (4.053)
inc_q[4]	1.069 (.717)	.886 (.248)	-.683 (.499)	.99 (.681)	.753 (1.165)	3.431 (1.642)	5.552 (4.231)
inc_q[5]	.601 (.833)	.61 (.259)	-.968 (.535)	.405 (.710)	.347 (1.221)	2.279 (1.807)	5.598 (5.345)
pai[2]	-.599 (.459)	.324 (.137)	.07 (.24)	-.245 (.267)	-1.558 (.535)	-1.175 (.964)	-1.47 (2.66)
pai[3]	-1.889 (.419)	.14 (.167)	-.348 (.274)	-1.205 (.302)	-2.672 (.590)	-4.188 (.891)	-9.198 (3.81)
cons	26.99 (1.038)	18.50 (.352)	22.12 (.547)	25.7 (.75)	30.32 (1.39)	36.63 (2.247)	46.5 (5.896)

Table 2.8: Percentage Distribution of BMI

Year	Sex	Under weight	Normal	Overweight	Obese I	Obese II	Obese III
1978	Male	0	48%	40.7%	10.3%	1%	%
	Female	4.1%	56.9%	28.7%	8.4%	1.9%	%
1994	Male	0	41%	46.8%	10.7%	1,5%	%
	Female	2.5%	57.8%	27.7%	9.6%	2.4%	%
2004	Male	0	32.4%	41.1%	18.6%	6.5%	1.4%
	Female	2%	43.5%	30.6%	13.6%	5.8%	4.5%
1978*	Male	1.3	44.6%	42.5%	9.5%	F	F
	Female	3.4%	52%	28.7%	11.5%	2.9%	1.5%
2004*	Male	1.4	33.6%	42%	16.5%	4.8%	1.6%
	Female	2.5%	44.1%	30.2%	14%	5.5%	3.8%

The data of 1978* and 2004* are adopted from Table 2 of Tjepkema (2006)

Table 2.9: Ranking of Factors by Degree of Importance ---Male

Quantile	1978/79							1994/95							2004						
	1	2	3	4	5	6	7	1	2	3	4	5	6	7	1	2	3	4	5	6	7
10	A	R	P	I	M	W	E	A	R	M	W	I	E	P	A	I	M	E	R	W	P
25	A	R	M	P	I	W	E	A	M	R	E	I	W	P	A	I	M	P	R	W	E
50	A	R	M	I	P	W	E	A	M	E	R	I	W	P	M	R	E	I	A	W	P
75	M	I	P	A	R	W	E	E	M	R	A	P	I	W	A	P	W	W	E	R	M
90	I	W	M	P	A	R	E	E	R	A	I	P	W	M	E	P	R	R	A	M	W
95	I	W	P	M	A	R	E	E	R	A	P	M	I	W	E	P	R	A	I	W	M
99	P	R	I	W	A	E	M	E	R	W	A	P	I	M	R	E	P	I	A	M	W

A: age R: region M: marital status W: working status E: education P: physical activity I: family income

Table 2.10: Ranking of Factors by Degree of Importance —Female

Quantile	1978/79							1994/95							2004						
	1	2	3	4	5	6	7	1	2	3	4	5	6	7	1	2	3	4	5	6	7
10	A	R	W	P	I	M	E	A	R	M	W	I	E	P	A	R	I	E	E	P	W
25	A	R	W	P	I	M	E	A	R	M	W	I	P	E	A	R	E	P	I	P	W
50	A	I	R	P	W	M	E	A	R	I	M	P	E	W	A	E	R	I	I	M	W
75	A	W	R	I	P	M	E	A	R	W	P	I	E	M	P	A	R	W	I	M	W
90	A	I	P	R	W	M	E	A	I	R	P	W	M	E	P	R	E	R	I	M	W
95	A	I	P	W	R	M	E	I	P	R	A	M	W	E	P	E	I	A	A	M	W
99	P	R	A	M	W	I	E	P	I	R	E	M	W	A	P	W	R	I	A	M	E

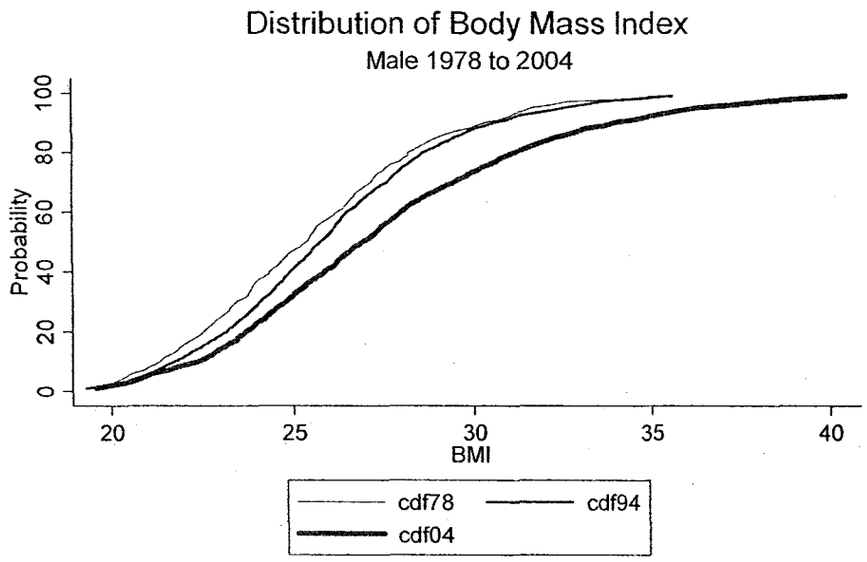
A: age R: region M: marital status W: working status E: education P: physical activity I: family income

Table 2.11: Predicted Percentage Distributions of BMI

Sex	Male			Female		
Year	2007^A	2007^P	2014^P	2007^A	2007^P	2014^P
Under weight	1.2%	0	0	3.9%	1.1%	1.8%
Normal	38.4%	28.8%	23.5%	50.2%	41%	36%
Overweight	39.3%	40.5%	38.8%	25.7%	31.8%	30.5%
Obese I	13.3%	20.8%	21.8%	9.8%	15.7%	17.4%
Obese II	2.7%	7.2%	10.5%	3.4%	5.9%	6.5%
Obese III	1.2%	2.7%	5.4%	1.8%	5.6%	9.6%

A denote estimation from Statistics Canada (CANSIM, Table 105-4009) and *P* denote the predicted percentage distribution of BMI from this study. Our prediction is for people aged 20 and 64, while estimation from Statistics Canada is for people aged 18 and over.

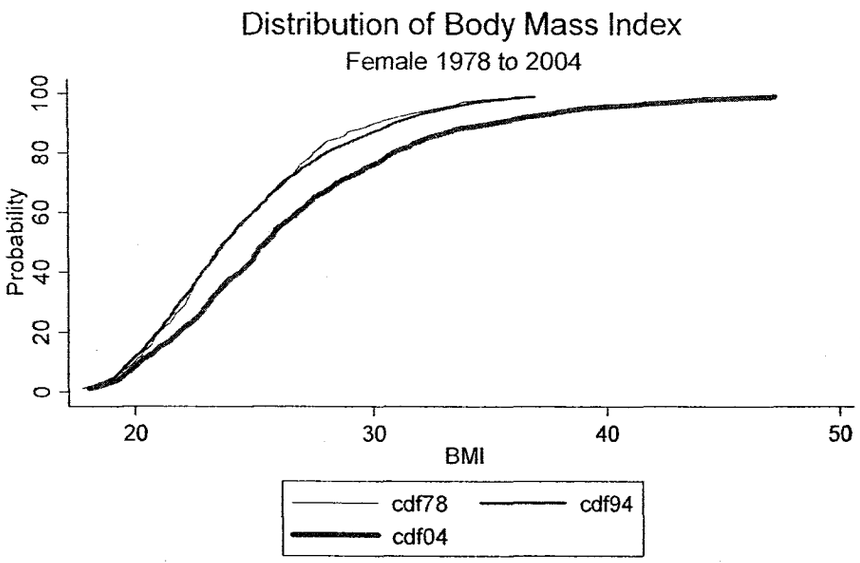
Figure 2.1



This research is based on respondents aged from 20 to 64

Data from CHS 1978 NPHS 1994 and CCHS 2004 Nutrition

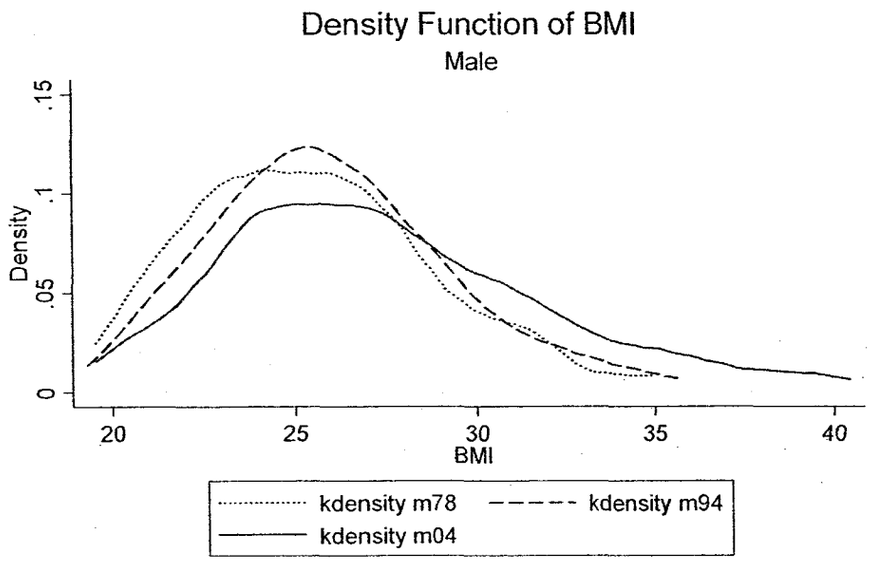
Figure 2.2



This research is based on respondents aged from 20 to 64

Data from CHS 1978 NPHS 1994 and CCHS 2004 Nutrition

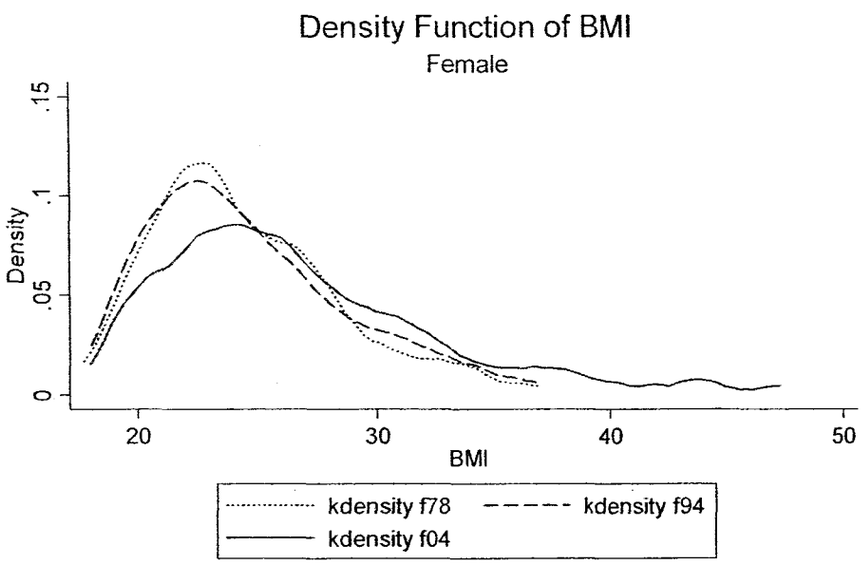
Figure 2.3



This research is based on respondents aged from 20 to 64

Data from CHS 1978 NPHS 1994 and CCHS 2004-Nutrition

Figure 2.4



This research is based on respondents aged from 20 to 64

Data from CHS 1978 NPHS 1994 and CCHS 2004-Nutrition

Figure 2.5

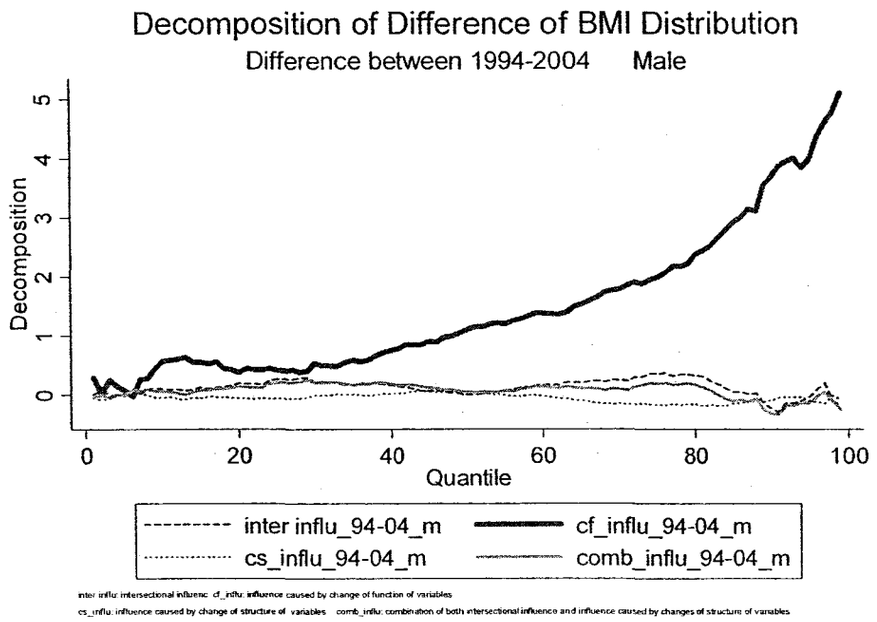
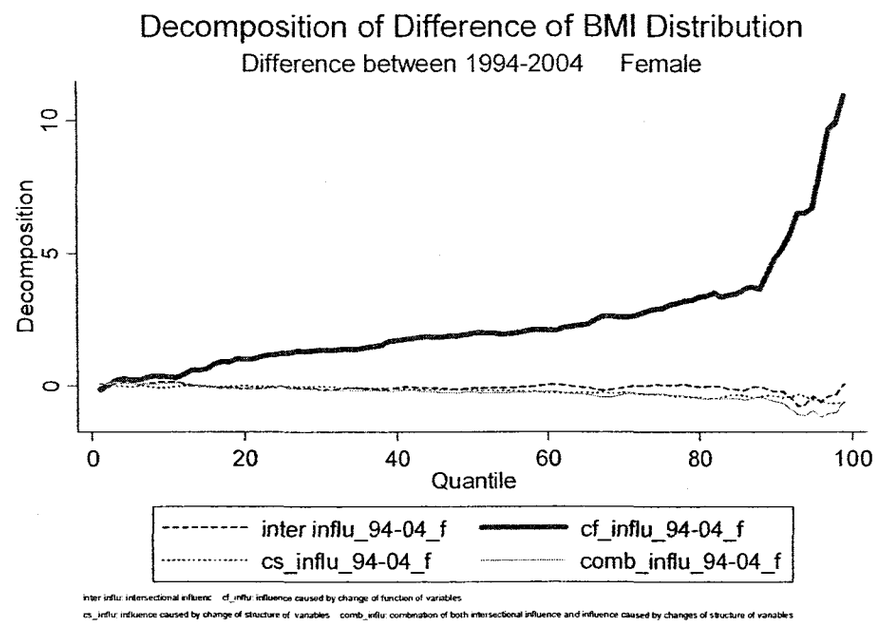


Figure 2.6



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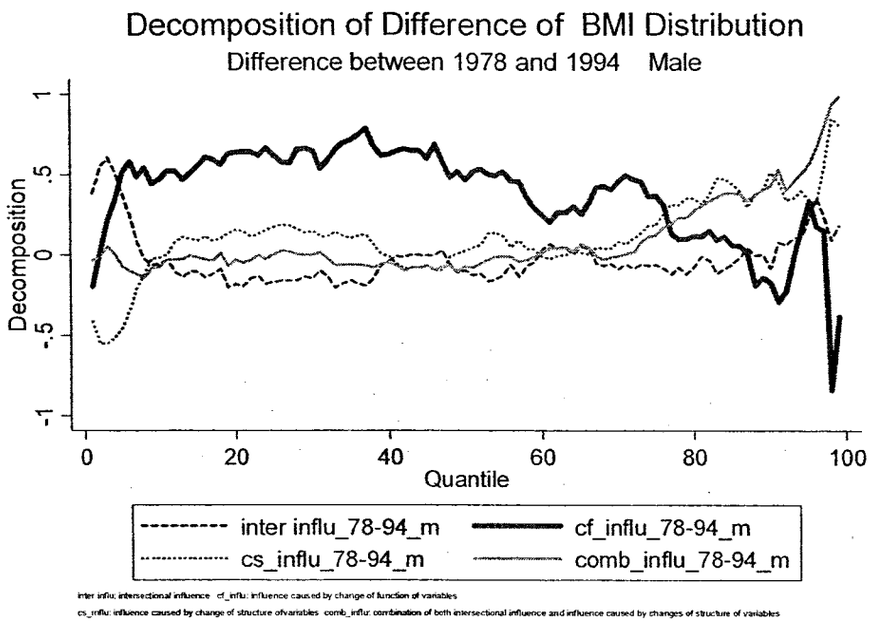


Figure 2.8

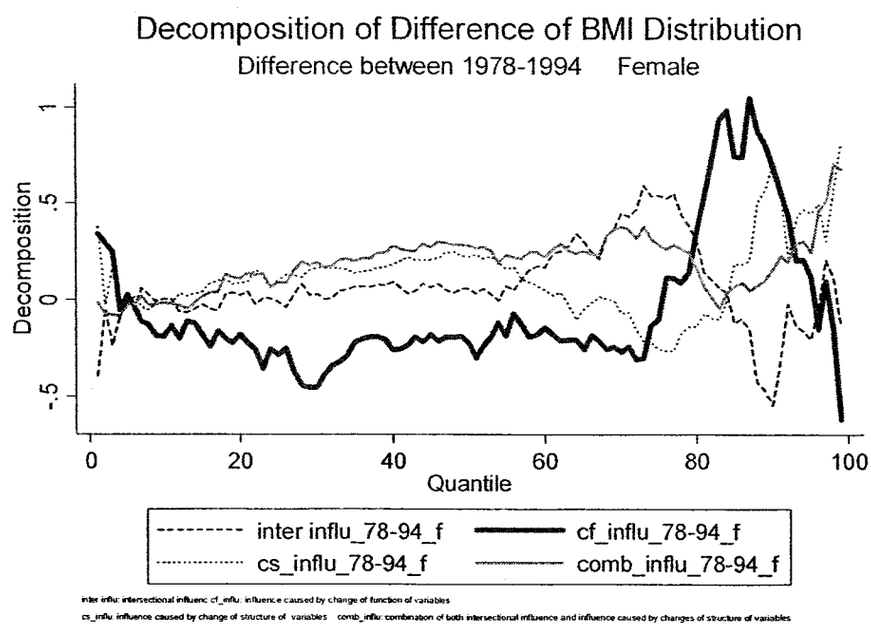


Figure 2.9

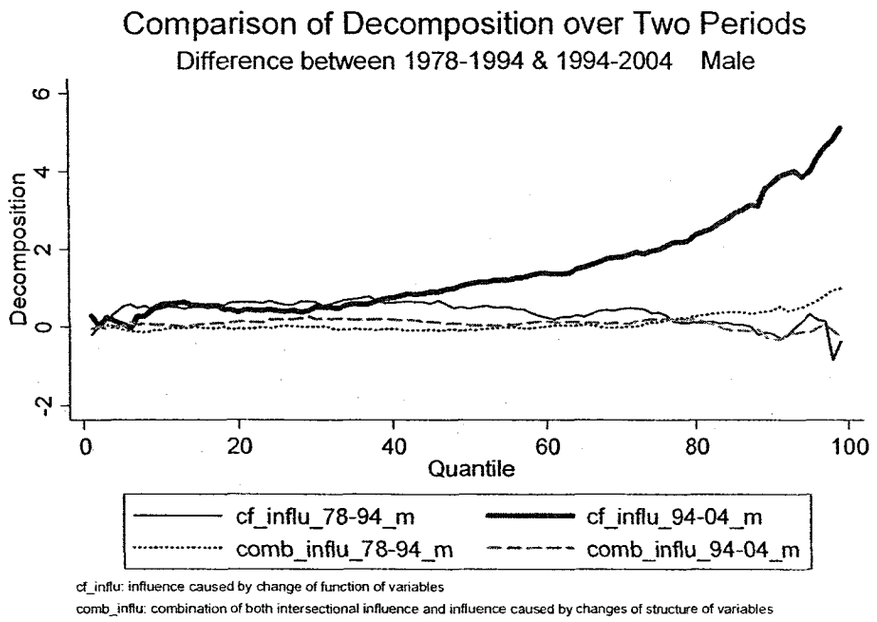


Figure 2.10

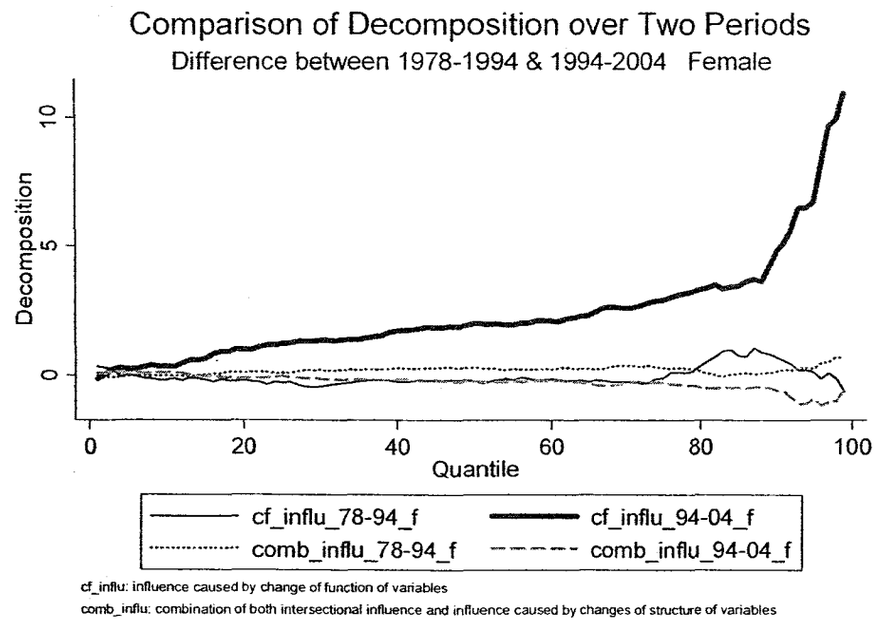
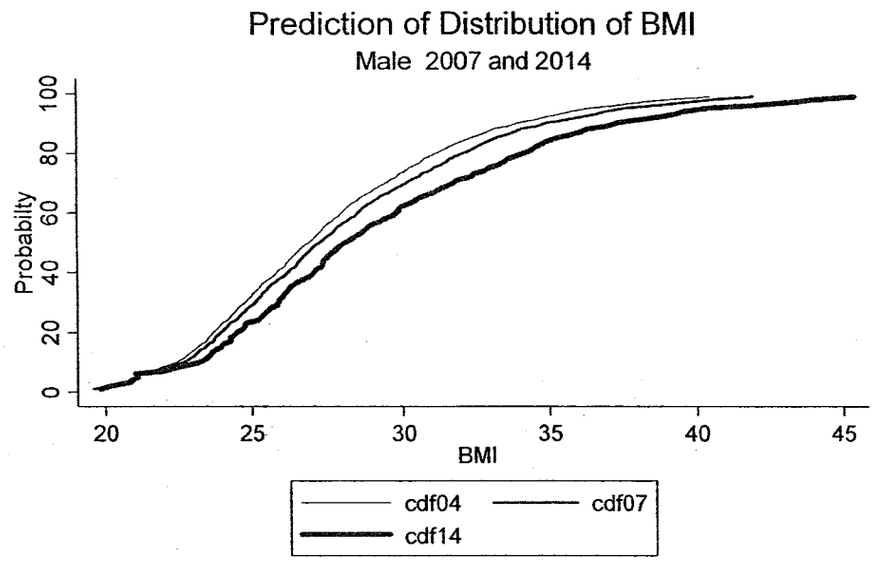


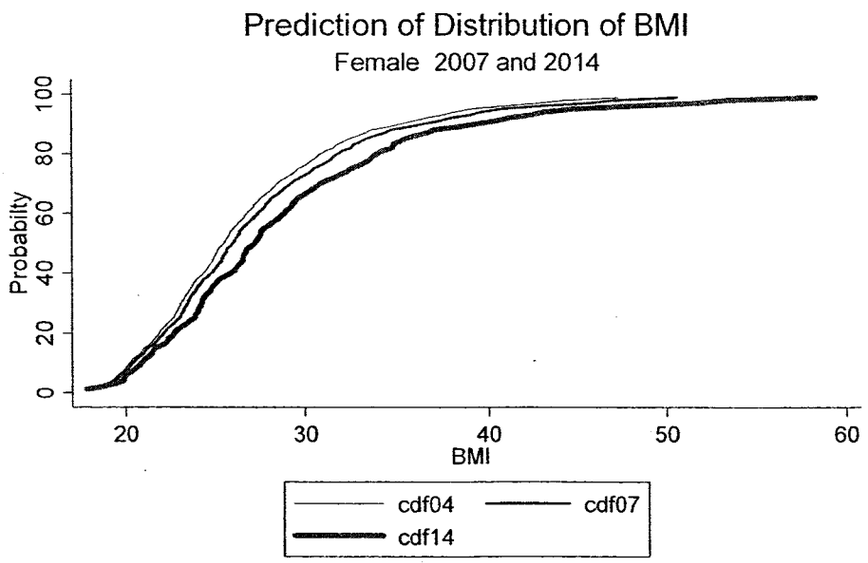
Figure 2.11



This research is based on respondents aged from 20 to 64

Prediction is based on data from NPHS 1994 and CCHS 2004 Nutrition

Figure 2.12



This research is based on respondents aged from 20 to 64

Prediction is based on data from NPHS 1994 and CCHS 2004 Nutrition

CHAPTER 3

The Pattern of Shifts in the BMI for Canadian Adults, 1994-2007

3.1. Introduction

The BMI is defined as weight in kilograms divided by the corresponding height in meters squared. According to the standard for obesity defined by the WHO (World Health Organization, 1997), the BMI for adults is classified into six categories: underweight, normal, overweight, obese I, obese II and obese III [1]. Each category of the BMI represents a different level of health risk (Flegal et al., 2005). Although underweight is also frequently associated with poor health, the sharp increase of obesity in many countries in the last two decades has attracted much attention in research, policy making and the health industry. An obesity epidemic implies that the number of people who shift from normal and overweight to the obese categories over time exceeds the number of people whose shift in the opposite direction over the same time.

Movements between different BMI categories could provide very important information on the obesity problem such as the characteristics of shift patterns among different groups, the spectrum of transition probabilities, and the long-run dynamic equilibrium. This information should prove useful, not only for health care and other

[1] Underweight is defined as $BMI < 18.5$, normal as $18.5 \leq BMI < 25$, overweight as $25 \leq BMI < 30$, obese I as $30 \leq BMI < 35$, obese II as $35 \leq BMI < 40$ and obese III as $BMI \geq 40$.

health related issues, but also for designing more efficient policies for containing and mitigating the obesity problem. For example, better understanding of the obesity determinants and dynamics would prove valuable for predicting the prevalence of some diseases such as cardiovascular disease, hypertension and diabetes II, and evaluating the demand for health care and other medical resources related to these diseases. Despite its important economic and policy implications, there are only a few papers that study the patterns of the shift from one BMI category to another. In this study, we explore the dynamic characteristics of the BMI shift[2] of Canadian adults from the longitudinal National Population Health Survey (NPHS) using Markov Chain analysis.

Markov Chain analysis has a long history in economics. In an early application of this method, Adelman et al. (1958) investigated trends in the concentration and mobility of firms in the U.S. iron and steel industry. Salkin et al.(1975) predicted the population of the western United States by studying the structure of population movements. These early studies assume stationarity, which requires that the transition probability from one state or category to another is constant over time. In order to build the model on a solid footing and to reflect better the dynamics of the actual process, time varying or nonstationary Markov Chains have also been used in studies of social mobility and industry concentration. For example, Zepeda (1995) employed aggregate time varying Markov chain methods to examine the size distributions of dairy farms.

[2]In this paper, the BMI shift is defined as movements among different categories of the BMI.

Whether the BMI shift process is stationary has important implications for future trends in obesity. In particular, stationary shifts in the BMI distribution can generate more reliable predictions of obesity prevalence in the long run. These predictions can then be used to evaluate the impact of obesity on society and the economy. On the other hand, if the BMI shift is not stationary, factors that affect people's weight are not balanced and their influence on weight varies over time. Hence, measures for obesity prevention and control should be adjusted accordingly.

Studies of obesity show that, before the early of 1990s, the prevalence of obesity in Canada has changed slowly, but it has increased dramatically during the 1990s and 2000s. This fact may imply that the BMI shift (or, more precisely, the transition probability matrix corresponding to the BMI shift) before and after 1990s, is not stationary. While this hypothesis can be tested using aggregate data on the prevalence of each category of BMI, it seems more interesting to study the stationarity of the BMI shift since the beginning of the 1990s, when the obesity prevalence in Canada has exhibited substantial increases (Tjepkema, 2006).

Based on micro-level biennial data from the longitudinal NPHS survey from 1994/95 to 2006/07, this study shows that men and women have different patterns of BMI shift. However, the shift patterns for both groups in all two-year periods exhibit a-common positive trend, i.e. people who move to higher categories of BMI are more than those who move to lower categories. Stationary tests show that for men and for women, BMI shifts are not stationary over this period. By conjecturing that people who have different lifestyles may differ in shift patterns, the Markov chain method is also applied to subgroups of men and women based on their activity level. More

specifically, these subgroups are formed by men and women who are active, moderate and inactive in physical activity. The results of the stationary test for these subgroups show that, for both men and women, the BMI shifts of the active and inactive groups are nonstationary but the BMI shifts of the moderate group are stationary. Furthermore, the BMI shifts for the moderate group are ergodic, which is to say there exist unique steady states of BMI for men and for women who are moderate in physical activity. According to the estimates of the transition probability matrices of these two moderate groups, in the steady state, the obesity prevalence for men who are moderate in physical activity is 29.61%, while for women who are at the same level of physical activity, obesity prevalence is 23.37%[3].

These results offer some interesting policy recommendations. While it may be difficult to induce people who are physically inactive to become fully active, it might be feasible to encourage them to switch from being inactive to moderately active. This could be achieved by walking 30 to 60 minutes a day, or taking an hour-long exercise class three times a week (Gilmour, 2007). If all inactive people succeed in this transformation and if the obesity prevalence for active and moderate groups remains unchanged, the obesity prevalence in Canada could be contained below 25% in the long run[4].

[3]The empirical results of this study reveal that the stationary hypothesis for the moderate group is strongly supported for both men and women.

[4]This prediction is based on the reasonable assumption that the obesity prevalence for the moderate group is less than that for the inactive group but higher than prevalence for the active group.

3.2. Data Description

The data used in this study are from household component of the longitudinal NPHS. The NPHS is conducted every two years starting in 1994/1995. The first three cycles (1994/1995. 1996/1997 and 1998/1999) were both cross-sectional and longitudinal. From Cycle 4 (2000/2001) onwards, the survey became strictly longitudinal (collecting health information from the same individuals in each cycle).

The NPHS was designed to provide measures of the level, trend and distribution of the health status of the population, collect data on the economic, social, demographic, occupational and environmental correlates of health, and provide information on a panel of people, who are followed over time, to reflect the dynamic process of health and illness. The data were collected primarily through computer-assisted personal interviews in 1994/1995 and primarily through computer-assisted telephone interviews thereafter. Telephone interviews comprised over 96% of all interviews in 1996/1997 and 1998/1999; over 98% in 2000/2001 and 2002/2003; and more than 99% in 2004/2005 and 2006/2007. The survey does not cover members of the Canadian Forces, people living on Indian reserves or in some remote areas.

The NPHS longitudinal sample includes 17,276 persons of all ages in 1994/1995. These same persons are set to be interviewed every two years over a period of 18 years, i.e. 10 cycles. This study collects data from all seven conducted and released cycles of the NPHS from 1994/1995 to 2006/2007. These seven cycles form six two-year periods, and each of them is between two consecutive cycles. This study focuses mainly on the respondents' BMI shift from 1994/1995 to 2006/2007 and compares

the shifts in each two-year period. Two years is a long enough period to allow adults' weight changes from one category of BMI to another.

Although the NPHS longitudinal sample includes persons of all ages, this study considers only respondents from 18 to 64 years old at each cycle from the first cycle to the sixth cycle of the survey. This means that, for each cycle from the second to the sixth, our data set adds some new candidates who are 18 or 19 and drops some old candidates who are 65 or 66. The reasons for limiting respondents from 18 to 64 years old at each cycle are: first, we are mainly interested in the BMI shift patterns of adults; second, we want to observe Canadians' BMI shift patterns among groups with the same age structure in different periods; and third, compared to children and teenagers younger than 18 and elderly people older than 64, people aged 18 to 64 are stable in their height, and their BMI changes are mainly due to changes in their weight. On the other hand, if we trace all respondents in all seven cycles, then, at each cycle after the second cycle, there are always some respondents who are taken out of the survey because of nonresponse or death which makes the number of respondents at later periods decrease dramatically. Up to the seventh cycle, there is a high percentage leakage of respondents. This may cause serious structural problems when we compare the shift patterns in different periods. In addition, if we trace all respondents in all seven cycles, the respondents' age increases two years at each periods, and there is a 12-year difference in age structure between the sample in the first period and the sample in the last period. Besides keeping the age structure unchanged, our data collecting method has another advantage since it allows the respondents who drop

out of the sample in early periods to return back to the sample later, as long as they participate in any two consecutive cycles.

By using the proposed data collecting method, this study includes 8935 respondents in the first period and 8316, 7995, 7514, 6967 and 6437 respondents in the second to sixth periods, respectively. However, respondents in each sample are not treated equally. In order to make the sample at each period to reflect the structure of the population, same individual may be assigned different weights in different periods[5].

NPHS contains the only longitudinal health survey that has been conducted for more than 12 years in Canada. Since the data in the survey are self-reported, the BMI based on the self-reported weight and height is generally expected to be underestimated. Nevertheless, the estimated probability transition matrices in this study are highly consistent with some previously reported findings that are based on measured data. This observation suggests that the usefulness of the results is not compromised by the self-reported nature of the data. Of course, future research on quantifying the magnitude and the importance of the bias arising from underreporting seems necessary in order to assess how the obesity prevalence estimates and predictions are affected.

3.3. Markov chain Model

Let p_i^t denote the proportion of people who belong to category i of the BMI at cycle t of the survey and let p_{ij}^t be the probability of moving to the BMI in category

[5]In this study, both the number of repondents and weight corresponding to them are used to describe their BMI shift.

j at cycle t, given being in category i of the BMI at cycle $(t-1)$. Then, it follows that

$$(3.1) \qquad \begin{pmatrix} p_1^t \\ p_2^t \\ p_3^t \\ p_4^t \\ p_5^t \\ p_6^t \end{pmatrix} = \begin{pmatrix} p_{11}^t & p_{21}^t & p_{31}^t & p_{14} & p_{15} & p_{61}^t \\ p_{12}^t & p_{22}^t & p_{32}^t & p_{24} & p_{25} & p_{62}^t \\ p_{13}^t & p_{23}^t & p_{33}^t & p_{34} & p_{35} & p_{63}^t \\ p_{14}^t & p_{24}^t & p_{34}^t & p_{44} & p_{45} & p_{64}^t \\ p_{15}^t & p_{25}^t & p_{35}^t & p_{54} & p_{55} & p_{65}^t \\ p_{16}^t & p_{26}^t & p_{36}^t & p_{64} & p_{65} & p_{66}^t \end{pmatrix} \begin{pmatrix} p_1^{t-1} \\ p_2^{t-1} \\ p_3^{t-1} \\ p_4^{t-1} \\ p_5^{t-1} \\ p_6^{t-1} \end{pmatrix}.$$

where $i = 1, 2, 3, 4, 5, 6$ represents the six categories of BMI - underweight, normal, overweight, obese I, obese II and obese III, respectively. Note that in the above probability transition matrix of elements p_{ij}^t, $\sum_{j=1}^{6} p_{ij}^t = 1$, for all i.

Based on the relationship between the BMI states in different cycles of the survey, maximum likelihood estimation can be used to obtain estimates of the probability transition matrix of BMI shift between two survey cycles. If n^t respondents are interviewed in both cycles $(t-1)$ and t of the survey, and if n_i^t is the number of respondents in BMI category i at cycle $(t-1)$, then $\sum_{i=1}^{6} n_i^t = n^t$. Moreover, if n_{ij}^t denotes the number of respondents whose BMI state shifts from category i to category j from cycle $t-1$ to cycle t, then $\sum_{j=1}^{6} n_{ij}^t = n_i^t$.

Assuming that everyone's BMI status is independent, the probability of the above BMI transition of the respondents across all cycles can be written as

$$(3.2) \qquad L = \Pi_{t=2}^{7} \Pi_{i=1}^{6} \Pi_{j=1}^{6} \left(p_{ij}^t\right)^{n_{ij}^t}$$

and the log maximum likelihood function is given by

$$\log L = \sum_{t=2}^{7} \sum_{i=1}^{6} \sum_{j=1}^{6} n_{ij}^t \log p_{ij}^t. \tag{3.3}$$

Using (3.3), the ML estimate of p_{ij}^t can be obtained in a straightforward manner as[6]

$$\hat{p}_{ij}^t = \frac{n_{ij}^t}{n_i^t} \text{ for } i, j = 1, 2, ...6. \tag{3.4}$$

The independence of the transitions in different periods implies that probability transition matrix in each period is only determined by the information from that period. More specifically, the estimate of $\hat{p}_{ij}^t$ depends only on n_{ij}^t and n_i^t and the probability transition matrix from cycle $(t-1)$ to cycle t can be obtained from these estimates. Intuitively, if there are n_{ij}^t out of n_i^t respondents who shift from category i to category j when moving from cycle $(t-1)$ to cycle t, the probability for this shift should be $\frac{n_{ij}^t}{n_i^t}$.

The likelihood function in (3.3) is valid under the assumption that each observation has the same weight. This is typically not the case for survey data where different observations or respondents have different weights and should not be treated equally. In order to account for this fact, weight information should be incorporated into the model. Instead of using the number of respondents in the sample, each observations' weight is used to reassign the frequency that each respondent appears in the sample.

[6]From $\log L = \sum_{i=1}^{6} \sum_{j=1}^{6} n_{ij} \log p_{ij}^k$ and $p_{i6} = 1 - \sum_{j=1}^{5} p_{ij}$, the first order condition can be written as $\frac{\partial \log L}{\partial p_{ij}^k} = \frac{n_{ij}}{p_{ij}^k} - \frac{n_i - \sum_{j=1}^{5} n_{ij}}{1 - \sum_{j=1}^{5} p_{ij}} = 0$ for or $\frac{n_{ij}}{p_{ij}^k} = \frac{n_i - \sum_{j=1}^{5} n_{ij}}{1 - \sum_{j=1}^{5} p_{ij}}$. Since the right side is same for each j, it follows that $\frac{n_{ij}}{n_{il}} = \frac{p_{ij}^k}{p_{il}^k}$ for $j, l = 1, 2, 3, 4, 5$, which implies that $p_{ij}^k = \frac{n_{ij}}{n_i}$ for $i, j = 1, 2, 3, 4, 5, 6$.

If n^t respondents are interviewed in both cycles $(t-1)$ and t of the survey, and ω_{ij}^t[7] is the sum of weights of all respondents whose BMI status shifts from category i to category j from cycle $(t-1)$ to cycle t, we can regard $n^t\omega_i j^t$ as the number of respondents whose BMI shifts from category i to category j from cycle $(t-1)$ to cycle t. The likelihood function under this setting can be expressed as

$$L = \Pi_{t=2}^{7}\Pi_{i=1}^{6}\Pi_{j=1}^{6}\left(p_{ij}^t\right)^{n^t\omega_{ij}^t}. \tag{3.5}$$

The corresponding log maximum likelihood function is

$$\log L = \sum_{t=2}^{7}\sum_{i=1}^{6}\sum_{j=1}^{6} n^t\omega_{ij}^t \log p_{ij}^t \tag{3.6}$$

and the ML estimate of $p_i j^t$ can be written as

$$\hat{p}_{ij}^t = \frac{\omega_{ij}^t}{\sum_{j=1}^{6}\omega_{ij}^t} \text{ for } i, j = 1, 2, ...6. \tag{3.7}$$

3.4. Estimate Results

The probability transition matrix for the six periods described in the data section are estimated by applying the Markov chain method to NPHS longitudinal data. *Table* 3.1 to *Table* 3.6 report the estimated probability transition matrices for all the six periods. Each table contains probability transition matrices for the whole sample and subsamples for men and women.

From the probability transition matrices, it is easy to see that, except for underweight people, people in the other five categories are more likely to stay in their

[7]For each period or t, $\sum_{i=1}^{6}\sum_{j=1}^{6}\omega_{ij}^t = 1$.

own categories[8]. Although most people with normal weight are still normal after 2 years, more than 90% of the people who left this group become overweight or obese. For overweight people, the possibility of becoming obese is not very different from that of going to normal or underweight; their differences are less than 1.5% in most of the periods. In addition, in three of the six periods, more overweight people become obese, and in the other three periods, more overweight people become normal or underweight. Obese people who left their categories, regardless of their obese category, are more likely to shift to the lower categories of BMI. However, because the proportion of normal and overweight people is much higher than the proportion of obese people[9], the number of people who transfer from the normal or overweight categories to the obese categories is much more than people who move in the opposite direction. Hence, the prevalence of obesity keeps increasing during the period under investigation.

For both men and women, the normal and overweight categories are the most stable categories; however, if we observe men and women in these two categories separately, their tendencies for staying in and leaving their original categories are different. Normal weight women are more likely to preserve their BMI status than normal weight men; in contrast, overweight men are more likely to remain in their BMI category than overweight women. Furthermore, overweight women are more likely to go back to normal weight than overweight men and overweight men are more likely to be obese than overweight women[10]. Compared to men who are in

[8]Normal and overweight people are most stable in their weight with more than 81% normal-weight people and 75% overweight people do not change their categories.
[9]The proportion of people in the lower obese category is higher than that in the higher obese category.
[10]All these conclusions on BMI shift are obtained from two-year-period shifts.

obese classes I and II, women who belong to these categories are more likely to be more obese; however, compared to men who are in obese class III, women in this category are more likely to go back to lower obese classes.

With these BMI shifts, what BMI distribution can we expect in the next period? If we look at the BMI percentage distribution at the beginning of each period in *Table* 3.7, it is easy to see that, for both men and women, the prevalence of normal weight declines over time, while the prevalence of each obesity category continually increases. Because overweight men are more likely to be obese than overweight women, men have higher prevalence to be in obese class I than women. Women in obese classes I and II are more likely to be more obese than men in these two classes and the women's group has a higher prevalence to be in obese class II and III than men.

3.5. Future Obesity Prevalence in Canada

Studies of obesity show that prior to the beginning of 1990s', the prevalence of obesity in Canada changes slowly; in contrast, after 1990s', obesity prevalence increases dramatically (Tjepkema, 2006). This fact may imply that the BMI shift differs in the periods before and after 1990, and the BMI shifts after 1990 reflect the BMI shift patterns of the obesity epidemic in Canada.

How worse could the obesity prevalence be if the current BMI shift were sustained? Will the prevalence of obesity continuously increase? Answers to these questions have very important implications for the economy; for example, how many resources need to be allocated to the treatment of obesity-related illness and how much output would the economy lose because of the absence from work due to the increasing prevalence of obesity.

The answer to the first question is meaningful only in the short run. To predict obesity prevalence in the short run (ten years, for instance) it is convenient and reasonable to assume that the BMI shift will remain unchanged, even if it is actually a nonstationary process. This setup is often used in the application of the Markov chain analysis in the literature. Based on the assumption that the BMI probability transition matrices for men and women are unchanged in the ten years from 2006, the percentage distribution of BMI for men and women in 2008, 2010, 2012, 2014 and 2016 are given in *Table* 3.8[11].

The prediction reported in *Table* 3.8 shows that in 2016 the Canadian men's and women's obesity prevalence would reach 32.2% and 25.2% respectively. In the next 10 years, the prevalence of all three obese classes will keep increasing for both men and women while the prevalence of normal weight will keep decreasing. For women, the prevalence of overweight will also continually increase in this period; however, for men, the prevalence increases initially but then starts to decrease.

Will the prevalence of obesity continuously increase? This question concerns obesity prevalence in the long run. If a Markov chain is ergodic, then there exists a steady state or an equilibrium and the equilibrium distribution is unique and independent of the initial configuration (see Chapter 11 in Hamilton, 1994). Although nonstationary Markov chains can also be ergodic, the small number of observations makes it difficult to evaluate whether the system is ergodic or not. In contrast, if we

[11]The prediction is based on the estimated probability transition matrix from 2004/05 to 2006/07 and the initial BMI percentage distribution in 2006.

know that a Markov chain is stationary, as long as its probability transition matrix is irreducible and aperiodic[12], it is ergodic.

3.5.1. Stationarity Test and Obesity Prevalence in The Long Run

Is the BMI shift in Canada stationary after 1990? To answer this question, the stationarity of the BMI shift is tested using the test that was proposed by Anderson and Goodman (1957). The test is based on estimates of the six probability transition matrices in the period from 1994/95 to 2006/07 for men, women and the whole sample.

Suppose that the probability transition matrices are stationary over the survey period from 1994/95 to 2006/07, and p_{ij} denotes the probability of an individual's BMI moving from category i to category j. Then the null hypothesis can be written as $H_0 : p_{ij}^t = p_{ij}$ $(t = 2, 3, ..7)$[13]. Under the null hypothesis, the likelihood function (3.5) takes the modified form

$$L = \Pi_{t=2}^{7}\Pi_{i=1}^{6}\Pi_{j=1}^{6}\left(p_{ij}\right)^{n^t\omega_{ij}^t} \tag{3.8}$$

and the estimates corresponding to this new likelihood function are given by

$$\hat{p}_{ij} = \frac{\sum_{t=2}^{7} n^t\omega_{ij}^t}{\sum_{k=1}^{6}\sum_{t=2}^{7} n^t\omega_{ik}^t}. \tag{3.9}$$

[12]If one of the eigenvalues of the probability transition matrix is unity and the remaning eigenvalues are less than one, then the Markov chain is ergodic.

[13]In order to make the notation consistent, p_{ij}^t that starts with (2) represents the transitional probability between cycle $t-1$ and cycle t.

The estimate $\hat{p}_{ij}$ is constructed as the ratio of the total number of respondents whose BMI shifts from category i to category j ($\sum_t = 2^7 n^t \omega_i j^t$) and the total number of respondents whose BMI is i at the beginning of each period ($\sum_k = 1^6 \sum_t = 2^7 n^t \omega_i k^t$).

Following Anderson and Goodman (1957), the stationary test for the BMI shift has the form

$$\chi^2 = \sum_{i=1}^{6} \sum_{t=2}^{7} \sum_{j=1}^{6} n^t \omega_{ij}^t (\hat{p}_{ij}^t - \hat{p}_{ij})^2 / \hat{p}_{ij} = \sum_{i=1}^{6} \chi_i^2. \tag{3.10}$$

If the null hypothesis is true, χ^2 and χ_i^2 are distributed as chi-square with $(T-2)[m(m-1)]$ and $(T-2)(m-1)$ degrees of freedom, respectively, where T is the number of cycles of the survey, and m is the number of states. The test statistics (3.10) covers the whole period from 1994/1995 to 2006/2007 and T and m are equal to 7 and 6, respectively[14].

Table 3.9 presents the results of the stationary tests for men, women and their combined sample over the six periods. The results of the stationary tests show that the BMI shifts appear to be nonstationary. The BMI shift patterns for men and the combined sample tend to be more unstable than those for women. Since it is difficult to verify if the BMI shifts are ergodic when they are nonstationary, this means that the future prevalence of obesity obtained from the man's and women's nonstationary BMI shifts would be unreliable.

It is natural to think that people with different characteristics may have different BMI shift patterns which might help us to identify subgroups with stationary BMI

[14]Tests for stationarity over several periods can be tested in the same way. For example, if we want to test the stationarity over the second to the fourth period, we just need to change t in both (3.9) and (3.10) from 3 to 5.

shifts. For example, lack of exercise and energy expense is believed to be one of the direct causes that leads to obesity. As a result, it seems reasonable to assume that people who differ in physical activity may also differ in their BMI shift. Using levels of the energy expense, people can be categorized as active, moderate and inactive in physical activity[15]. Taking a closer look at these three subgroups separately for men and women, we find that, for the overweight and obese men and women, the more active they are in physical activity, the more likely they are to move to the lower BMI categories. The stationary tests corresponding to these subgroups show that, for both men and women, the BMI shifts of the active and inactive subgroups are nonstationary; however, the BMI shifts of the moderate subgroups are stationary[16]. Furthermore, the probability transition matrices of the BMI shift for these two moderate subgroups are irreducible and aperiodic; hence, their BMI shifts are ergodic. This implies that there exist unique steady states for men and women who are moderate in physical activity. The BMI percentage steady-state distribution for these subgroups are given in *Table* 3.11. At steady state, the obesity prevalence for men who are physically moderately active is 29.61%, while for women who are also physically moderately active, it is 23.37%.

Both previous studies and our analysis of the longitudinal NPHS data indicate that, for both men and women, obesity prevalence is negatively related to their physical activity index (*Tables* 3.12 and 3.13). Based on this fact, it is reasonable to assume

[15]Acoording to the difinition that was adopted by NPHS, active in physical activity is defined as using 3 or more kilocalories per kilogram of body weight per day; moderately active is defined as using 1.5 to less than 3 kilocalories per kilogram of body weight per day; inactive is defined as using less than 1.5 kilocalories per kilogram of body weight per day.

[16]*Table* 3.10 presents the results of the stationary tests for the active, moderate and inactive men, women and their combined sample over the six periods.

that, in the long run, even men or women who are physically moderately active would reach their steady state and their obesity prevalence would still be higher than that of the active group and lower than that of the inactive group. Hence, in the long run, the obesity prevalence for active Canadian men and women can be kept below 29.61% and 23.37%, respectively, while obesity prevalence for inactive Canadian men and women is expected to be higher.

People who are inactive in physical activity account for the biggest portion of the Canadian population. Close to half (48%) of Canadians aged 12 or older (12.7 million people) were inactive in their leisure time in 2005 (Gilmour, 2007). This means that obesity prevalence in Canada is predominantly influenced by physically inactive people.

A policy recommendation that naturally emerges from these findings is to encourage or provide incentives to inactive obese people to become moderately physically active. This could be achieved by walking 30 to 60 minutes a day, or taking an hour-long exercise class three times a week. If these people could become moderately active, then obesity prevalence could be decreased gradually. We could evaluate this effect by applying the BMI shift pattern of moderate group to the inactive group. More specifically, the estimated probability transition matrices (which do not reject the stationary hypothesis) are used for the inactive group BMI distribution at the initial year, 1994/95. Applying the formula (3.1) recursively, the BMI percentage distributions at each of the following periods are given in *Table* 3.14 for men and *Table* 3.15 for women. In order to verify if physical activity is negatively related to \obesity prevalence, the probability transition matrices of moderate groups are also

applied to the active groups and the resulting BMI distributions are also reported in *Table* 3.14 and *Table* 3.15. Comparing these calibrated BMI percentage distributions to the actual BMI distributions in *Table* 3.12 and *Table* 3.13, it is easy to see that for the inactive group the calculated obesity prevalence is always lower than their actual counterpart in the periods following 1998/99. For example, if women who were physically inactive in 1994 could become physically moderately active, then their obesity prevalence in 2004 would decrease by more than 2%. In contrast, the calculated obesity prevalence for the active group is higher than its actual counterpart in most of the subsequent periods which lends support to the hypothesis that the relationship between physical activity and obesity prevalence is negative.

From these results, we can conclude that imposing the moderate group's shift pattern will slow down the increase of obesity prevalence of the inactive group although its obesity prevalence would still increase. If they are successfully shifted from inactive to moderately active, their obesity prevalence would approach that of moderate people in the long run. Given the low obesity prevalence of the active group and the difference in obesity prevalence between moderate men and women, the obesity prevalence among Canadian adults could be confined to around 25% in the long run as long as all inactive Canadian adults become moderately active.

In this paper, only BMI shift patterns for active, moderate and inactive subgroups are studied[17]. However, this method could be applied to any other subgroups which are formed using a different categorization such as race, age, resident region, and socioeconomic status such as family income and education.

[17]The respondents included in this study are between 18 to 64 years old.

3.6. Discussion

It is often acknowledged that self-reported BMI is not reliable because a large part of respondents, especially heavy respondents, tend to understate their weight or overstate their height. For example, Gorber et al. (2007) found that, in the Canadian Community Health Survey, the proportion of respondents reporting being obese was estimated to be 18% for those who were interviewed in person compared with an estimate of only 13% for those who were asked the same questions by phone. In NPHS, height and weight are self-reported and they are obtained by phone. While this may induce some biases in the estimation results, the obtained probability transition matrices are fairly consistent with some previous findings that are based on measured data.

The probability transition matrices from this study show that women with normal weight are more likely to keep their BMI status than normal weight men while overweight men are more likely to keep their BMI status than overweight women. Further, overweight women are more likely to go back to normal weight than overweight men, and overweight men are more likely to become obese than overweight women. Finally, women who are in obese class I and class II are more likely to become more obese than men who belong to the same categories. This is highly consistent with the previous findings on the BMI distribution of the Canadian population which can be summarized as follows: (i) normal-weight women are a higher percentage than normal-weight men; (ii) men who are in the overweight and obese class I categories are a higher percentage than women in these two categories; and (iii) women have a

higher prevalence in obese classes II and III than men[18]. Why is the BMI shift based on the understated BMI still consistent with the objectively measured tendency in the BMI distribution? A possible explanation is that overweight and obese people use similar rates to underreport their weight and over-report their height, and these rates are stable over time. Although the estimate of the BMI shift may not yield an exact estimation of the BMI distribution, it does reflect the relative movements across the different BMI categories.

Stationary tests show that the BMI shifts for the active and inactive groups are nonstationary but the BMI shift for the moderate group is stationary. This result still holds when the categories of BMI are reduced to four categories which are not obese, obese class I, obese class II and obese class III. Compared to the moderate group, people in the active group do not easily keep active at the same level all the time, especially those who are very active in physical activity. When there is an unexpected change in their life or in the environment, these people may reduce their physical activities dramatically while their food consumption do not change much. In other words, the BMI of these people are more sensitive to unexpected changes than people in the moderate and inactive groups. The stationary tests for both active men and women show that χ_2^2 is the component that leads to significantly rejections of the null hypothesis[19]. This means that, in the active group, people with normal weight are the most unstable subgroup in terms of BMI shift and their mutableness is the main source of the nonstationarity of the BMI shift for the active group. This result

[18]See Table 2 of Tjepkema (2006). The results in the table show that, in 2004, the prevalence of normal weight, overweight, obese class 1, 2 and 3 for man are 33.6%, 42%, 16.5%, 4.8% and 1.6%, respectively, while the cooresponding percentages for women are 44.1%, 30.2%, 14%, 5.5% and 3.8%.

[19]As mentioned above, under the null hypothesis, χ_i^2 $(i = 1, 2, 3, 4, 5, 6)$ in the stationarity test (3.10) are distributed as chi-square with $(T - 2)(m - 1)$ degrees of freedom.

supports the previous interpretation of the nonstationarity for active group which is that people who are very physically active are unstable in their BMI shift.

A closer inspection of the stationary test for physically inactive people reveals that χ_3^2 is the largest part and χ_2^2 is the second largest part that contribute to the nonstationarity of BMI shift for the inactive group. This implies that, in the inactive group, overweight and normal weight people are the most unstable subgroups in terms of BMI shift. This finding may be difficult to rationalize but one possible reason is that inactive people who are in the overweight and normal categories are sensitive to changes in their weight. They may often struggle with their food intake and live in a process of frequent transformation of diet control and indulging in eating which leads to irregular changes in their weight.

In fact, even the stationary test results for the moderate group show that, χ_2^2 for men and χ_3^2 for women contribute most to the significant rejections of the null hypothesis. This indicates that the BMI shift of normal and overweight people is the main source of the changes in the BMI distribution. This means that, as a strategy to address the obesity epidemic, effort to prevent the occurrence of new obese cases is much more effective and important than effort to reduce the number of existing obese people.

3.7. Conclusion

By applying the Markov chain method to the seven cycles of the longitudinal National Population Health Survey (NPHS), this study investigates the BMI shift patterns of Canadian adults aged 18 to 64 during the period 1994/95 - 2006/07 which is characterized by a dramatic increase of obesity prevalence. The estimated

probability transition matrices indicate that men and women are different in BMI shift patterns. However, the shift patterns for both groups in all periods exhibit a common positive trend, i.e., the people who move to higher categories of BMI are more than those who move to lower categories. Stationary tests show that for both men and women, BMI shifts are not stationary over this period. Interestingly though, the BMI shifts of moderately active subgroups of both men and women are stationary and ergodic. Based on the estimated probability transition matrices for the moderately active groups, the obesity prevalence at steady state (long run) is predicted to be 29.61% for men and 23.37% for women.

Some of the main findings can be summarized as follows. First, women with normal weight and overweight men have the highest probability to remain in their BMI category. Second, because the proportion of normal and overweight people is much higher than the proportion of obese people, the number of people who move from the normal or overweight categories to the obese category is much more than people moving in the opposite direction. Hence, the prevalence of obesity keeps increasing over time. Finally, the stationary tests show that the BMI shift of normal and overweight people is the main source of the changes in the BMI shift and the effort to prevent the occurrence of new obese cases would prove much more effective than the effort to reduce the number of existing obese people.

People who are physically inactive account for the biggest portion of the Canadian population. Compared with people who are physically active or moderate, the obesity prevalence for this group is substantially higher. Hence, obesity prevalence in Canada is most closely associated with physically inactive people. Health policies that aim at

dealing with the obesity problem should focus more on physically inactive people and design incentives that would encourage them to become moderately or fully physically active. Our calculations show that if all inactive people become moderately active, assuming that obesity prevalence for active and moderate groups remains unchanged, obesity prevalence in Canada could be confined below 25% in the long run.

Table 3.1: Probability Transition Matrix 1994/95 to 1996/97

Whole

	Underweight	Normal	Overweight	Obese I	Obese II	Obese III
Underweight	43.43%	1.55%	0.01%	0.01%	0	0
Normal	54.97%	83.93%	13.1%	2.56%	0.92%	1.14%
Overweight	1.13%	13.85%	78.25%	24.16%	2.67%	1.96%
Obese I	0.19%	0.64%	8.29%	64.51%	33.85%	6.89%
Obese II	0.28%	0.02%	0.28%	8.2%	49.98%	30.08%
Obese III	0	0.01%	0.07%	0.56%	12.58%	59.93%

Men

	Underweight	Normal	Overweight	Obese I	Obese II	Obese III
Underweight	51.83%	0.8%	0	0	0	0
Normal	45.73%	80.17%	11.3%	2.07%	0	0
Overweight	2.44%	18.27%	81.07%	27.50%	3.08%	0
Obese I	0	0.76%	7.53%	64.36%	32.55%	1.7%
Obese II	0	0.01%	0.11%	56.91%	53.05%	25.61%
Obese III	0	0	0	0.37%	11.32%	72.69%

Women

	Underweight	Normal	Overweight	Obese I	Obese II	Obese III
Underweight	41.95%	2.13%	0.03%	0.02%	0	0
Normal	56.6%	86.84%	16.42%	3.17%	1.62%	1.78%
Overweight	0.89%	10.43%	73.09%	19.9%	2.36%	3.07%
Obese I	0.23%	0.56%	9.68%	64.71%	34.86%	9.83%
Obese II	0.33%	0.04%	0.59%	11.4%	47.61%	32.6%
Obese III	0	0.01%	0.19%	0.8%	13.54%	52.7%

Table 3.2: Probability Transition Matrix 1996/97 to 1998/99

Whole						
	Underweight	**Normal**	**Overweight**	**Obese I**	**Obese II**	**Obese III**
Underweight	51.18%	1.17%	0.04%	0	0	0
Normal	48.54%	82.94%	12.92%	1.72%	0.38%	0
Overweight	0.28%	15.52%	75.52%	20.92%	3.04%	2.82%
Obese I	0	0.20%	11.25%	65.07%	27.15%	7.13%
Obese II	0	0.07%	0.26%	11.33%	59.45%	18.55%
Obese III	0	0.11%	0.01%	0.1%	9.98%	71.5%
Men						
	Underweight	**Normal**	**Overweight**	**Obese I**	**Obese II**	**Obese III**
Underweight	39.94%	0.7%	0.03%	0	0	0
Normal	60.06%	77.61%	11.58%	1.72%	0.25%	0
Overweight	0	21.41%	78.28%	21.58%	3%	0
Obese I	0	0.2%	9.94%	64.58%	28.86%	10.36%
Obese II	0	0	0.16%	11.62%	56.81%	13.29%
Obese III	0	0.08%	0	0.49%	11.07%	76.34%
Women						
	Underweight	**Normal**	**Overweight**	**Obese I**	**Obese II**	**Obese III**
Underweight	54.5%	1.52%	0.03%	0	0	0
Normal	45.13%	86.97%	15.45%	1.73%	0.44%	0
Overweight	0.37%	11.07%	70.3%	20.07%	30.66%	4.77%
Obese I	%	0.21%	13.72%	65.7%	26.95%	4.88%
Obese II	%	0.1%	0.46%	10.95%	61.09%	22.22%
Obese III	%	0.12%	0.03%	1.55%	9.31%	68.13%

Table 3.3: Probability Transition Matrix 1998/99 to 2000/01

Whole

	Underweight	Normal	Overweight	Obese I	Obese II	Obese III
Underweight	44.87%	1.95%	0.29%	0.46%	0	0
Normal	53.83%	81.1%	11.96%	1.94%	1.24%	1.48%
Overweight	1.29%	16.24%	75.35%	24%	1.09%	3%
Obese I	0	0.39%	11.57%	63.32%	23.05%	7.77%
Obese II	0	0.24%	0.66%	9.51%	56.59%	13.8%
Obese III	0	0.08%	0.15%	0.76%	18.03%	73.95%

Men

	Underweight	Normal	Overweight	Obese I	Obese II	Obese III
Underweight	13.06%	1.56%	0.35%	0.44%	0	0
Normal	81.4%	77.19%	9.62%	1.79%	1.75%	0
Overweight	5.54%	20.41%	78.98%	23.66%	0.37%	4.55%
Obese I	0	0.48%	10.23%	64.75%	24.17%	4.04%
Obese II	0	0.36%	0.66%	8.96%	64.23%	6.14%
Obese III	0	0	0.15%	0.39%	9.47%	85.28%

Women

	Underweight	Normal	Overweight	Obese I	Obese II	Obese III
Underweight	54.62%	2.22%	0.18%	0.5%	0	0
Normal	45.38%	83.83%	16.34%	2.13%	0.86%	2.44%
Overweight	0	13.33%	68.55%	24,42%	1.63%	2%
Obese I	0	0.33%	14.1%	61.56%	22.1%	10.17%
Obese II	0	0.15%	0.66%	10.18%	50.74%	18.74%
Obese III	0	0.14%	0.13%	1.22%	24.57%	66.65%

Table 3.4: Probability Transition Matrix 2000/01 to 2002/03

Whole						
	Underweight	**Normal**	**Overweight**	**Obese I**	**Obese II**	**Obese III**
Underweight	40.83%	1.67%	0.17%	0	0	0
Normal	50.3%	81.54%	11.14%	1.54%	0.68%	5.4%
Overweight	4.75%	15.75%	75.99%	20.47%	4.8%	4.29%
Obese I	3.22%	0.6%	11.75%	66.41%	27.62%	3.26%
Obese II	0.9%	0.34%	0.89%	10.6%	47.99%	19.3%
Obese III	0	0.11%	0.07%	0.97%	18.91%	67.76%
Men						
	Underweight	**Normal**	**Overweight**	**Obese I**	**Obese II**	**Obese III**
Underweight	28.42%	1.25%	0.25%	0	0	0
Normal	51.08%	77.23%	10%	0.88%	0	5.31%
Overweight	15.4%	19.87%	77.23%	20.28%	5.27%	7.24%
Obese I	4.97%	0.55%	12.12%	69.13%	32.14%	4.96%
Obese II	0.13%	0.83%	0.39%	9.23%	48.07%	13.23%
Obese III	0	0.26%	0	0.48%	14.52%	69.25%
Women						
	Underweight	**Normal**	**Overweight**	**Obese I**	**Obese II**	**Obese III**
Underweight	46.09%	1.95%	0	0	0	0
Normal	50%	84.52%	13.3%	2.37%	1.35%	5.44%
Overweight	0.23%	12.89%	73.61%	20.7%	4.34%	2.7%
Obese I	2.48%	0.63%	11.05%	63.02%	23.21%	2.34%
Obese II	1.24%	0	1.84%	12.32%	47.92%	22.57%
Obese III	0	0	0.2%	1.58%	23.18%	66.95%

Table 3.5: Probability Transition Matrix 2002/03 to 2004/05

Whole						
	Underweight	**Normal**	**Overweight**	**Obese I**	**Obese II**	**Obese III**
Underweight	39.58%	1.39%	0.03%	0	0	0
Normal	59.63%	82.48%	13.06%	1.14%	0.86%	1.22%
Overweight	0.79%	15.47%	76.05%	19.41%	2.55%	1.69%
Obese I	0	5.61%	10.3%	68.2%	33.03%	8.59%
Obese II	0	0.34%	0.32%	10.1%	50.72%	15.72%
Obese III	0	0.1%	0.23%	1.13%	12.84%	72.77%
Men						
	Underweight	**Normal**	**Overweight**	**Obese I**	**Obese II**	**Obese III**
Underweight	36.34%	1.19%	0	0	0	0
Normal	62.25%	77.3%	9.08%	1.3%	0.64%	1.23%
Overweight	1.42%	20.73%	82.04%	18.71%	0.98%	0
Obese I	0	0.69%	8.68%	72.75%	29.01%	3.46%
Obese II	0	0	0.1%	6.79%	56.38%	19.1%
Obese III	0	0.09%	0.11%	0.46%	13%	76.22%
Women						
	Underweight	**Normal**	**Overweight**	**Obese I**	**Obese II**	**Obese III**
Underweight	40.49%	1.53%	0.08%	0	0	0
Normal	58.9%	86.15%	20.02%	0.9%	1.07%	1.21%
Overweight	0.61%	11.74%	65.64%	20.57%	4.06%	2.45%
Obese I	0	0.47%	13.13%	61.14%	36.89%	10.91%
Obese II	0	0	0.7%	15.23%	45.29%	14.2%
Obese III	0	%	0.4%	2.16%	12.68%	71.22%

Table 3.6: Probability Transition Matrix 2004/05 to 2006/07

Whole						
	Underweight	**Normal**	**Overweight**	**Obese I**	**Obese II**	**Obese III**
Underweight	46.84%	0.99%	0.29%	0	0	0
Normal	44.32%	81.99%	10%	1.03%	0	1.11%
Overweight	8.84%	15.88%	78.28%	17.54%	1.75%	2.23%
Obese I	0	0.99%	10.87%	69.83%	22.95%	8.7%
Obese II	0	0.07%	0.26%	10.91%	59.66%	23.65%
Obese III	0	0.08%	0.27%	0.68%	15.68%	64.31%
Men						
	Underweight	**Normal**	**Overweight**	**Obese I**	**Obese II**	**Obese III**
Underweight	14.31%	0.78%	0.35%	0	0	0
Normal	51.29%	77.5%	6.34%	1.21%	0	0
Overweight	34.4%	20.42%	82.79%	15.83%	0.21%	4.57%
Obese I	0	1.23%	10.07%	72.8%	26.31%	9.42%
Obese II	0	0.06%	0.32%	9.85%	61.54%	3.61%
Obese III	0	%	0.13%	0.3%	11.94%	82.4%
Women						
	Underweight	**Normal**	**Overweight**	**Obese I**	**Obese II**	**Obese III**
Underweight	58.09%	1.13%	0.18%	0	0	0
Normal	41,91%	85.15%	17.46%	0.74%	0	1.82%
Overweight	0	12.69%	69.14%	20.35%	3%	0.72%
Obese I	0	0.81%	12.51%	64.96%	20.1%	8.25%
Obese II	0	0.08%	0.12%	12.66%	58.05%	36.56%
Obese III	0	0.14%	0.6%	1.29%	18.86%	52.66%

Table 3.7: Percentage distribution of BMI at the beginning of each period

	Underweight	Normal	Overweight	Obese I	Obese II	Obese III
Whole						
1994/1995	2.11%	49.36%	35.88%	09.72%	1.99%	0.93%
1996/1997	1.77%	48.31%	36.69%	10.05%	2.23%	0.94%
1998/1999	1.61%	46.88%	36.27%	11.38%	2.89%	0.96%
2000/1001	1.91%	44.78%	36.13%	12.37%	3.31%	1.5%
2002/2003	1.54%	42.7%	36.75%	13.83%	3.4%	1.77%
2004/2005	1.46%	42.42%	36.6%	14.2%	3.4%	1.92%
Men						
1994/1995	0.61%	41.62%	44.92%	10.52%	1.67%	0.65%
1996/1997	0.78%	39.93%	46.08%	10.82%	1.64%	0.74%
1998/1999	0.73%	37.62%	46.20%	12.27%	2.45%	0.74%
2000/1001	1.10%	35.48%	45.95%	13.28%	3.16%	1.02%
2002/2003	0.65%	34.04%	44.87%	16.17%	3.2%	1.06%
2004/2005	0.07%	32.70%	45.78%	16.49%	2.92%	1.40%
Women						
1994/1995	3.72%	57.68%	26.17%	8.86%	2.34%	1.23%
1996/1997	2.85%	57.40%	26.51%	9.21%	2.86%	1.16%
1998/1999	2.52%	56.61%	25.86%	10.44%	3.36%	1.20%
2000/1001	2.78%	54.71%	25.63%	11.38%	3.46%	2.02%
2002/2003	2.51%	52.09%	27.94%	11.30%	3.62%	2.55%
2004/2005	2.34%	53.62%	26.02%	11.56%	3.95%	2.51%

Table 3.8: Prediction of Percentage distribution of BMI

	Men					
	Underweight	**Normal**	**Overweight**	**Obese I**	**Obese II**	**Obese III**
2006	0.52%	28.8%	45.71%	17.92%	3.64%	1.61%
2008	0.47%	25.81%	48.31%	19.3%	4.23%	1.88%
2010	0.44%	23.54%	48.58%	20.52%	4.74%	2.18%
2012	0.42%	21.8%	48.54%	21.58%	5.19%	2.48%
2014	0.04%	20.44%	48.32%	22.46%	5.58%	2.8%
2016	0.39%	19.38%	48.02%	23.2%	5.91%	3.1%
	Women					
2006	2.01%	51.31%	27.28%	12.2%	4.75%	2.45%
2008	1.8%	49.43%	28.01%	12.91%	5.27%	2.58%
2010	1.66%	47.87%	28.44%	13.56%	5.71%	2.76%
2012	1.55%	46.57%	28.69%	14.13%	6.11%	2.94%
2014	1.48%	45.47%	28.82%	14.62%	6.48%	3.12%
2016	1.43%	44.53%	28.89%	15.03%	6.83%	3.29%

Table 3.9: Test for Stationary Hypothesis

	Men	Women	Whole
χ^2_{150}	283.11	232.49	322.01
p value	0.01%	0.01%	0.01%
H_0	R	R	R

R denotes rejection of stationary hypothesis.

Table 3.10: Test for Stationary Hypothesis (Subgroups)

	Active			Moderate			Inactive		
	Men	**Women**	**Whole**	**Men**	**Women**	**Whole**	**Men**	**Women**	**Whole**
χ^2_{150}	222.39	190.72	248.73	155.92	156.85	189.93	256.42	217.72	305.83
p value	0.01%	1.38%	0.01%	35.35%	33.43%	1.52%	0.01%	0.03%	0.01%
H_0	R	R	R	A	A	R	R	R	R

A denotes acceptance of stationary hypothesis while R denotes rejection of it.

Table 3.11: Percentage distribution of BMI at steady state (Moderate subgroups)

	Underweight	Normal	Overweight	Obese I	Obese II	Obese III
Men	0.12%	18.68%	51.59%	23.8%	4.31%	1.5%
Women	1.08%	42.46%	33.09%	15.09%	5.52%	2.76%

The results are just for men and women who are moderate in physical activity

Table 3.12: Percentage distribution of BMI at the beginning of each period (Men)

	Underweight	Normal	Overweight	Obese I	Obese II	Obese III
Active						
1994/1995	0.1%	40.6%	47.8%	10.2%	0.8%	0.5%
1996/1997	0.4%	43 %	46%	8.7%	1.9%	0
1998/1999	0.4%	40.4%	47%	10.7%	1%	0.4%
2000/1001	1.7%	34.8%	49.3%	11.6%	2%	0.6%
2002/2003	0.5%	39.7%	44.6%	13.4%	1.1%	0.7%
2004/2005	0.8%	32.6%	48.2%	15.7%	1.8%	0.9%
Moderate						
1994/1995	0.4%	37.8%	49.6%	9.9%	1.9%	0.4%
1996/1997	0.6%	36.6 %	50.2%	10.7%	1%	0.9%
1998/1999	0.9%	36.3%	48.1%	12.1%	2.6%	0
2000/1001	0.5%	34.8%	48.3%	13.3%	2.2%	0.9%
2002/2003	0.6%	31.2%	46.6%	18%	2.4%	1.1%
2004/2005	0.9%	33.4%	45.9%	16.5%	1.5%	1.8%
Inactive						
1994/1995	0.9%	43.8%	41.8%	10.7%	1.9%	0.8%
1996/1997	0.1%	40.5 %	44.1%	11.4%	1.9%	1%
1998/1999	0.8%	37.2%	45%	12.7%	3%	1.2%
2000/1001	1.1%	35.8%	43.2%	14.5%	4.2%	1.2%
2002/2003	0.6%	32.3%	44.3%	16.8%	4.7%	1.3%
2004/2005	0.4%	32%	44.5%	17.1%	4.5%	1.6%

Table 3.13: Percentage distribution of BMI at the beginning of each period (Women)

	Underweight	Normal	Overweight	Obese I	Obese II	Obese III
Active						
1994/1995	4%	64.6%	23.4%	6.4%	1.2%	0.3%
1996/1997	2.1%	61.5 %	26.7%	6.6%	2.4%	0.6%
1998/1999	2.2%	66.3%	21.9%	7.8%	1.7%	0.2%
2000/1001	2.3%	61%	23.7%	9.5%	2.8%	0.8%
2002/2003	2.7%	61.7%	22.1%	8.7%	3.9%	0.9%
2004/2005	1.8%	60.4%	23.2%	10.5%	2.9%	1.3%
Moderate						
1994/1995	2.8%	61.8%	25%	7.2%	2.2%	0.1%
1996/1997	2.7%	62.1 %	23.6%	8.4%	2.7%	0.6%
1998/1999	1.7%	58.1%	27.1%	10.7%	1.5%	0.1%
2000/1001	2.3%	55.8%	28%	9.6%	2.7%	1.7%
2002/2003	2.4%	51.5%	31.7%	11.3%	2%	1.1%
2004/2005	2.5%	55.3%	28%	10.2%	2.9%	1.1%
Inactive						
1994/1995	4%	54.3%	27.3%	10.2%	2.7%	1.5%
1996/1997	3.2%	54.1 %	27.9%	10.1%	3.1%	1.6%
1998/1999	3%	53.4%	26.4%	11.2%	4.4%	1.6%
2000/1001	3.2%	51.5%	25.3%	13.1%	4.3%	2.6%
2002/2003	2.5%	48.1%	28.2%	12.8%	4.4%	3.9%
2004/2005	2.6%	49.7%	26.4%	12.1%	5.2%	4%

Table 3.14: BMI Percentage distribution by taking moderate shift pattern (Men)

	Underweight	Normal	Overweight	Obese I	Obese II	Obese III
			Active			
1996/1997	0.24%	34.94 %	50.54%	12.55%	1.24%	0.49%
1998/1999	0.22%	30.98%	51.99%	14.63%	1.67%	0.51%
2000/1001	0.19%	28.09%	52.72%	16.39%	2.05%	0.56%
2002/2003	0.18%	25.94%	53.02%	17.84%	2.4%	0.62%
2004/2005	0.16%	24.33%	53.1%	19.01%	2.7%	0.70%
2006/2007	0.15%	23.11%	53.04%	19.96%	2.97%	0.77%
			Inactive			
1996/1997	0.31%	37.45%	46.67%	12.79%	1.98%	0.8%
1998/1999	0.24%	32.61 %	49.5%	14.7%	2.14%	0.8%
2000/1001	0.21%	29.13%	51.09%	16.38%	2.37%	0.82%
2002/2003	0.18%	26.61%	51.96%	17.8%	2.61%	0.84%
2004/2005	0.17%	24.75%	52.39%	18.97%	2.85%	0.88%
2006/2007	0.16%	23.37%	52.56%	19.92%	3.07%	0.92%

Table 3.15: BMI Percentage distribution by taking moderate shift pattern (women)

	Active					
	Underweight	**Normal**	**Overweight**	**Obese I**	**Obese II**	**Obese III**
1996/1997	2.59%	61.21 %	26.87%	7.09%	1.58%	0.65%
1998/1999	1.95%	58.04%	29.07%	8.02%	1.99%	0.91%
2000/1001	1.64%	55.37%	30.46%	8.98%	2.38%	1.14%
2002/2003	1.48%	53.16%	31.35%	9.87%	2.76%	1.34%
2004/2005	1.38%	51.36%	31.92%	10.65%	3.12%	1.52%
2006/2007	1.31%	49.89%	32.29%	11.33%	3.44%	1.68%
	Inactive					
1996/1997	2.44%	53.16%	29.23%	10.43%	3.13%	1.6%
1998/1999	1.78%	51.58 %	30.55%	10.93%	3.42%	1.71%
2000/1001	1.48%	50.1%	31.42%	11.48%	3.67%	1.82%
2002/2003	1.34%	48.81%	31.98%	12%	3.9%	1.93%
2004/2005	1.26%	47.75%	32.34%	12.46%	4.11%	2.03%
2006/2007	1.22%	46.87%	32.58%	12.86%	4.29%	2.13%

Concluding Remarks

This thesis contains two different topics which study issues on time series econometrics and health economics respectively.

The first chapter proposes a bootstrap test for a unit root in processes with GARCH errors and shows its asymptotic validity under very weak moment and distributional assumptions. The proposed method offers several important advantages over the existing tests that do not exploit the information in the conditional variance and its asymptotic counterpart. First, the test delivers impressive power gains by explicitly incorporating the GARCH structure of the errors, especially for highly persistent GARCH specifications with power improvements over the DF-type tests. While the asymptotic counterpart of the test requires the computation of nuisance parameters and suffers from relatively large size distortions, the proposed bootstrap procedure is straightforward to implement and appears to control the size uniformly over all possible GARCH specifications that guarantee the existence of second moments of the errors. Finally, while generalizing the asymptotic theory to more complicated setups would be quite involved, our bootstrap method can be easily adapted to models with a lag length that goes to infinity at certain rate, asymmetric errors and other types of conditional heteroskedasticity.

Chapter 2 has applied quantile regression to determine the quantitative influence of several observable factors at different points on the Canadian BMI distribution,

using data from three distinct Canadian surveys undertaken in 1978, 1994 and 2004. The results obtained strongly suggest that the epidemic of obesity in Canada is mainly developed during the period 1994 to 2004. This epidemic is more closely related to lifestyle and socioeconomic factors than demographic factors. Educational achievement has a negative impact, that is to say, the higher the educational level a person has achieved, the lower the likelihood that person will be obese. The role of the education has shifted over the years from the weakest to the strongest factor affecting the BMI. This, in turn, implies that the relationship between the capacity to avoid obesity and education is strong. Among the seven factors in this study, being physically inactive is the most important reason for women to be obese. On the other hand, working status is the least important factor related to the BMI for both men and women. This not only indicates the dominance of sedentary work and continually decreasing expenditure of energy in the workplace, but also reveals that physical activity is the main way for people to expend energy. The fact that age becomes less important in affecting the BMI at higher quantiles indicates that a change in the age structure of Canadians is not a reason for the obesity epidemic in Canada. A decomposition by quantiles has been introduced, in an effort to explore the development over time of the Canadian BMI distribution, especially in the future. The calculation reveals that, after 1994, the component of most importance in changing the BMI distribution has been the *function* of factors, that is, the changing intensity with which factors impinge on BMI. *Structural* changes, that is, changes in the factors themselves, and the *interaction* of *structure* and *function* are of trivial importance. To put the matter technically: in a quantile regression analysis of the BMI by cross-sections over time,

the most significant changes have been those in the coefficients, not changes in the variables or changes in the interaction between the two.

Chapter 3 investigates the BMI shift patterns of Canadian adults aged 18 to 64 by applying the Markov chain method to the seven cycles of the longitudinal National Population Health Survey (NPHS, 1994/95 - 2006/07). The estimated probability transition matrices indicate that men and women are different in BMI shift patterns. However, the shift patterns for both groups in all periods exhibit a common positive trend. Stationary tests show that for both men and women, BMI shifts are not stationary over this period. Interestingly though, the BMI shifts of moderate active subgroups of both men and women are stationary and ergodic. Some of the main finding can be summarized as follows. First, women with normal weight and overweight men have the highest probability to remain in their BMI category. Second, because the proportion of normal and overweight people is much higher than the proportion of obese people, the number of people who move from the normal or overweight categories to the obese category is much more than people moving in the opposite direction. Hence, the prevalence of obesity keeps increasing over time. Finally, the stationary tests show that the BMI shift of normal and overweight people is the main source of the changes in the BMI shift and the effort to prevent the occurrence of new obese cases would prove much more effective than the effort to reduce the number of existing obese people. People who are inactive in physical activity account for the biggest portion of the Canadian population. Compared with people who are active or moderate in their physical activity, the obesity prevalence for this group is substantially higher. Hence, the obesity prevalence in Canada is most closely associated with

the physically inactive people. Health policies that aim at dealing with the obesity problem should focus more on the physically inactive people and design incentives that would encourage them to become moderately or fully physically active. Our calculations show that if all inactive people become moderately active, assuming that the obesity prevalence for active and moderate groups remains unchanged, the obesity prevalence in Canada could be confined below 25% in the long run.

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Appendix

A.1 Mathematical Proofs and Auxiliary Lemmas of Chapter 1

(1) Auxiliary Lemmas

Auxiliary Lemma 1

Under Assumption 1,

(a) $\widehat{h}_t - h_t = o_p(1) + O(\beta^t)$

(b) $\frac{\varepsilon_t^2}{h_t \widehat{h}_t} = O_p(1)$.

PROOF. For proof of part (a), see Gospodinov (2008). For part (b) note that

$$\frac{\varepsilon_t^2}{h_t \widehat{h}_t} < \frac{\varepsilon_t^2}{\omega \widehat{\omega}} \leq \frac{\varepsilon_t^2}{\gamma^2},$$

where $\gamma = \min\{\omega, \widehat{\omega}\} > 0$. Since $E\left(\frac{\varepsilon_t^2}{\gamma^2}\right) = \frac{\sigma^2}{\gamma^2}$ and $\frac{\varepsilon_t^2}{h_t \widehat{h}_t} \geq 0$, it is easy to show that $\frac{\varepsilon_t^2}{h_t \widehat{h}_t} = O_p(1)$.

□

Auxiliary Lemma 2

Let $h_t^* = \widehat{\omega}\left[1 + \sum_{k=1}^{\infty} \Pi_{i=1}^{k} \left(\hat{\alpha}\eta_{t-i}^{*2} + \hat{\beta}\right)\right]$ *and* $\varepsilon_t^* = \eta_t^* \sqrt{\widehat{\omega}\left[1 + \sum_{k=1}^{\infty} \Pi_{i=1}^{k} \left(\hat{\alpha}\eta_{t-i}^{*2} + \hat{\beta}\right)\right]}$ *and suppose that* η_0^* *is drawn from* $\widetilde{F}_T^{sym}(\eta)$ *and the sequence* $\{h_t^*\}$ *is initialized from its invariant measure. Then,* $\{h_t^*\}$ *and* $\{\varepsilon_t^*\}$ *are strictly stationary and ergodic processes.*

PROOF. The proof follows directly from Theorem 2 in Nelson (1990).

□

Auxiliary Lemma 3

Let $\xi_t = \lambda_1\varepsilon_t^* + \lambda_2[\frac{\varepsilon_t^*}{h_t^*} - \frac{\hat{\alpha}}{h_t^*}(\frac{\varepsilon_t^{*2}}{h_t^*} - 1)\sum_{k=1}^{t-1}\hat{\beta}^{k-1}\varepsilon_{t-k}^*]$ *and* $\xi_t' = \lambda_1\varepsilon_t^* + \lambda_2[\frac{\varepsilon_t^*}{h_t^*} - \frac{\hat{\alpha}}{h_t^*}(\frac{\varepsilon_t^{*2}}{h_t^*} - 1)\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\varepsilon_{t-k}^*]$, *and denote* $S_{[Tr]} = \frac{1}{\sqrt{T}}\sum_{t=1}^{[Tr]}\xi_t$ *and* $S'_{[Tr]} = \frac{1}{\sqrt{T}}\sum_{t=1}^{[Tr]}\xi_t'$ *for* $\{0 \le r \le 1\}$. *Then,* $E^*\left|S_{[Tr]} - S'_{[Tr]}\right| \to 0$.

PROOF. The proof is similar to the proof of (4.6) in Lemma 4.2 in Ling and Li (2003). More specifically,

$$
\begin{aligned}
E^*\left|S_{[Tr]} - S'_{[Tr]}\right| &\le \frac{1}{\sqrt{T}}\sum_{t=1}^{[Tr]} E^*\left|\frac{\lambda_2}{h_t^*}(\frac{\varepsilon_t^{*2}}{h_t^*} - 1)\left(\hat{\alpha}\sum_{k=t}^{\infty}\hat{\beta}^{k-1}\varepsilon_{t-k}^*\right)\right| \\
&\le \frac{1}{\sqrt{T}}\sum_{t=1}^{[Tr]} E^*\left|\frac{\lambda_2}{h_t^*}(\frac{\varepsilon_t^{*2}}{h_t^*} - 1)\right| E^*\left|\hat{\alpha}\sum_{k=t}^{\infty}\hat{\beta}^{k-1}\frac{\varepsilon_{t-k}^*}{\sqrt{h_t^*}}\right| \\
&\le \frac{1}{\sqrt{T}}\sum_{t=1}^{[Tr]} E^*\left|\frac{\lambda_2}{\sqrt{\hat{\alpha}_0}}(\eta_t^{*2} - 1)\right| E^*\left|\hat{\alpha}\sum_{k=t}^{\infty}\hat{\beta}^{k-1}\frac{\varepsilon_{t-k}^*}{\sqrt{h_t^*}}\right| \\
&\le \frac{c}{\sqrt{T}}\sum_{t=1}^{[Tr]}\left(\sum_{k=t}^{\infty}\hat{\beta}^{(k-1)/2}\right) \\
&= \frac{c}{\sqrt{T}}\sum_{t=1}^{[Tr]} O\left(\hat{\beta}^{t/2}\right) \to 0 \text{ as } T \to \infty,
\end{aligned}
$$

where c is a constant.

□

Lemmas and Theorems

Proof of Lemma 2

part (a)

For any $\eta \in \mathbb{R}$,

$$F_T^{sym}(\eta) = \frac{1}{2T}\sum_{i=1}^{2T} I(\eta_i \leq \eta) \tag{3.11}$$

$$= \frac{1}{2T}\sum_{i=1}^{T} I(\eta_i \leq \eta) + \frac{1}{2T}\sum_{i=1}^{T} I(-\eta_i \leq \eta)$$

$$= \frac{1}{2}\left[F_T(\eta) + (1 - F_T(-\eta))\right]$$

$$\longrightarrow \frac{1}{2}\left[F(\eta) + (1 - F(-\eta))\right] = F(\eta) \text{ as } T \longrightarrow \infty \tag{3.12}$$

and by the symmetry of F. Because d_2 is a metric, $d_2\left(\widetilde{F}_T^{sym}, F\right)^2 \leq d_2\left(\widetilde{F}_T^{sym}, F_T^{sym}\right)^2 + d_2\left(F_T^{sym}, F\right)^2$ and $d_2\left(F_T^{sym}, F\right)^2 \to 0$ from (3.11) and Bickel and Freedman (1981). Next,

$$d_2(\widetilde{F}_T^{sym}, F_T^{sym})^2 \leq E^*\left|\widetilde{\eta}_j - \eta_j\right|^2 \tag{3.13}$$

$$= \frac{1}{T}\sum_{j=1}^{T}\left[\widetilde{\eta}_j - \eta_j - \frac{1}{T}\sum_{i=1}^{T}\widetilde{\eta}_i\right]^2$$

$$= \frac{1}{T}\sum_{j=1}^{T}\left[(\widetilde{\eta}_j - \eta_j) - \frac{1}{T}\sum_{i=1}^{T}(\widetilde{\eta}_i - \eta_i) - \frac{1}{T}\sum_{i=1}^{T}\eta_i\right]^2$$

$$\leq \frac{1}{T}\sum_{j=1}^{T}\left[3(\widetilde{\eta}_j - \eta_j)^2 + \frac{3}{T}\sum_{i=1}^{T}(\widetilde{\eta}_j - \eta_j)^2 + 3\left(\frac{1}{T}\sum_{i=1}^{T}\eta_i\right)^2\right] \tag{3.14}$$

$$= \frac{6}{T}\sum_{j=1}^{T}(\widetilde{\eta}_j - \eta_j)^2 + \frac{3}{T^2}\left(\sum_{i=1}^{T}\eta_i\right)^2 \to 0$$

since $T^{-1/2}\sum_{i=1}^{T}\eta_i = O_p(1)$ and $\frac{6}{T}\sum_{j=1}^{T}(\widetilde{\eta}_j - \eta_j)^2 = \frac{6}{T}\sum_{j=1}^{T}\frac{\varepsilon_j^2}{h_j\widehat{h}_j}\left(\widehat{h}_j^{1/2} - h_j^{1/2}\right)^2 \to 0$ from the results in Auxiliary Lemma A1 (see also Pascual *et al.*, 2000).

part (b)

Note that the k^{th} moment of the symmetrized residuals $\{\widetilde{\eta}_1, \widetilde{\eta}_2, ..., \widetilde{\eta}_T, -\widetilde{\eta}_1, -\widetilde{\eta}_2, ..., -\widetilde{\eta}_T\}$ is given by $(2T)^{-1}\left[\sum_{t=1}^{T}(\widetilde{\eta}_t)^k + \sum_{t=1}^{T}(-\widetilde{\eta}_t)^k\right] = T^{-1}\sum_{t=1}^{T}(\widetilde{\eta}_t)^p$ if $p \geq 1$ is even and 0 if p is odd. Then, since $\{\eta_t^*\}$ is an *iid* sample from the symmetrized empirical distribution function of the recentered standardized residuals, $E^*(\eta_t^*) = 0$ and hence $E^*(\varepsilon_t^*) = 0$.

part (c)

Because $E^*(\varepsilon_t^*) = 0$ and η_t^* are *iid* conditionally on the sample, $Var^*(\varepsilon_t^*) = E^*(\varepsilon_t^*)^2 = E^*(h_t^*)E^*(\xi_t^*)^2$ and

$$Var^*(\varepsilon_t^*) = \left(\frac{1}{T}\sum_{t=1}^{T}\widehat{h}_t\right)\left(\frac{1}{T}\sum_{t=1}^{T}\widetilde{\eta}_t^2\right). \tag{3.15}$$

First, from part (a) of Auxiliary Lemma 1 and $\sum_{t=1}^{T}\beta^t = O_p(1)$, it follows that that $\frac{1}{T}\sum_{t=1}^{T}\left(\widehat{h}_t - h_t\right) = o_p(1)$ and $\frac{1}{T}\sum_{t=1}^{T}\widehat{h}_t \xrightarrow{p} E(h_t)$.

Combining this result with $\frac{1}{T}\sum_{t=1}^{T}\widetilde{\eta}_t^2 \xrightarrow{p} 1$, we have

$$Var^*(\varepsilon_t^*) \xrightarrow{p} \sigma^2 \text{ as } T \to \infty.$$

part (d)

Finally, since $E^*(\eta_t^*)^3 = 0$ by construction, η_t^* are *iid* conditionally on the sample, and $\frac{1}{T}\sum_{t=1}^{T}\widehat{h}_t = O_p(1)$, we obtain that $E^*(\varepsilon_t^*)^3 = 0$.

□

Proof of Lemma 3

part (a)

By recursive substitution,

$$\begin{aligned} h_t^* &= \widehat{\omega} + \widehat{\alpha}\varepsilon_{t-1}^{*2} + \widehat{\beta}h_{t-1}^* \\ &= \widehat{\omega} + \widehat{\omega}\left(\widehat{\alpha}\eta_{t-1}^{*2} + \widehat{\beta}\right) + \widehat{\omega}\left(\widehat{\alpha}\eta_{t-1}^{*2} + \widehat{\beta}\right)\left(\widehat{\alpha}\eta_{t-2}^{*2} + \widehat{\beta}\right) + ... \\ &= \widehat{\omega}[1 + \sum_{k=1}^{t-1}\prod_{i=1}^{k}\left(\widehat{\alpha}\eta_{t-i}^{*2} + \widehat{\beta}\right)] + \widehat{\omega}\prod_{i=1}^{t}\left(\widehat{\alpha}\eta_{t-i}^{*2} + \widehat{\beta}\right)h_0^*. \end{aligned}$$

If the two candidate initial values are h_{01}^* and h_{02}^*, then the difference between the corresponding sequences h_{t1}^* and h_{t2}^* is given by

$$|h_{t1}^* - h_{t2}^*| = \widehat{\omega}\prod_{i=1}^{t}\left(\widehat{\alpha}\eta_{t-i}^{*2} + \widehat{\beta}\right)|h_{01}^* - h_{02}^*|$$

and

$$\begin{aligned} E^*|h_{t1}^* - h_{t2}^*| &= \widehat{\omega}\,|h_{01}^* - h_{02}^*|\,E^*\left[\prod_{i=1}^{t}\left(\widehat{\alpha}\eta_{t-i}^{*2} + \widehat{\beta}\right)\right] \\ &= \widehat{\omega}\,|h_{01}^* - h_{02}^*|\left(\widehat{\alpha} + \widehat{\beta}\right)^t, \end{aligned}$$

using that $E^*(\eta_t^*)^2 \to 1$. Since $\widehat{\alpha} + \widehat{\beta} < 1$ by construction, $E^*|h_{t1}^* - h_{t2}^*| \to 0$ as $t \to \infty$.

part (b)

Rewrite $\left|\sqrt{h_{t1}^*} - \sqrt{h_{t2}^*}\right|$ as

$$\begin{aligned} \left|\sqrt{h_{t1}^*} - \sqrt{h_{t2}^*}\right| &= \left|\frac{h_{t1}^* - h_{t2}^*}{\sqrt{h_{t1}^*} + \sqrt{h_{t2}^*}}\right| \\ &\leq \left|\frac{h_{t1}^* - h_{t2}^*}{2\widehat{\omega}}\right|. \end{aligned}$$

Then,

$$\begin{aligned} E^* |\varepsilon_{t1}^* - \varepsilon_{t2}^*| &= E\left[\left(\left|\sqrt{h_{t1}^*} - \sqrt{h_{t2}^*}\right|\right)|\eta_t^*|\right] \\ &\leq E^*\left(\left|\frac{h_{t1}^* - h_{t2}^*}{2\widehat{\omega}}\right||\eta_t^*|\right) \\ &= \frac{1}{2\widehat{\omega}} E^* |h_{t1}^* - h_{t2}^*| E|\eta_t^*| \\ &= \frac{1}{2}|h_{01}^* - h_{02}^*|\left(\hat{\alpha}+\hat{\beta}\right)^t E^*|\eta_t^*| \\ &= \frac{1}{2}|h_{01}^* - h_{02}^*|\left(\hat{\alpha}+\hat{\beta}\right)^t E^*|\eta_t^*| \end{aligned}$$

using that $E^*(\eta_t^*)^2 \to 1$. Since $E^*|\eta_t^*| < \infty$ and $\widehat{\alpha}+\widehat{\beta} < 1$, $E|\varepsilon_{t1}^* - \varepsilon_{t2}^*| \to 0$ as $t \to \infty$.

part (c)

Note that

$$\begin{aligned} E^*\left|\frac{1}{\sqrt{T}}\sum_{i=1}^{T}\varepsilon_{i1}^* - \frac{1}{\sqrt{T}}\sum_{i=1}^{T}\varepsilon_{i2}^*\right| &\leq \frac{1}{\sqrt{T}}\sum_{i=1}^{T} E^*|\varepsilon_{i1}^* - \varepsilon_{i2}^*| \\ &\leq \frac{1}{\sqrt{T}}\sum_{i=1}^{T}\frac{1}{2}|h_{01}^* - h_{02}^*|\left(\hat{\alpha}+\hat{\beta}\right)^i E^*|\eta_t^*| \\ &= \frac{|h_{01}^* - h_{02}^*| E^*|\eta_t^*|}{2\sqrt{T}}\sum_{i=1}^{T}\left(\hat{\alpha}+\hat{\beta}\right)^i \\ &= \frac{|h_{01}^* - h_{02}^*| E^*|\eta_t^*|}{2\sqrt{T}}\frac{1-\left(\hat{\alpha}+\hat{\beta}\right)^{T+1}}{1-\left(\hat{\alpha}+\hat{\beta}\right)} \\ &\leq \frac{1}{2\sqrt{T}}\frac{|h_{01}^* - h_{02}^*| E^*|\eta_t^*|}{1-\left(\hat{\alpha}+\hat{\beta}\right)} = O\left(\frac{1}{\sqrt{T}}\right). \end{aligned}$$

part (d)

The two partial sums have the following form

$$S^{(1)}_{[Tr]} = \frac{1}{\sqrt{T}}\sum_{t=1}^{[Tr]}\xi_{t1} = \frac{1}{\sqrt{T}}\sum_{t=1}^{[Tr]}\{\lambda_1\varepsilon^*_{t1} + \lambda_2[\frac{\varepsilon^*_{t1}}{h^*_{t1}} - \frac{\hat{\alpha}}{h^*_{t1}}(\frac{\varepsilon^{*2}_{t1}}{h^*_{t1}} - 1)\sum_{k=1}^{t-1}\hat{\beta}^{k-1}\varepsilon^*_{t-k1}]\}$$

$$S^{(2)}_{[Tr]} = \frac{1}{\sqrt{T}}\sum_{t=1}^{[Tr]}\xi_{t2} = \frac{1}{\sqrt{T}}\sum_{t=1}^{[Tr]}\{\lambda_1\varepsilon^*_{t2} + \lambda_2[\frac{\varepsilon^*_{t2}}{h^*_{t2}} - \frac{\hat{\alpha}}{h^*_{t2}}(\frac{\varepsilon^{*2}_{t2}}{h^*_{t2}} - 1)\sum_{k=1}^{t-1}\hat{\beta}^{k-1}\varepsilon^*_{t-k2}]\}$$

Taking expectations under P^* of the difference yields

$$\begin{aligned}
E^*\left|S^{(1)}_{[Tr]} - S^{(2)}_{[Tr]}\right| &\le \frac{\lambda_1}{\sqrt{T}}E^*\sum_{t=1}^{[Tr]}|\varepsilon^*_{t1} - \varepsilon^*_{t2}| + \frac{\lambda_2}{\sqrt{T}}E^*\sum_{t=1}^{[Tr]}\left|\frac{\varepsilon^*_{t1}}{h^*_{t1}} - \frac{\varepsilon^*_{t2}}{h^*_{t2}}\right| \\
&+ \frac{\lambda_2\hat{\alpha}}{\sqrt{T}}E^*\sum_{t=1}^{[Tr]}\left|(\frac{\varepsilon^{*2}_{t1}}{h^*_{t1}} - 1)\sum_{k=1}^{t-1}\hat{\beta}^{k-1}\frac{\varepsilon^*_{t-k1}}{h^*_{t1}} - (\frac{\varepsilon^{*2}_{t2}}{h^*_{t2}} - 1)\sum_{k=1}^{t-1}\hat{\beta}^{k-1}\frac{\varepsilon^*_{t-k2}}{h^*_{t2}}\right| \\
&= \frac{\lambda_1}{\sqrt{T}}E^*\sum_{t=1}^{[Tr]}|\varepsilon^*_{t1} - \varepsilon^*_{t2}| + \frac{\lambda_2}{\sqrt{T}}E^*\sum_{t=1}^{[Tr]}\left|\frac{\varepsilon^*_{t1}}{h^*_{t1}} - \frac{\varepsilon^*_{t2}}{h^*_{t2}}\right| \\
&+ \frac{\lambda_2\hat{\alpha}}{\sqrt{T}}E^*\sum_{t=1}^{[Tr]}\left|(\eta^{*2}_t - 1)\sum_{k=1}^{t-1}\hat{\beta}^{k-1}\frac{\varepsilon^*_{t-k1}}{h^*_{t1}} - (\eta^{*2}_t - 1)\sum_{k=1}^{t-1}\hat{\beta}^{k-1}\frac{\varepsilon^*_{t-k2}}{h^*_{t2}}\right| \\
&= \frac{\lambda_1}{\sqrt{T}}E^*\sum_{t=1}^{[Tr]}|\varepsilon^*_{t1} - \varepsilon^*_{t2}| + \frac{\lambda_2}{\sqrt{T}}E\sum_{t=1}^{[Tr]}\left|\frac{\varepsilon^*_{t1}}{h^*_{t1}} - \frac{\varepsilon^*_{t2}}{h^*_{t2}}\right| \\
&+ \frac{\lambda_2\hat{\alpha}}{\sqrt{T}}\sum_{t=1}^{[Tr]}E^*\left|\sum_{k=1}^{t-1}\hat{\beta}^{k-1}\frac{\varepsilon^*_{t-k1}}{h^*_{t1}} - \sum_{k=1}^{t-1}\hat{\beta}^{k-1}\frac{\varepsilon^*_{t-k2}}{h^*_{t2}}\right| E^*\left|(\eta^{*2}_t - 1)\right| \\
&= \lambda_1 I_1 + \lambda_2 I_2 + \lambda_2\hat{\alpha}I_3.
\end{aligned}$$

From part (c) we know $I_1 = O\left(T^{-1/2}\right)$. Furthermore,

$$\begin{aligned}
I_2 &= \frac{1}{\sqrt{T}} E^* \sum_{t=1}^{[Tr]} \left| \frac{\varepsilon_{t1}^*}{h_{t1}^*} - \frac{\varepsilon_{t2}^*}{h_{t2}^*} \right| = \frac{1}{\sqrt{T}} E^* \sum_{t=1}^{[Tr]} \left| \left(\frac{1}{\sqrt{h_{t1}^*}} - \frac{1}{\sqrt{h_{t2}^*}} \right) \eta_t^* \right| \\
&\leq \frac{1}{\sqrt{T}} E^* \sum_{t=1}^{[Tr]} \left| \left(\frac{\sqrt{h_{t2}^*} - \sqrt{h_{t1}^*}}{\sqrt{h_{t1}^* h_{t2}^*}} \right) \eta_t^* \right| \\
&\leq \frac{1}{\widehat{\omega}\sqrt{T}} E^* \sum_{t=1}^{[Tr]} \left| \left(\sqrt{h_{t2}^*} - \sqrt{h_{t1}^*} \right) \eta_t^* \right| = \frac{1}{\widehat{\omega}\sqrt{T}} E^* \sum_{t=1}^{[Tr]} \left| \varepsilon_{t1}^* - \varepsilon_{t2}^* \right| \\
&\leq \frac{1}{\widehat{\omega}\sqrt{T}} E^* \sum_{t=1}^{T} \left| \varepsilon_{t1}^* - \varepsilon_{t2}^* \right| = O\left(\frac{1}{\sqrt{T}} \right)
\end{aligned}$$

and

$$\begin{aligned}
I_3 &= \frac{1}{\sqrt{T}} \sum_{t=1}^{[Tr]} E^* \left| \sum_{k=1}^{t-1} \hat{\beta}^{k-1} \frac{\varepsilon_{t-k1}^*}{h_{t1}^*} - \sum_{k=1}^{t-1} \hat{\beta}^{k-1} \frac{\varepsilon_{t-k2}^*}{h_{t2}^*} \right| E^* \left| \left(\eta_t^{*2} - 1 \right) \right| \\
&= \frac{C}{\sqrt{T}} \sum_{t=1}^{[Tr]} E^* \left| \sum_{k=1}^{t-1} \hat{\beta}^{k-1} \frac{\varepsilon_{t-k1}^*}{h_{t1}^*} - \sum_{k=1}^{t-1} \hat{\beta}^{k-1} \frac{\varepsilon_{t-k2}^*}{h_{t2}^*} \right| \\
&\leq \frac{C}{\sqrt{T}} \sum_{t=1}^{[Tr]} \sum_{k=1}^{t-1} \hat{\beta}^{k-1} E^* \left| \frac{\varepsilon_{t-k1}^*}{h_{t1}^*} - \frac{\varepsilon_{t-k2}^*}{h_{t2}^*} \right| \\
&= \frac{C}{\sqrt{T}} \sum_{t=1}^{[Tr]} \sum_{k=1}^{t-1} \hat{\beta}^{k-1} E^* \left| \frac{h_{t2}^* \left(\varepsilon_{t-k1}^* - \varepsilon_{t-k2}^* \right) + \varepsilon_{t-k2}^* \left(h_{t2}^* - h_{t1}^* \right)}{h_{t1}^* h_{t2}^*} \right| \\
&< \frac{C}{\sqrt{T}} \sum_{t=1}^{[Tr]} \sum_{k=1}^{t-1} \hat{\beta}^{k-1} \left(E^* \left| \frac{\left(\varepsilon_{t-k1}^* - \varepsilon_{t-k2}^* \right)}{h_{t1}^*} \right| + E^* \left| \frac{\varepsilon_{t-k2}^* \left(h_{t2}^* - h_{t1}^* \right)}{h_{t1}^* h_{t2}^*} \right| \right) \\
&= \frac{C}{\sqrt{T}} \sum_{t=1}^{[Tr]} \sum_{k=1}^{t-1} \hat{\beta}^{k-1} \left(E^* \left| \frac{\left(\varepsilon_{t-k1}^* - \varepsilon_{t-k2}^* \right)}{h_{t1}^*} \right| + E^* \left| \frac{\varepsilon_{t-k2}^*}{\sqrt{h_{t2}^*}} \frac{\left(h_{t2}^* - h_{t1}^* \right)}{h_{t1}^* \sqrt{h_{t2}^*}} \right| \right) \\
&\leq \frac{C}{\sqrt{T}} \sum_{t=1}^{[Tr]} \sum_{k=1}^{t-1} \hat{\beta}^{k-1} \left[\frac{1}{\widehat{\omega}} E^* \left| \left(\varepsilon_{t-k1}^* - \varepsilon_{t-k2}^* \right) \right| + \frac{1}{\widehat{\omega}\sqrt{\widehat{\omega}}} E^* \left| \left(\beta^{-\frac{k-1}{2}} \right) \left(h_{t2}^* - h_{t1}^* \right) \right| \right]
\end{aligned}$$

$$= \frac{C}{\widehat{\omega}\sqrt{T}} \sum_{t=1}^{[Tr]} \sum_{k=1}^{t-1} \hat{\beta}^{k-1} \left[E^* \left| \left(\varepsilon^*_{t-k1} - \varepsilon^*_{t-k2} \right) \right| + \frac{1}{\sqrt{\widehat{\omega}}} E^* \left| \left(\beta^{-\frac{k-1}{2}} \right) \left(h^*_{t2} - h^*_{t1} \right) \right| \right]$$

$$\leq \frac{C}{\widehat{\omega}\sqrt{T}} \sum_{t=1}^{[Tr]} \sum_{k=1}^{t-1} \hat{\beta}^{k-1} [\frac{1}{2} \left| h^*_{01} - h^*_{02} \right| \left(\hat{\alpha} + \hat{\beta} \right)^{t-k} E^* \left| \eta^*_t \right|$$

$$+ \frac{1}{\sqrt{\widehat{\omega}}} \left(\beta^{-\frac{k-1}{2}} \right) \hat{\alpha}_0 \left| h^*_{01} - h^*_{02} \right| \left(\hat{\alpha} + \hat{\beta} \right)^t]$$

$$= \frac{E^* \left| \eta^*_t \right| C \left| h^*_{01} - h^*_{02} \right|}{2\widehat{\omega}\sqrt{T}} \sum_{t=1}^{[Tr]} \sum_{k=1}^{t-1} \hat{\beta}^{k-1} \left(\hat{\alpha} + \hat{\beta} \right)^{t-k}$$

$$+ \frac{C \left| h^*_{01} - h^*_{02} \right|}{\sqrt{\widehat{\omega}}\sqrt{T}} \sum_{t=1}^{[Tr]} \sum_{k=1}^{t-1} \left(\beta^{\frac{k-1}{2}} \right) \left(\hat{\alpha} + \hat{\beta} \right)^t$$

$$= \frac{E^* \left| \eta^*_t \right| C \left| h^*_{01} - h^*_{02} \right|}{2\widehat{\omega}} T_1 + \frac{C \left| h^*_{01} - h^*_{02} \right|}{\sqrt{\widehat{\omega}}} T_2.$$

where $C = E \left| (\eta^{*2}_t - 1) \right|$.

The terms T_1 and T_2 are both of order $O\left(T^{-1/2}\right)$ since

$$\begin{aligned}
T_1 &= \frac{1}{\sqrt{T}} \sum_{t=1}^{[Tr]} \sum_{k=1}^{t-1} \hat{\beta}^{k-1} \left(\hat{\alpha}+\hat{\beta}\right)^{t-k} \\
&= \frac{1}{\hat{\beta}\sqrt{T}} \sum_{t=1}^{[Tr]} \sum_{k=1}^{t-1} \frac{\hat{\beta}^{k}}{\left(\hat{\alpha}+\hat{\beta}\right)^{k}} \left(\hat{\alpha}+\hat{\beta}\right)^{t} \\
&= \frac{1}{\hat{\beta}\sqrt{T}} \sum_{t=1}^{[Tr]} \frac{1-r^{t}}{1-r} \left(\hat{\alpha}+\hat{\beta}\right)^{t} \\
&< \frac{1}{\hat{\beta}\left(1-r\right)\sqrt{T}} \sum_{t=1}^{[Tr]} \left(\hat{\alpha}+\hat{\beta}\right)^{t} \\
&= \frac{1}{\hat{\beta}\left(1-r\right)\sqrt{T}} \frac{1-\left(\hat{\alpha}+\hat{\beta}\right)^{[Tr]+1}}{1-\left(\hat{\alpha}+\hat{\beta}\right)} \\
&< \frac{1}{\hat{\beta}\left(1-r\right)\left[1-\left(\hat{\alpha}+\hat{\beta}\right)\right]} \frac{1}{\sqrt{T}} = O\left(\frac{1}{\sqrt{T}}\right)
\end{aligned}$$

and

$$\begin{aligned}
T_2 &= \frac{1}{\sqrt{T}} \sum_{t=1}^{[Tr]} \sum_{k=1}^{t-1} \left(\beta^{\frac{K-1}{2}}\right) \left(\hat{\alpha}+\hat{\beta}\right)^{t} \\
&= \frac{1}{\sqrt{T}} \sum_{t=1}^{[Tr]} \left(\hat{\alpha}+\hat{\beta}\right)^{t} \sum_{k=1}^{t-1} \beta^{\frac{K-1}{2}} \\
&= \frac{1}{\sqrt{T}} \sum_{t=1}^{[Tr]} \left(\hat{\alpha}+\hat{\beta}\right)^{t} \frac{1-\beta^{\frac{t-1}{2}}}{1-\beta^{\frac{1}{2}}} \\
&< \frac{1}{\left(1-\beta^{\frac{1}{2}}\right)\sqrt{T}} \sum_{t=1}^{[Tr]} \left(\hat{\alpha}+\hat{\beta}\right)^{t} \\
&< \frac{1}{\left[1-\left(\hat{\alpha}+\hat{\beta}\right)\right]\left(1-\beta^{\frac{1}{2}}\right)} \frac{1}{\sqrt{T}} = O\left(\frac{1}{\sqrt{T}}\right).
\end{aligned}$$

Then, it follows that $I_3 = O\left(T^{-1/2}\right)$ and combining this with $I_1 = O\left(T^{-1/2}\right)$ and $I_2 = O\left(T^{-1/2}\right)$, we obtain that $E^*\left|S^{(1)}_{[Tr]} - S^{(2)}_{[Tr]}\right| = O\left(T^{-1/2}\right)$.

□

Proof of Lemma 4

part (a)

As in part (a) of Auxiliary Lemma 1 and part (c) of Lemma 2, we can show that $h^*_t - h_t = o_p(1) + O(\beta^t)$ and $\frac{1}{T}\sum_{t=1}^{T}(h^*_t - h_t) = o_p(1)$ which implies that $E^*(h^*_t) \to^p E(h_t)$.

part (b)

Since both ε^*_t and h^*_t are stationary and ergodic (Auxiliary Lemma 2), $\frac{1}{h^*_t}$ and $\frac{\varepsilon^{*2}_{t-k}}{h^{*2}_t}$ are also stationary and ergodic. Using

$$E^*\left(1/h^*_t\right) = \frac{1}{T}\sum_{t=1}^{T}\frac{1}{h^*_t} + o_p(1)$$

$$E(1/h_t) = \frac{1}{T}\sum_{t=1}^{T}\frac{1}{h_t} + o_p(1)$$

we have

$$\begin{aligned}
\left|\frac{1}{T}\sum_{t=1}^{T}\frac{1}{h^*_t} - \frac{1}{T}\sum_{t=1}^{T}\frac{1}{h_t}\right| &= \left|\frac{1}{T}\sum_{t=1}^{T}\left(\frac{1}{h^*_t} - \frac{1}{h_t}\right)\right| \\
&= \left|\frac{1}{T}\sum_{t=1}^{T}\frac{\hat{h}_t - h^*_t}{h^*_t\hat{h}_t}\right| \\
&\leq \frac{1}{\omega\widehat{\omega}}\left|\frac{1}{T}\sum_{t=1}^{T}(h_t - h^*_t)\right| \\
&= \frac{1}{\omega\widehat{\omega}}\left|\frac{1}{T}\sum_{t=1}^{T}h_t - \frac{1}{T}\sum_{t=1}^{T}h^*_t\right|
\end{aligned}$$

$$= \frac{1}{\omega\widehat{\omega}} \left| Eh_t - E^*(h_t^*) + o_p(1) \right| = o_p(1) \text{ as } T \to \infty.$$

part (c)

From part (b) we already have that $\left| \frac{1}{T}\sum_{t=1}^{T} \frac{1}{h_t} - \frac{1}{T}\sum_{t=1}^{T} \frac{1}{h_t^*} \right| = o_p(1)$. Next,

$$\begin{aligned}
&\left| \frac{1}{T}\sum_{t=1}^{T} \varepsilon_{t-k}^{*2}/h_t^{*2} - \frac{1}{T}\sum_{t=1}^{T} \varepsilon_{t-k}^{2}/h_t^{2} \right| \\
&= \left| \frac{1}{T}\sum_{t=1}^{T} \left(\varepsilon_{t-k}^{*2}/h_t^{*2} - \varepsilon_{t-k}^{2}/h_t^{2} \right) \right| \\
&= \left| \frac{1}{T}\sum_{t=1}^{T} \frac{h_t^2\varepsilon_{t-k}^{*2} - h_t^{*2}\varepsilon_{t-k}^{2}}{h_t^2 h_t^{*2}} \right| \\
&= \left| \frac{1}{T}\sum_{t=1}^{T} \frac{h_t^2\varepsilon_{t-k}^{*2} - h_t^2\varepsilon_{t-k}^{2} + h_t^2\varepsilon_{t-k}^{2} - h_t^{*2}\varepsilon_{t-k}^{2}}{h_t^2 h_t^{*2}} \right| \\
&= \left| \frac{1}{T}\sum_{t=1}^{T} \frac{h_t^2\left(\varepsilon_{t-k}^{*2} - \varepsilon_{t-k}^{2}\right) + \left(h_t^2 - h_t^{*2}\right)\varepsilon_{t-k}^{2}}{h_t^2 h_t^{*2}} \right| \\
&\leq \left| \frac{1}{T}\sum_{t=1}^{T} \frac{\left(\varepsilon_{t-k}^{*2} - \varepsilon_{t-k}^{2}\right)}{h_t^{*2}} \right| + \left| \frac{1}{T}\sum_{t=1}^{T} \frac{\left(h_t^2 - h_t^{*2}\right)\varepsilon_{t-k}^{2}}{h_t^2 h_t^{*2}} \right| \\
&\leq \frac{1}{T\widehat{\omega}^2} \left| \sum_{t=1}^{T} \left(\varepsilon_{t-k}^{*2} - \varepsilon_{t-k}^{2}\right) \right| + \frac{1}{T\widehat{\omega}} \left| \sum_{t=1}^{T} \frac{\left(h_t + h_t^*\right)\left(h_t - h_t^*\right)}{h_t h_t^*} \frac{\varepsilon_{t-k}^{2}}{h_t} \right| \\
&\leq \frac{1}{\widehat{\omega}^2} \left| \frac{1}{T}\sum_{t=1}^{T} \varepsilon_{t-k}^{*2} - \frac{1}{n}\sum_{t=1}^{n} \varepsilon_{t-k}^{2} \right| + \frac{\beta^{-(k-1)}}{T\widehat{\omega}} \left| \sum_{t=1}^{T} \left(\frac{1}{h_t^*} + \frac{1}{h_t} \right)\left(h_t - h_t^*\right) \right| \\
&\leq \frac{1}{\widehat{\omega}^2} \left| \frac{1}{T}\sum_{t=1}^{T} \varepsilon_{t-k}^{*2} - \frac{1}{T}\sum_{t=1}^{T} \varepsilon_{t-k}^{2} \right| + \frac{\left(\omega + \widehat{\omega}\right)\beta^{-(k-1)}}{\omega\widehat{\omega}^2} \left| \frac{1}{T}\sum_{t=1}^{T} h_t - \frac{1}{T}\sum_{t=1}^{T} h_t^* \right| \\
&= \frac{1}{\widehat{\omega}^2} \left| E^*\left(\varepsilon_{t-i}^{*2}\right) - E\left(\varepsilon_{t-i}^{2}\right) + o_p(1) \right| + \frac{\left(\omega + \widehat{\omega}\right)\beta^{-(k-1)}}{\omega\widehat{\omega}^2} \left| E\left(h_t\right) - E^*\left(h_t^*\right) + o_p(1) \right| \\
&= \beta^{-(k-1)} o_p(1) \text{ as } T \to \infty.
\end{aligned}$$

Then,

$$
\begin{aligned}
|K-K^*| \leq & \left|\frac{1}{T}\sum_{t=1}^{T}\frac{1}{h_t}-\frac{1}{T}\sum_{t=1}^{T}\frac{1}{h_t^*}\right| + |\,\kappa\alpha^2\sum_{k=1}^{\infty}\beta^{2(k-1)}\left(\frac{1}{T}\sum_{t=1}^{T}\varepsilon_{t-k}^2/h_t^2+o_p(1)\right) \\
& -\kappa^*\hat{\alpha}^2\sum_{k=1}^{\infty}\hat{\beta}^{2(k-1)}\left(\frac{1}{T}\sum_{t=1}^{T}\varepsilon_{t-k}^{*2}/h_t^{*2}+o_p(1)\right)| \\
= & \left|\frac{1}{T}\sum_{t=1}^{T}\frac{1}{h_t}-\frac{1}{T}\sum_{t=1}^{T}\frac{1}{h_t^*}\right| + \left|\left(\kappa\alpha^2-\kappa^*\hat{\alpha}^2\right)\sum_{k=1}^{\infty}\beta^{2(k-1)}E(\varepsilon_{t-k}^2/h_t^2)\right| \\
& + \left|\kappa^*\hat{\alpha}^2\left(\sum_{k=1}^{\infty}\beta^{2(k-1)}\left(\frac{1}{T}\sum_{t=1}^{T}\varepsilon_{t-k}^2/h_t^2+o_p(1)\right)-\sum_{k=1}^{\infty}\hat{\beta}^{2(k-1)}\left(\frac{1}{T}\sum_{t=1}^{T}\varepsilon_{t-k}^{*2}/h_t^{*2}\right)\right)\right| \\
& + o_p(1). \\
= & K_1 + \left|\kappa\alpha^2-\kappa^*\hat{\alpha}^2\right| K_2 + \kappa^*\hat{\alpha}^2 K_3
\end{aligned}
$$

The first term K_1 is $o_p(1)$ (see part (b) above) and from the results of Lemma 2 and the properties of the MLE, it follows that $|\kappa\alpha^2-\kappa^*\hat{\alpha}^2| = o_p(1)$. Furthermore,

$$
\begin{aligned}
K_3 = & \left|\sum_{k=1}^{\infty}\beta^{2(k-1)}\left(\frac{1}{T}\sum_{t=1}^{T}\left(\varepsilon_{t-k}^2/h_t^2-\varepsilon_{t-k}^{*2}/h_t^{*2}\right)\right)+\sum_{k=1}^{\infty}\left(\frac{1}{T}\sum_{t=1}^{T}\left(\beta^{2(k-1)}-\hat{\beta}^{2(k-1)}\right)\varepsilon_{t-k}^{*2}/h_t^{*2}\right)\right| \\
\leq & \left|\sum_{k=1}^{\infty}\beta^{2(k-1)}\cdot\beta^{-(k-1)}o_p(1)\right| + \frac{1}{\widehat{\omega}}\left|\sum_{k=1}^{\infty}\left(\frac{1}{T}\sum_{t=1}^{T}\left(\beta^{2(k-1)}-\hat{\beta}^{2(k-1)}\right)\hat{\beta}^{-(k-1)}\right)\right| \\
\leq & \left|\sum_{k=1}^{\infty}\beta^{k-1}\right| o_p(1) + \frac{1}{\widehat{\omega}}\left|\sum_{k=1}^{\infty}\left(\left(\frac{\beta^2}{\hat{\beta}}\right)^{(k-1)}-\hat{\beta}^{(k-1)}\right)\right| \\
= & \frac{1}{\widehat{\omega}}\left|\sum_{k=1}^{\infty}\left(\left(\frac{\beta^2}{\hat{\beta}}-\hat{\beta}\right)\left(\left(\frac{\beta^2}{\hat{\beta}}\right)^{(k-2)}+\left(\frac{\beta^2}{\hat{\beta}}\right)^{(k-3)}\hat{\beta}+\ldots+\hat{\beta}^{(k-2)}\right)\right)\right| + o_p(1) \\
\leq & \frac{1}{\widehat{\omega}}\left|\sum_{k=1}^{\infty}\left(\frac{\beta^2}{\hat{\beta}}-\hat{\beta}\right)(k-1)\lambda^{(k-2)}\right| + o_p(1) \\
= & o_p(1)\lim_{T\to\infty}\left(\frac{1-\lambda^{T-1}}{1-\lambda}+(T-1)\lambda^{T-1}\right)+o_p(1) = o_p(1),
\end{aligned}
$$

where $\lambda = \beta^2/\hat{\beta} < 1$. Therefore, $|K - K^*| = o_p(1)$ as $T \to \infty$.

part (d)

We can use similar arguments as in part (c) to prove $F^* \to^p F$.

□

Proof of Lemma 5

The structure of the proof for $T^{-1/2}\sum_{t=1}^{[Tr]} \varepsilon_t^* \Rightarrow W_1(r)$ is similar to the proof of Lemma 3 in Gospodinov (2008) for two-parameter partial sum processes. To establish the invariance principle for the partial sum process $W^*_{[Tr]} = T^{-1/2}\sum_{t=1}^{[Tr]} \varepsilon_t^*$, we need to show that, conditionally on the sample, $W^*_{[Tr]} \Rightarrow W_1(r)$ by demonstrating the convergence of the finite-dimensional distributions and verifying the tightness condition.

Let, for any $0 \leq r_{k-1} < r_k \leq 1$,

$$Y^*_{T,k} = W^*_{[Tr_k]} - W^*_{[Tr_{k-1}]} = T^{-1/2} \sum_{t=[Tr_{k-1}]}^{[Tr_k]} \varepsilon_t^*$$

and $Z^*_{T,m} = \{Y^*_{T,1}, ..., Y^*_{T,i}, ..., Y^*_{T,m}\}$ with $Y^*_{T,1} = W^*_{[Tr_1]}$ and $0 < r_1 < ... < r_i < ... < r_m \leq 1$ for $m \geq 1$. Similarly, let $W_{[Tr]} = T^{-1/2}\sum_{t=1}^{[Tr]} \varepsilon_t$, $Y_{T,k} = T^{-1/2}\sum_{t=[Tr_{k-1}]}^{[Tr_k]} \varepsilon_t$ and $Z_{T,m} = \{Y_{T,1}, ..., Y_{T,i}, ..., Y_{T,m}\}$ with $Y_{T,1} = W_{[Tr_1]}$.

From the definition of the Mallows metric, $d_2(Z_{T,m}, Z^*_{T,m}) = \inf\left(E^*\sum_{i=1}^{m} \left|Y_{T,i} - Y^*_{T,i}\right|^2\right)^{1/2}$ conditionally on the sample, where the infimum is taken over all pairs (η_t, η_t^*) with

marginals F and $\widetilde{F}_T^{sym}$ as in Miguel and Olave (1999). Then,

$$\begin{aligned} d_2(Z_{T,m}, Z^*_{T,m})^2 &\leq \sum_{i=1}^{m} E^*(Y_{T,i} - Y^*_{T,i})^2 \\ &= \frac{1}{T}\sum_{i=1}^{m} \sum_{t=[Tr_{i-1}]}^{[Tr_i]} E^*(\varepsilon_t - \varepsilon^*_t)^2 \\ &= \frac{1}{T}\sum_{i=1}^{m} \sum_{t=[Tr_{i-1}]}^{[Tr_i]} E^*(\sqrt{h^*_t}\eta_t - \sqrt{h^*_t}\eta^*_t + (\sqrt{h_t} - \sqrt{h^*_t})\eta_t)^2 \\ &\leq \frac{\sigma^2}{T}\sum_{t=1}^{[Tr_m]} E^*(\eta_t - \eta^*_t)^2 + o_p(1) \end{aligned}$$

using that $h^*_t = h_t + o_p(1)$, $E^*(h^*_t) \xrightarrow{p} \sigma^2$ and h^*_t is independent of $(\eta^*_t, \eta^*_{t+1}, ...)$. Since the infimum in the Mallows metric definition is always attained (Bickel and Freedman, 1981) and $d_2(\widetilde{F}_T^{sym}, F_T^{sym})^2 \to 0$ from part (a) of Lemma 2,

$$d_2(Z_{T,m}, Z^*_{T,m})^2 \leq d_2(\widetilde{F}_T^{sym}, F_T^{sym})^2 \to 0 \text{ as } T \to \infty.$$

Finally, because the Mallows metric convergence implies convergence in distribution and $(W_{[Tr_1]}, ..., W_{[Tr_i]}, ..., W_{[Tr_m]}) \Rightarrow (W_1(r_1), ..., W_1(r_i), ..., W_1(r_m))$ for any m-tuples $0 < r_1 < ... < r_i < ... < r_m \leq 1$, $m \geq 1$,

$$(W^*_{[Tr_1]}, ..., W^*_{[Tr_i]}, ..., W^*_{[Tr_m]}) \Rightarrow (W_1(r_1), ..., W_1(r_i), ..., W_1(r_m))$$

conditionally on $(y_1, ..., y_T)$ and for almost all sample paths $(y_1, y_2, ...)$. This establishes the weak convergence of the finite-dimensional distributions.

To establish the tightness of $W^*_{[Tr]}$, it suffices to show that for almost all sample paths and all $\epsilon > 0$

$$\limsup_{T\to\infty} P^* \left\{ \sup_{|r'-r|<\nu} \left| W^*_{[Tr']} - W^*_{[Tr]} \right| \geq \epsilon \right\} = 0$$

as $\nu \to 0$ for $0 \leq r < r' \leq 1$.

By Chebyshev's inequality,

$$P^* \left\{ \left| W^*_{[Tr']} - W^*_{[Tr]} \right| \geq \epsilon \right\} \leq \frac{E^* \left| W^*_{[Tr']} - W^*_{[Tr]} \right|^2}{\epsilon^2} = \frac{(r'-r)}{\epsilon^2}$$

using that

$$E^* \left[W^*_{[Tr]} W^*_{[Tr']} \right] = T^{-1} \sum_{t=1}^{[Tr]\wedge[Tr']} E^* \left[(\varepsilon^*_t)^2 \right]$$

$$= (r \wedge r') \text{ as } T \to \infty.$$

This completes the proof of $T^{-1/2} \sum_{t=1}^{[Tr]} \varepsilon^*_t \Rightarrow W_1(r)$.

For obtaining the limit of $T^{-1/2} \sum_{t=1}^{[Tr]} \left[\frac{\varepsilon^*_t}{h^*_t} + (1-\eta^{*2}_t) \frac{\widehat{\alpha}}{h^*_t} \sum_{j=1}^{t} \widehat{\beta}^{j-1} \varepsilon^*_{t-j} \right]$, we use similar arguments as in the proof of Lemma 4.2 in Ling and Li (2003). Define the process $S_{[Tr]} = T^{-1/2} \sum_{t=1}^{[Tr]} \xi_t$, where $\xi_t = \lambda_1 \varepsilon^*_t + \lambda_2 [\frac{\varepsilon^*_t}{h^*_t} - \frac{\hat{\alpha}}{h^*_t} (\frac{\varepsilon^{*2}_t}{h^*_t} - 1) \sum_{k=1}^{t-1} \hat{\beta}^{k-1} \varepsilon^*_{t-k}]$. First, we show that ξ_t is a martingale difference sequence with respect to $\mathcal{F}^*_t$, where $\mathcal{F}^*_t$ is a σ-field generated by $\{\eta^*_t, \eta^*_{t-1}, ...\}$.

Taking expectations under P^* of S_T^2, we have

$$
\begin{aligned}
E^*(S_T^2) &= E^*\left(\frac{1}{\sqrt{T}}\sum_{i=1}^{T}\xi_i\frac{1}{\sqrt{T}}\sum_{j=1}^{T}\xi_j\right) = \frac{1}{T}E^*\left(\sum_{i=1}^{T}\sum_{j=1}^{T}\xi_i\xi_j\right) \\
&= \frac{1}{T}\sum_{i=1}^{T}\sum_{j=1}^{T}E^*\{\lambda_1^2\varepsilon_i^*\varepsilon_j^* + \lambda_1\lambda_2\varepsilon_i^*[\frac{\varepsilon_j^*}{h_j^*} - \frac{\hat{\alpha}}{h_j^*}(\frac{\varepsilon_j^{*2}}{h_j^*} - 1)\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\varepsilon_{j-k}^*] \\
&+ \lambda_1\lambda_2\varepsilon_j^*[\frac{\varepsilon_i^*}{h_i^*} - \frac{\hat{\alpha}}{h_i^*}(\frac{\varepsilon_i^{*2}}{h_i^*} - 1)\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\varepsilon_{i-k}^*] \\
&+ \lambda_2^2[\frac{\varepsilon_i^*}{h_i^*} - \frac{\hat{\alpha}}{h_i^*}(\frac{\varepsilon_i^{*2}}{h_i^*} - 1)\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\varepsilon_{i-k}^*][\frac{\varepsilon_j^*}{h_j^*} - \frac{\hat{\alpha}}{h_j^*}(\frac{\varepsilon_j^{*2}}{h_j^*} - 1)\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\varepsilon_{j-k}^*]\} \\
&= \frac{1}{T}\sum_{i=1}^{T}\sum_{j=1}^{T}E^*\left(\lambda_1^2 I_{ij}^1 + \lambda_1\lambda_2 I_{ij}^2 + \lambda_1\lambda_2 I_{ij}^3 + \lambda_2^2 I_{ij}^4\right).
\end{aligned}
$$

If $i \neq j$ and $i > j$, then

$$
E^*\left(I_{ij}^1\right) = E^*\left(\varepsilon_i^*\varepsilon_j^*\right) = E^*\left(\sqrt{h_i^* h_j^*}\eta_i^*\eta_j^*\right) = E^*\left(\eta_i^*\right)E^*\left(\sqrt{h_i^* h_j^*}\eta_j^*\right) = 0
$$

and

$$
\begin{aligned}
E^*\left(I_{ij}^2\right) &= E^*\left\{\varepsilon_i^*[\frac{\varepsilon_j^*}{h_j^*} - \frac{\hat{\alpha}}{h_j^*}(\frac{\varepsilon_j^{*2}}{h_j^*} - 1)\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\varepsilon_{j-k}^*]\right\} \\
&= E^*\left\{\sqrt{h_i^*}\eta_i^*[\frac{\varepsilon_j^*}{h_j^*} - \frac{\hat{\alpha}}{h_j^*}(\frac{\varepsilon_j^{*2}}{h_j^*} - 1)\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\varepsilon_{j-k}^*]\right\} \\
&= E^*\left(\eta_i^*\right)E\left\{\sqrt{h_i^*}[\frac{\varepsilon_j^*}{h_j^*} - \frac{\hat{\alpha}}{h_j^*}(\frac{\varepsilon_j^{*2}}{h_j^*} - 1)\sum_{k=1}^{j-1}\hat{\beta}^{k-1}\varepsilon_{j-k}^*]\right\} = 0
\end{aligned}
$$

using that $E^*\left(\eta_i^*\right) = 0$.

Also.

$$
\begin{aligned}
E^*\left(I_{ij}^3\right) &= E^*\left\{\varepsilon_j^*[\frac{\varepsilon_i^*}{h_i^*} - \frac{\hat{\alpha}}{h_i^*}(\frac{\varepsilon_i^{*2}}{h_i^*} - 1)\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\varepsilon_{i-k}^*]\right\} \\
&= E^*\left[\frac{\varepsilon_i^*\varepsilon_j^*}{h_i^*} - \hat{\alpha}(\frac{\varepsilon_i^{*2}}{h_i^*} - 1)\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\frac{\varepsilon_j^*\varepsilon_{i-k}^*}{h_i^*}\right] \\
&= -\hat{\alpha}E^*\left[(\frac{\varepsilon_i^{*2}}{h_i^*} - 1)\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\frac{\varepsilon_j^*\varepsilon_{i-k}^*}{h_i^*}\right] \\
&= -\hat{\alpha}E^*\left[(\eta_i^{*2} - 1)\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\frac{\varepsilon_j^*\varepsilon_{i-k}^*}{h_i^*}\right] \\
&= -\hat{\alpha}E^*\left(\eta_i^{*2} - 1\right)E^*\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\frac{\varepsilon_j^*\varepsilon_{i-k}^*}{h_i^*} \to 0 \text{ as } T \to \infty
\end{aligned}
$$

and

$$
\begin{aligned}
E^*\left(I_{ij}^4\right) &= E^*\left\{[\frac{\varepsilon_i^*}{h_i^*} - \frac{\hat{\alpha}}{h_i^*}(\frac{\varepsilon_i^{*2}}{h_i^*} - 1)\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\varepsilon_{i-k}^*][\frac{\varepsilon_j^*}{h_j^*} - \frac{\hat{\alpha}}{h_j^*}(\frac{\varepsilon_j^{*2}}{h_j^*} - 1)\sum_{k'=1}^{\infty}\hat{\beta}^{k'-1}\varepsilon_{j-k'}^*]\right\} \\
&= E^*\left\{\frac{\varepsilon_i^*}{h_i^*}[\frac{\varepsilon_j^*}{h_j^*} - \frac{\hat{\alpha}}{h_j^*}(\frac{\varepsilon_j^{*2}}{h_j^*} - 1)\sum_{k'=1}^{\infty}\hat{\beta}^{k'-1}\varepsilon_{j-k'}^*]\right\} \\
&- E^*\left\{[\frac{\hat{\alpha}}{h_i^*}(\frac{\varepsilon_i^{*2}}{h_i^*} - 1)\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\varepsilon_{i-k}^*][\frac{\varepsilon_j^*}{h_j^*} - \frac{\hat{\alpha}}{h_j^*}(\frac{\varepsilon_j^{*2}}{h_j^*} - 1)\sum_{k'=1}^{\infty}\hat{\beta}^{k'-1}\varepsilon_{j-k'}^*]\right\} \\
&= -E^*\left\{[\frac{\hat{\alpha}}{h_i^*}(\frac{\varepsilon_i^{*2}}{h_i^*} - 1)\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\varepsilon_{i-k}^*][\frac{\varepsilon_j^*}{h_j^*} - \frac{\hat{\alpha}}{h_j^*}(\frac{\varepsilon_j^{*2}}{h_j^*} - 1)\sum_{k'=1}^{\infty}\hat{\beta}^{k'-1}\varepsilon_{j-k'}^*]\right\} \\
&= -E^*\{\hat{\alpha}[(\frac{\varepsilon_i^{*2}}{h_i^*} - 1)\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\frac{\varepsilon_j^*\varepsilon_{i-k}^*}{h_i^*h_j^*}] \\
&- \hat{\alpha}^2[(\frac{\varepsilon_i^{*2}}{h_i^*} - 1)(\frac{\varepsilon_j^{*2}}{h_j^*} - 1)\sum_{k=1}^{\infty}\sum_{k'=1}^{\infty}\hat{\beta}^{k-1}\hat{\beta}^{k'-1}\frac{\varepsilon_{i-k}^*\varepsilon_{j-k'}^*}{h_i^*h_j^*}]\}.
\end{aligned}
$$

$$= -E^*\{\hat{\alpha}[(\eta_i^{*2}-1)\eta_j^* \sum_{k=1}^{\infty} \hat{\beta}^{k-1} \frac{\varepsilon_{i-k}^*}{h_i^* \sqrt{h_j^*}}]$$

$$- \hat{\alpha}^2[(\eta_i^{*2}-1)(\eta_j^{*2}-1) \sum_{k=1}^{\infty}\sum_{k'=1}^{\infty} \hat{\beta}^{k-1}\hat{\beta}^{k'-1} \frac{\varepsilon_{i-k}^*\varepsilon_{j-k'}^*}{h_i^* h_j^*}]\}$$

$$\to 0 \text{ as } T \to \infty$$

from Lemma 2.

Combining the above results imply that

$$E^*(S_T^2) = \frac{1}{n}\sum_{i=1}^{n} E^*\left(\lambda_1^2 I_{ii}^1 + \lambda_1\lambda_2 I_{ii}^2 + \lambda_1\lambda_2 I_{ii}^3 + \lambda_2^2 I_{ii}^4\right) + o_p(1)$$

$$= \frac{1}{n}\sum_{i=1}^{n}[\lambda_1^2 E^*\left(\varepsilon_i^{*2}\right) + 2\lambda_1\lambda_2 E^*\left(\frac{\varepsilon_i^{*2}}{h_i^*}\right)$$

$$+ \lambda_2^2 E^*\left(\frac{\varepsilon_i^{*2}}{h_i^{*2}} + \hat{\alpha}^2\left(\frac{\varepsilon_i^{*2}}{h_i^*} - 1\right)^2 \sum_{k=1}^{\infty} \hat{\beta}^{2(k-1)} \frac{\varepsilon_{i-k}^{*2}}{h_i^{*2}}\right)] + o_p(1)$$

$$= \frac{1}{n}\sum_{i=1}^{n}\{\lambda_1^2 E^*\left(\varepsilon_i^{*2}\right) + 2\lambda_1\lambda_2 E^*\left(\eta_i^{*2}\right)$$

$$+ \lambda_2^2[E^*(\frac{\eta_i^{*2}}{h_i^*}) + \hat{\alpha}^2 E^*\left(\eta_i^{*4} - 2\eta_i^{*2} + 1\right)^2 \sum_{k=1}^{\infty} \hat{\beta}^{2(k-1)} E^*(\frac{\varepsilon_{i-k}^{*2}}{h_i^{*2}})]\} + o_p(1)$$

$$= \frac{1}{n}\sum_{i=1}^{n}\left[\lambda_1^2\sigma^{*2} + 2\lambda_1\lambda_2 + \lambda_2^2\left(E\left(1/h_i^*\right) + \kappa^*\hat{\alpha}^2 \sum_{k=1}^{\infty} \hat{\beta}^{2(k-1)} E^*\left(\varepsilon_{i-k}^{*2}/h_i^{*2}\right)\right) + o(1)\right]$$

$$+ o_p(1)$$

$$= \lambda_1^2\sigma^{*2} + 2\lambda_1\lambda_2 + \lambda_2^2\left(E\left(1/h_t^*\right) + \kappa^*\hat{\alpha}^2 \sum_{k=1}^{\infty} \hat{\beta}^{2(k-1)} E^*\left(\varepsilon_{t-k}^{*2}/h_t^{*2}\right)\right) + o_p(1)$$

$$\to \lambda' \Omega^* \lambda < \infty \text{ as } T \to \infty,$$

where $\sigma^{*2} = E^*(\varepsilon_t^{*2}) = E^*(h_t^*)$.

Note that $E^*\left(\xi_t^2|\mathcal{F}_{t-1}^*\right) = \lambda_1^2 h_t^* + 2\lambda_1\lambda_2 + \lambda_2^2\left(\frac{1}{h_t^*} + \kappa^*\left(\frac{\hat{\alpha}}{h_t^*}\sum_{k=1}^{\infty}\hat{\beta}^{k-1}\varepsilon_{t-k}^*\right)^2\right)$ as $T \to \infty$. Since ε_t^* is strictly stationary and ergodic and from Lemma 2, it follows that $E^*\left(\xi_t|\mathcal{F}_{t-1}^*\right) = 0$ and ξ_t is a strictly stationary and ergodic martingale difference sequence. Then, from the ergodic theorem, $[E^*(S_T^2]^{-1}\left[\frac{1}{T}\sum_{t=1}^{T}E^*\left(\xi_t^2|\mathcal{F}_{t-1}^*\right)\right] \to 1$ a.s. Furthermore, it implies that $\{\xi_t^2\}$ is uniformly integrable and for any $\epsilon > 0$

$$\frac{1}{\sigma_T^2}\sum_{i=1}^{T}E^*\left[\xi_i^2 I(\xi_i^2 > \sigma_T^2\epsilon)\right] \to 0 \text{ as } T \to \infty, \tag{3.16}$$

where $\sigma_T^2 = E^*\left[\left(\sum_{t=1}^{T}\xi_t\right)^2\right]$. Also,

$$\begin{aligned}\frac{1}{\sigma_T^2}\max_{0\le i\le T}\xi_i^2 &= \frac{1}{\sigma_T^2}\max_{0\le i\le T}\left[\xi_i^2 I(\varepsilon_i^2 \le \sigma_T^2\epsilon) + \xi_i^2 I(\xi_i^2 > \sigma_T^2\epsilon)\right]\\ &\le \frac{1}{\sigma_T^2}\max_{0\le i\le T}\left[\sigma_T^2\epsilon + \xi_i^2 I(\xi_i^2 > \sigma_T^2\epsilon)\right] = \epsilon + \frac{1}{\sigma_T^2}\max_{0\le i\le T}\xi_i^2 I(\xi_i^2 > \sigma_T^2\epsilon)\\ &\le \epsilon + \frac{1}{\sigma_T^2}\sum_{i=1}^{T}\xi_i^2 I(\xi_i^2 > \sigma_T^2\epsilon)\end{aligned}$$

and $E\left(\sigma_T^{-2}\max_{0\le i\le T}\xi_i^2\right) \to 0$ from (3.16) and since ϵ is arbitrary small. Then, from Markov's inequality,

$$\frac{1}{\sigma_T}\max_{0\le i\le T}|\xi_i| \to 0 \text{ as } T \to \infty.$$

Therefore, the conditions for the FCLT for martingale difference sequences are satisfied (Theorem 4.1 in Hall and Heyde, 1980; Theorem 27.14 in Davidson, 1994) and from $T^{-1/2}\sum_{t=1}^{[Tr]}\varepsilon_t^* \Rightarrow W_1(r)$ and Lemma 4,

$$S_{[Tr]} \Rightarrow W_2(r).$$

□

Proof of Theorem 1

Recall that

$$\phi^*_{ML} = \widetilde{\phi}^* - \left[\sum_{t=1}^{T} \frac{\partial^2 l_t^*(\phi, \delta^*)}{\partial \phi^2}\right]^{-1}_{\phi=\widetilde{\phi}^*} \left[\sum_{t=1}^{T} \frac{\partial l_t^*(\phi, \delta^*)}{\partial \phi}\right]_{\phi=\widetilde{\phi}^*}$$

and

$$T(\phi^*_{ML} - 1) = -\left[\frac{1}{T^2}\sum_{t=1}^{T} \frac{\partial^2 l_t^*(\phi, \delta^*)}{\partial \phi^2}\right]^{-1}_{\phi=1} \left[\frac{1}{T}\sum_{t=1}^{T} \frac{\partial l_t^*(\phi, \delta^*)}{\partial \phi}\right]_{\phi=1}.$$

Following Ling and Li (2003), the first two derivatives of the likelihood for observation t with respect to ϕ can be expressed as

$$\left[\frac{1}{T}\sum_{t=1}^{T} \frac{\partial l_t^*(\phi, \delta^*)}{\partial \phi}\right]_{\phi=1} = \frac{1}{T}\sum_{t=1}^{T} y^*_{t-1}\left[\frac{\varepsilon_t^*}{h_t^*} + (1-\eta_t^{*2})\frac{\widehat{\alpha}}{h_t^*}\sum_{j=1}^{t}\widehat{\beta}^{j-1}\varepsilon^*_{t-j}\right] + o_p(1)$$

$$-\left[\frac{1}{T^2}\sum_{t=1}^{T} \frac{\partial^2 l_t^*(\phi, \delta^*)}{\partial \phi^2}\right]_{\phi=1} = T^{-2}\sum_{t=1}^{T} y^{*2}_{t-1}\left[\frac{1}{h_t^*} + 2\widehat{\alpha}^2\frac{\varepsilon_t^{*2}}{h_t^*}\sum_{j=1}^{t}\widehat{\beta}^{2(j-1)}\frac{\varepsilon^{*2}_{t-j}}{h_t^{*2}}\right] + o_p(1)$$

$$= \left[T^{-2}\sum_{t=1}^{T} y^2_{t-1}\right]\left[E^*\left(\frac{1}{h_t^*}\right) + 2\widehat{\alpha}^2\frac{\varepsilon_t^{*2}}{h_t^*}\sum_{j=1}^{t}\widehat{\beta}^{2(j-1)}E^*\left(\frac{\varepsilon^{*2}_{t-j}}{h_t^{*2}}\right)\right]$$

$$+ o_p(1).$$

From Lemmas 4 and 5 and the continuous mapping theorem

$$\left[\frac{1}{T}\sum_{t=1}^{T} \frac{\partial l_t^*(\phi, \delta^*)}{\partial \phi}\right]_{\phi=1} \Rightarrow \int_0^1 W_1(r)dW_2(r)$$

and

$$-\left[\frac{1}{T^2}\sum_{t=1}^{T}\frac{\partial^2 l_t^*(\phi,\delta^*)}{\partial\phi^2}\right]_{\phi=1} \Rightarrow F\int_0^1 W_1(r)^2 dr.$$

Thus,

$$\left[-\sum_{t=1}^{T}\frac{\partial^2 l_t^*(\phi,\delta^*)}{\partial\phi^2}\right]^{1/2}_{\phi=\phi^*_{ML}} (\phi^*_{ML}-1) \Rightarrow \frac{1}{\sqrt{F}}\frac{\int_0^1 W_1(r)dW_2(r)}{\left(\int_0^1 W_1(r)^2dr\right)^{1/2}}.$$

Define $W_1(r)=\sigma B_1(r)$, $\rho = 1/\sigma\sqrt{K}$ and $W_2(r)=\sqrt{K}[\rho B_1(r)+\sqrt{1-\rho^2}B_2(r)]$, where $B_1(r)$ and $B_2(r)$ are two independent standard Brownian motions. Substituting for $W_1(r)$ and $W_2(r)$ in the above expression, we obtain

$$\left[-\sum_{t=1}^{T}\frac{\partial^2 l_t^*(\phi,\delta^*)}{\partial\phi^2}\right]^{1/2}_{\phi=\phi^*_{ML}} (\phi^*_{ML}-1) \Rightarrow \sqrt{\frac{K}{F}}\left[\rho\frac{\int_0^1 B_1(r)dB_2(r)}{\left(\int_0^1 B_1^2(r)dr\right)^{1/2}}+\sqrt{1-\rho^2}\frac{\int_0^1 B_1(r)dB_2(r)}{\left(\int_0^1 B_1^2(r)dr\right)^{1/2}}\right].$$

Noting that $\left(\int_0^1 B_1^2(r)dr\right)^{-1/2}\int_0^1 B_1(r)dB_2(r)$ is distributed as a standard normal random variable yields the desired result.

□