MONASH University
Business and Economics

Centre for Health Economics

Research Paper 2007 (20)

Does Exercise Reduce Obesity? Evidence from Australia

Professor Pushkar Maitra
Professor, Department of Economics,
Monash University

Dr Anurag Sharma
Research Fellow, Centre for Health Economics,
Monash University

October, 2007

ISSN 1833 – 1173
ISBN 1 921187 19 0

Does Exercise Reduce Obesity? Evidence from Australia *

Pushkar Maitra† and Anurag Sharma‡

October 2007

Abstract

The International Obesity Taskforce calls obesity *one of the most important medical and public health problems of our time.* An estimated 1 billion people around the world are over weight, of whom around 300 million are clinically obese. Estimates suggest that obesity levels will continue to rise in the early 21^{st} century - with severe health consequences in the absence of quick and directed intervention. Leaving genetics aside, obesity is essentially due to an imbalance between caloric intake and expenditures i.e, too high caloric intake and too low caloric expenditure. A large part of the economic research on obesity has focused on factors that lead to this imbalance. In this paper we examine the relationship between obesity (as measured by BMI) and the duration of exercise. Single equation estimates show that exercise duration has a negative and statistically significant effect on the probability of being overweight or obese. However when we take into account the potential endogeneity of exercise duration in the BMI regressions (arising from a standard problem of reverse causation), we no longer find a negative relationship between exercise duration and BMI. There is either no effect or the effect is actually positive indicating that the results are essentially driven by individuals who are and who perceive themselves to be overweight and obese conducting more exercise.

Keywords: Obesity, Exercise, Australia
JEL Codes: I10, I12, C25

*Funding provided by the Australian Research Council Discovery Grant Scheme. We would like to thank participants at the AHES Annual conference at Brisbane for their comments on an earlier version of the paper. The usual caveat applies.

†Pushkar Maitra, Department of Economics, Monash University, Clayton Campus, VIC 3800. Email: Pushkar.Maitra@Buseco.monash.edu.au

‡Anurag Sharma, Centre for Health Economics, Monash University, Clayton Campus, VIC 3800. Email: Anurag.Sharma@Buseco.monash.edu.au

1 Introduction

The International Obesity Taskforce (http:/lwww.iotf.org) calls obesity *one of the most important medical and public health problems of our time*. Obesity is defined as a condition of excess body fat and is associated with a large number of debilitating and life-threatening disorders. Health experts argue that given the height of an individual, the weight of that individual should lie within a certain range for the individual to he of 'normal' weight. The measure most commonly used to assess if an individual is obese is that of the Body Mass Index (BMI). This is a simple ratio of weight divided by the square of the height (kg/m^2). An individual with a BMI of less than 18.5 is termed as *underweight*, an individual with a BMI in the range $18.5 - 25.0$ is termed as *normal*, an individual with a BMI in the range $25.0 - 30.0$ is said to be *overweight* and finally an individual with a BMI greater than 30.0 is said to be *obese*.

An estimated 1 billion people around the world are over weight, of whom around 300 million are clinically obese (WHO, 2004). Estimates suggest that obesity levels will continue to rise in the early 21^{st} century - with severe health consequences in the absence of quick and directed intervention. Obesity levels in some countries have doubled in recent years. The problem is unfortunately not restricted to developed countries. Even in many developing countries, where chronic malnutrition is still a major problem facing large sections of the population (overall around 800 million people worldwide suffer from malnutrition), other sections of population are experiencing significant increases in obesity levels. The consequences of ignoring obesity are increasing levels of serious illness and rising health costs. For example In the US more people are obese than the number who smoke, use illegal drugs or are in ill health for reasons unrelated to obesity. Finkelstein et al. (2003) estimate that in the US medical expenditure attributable to obesity reached $75 billion in 2003. Obesity has been viewed as a major risk factor for non-communicable diseases like heart problems, cancer and diabetes; it now rivals asthma in terms of chronic disease burden. It has been proved to have significant economic and psychological costs relating to low wages, productivity, absenteeism and low self-esteem (Hamermesh and Biddle, 1994; Averett and Korenman, 1999; Cawley, 2004; Baum and Ford, 2004; Morris, 2006).

Australia is not immune to this global problem of excessive weight. Australians are at the point where it is almost more "normal" to have a weight problem than not. It has been estimated that obesity and its associated illnesses cost the Australian society and governments a total of $21 billion in 2005 (AE, 2006). The 1999 - 2000 Australian Diabetes, Obesity and Lifestyle Study indicated over seven million adult Australians aged 25 years and over (60%) were overweight. Of these, over two million (21%) were obese. Men were more likely than women to be overweight, with 67% of men compared with 52% of women aged 25 years and over being overweight. There have been significant increases in the proportion of overweight and obese Australians over the last 20 years. From 1980 to 2000, for people aged 25-64 years, the proportion of overweight women increased from 27% to 47%, and the proportion of overweight men from 47% to 66%. On average, women in 1999 weighed 4.8 kg more than their counterparts in 1980 and men 3.6 kg more.

Leaving genetics aside, obesity is due to an imbalance between caloric intake and expenditures i.e, too high caloric intake and too low caloric expenditure. A large part of the economic research on obesity has focused on factors that lead to this imbalance. For example Cutler et al. (2003) argue that Americans have become more obese over the past 25 years primarily because of increased caloric intake outside of the main meals (more snacking). Chou et al. (2004) argue that this increased obesity rate in the US is the result of changes in social structures and forces that have altered an individual's time allocation for food preparation and consumption in an industrialized society like the US. They argue that given the high value of time in industrialized societies, individuals typically end up devoting more time to the labour market and thereby having less time available for food preparation and leisure. Philipson and Posner (2003), Philipson (2002) and Lakdawalla and Philipson (2002a) on the other hand argue that increases in BMI over time are related to a lower use of calories, arising primarily from reductions in the strenuousness of work. They argue that changes in the strenuousness of work or physical activity, resulting from economic development is a major factor contributing to BMI growth in recent years (Lakdawalla and Phillipson, 2006).

Whatever might be the cause, the important question facing health experts and policy makers is how to combat this serious problem. Regular exercise is increasingly regarded as an effective option to address the problem of obesity. Primarily, the research focuses on two strategies: (1) Exercise for weight loss, with the philosophy that lower body weight can lead to better health; and (2) exercise for health and fitness, with the philosophy that correct exercise can lead to better health, no matter what the resulting weight change. In order to lose weight an energy deficit must be met. This means that throughout the day and individual must burn (expend) more calories than they consume (absorb) from food.[1] If the goal is to lose weight, the combination of energy intake reductions and energy expenditure increases through structured exercise and other forms of physical activity are recommended as the ideal weight loss intervention. Exercise can be a key component for inducing an energy deficit. When individuals exercise, they expend more calories compared to when they are sedentary. Structured exercise interventions alone can produce weight loss as well as changes in body composition. So great is the emphasis on exercise, that in 2006 the Prime Minister of Australia, John Howard, introduced legislation in the federal parliament, which stipulated a minimum physical activity requirement of two hours a week for primary and junior secondary schools for schools to have a slice of a federal funding. This, in his opinion, is an important step in addressing childhood obesity.[2] For adults, exercise has been associated with decreased instances of heart disease, dyslipidemia (cholesterol abnormalities), hypertension (high blood pressure), renal (kidney) disease, hyperinsulinemia, type II diabetes, cancer, osteoarthritis, and mortality. People that exercise not only experience lower disease risk, but also report mental differences such as improved mood.

In this paper we will re-examine the relationship between obesity (as measured by BMI) and the duration of exercise. The simplest way of analyzing this relationship would be to include the duration of exercise as an explanatory variable in regressions where the

[1] Many attempt to lose weight by dietary modification alone. This, when done appropriately can generate positive results. However, many individuals that modify their diet do so inappropriately by either not getting enough calories or choosing diets that lack proper nutrients and balance. The *cliche* term, yo-yo dieting, is often used to describe the up-and-down weight fluctuations that can occur with drastic dietary plans. Some studies have implied that weight fluctuations may be more harmful than remaining overweight. Diet alone is seldom recommended by health and medicine based professional organizations. One reason for this is that diet without exercise does not improve cardiorespiratory fitness, which is a necessary requirement for burning calories.

[2] This is a part of a five year $500 million program (the Australian Better Health Initiative) aimed at reducing the impacts of chronic disease, which includes a focus on promoting healthy weight.

dependent variable is BMI (or the propensity of being overweight or obese). If the duration of exercise has a negative and statistically significant effect on BMI we can say that increased exercise is associated with a reduction in BMI levels. However this is merely an association and we cannot say anything about causation. It is possible that overweight and obese individuals are more likely to conduct more exercise and the causation actually runs the other way. What we could have here is the standard problem of reverse causation (or feed back effect) where the dependent variable (in this case BMI) causes, either directly or indirectly, one of its causes. Lakdawalla and Phillipson (2006) argue that eating and exercise are complementary to each other. Most of the developed countries have faced a significant reduction in food prices in last three decades and this same period has also witnessed a significant increase in average BMI levels. Such a BMI growth has also increased the value of weight control activities like exercise for individuals who perceive themselves to be overweight. This has led to a rise in leisure based exercise along with BMI growth. The authors further observe that for the US, the growth in BMI exceeds the growth in exercise resulting in higher BMI *and* more exercise. In econometric terms such an interaction between BMI and exercise indicates towards a potential endogeneity problem and not taking into account could result us obtaining biased estimates. Indeed we find that in most cases the null hypothesis of exogeneity of exercise duration in the BMI regressions is rejected and most importantly a negative relationship between exercise duration and BMI no longer exists, once we account for this endogeneity. There is either no effect or the effect is actually positive, indicating that the results are essentially driven by individuals who are and who perceive themselves to be overweight and obese conducting more exercise.

2 Data and Descriptive Statistics

The data used in this paper come from the National Health Survey (NHS) data sets for Australia for 1989, 1995, 2001 and 2005. These were cross-sectional representative surveys conducted by the Australian Bureau of Statistics (ABS) as a part of a series of regular health surveys, which collect information about the health status of Australians, their use of health services and facilities, and health related aspects of their lifestyle. These surveys are designed to obtain national benchmark information on a range of health issues, enable trends in health to be monitored over time, and provide information on health indicators for national health priority areas and for important subgroups of the population. The surveys covered urban and rural areas across all States and Territories and included residents of private dwellings only. Visitors to private dwellings were excluded, as were persons in institutions such as hospitals and nursing homes and special dwellings such as hotels and boarding houses.

The content of the surveys in the NHS series has differed between surveys, around a common core. Typically however information was collected on the following set of variables: indicators of health status (self-assessed health status, health transition, quality of life, psychological distress, long-term conditions focussing in particular on asthma, diabetes, cardiovascular and cancer), and injuries; health-related actions taken (visits to hospitals and day clinics, consultations with doctors, dentists and other health professionals), use of medications (for national health priority area conditions only), days away from work and other days of reduced activity), health risk factors (smoking, alcohol consumption, diet, exercise, body mass, sun protection, breastfeeding, immunisation); supplementary women's health items (breast and cervical cancer screening practices, contraceptive/protective behaviours, hormone replacement therapy, breastfeeding history); and demographic and socio-economic characteristics (including Indigenous status, private health insurance and housing). The sample size varies across surveys. The focus of the paper is on the relationship between exercise and *adult* obesity and we therefore restrict our estimating sample to individuals aged 20 and higher.

Table 1 presents descriptive statistics for the variables of interest. Looking at the averages across the four survey years gives us a good idea of the broad trends in obesity and exercise patterns in Australia over the period 1989 - 2005. The average BMI (measured using a 4 point scale: BMI = 1 if underweight; BMI = 2 if normal; BMI = 3 if overweight; BMI = 4 if obese) has increased from 2.4 in 1989 to 2.7 in 2005. For men the average BMI has increased from 2.5 in 1989 to 2.8 in 2005, an increase of 13.25% over the 16 year period, compared to an increase of 16% for women over the same period (from 2.25 in 1989 to 2.6 in 2005). 60% of the sample did not engage in any exercise during the past fortnight, down from 62.4% in 1989. When we look at the numbers separately for men and women, we find that interestingly women are more likely to have not engaged in any exercise during the past fortnight compared to men and the gender difference is statistically significant in each of the survey years. Even for those who did engage in moderate or vigourous exercise over the past fortnight, the average duration of exercise has decreased over the period 1989 - 2005. Overall the duration of exercise in the past fortnight has gone down from 6.7 hours in 1989 to 5.9 hours in 2005, a decrease of 12.4%. For men this decline is 13.25% (down from 7.9 hours in 1989 to 6.8 hours in 2005), while for women this decline is 9% (down from 5.5 hours in 1989 to 4.9 hours in 2005). The gender difference in time spent on exercise is statistically significant in each of the four survey years. Thus though both BMI and proportion of individuals undertaking exercise has increased over the period 1989 - 2005, the duration of exercise per fortnight has fallen over the same period.

Table 2 examines the time patterns in obesity (and BMI) in greater detail. The percentage of the sample that is obese has increased monotonically from 9.3% in 1989 to 19% in 2005: a 105% increase over the 16 year period. The numbers are even more striking if we combine the overweight and the obese: 54.3% of the sample in 2005 are either overweight or obese. If we look at the percentages by gender, while the average BMI levels have had a greater increase in the case of women, obesity rates have increased at a faster rate for men: the percentage of men who are obese has increased from 8.7% in 1989 to 19.5% in 2005 while the percentage of women who are obese has increased from 9.8% in 1989 to 18.6% in 2005. The gender effect becomes stronger if we combine the overweight and the obese. 62.9% of the sample of men are either overweight or obese in the 2005 sample (up from 46% in 1989) compared to 46.65% of women in 2005 (up from 31.8% in 1989). Conversely being underweight appears to be a greater problem in the case of women. The percentage of women that are underweight is greater in each of the survey years, though there is a strong downward trend for both men and women.

When we looked at the data on the actual duration of exercise ($EXERCISE$) in minutes, we found that the data was characterized by large mass points at 30 mins, 60 mins, 90 mins, 120 mins and so on. We therefore compressed the duration of exercise into 7 categories

($EXCAT$) as follows:

$$EXCAT = \begin{cases} 0 & \text{if } EXERCISE = 0 \\ 1 & \text{if } EXERCISE \in (0, 60] \text{ mins} \\ 2 & \text{if } EXERCISE \in (60, 120] \text{ mins} \\ 3 & \text{if } EXERCISE \in (120, 240] \text{ mins} \\ 4 & \text{if } EXERCISE \in (240, 480] \text{ mins} \\ 5 & \text{if } EXERCISE \in (480, 840] \text{ mins} \\ 6 & \text{if } EXERCISE > 840 \text{ mins} \end{cases}$$

Table 3 presents the distribution in these different exercise duration categories. The gender difference is generally statistically significant in each of the survey years and in each of the exercise categories, with the proportion of men in each of the categories being greater compared to the proportion of women (except of course the no exercise category).

Figure 4 presents the raw correlation between exercise duration and BMI for the four survey years. It is worth noting that in general and irrespective of the duration of exercise, average BMI has increased over the period 1989 - 2005 and this is true for both men and women. Additionally there appears to be a negative correlation between exercise duration and BMI. For the full sample, the correlation between $EXCAT$ and BMI are -0.0574, -0.0485, -0.0734 and -0.0685 for the 1989, 1995, 2001 and 2005 surveys respectively and all are statistically significantly different from 0 at 1% level of significance.

Before proceeding to the regressions and the econometric analysis, some more discussion of the effect of the different socio-economic characteristics on BMI across the different survey years might be worthwhile. Figure 1 presents the average BMI across the different income deciles for the different survey years. There are some interesting common features across the different survey years: most importantly there is a jump in the average BMI for the households in the 2^{nd} (for the 2001 and 2005 surveys) or the 3^{rd} (for the 1989 and 1995 surveys) income deciles, but otherwise (with the exception of the 2005 survey) the average BMI is quite flat across the different income deciles. For the 2005 survey data we find an increasing trend in the average BMI as one goes up the income scale. This pattern is essentially driven by the rich, overweight/obese males: average BMI levels show a downward trend for females as one moves up the income deciles.

Figure 2 presents the average BMI by the different age categories. Looking at the cross-sectional data, for each of the survey years, we find an inverted u-shaped relationship between age and average BMI, with the peak being attained in the age category $55 - 64$. There are some interesting cohort effects as well. While the NHS collect data at only one point in time, it is possible to observe changes over time in the average BMI for a cohort of people born in the same 10 year period. For example, survey respondents aged $25 - 34$ in and those aged $35 - 44$ in 2005 essentially come from the same group born between 1960 and 1970. Average BMI has increased for every cohort: the only exception being those aged $55 - 64$ in 1995. For these individuals, average BMI shows a slight decline. The pattern is similar for males and females.

Figure 3 presents the average BMI for the population classified by educational attainment. In general the average BMI is lower for those with a post-graduate qualification, compared to those who have no formal schooling. The gender specific averages show the same pattern.

3 Analytical Framework

The theoretical frameworks that underlie the empirical analysis of obesity can be classified into two categories: neoclassical and behavioral. The neoclassical framework includes models where individuals compare lifetime costs and benefits of weight gain (Philipson and Posner, 2003; Lakdawalla and Philipson, 2002a). Weight is considered as a durable capital good and exercise and food intake are the main determinants of change in weight. An individual derives utility from eating and level of weight but might derive disutility from exercise. One of the main implications of the neoclassical theory is that being overweight lowers the value of eating and increases the value of exercise. This is because the marginal benefit of eating today is equal to the current pleasure of eating, plus the present-discounted marginal (dis)utility of weight gain. Cawley (1999) extends the basic neoclassical framework by modelling the effect of 'rational addiction' on weight: he argues that due to such addiction past eating can raise the marginal utility of current eating. The behaviorial models (see for example Cutler et al. (2003)) agree that eating is addictive but in addition assume that individuals with addiction have problems with self-control leading to non-commitment to the future consumption of food, exercise and weight. Thus in a behavioural framework it is assumed that individuals use a hyperbolic discount factor, while the neoclassical framework where individuals discount their future exponentially. Lakdawalla and Phillipson (2006) argue that despite the structural differences between neoclassical and behavioural models, both predict a similar impact of addiction on weight, i.e. lead to similar reduced form estimating equations. Unfortunately behavioural theories explain little about the cross sectional heterogeneity and dynamic trends of weight, which is the primary focus of this paper. Accordingly in this paper we base our analytical approach on the neoclassical framework.

Following Chou et al. (2004) and Rashad (2006), we assume that the body mass index (BMI) of an individual is the sum of energy balance in all periods and a set of variables ($\mathbf{X}$), which are specific to the individual and can be viewed as the individual's predisposition towards being overweight or obese. If we define the energy balance (E) at time t as the excess of calorie intake (C) over energy expenditure (A), we can write

$$BMI = f(\sum_t E; \mathbf{X}, \varepsilon) \quad (1)$$

where ε denotes a set of unobserved individual specific characteristics that affect the body mass index of the individual. Assuming a linear functional form for $f(.)$ in equation (1) the estimating equation can be written as:

$$BMI = \beta_0 + \beta_1 \boldsymbol{X} + \beta_2 E + \varepsilon \quad (2)$$

Note that the empirical model focuses on *current* energy balance rather than energy balance accumulated over time. While it is true that this could result in an underestimation of the effect of E on BMI, accounting for accumulated energy balance is difficult because of lack of adequate data.

Given the categorical nature of the BMI variable in our data (see details below), equation (2) is estimated using ordered probit, where the dependent variable takes the following values:

$$BMI = \begin{cases} 0 & \text{if BMI} < 18.5\text{: underweight} \\ 1 & \text{if BMI} \in [18.5,\ 25.0)\text{: normal} \\ 2 & \text{if BMI} \in [25.0,\ 30.0)\text{: overweight} \\ 3 & \text{if BMI} \geq 30.0)\text{: obese} \end{cases}$$

This also implies that an individual's BMI is an ordinal (as opposed to cardinal) measure of his/her weight related health status. This preserves the underlying ordinal nature of the BMI index but at the same time assumes that individuals in the same health category are of an approximately equivalent weight related health level.

While most individuals desire to be fit and of normal weight, it is an unavoidable fact of life that some individuals derive more utility out of food consumption compared to others. Individuals undertake different activities to try and restrict their weight. As noted earlier, the particular activity we will consider in this paper is the amount of physical activity undertaken reduce weight: the duration of moderate or vigorous exercise conducted by the individual in the past fortnight. One would expect an increase in the duration of exercise to have a negative effect on BMI i.e., we expect the coefficient estimate β_2 to be negative and statistically significant if this simple relationship between physical activity (exercise duration) and BMI is true.

That is however not the end of the story. Suppose estimation of equation (2) yields a negative and statistically significant $\hat{\beta}_2$. Can we then necessarily conclude that conducting more exercise *causes* the individual to be less likely to be overweight or obese? Is it not possible that overweight and obese individuals are more likely to conduct more exercise and the causation actually runs the other way?[3] What we have here is the standard problem of reverse causation (or feed back effect) where the dependent variable (in this case BMI) causes, either directly or indirectly, one of its causes. The causal variable (E) in this case is correlated with ε, the error term in equation (2) and we have a potential endogeneity problem. The standard estimation procedure in this case is to use instrumental variable estimation.

The main difficulty with instrumental variable estimation is to obtain proper instruments: variables which are correlated with the potentially endogenous variable (E), but are uncorrelated with the dependent variable BMI. We use as instruments variables, which are likely to be correlated with the magnitude of exercise conducted in the last fortnight but is not likely to have a direct effect on the individual's BMI. We assume that the duration

[3]Per se there is no problem with this: after all the ultimate aim is to lose weight. However it is the issue of causation that we are concerned with.

of moderate or vigorous exercise is an indicator of health consciousness. Accordingly we choose as instruments variables that are likely to represent health conscious behaviour on the part of individuals, thereby being correlated with exercise duration. However because of changes in the survey questionnaire we are unable to use the same set of variables as instruments across the four survey years.[4] For the 2001 and 2005 data, we use as instruments (i) whether the individual consumes two or more serves of fruit during the day and (ii) whether the individual consumes two or more serves of vegetables during the day. Health experts encourage consumption of fruits and vegetables as a part of healthy eating habits and we assume that individuals who consume at least two serves of fruits and vegetables a day are more health conscious and are more likely to spend more time conducting moderate or vigorous exercise. These instruments are not directly correlated with BMI. The correlation between fruit serves and BMI is -0.003 for 2005 and 0.003 for 2001. Similarly the correlation between vegetable serves and BMI is 0.002 for 2005 and 0.004 for 2001.[5] For the 1989 and 1995 data we use as instrument whether the individual is a member of a private health fund and has ancillary cover. A person who values exercise more is a health conscious person and is more likely to take the ancillary cover, which includes services like massage, chiropractic and dental. It should be noted that this argument for participation in private health insurance market is not valid for survey years 2001 and 2005. This is mainly because post- 2000 individuals decision to take private health insurance cover is determined by the federal government's policy of tax rebates for subscribers and penalties for non subscribers.

There is however one other issue that needs to be pointed out. There is an argument that riskier individuals are more likely to take up private insurance. Empirically however the evidence is mixed. For example Savage and Wright (2003) find evidence in favour of adverse selection. Doiron et al. (2007) find a positive relationship between risk and self-assessed health after using more objective health indicators. However Doiron et al. (2007) also find that individuals who engage in risk taking behaviour are less likely to be in good health and are less likely to buy private cover. Finally, Cawley and Philipson (1999); Chiappori and Salanie (2000) find a negative correlation between risk and private insurance.

[4] The validity of instruments is tested through Sargan test discussed later in the paper.

[5] These correlations are not statistically significantly different from 0.

The set of individual characteristics (**X**) includes a set of demographic and socio-economic characteristics like educational attainment, age, income, gender, marital status, employment status, whether the person is an ex-smoker, whether the person is a migrant, and whether the person has embarked on a weight loss program because of his/her medical condition. Individual characteristics have been used extensively as determinants of BMI in the existing empirical literature. For example, Lakdawalla and Philipson (2002b) argue that income has both a direct and an indirect effects on weight. Specifically income affects weight indirectly through the strenuousness of work and hence caloric expenditure. Similarly education and employment status (which determine the occupation of an individual) can also reflect the strenuousness of work and hence caloric expenditure. The dummy variable for ex-smoker controls for the weight changes as a result of individual's smoking behaviour. The income variable and the age variable are included as ten dummies indicating the quartile of the income and age distribution. Such a specification allows for income and age to have an inverted U-shape relation with BMI (Lakdawalla and Philipson, 2002b). However, NHS only reports persons age and income in categories and thus its not possible to use an individual's actual age and income. BMI and age can have inverted u-shape relationship for biological reasons: individuals gain weight until they hit their middle age and then tend to loose weight as they grow older. The relationship between weight and income could be u-shaped keeping the level of individual's strenuousness at the workplace constant. However, if the level of strenuousness reduces with income, then weight and income will have a positive relationship throughout.

4 Results

We now turn to the regression results. In case we present the results corresponding to a number of different specifications, for the pooled sample and separately for males and females. The upper panels in each of the tables present the results corresponding to the specification where exercise duration is categorical, measured by $EXCAT$, while in the lower panels we present the results corresponding to the specification where exercise duration is assumed to be continuous, measured by $EXERCISE$. In each case we present results assuming that exercise duration is exogenous in the BMI category regressions and assuming that they are endogenous (the IV estimates).

Before proceeding further, a note on the estimation methodology used. First, when exercise duration is continuous we use the methodology developed by Rivers and Vuong (1988) to correct for the potential endogeneity problem. The procedure may be described as follows. First $EXERCISE$ is regressed on the full set of exogenous variables (including the instruments) in the system. This is the first stage regression. The error terms from the first stage regression ($EX\widehat{ERCI}SE$) is included as an additional regressor in the second stage estimation. A significant coefficient on ($EX\widehat{ERCI}SE$) implies that the null hypothesis of exogeneity of exercise duration on BMI is rejected.

The results discussed above are robust to the alternative model specifications used for empirical analysis. The results from the model with categorical exercise variables and gender based equations remain qualitatively the same for all the survey years. Multiple instruments are used for survey years 2001 and 2005 for instrumental variable regression and Hansen-Sargan test is used to test for overidentifying restrictions in the probit models modelling likelihood of obese and overweight individuals. The Sargan test for both 2000 and 2005 fail to reject the null indicating that instruments are valid. The Sargan statistic for 2001 survey year is 0.059 distributed as $\chi^2(1)$ with p-value of 0.80. The Sargan test statistic for 2005 is 0.578 with a p-value of 0.44. The instrumental variable regressions for 1989 and 1995 data use single instrument and hence the model is exactly identified.[6]

Table 4 presents the first set of results, 2SLS or ordered probit as the case might be. It is clear from these results that failure to account for the potential endogeneity of exercise duration results in us obtaining biased estimates of the effect of exercise duration on health status (BMI). Exercise duration is always negative and statistically significant in the single equation estimates (exogenous specification). However when we correct for the potential endogeneity of exercise duration in the BMI regressions, exercise duration no longer has a statistically significant effect on BMI category. The lone exception is the 1989 data where, the coefficient estimate of exercise duration changes sign when we account for this potential

[6]Note that the Sargan tests are computed only for the 2SLS regressions.

endogeneity. Unlike in the single equation estimates, an increase in exercise duration (in the past fortnight) either has no effect or has a positive and statistically significant effect on the probability of being obese. This result holds irrespective of whether exercise duration is continuous or categorical and it holds for the pooled sample and separately for the sample of men and women. This general pattern holds for the gender specific regressions (see Table 8).[7]

What this possibly means is that there is a selection problem here - the sample of individuals who choose to exercise more is not a random subset of the population. They are likely to be those who have chosen to exercise more possibly because they are more concerned about their health: the results are (possibly) driven by the overweight and obese choosing to exercise more as a means to lose weight and this results in exercise duration no longer having a statistically significant effect on BMI once we correct for the potential endogeneity of exercise duration in the BMI regression. The negative direct effect of exercise duration on BMI is therefore matched by the indirect effect: those with a high BMI are the ones who are spending more time exercising (the reverse causality effect) and the final effect, when we account for this reverse causality, is that exercise duration does not have a statistically significant effect on BMI.

It is also worth noting that our results are similar to those obtained in the literature. Rashad (2006) in her analysis of the effect of activity adjusted caloric intake on BMI (using US data) finds that not accounting for the potential endogeneity of the caloric intake variable results in a negative and statistically significant effect of caloric intake on BMI. However when she does account for the endogeneity of this variable (by conducting a structural estimation), caloric intake no longer has a statistically significant effect on BMI (Table 2, page 274).

The rest of the paper tries to examine the relative strengths of the direct and indirect effects and how much is actually driven by the high risk. It is worth noting that for the rest of the paper we restrict the sample to include individuals who have a BMI of 18.5 or higher, i.e. ignore the underweight from our analysis. The main reason for this is that the

[7]The one exception is females in 2005: after correcting for potential endogeneity

underweight might contaminate the relationship between exercise duration and BMI - for this group, we might obtain a relationship between exercise duration and BMI for reasons completely unrelated to the health of the individuals.

In Table 5 we present the effects of exercise duration on the probability of being overweight or obese (BMI $\geq$ 25). As before we present the results corresponding to a number of different specifications. But the main result remains the same: exercise duration is negative and statistically significant in the single equation estimates (exogenous specification), but exercise duration generally does not have a statistically significant effect on the probability of being overweight or obese (again the sole exception being the 1989 data). The main story remains the same across the two genders (see Table 9). The result remains the same when we examine the effect of exercise duration on the probability of being obese (BMI $\geq$ 30, see Table 6 for the results for the pooled sample and Table 10 for the gender specific regressions).

It has often been argued that the relationship between exercise and health status is driven not by the obese, but more by the overweight who are often the group that seeks to reduce weight. To examine whether that is the case, in Table 7 we present the effects of exercise duration on the probability of being overweight (here the sample is restricted to individuals with BMI $\in (18.5, 30]$). The main story remains the same. Again if we do not account for the potential endogeneity or reverse causation effect, exercise duration has a negative and statistically significant effect on the probability of being overweight. Once we take the reverse causality into account, exercise duration does not have a statistically significant effect on the probability of being overweight. The gender specific regressions (Table 11) essentially tell the same story.

The regressions results discussed above control for other variables that might have an effect on BMI. Of particular interest, as we have noted earlier, are income, age of the individual and educational attainment. Table 12 presents a summary of the effects of these variables.[8]

First, controlling for other characteristics income has an inverted U-shaped relationship

[8]The discussion that follows is based on the results of endogenous model specification with continuous exercise variable. Due to space constraints, we do not discuss the results from alternative the model specifications with categorical exercise variable.

with BMI for overweight individuals in survey years 1989 and 2001 and a positive relationship at lower quartiles for survey year 2005. For example, in 2005 individuals in second income quartile are 9 percentage points more likely to be overweight relative to individuals with no income. The marginal effects for this cohort are 9 percentage points and 4 percentage points for the 1989 and 2001 data respectively. The relationship between income and BMI for obese individuals is positive at lower quartiles and insignificant at higher quartiles for all survey years. The marginal effects are consistently lower for obese individuals relative to overweight individuals. For example, in 2005 individuals in second income quartile are 5 percentage points more likely to be obese. According to neoclassical framework the effect of income on weight can be decomposed into earned income and unearned income effect which also differs for underweight and overweight individuals (Lakdawalla and Philipson, 2002a). For sedentary work, an increase in earned income reduces physical activity which might lead to an increase in weight. However, for overweight individual, an increase in income, above a certain threshold, also raises the marginal disutility of being overweight. This will induce such individuals to undertake weight control activities thus allowing for an inverted-u relationship between BMI and income. Our results show that we get an inverted-u shaped relationship between BMI and income for overweight individuals in 1989 and 2001. For obese individuals, the results from all survey years show a positive relationship between BMI and income at lower income quartiles and no significant relationship between weight and BMI at higher income quartiles. This finding is interesting as it indicates towards the response of obese and overweight individuals towards the marginal disutility of weight. The result of insignificant relation between BMI and income at higher income quartiles implies that either these individuals do not perceive themselves to reach an ideal weight which keeps their marginal disutility of weight very low *or* their response of undertaking weight control activities is not sufficient enough to significantly reduce the BMI. The results from the significance of exercise variable show that the second scenario is more likely where growth in BMI for obese and overweight individuals is exceeding the growth in exercise.

Turning to the effect of age on BMI levels, in general we find a positive relationship between age and BMI status. For example, in 2005 individuals aged $35-44$ are 9 percentage points more likely to be obese and 12 percentage points more likely to be overweight relative to the base cohort. The marginal effects for other survey years are broadly the same and the magnitude of these effects is consistently lower for obese individuals compared to overweight individuals.

Increased educational attainment either has a negative or no effect on the BMI status. The effect of education on BMI is negative for years 1989 and 2005 and is not statistically significant for 1995 and 2001 data. For example, in 2005, an individual with a Post Graduate (PG) degree is 3 percentage points less likely to be obese and 14 percentage points less likely to be overweight relative to an individual with no education. The corresponding effects for the 1989 data are 6 percentage points and 14 percentage points for the obese and overweight individuals respectively.

Individuals working part time are less likely to be obese and overweight compared to those working full time. For example, in 2005 an individual working part time is 7 percentage points less likely to be overweight and 4 percentage points less likely to be obese compared to a full time employee. The marginal effect for previous survey years are also negative and significant but are lower in magnitude. For example in 1989 a part time employee is just 2 percentage points less likely to be obese and 3 percentage points less likely to be overweight relative to full time employee. Such a trend is partly a reflection of the changing nature of work: over time the nature of work has become increasingly sedentary which requires less physical activity. As a consequence individuals working full time and therefore spending more time at work are more likely to have reduced physical activity and thus higher BMI.

The effect of migrant status on BMI is insignificant for 1989 and 1995 and negative for 2001 and 2005. For example, in 2005 a migrant is 4 percentage points less likely to be obese and overweight relative to individuals born in Australia. The effect of smoking status on BMI is positive and significant for ex-smokers for year 1989, 1995 and 2005 and insignificant for year 2001. For example in 2005 ex-smokers are 3 percentage points more likely to be overweight and 13 percentage points more likely to be obese. Marital status has no significant effect on probability of being obese but has a positive effect for overweight people. For example, in 2005 married individuals are 5 percentage points more likely to be overweight relative to never married individuals. The control variable capturing weight gained due to medical condition has a positive effect on BMI as expected. For example in 2005, an individual with a weight gain due to a medical condition is 22 percentage points more likely to be overweight and 25 percentage points more likely to be obese relative to individuals without such medical condition.

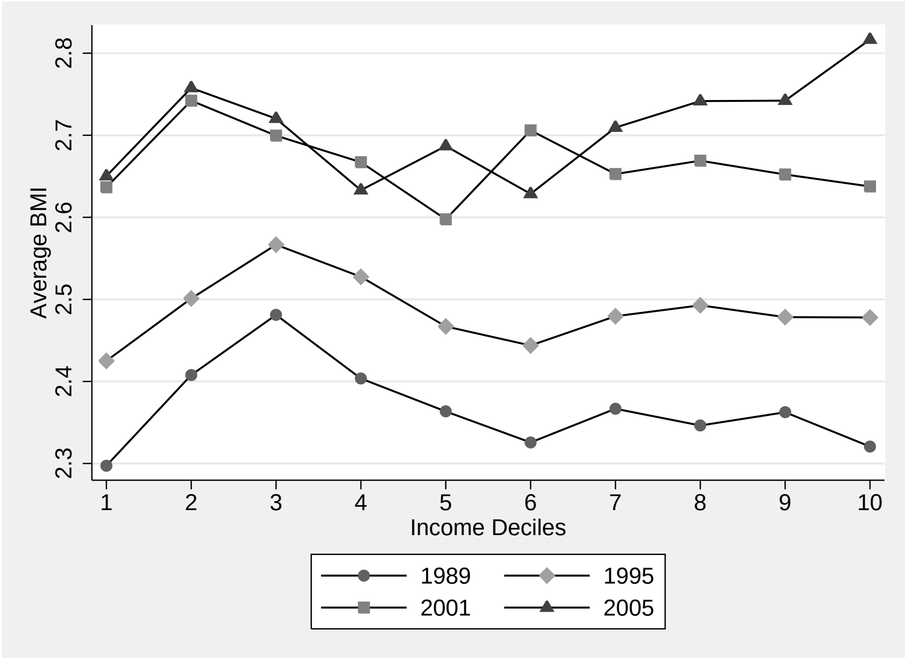
5 Conclusion

An estimated 1 billion people around the world are over weight, of whom around 300 million are clinically obese. Health experts argue that obesity levels will continue to rise in the early 21^{st} century - with severe health consequences in the absence of quick and directed intervention. Australia is not immune to this global problem of excessive weight. Australians are at the point where it is almost more normal to have a weight problem than not. It has been estimated that obesity and its associated illnesses cost the Australian society and governments a total of $21 billion in 2005. Regular exercise is increasingly regarded as an effective option for addressing the problem of obesity.

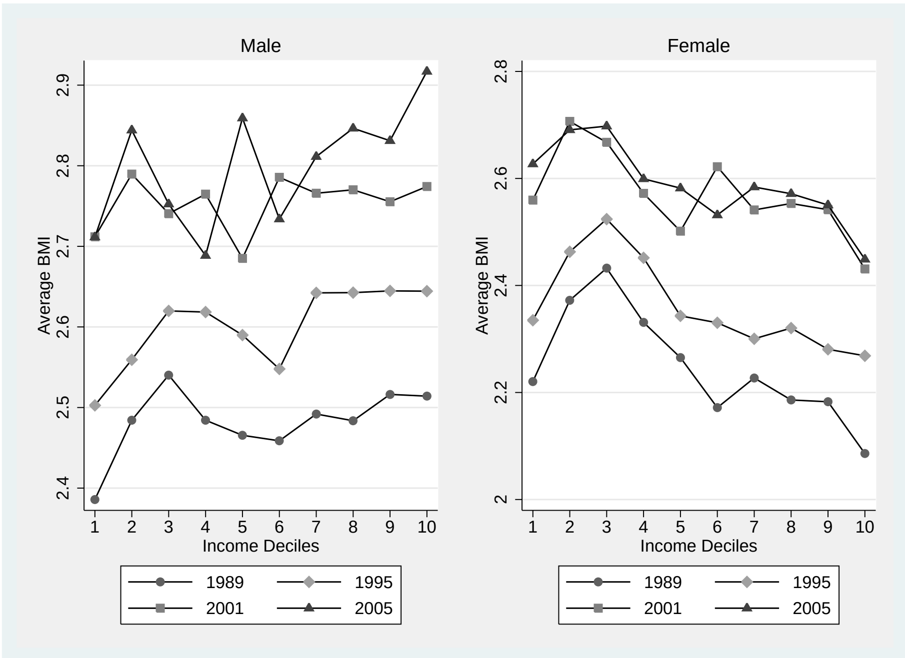
In this paper we examine the relationship between obesity (as measured by BMI) and the duration of exercise. Single equation estimates show that exercise duration has a negative and statistically significant effect on the probability of being overweight or obese. However when we take into account the potential endogeneity of exercise duration in the BMI regressions (arising from a standard problem of reverse causation), we no longer find a negative relationship between exercise duration and BMI. There is either no effect or the effect is actually positive indicating that the results are essentially driven by individuals who are and who perceive themselves to be overweight and obese conducting more exercise.

As discussed earlier the Australian government is spending a lot of money in information campaigns to highlight the importance of physical activity and healthy diet as tools for weight control. The results from our analysis show that despite these policy measures the growth in BMI has far exceeded the growth in exercise. Thus one policy suggestion could be to complement these information campaigns with some incentive schemes. For example, a subsidised gym membership or health insurance premium subsidies for physically active individuals could motivate people to undertake more physical activity. It should also be noted that according to our results income has either positive effect or no effect on BMI especially for obese individuals. Thus any incentive scheme which directly increases the disposable income of individuals (e.g. tax rebate) will not be effective in reducing BMI.

Figure 1: BMI by Income Decile

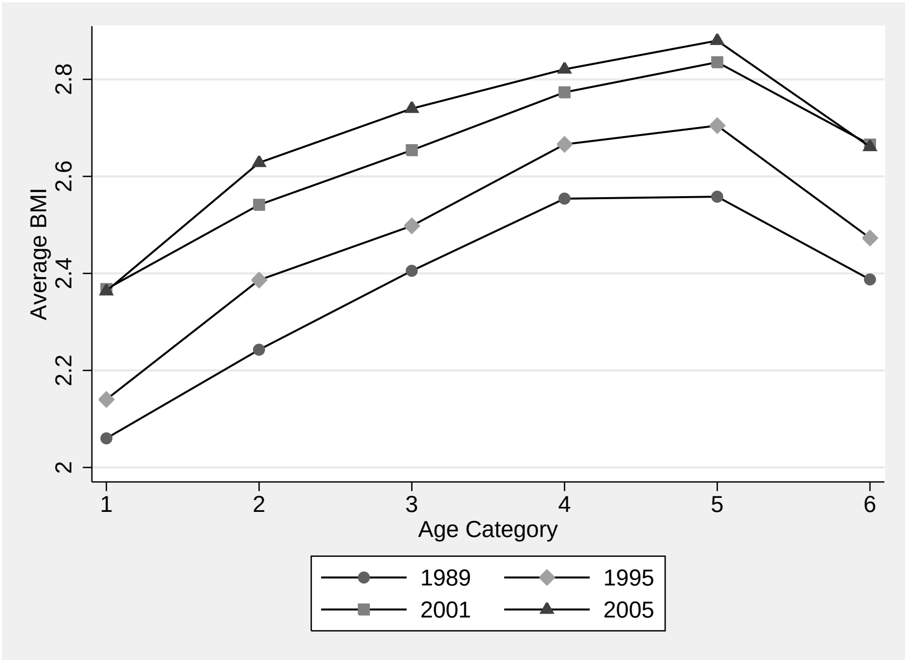


Full Sample

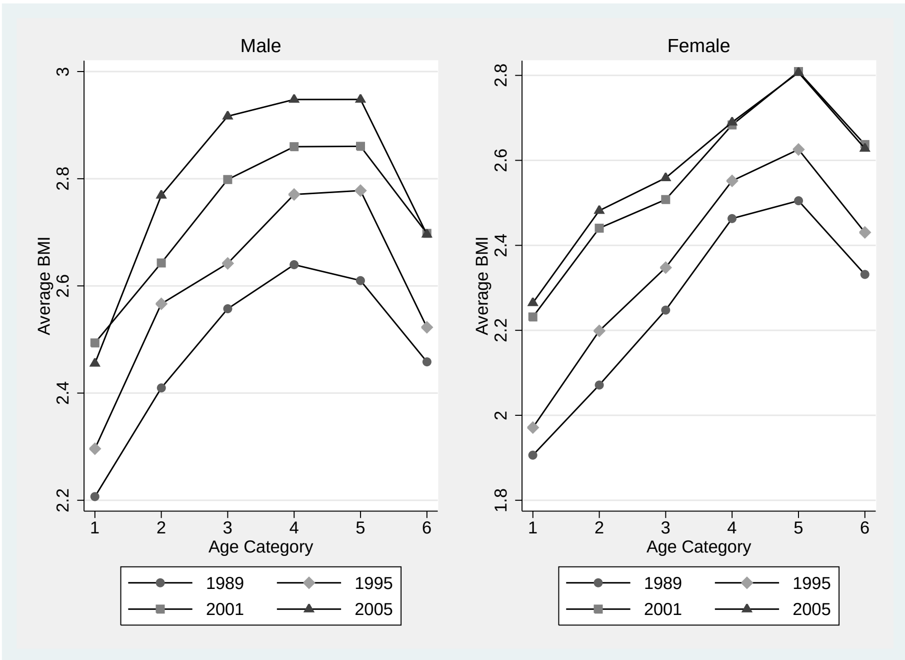


Gender Specific

Figure 2: BMI by Age Category



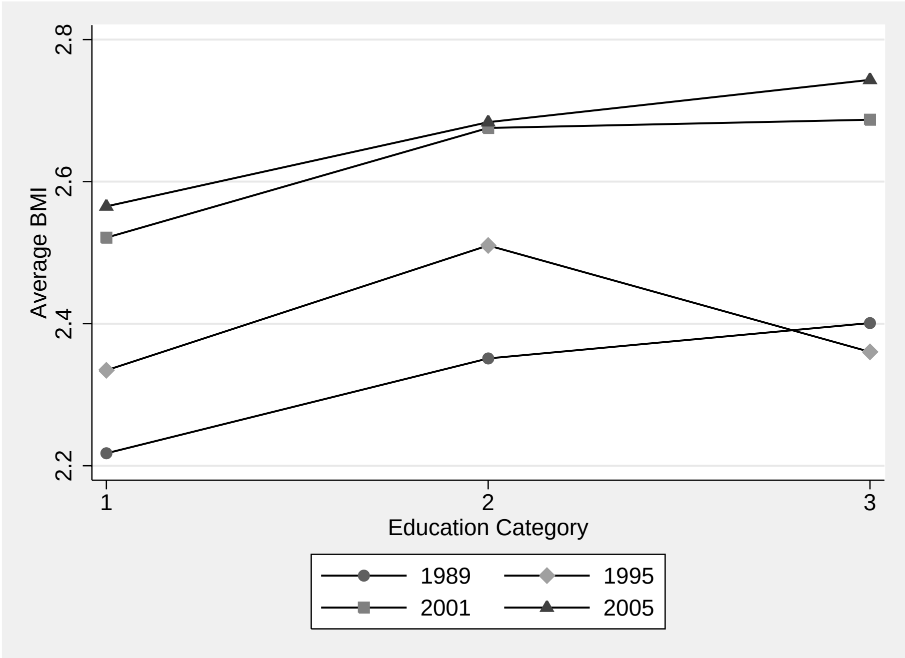
Full Sample



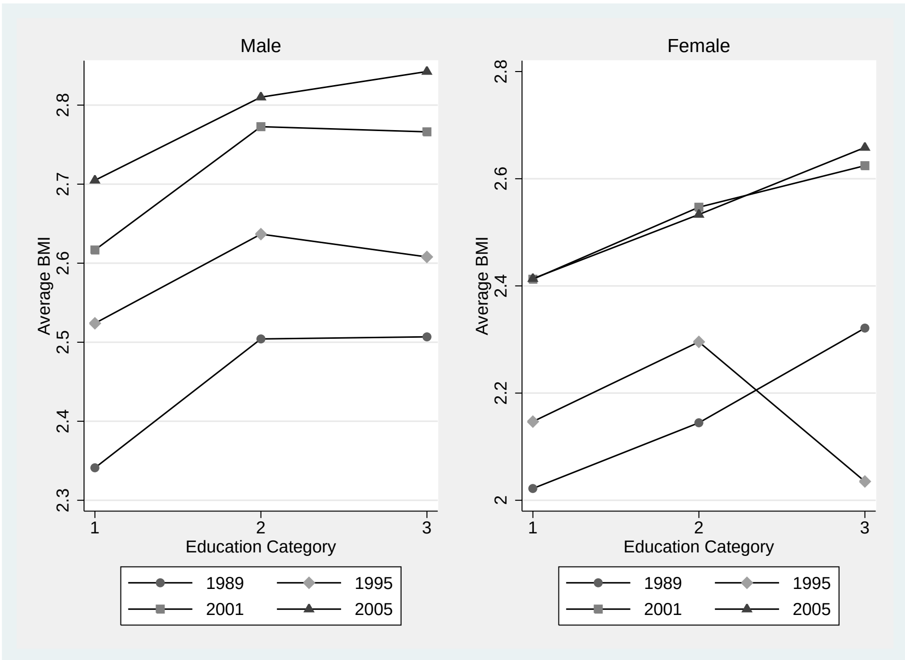
Gender Specific

Age Categories:- 1: 20-24 yrs, 2: 25-34 yrs, 3: 35-44 yrs, 4: 45-54 yrs, 5: 55-64 yrs, 6: 65+ yrs

Figure 3: BMI by Educational Attainment



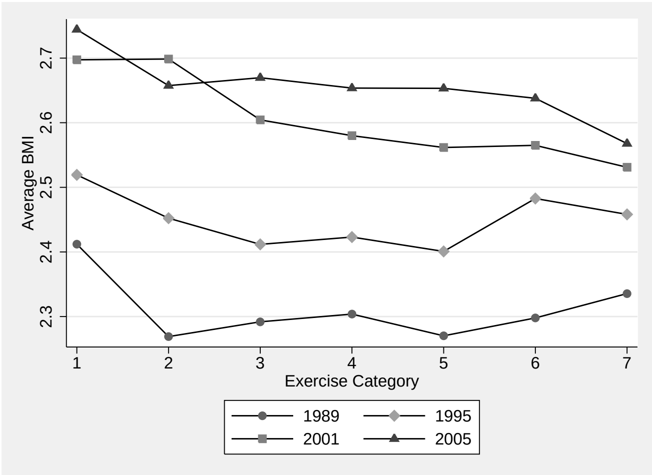
Full Sample



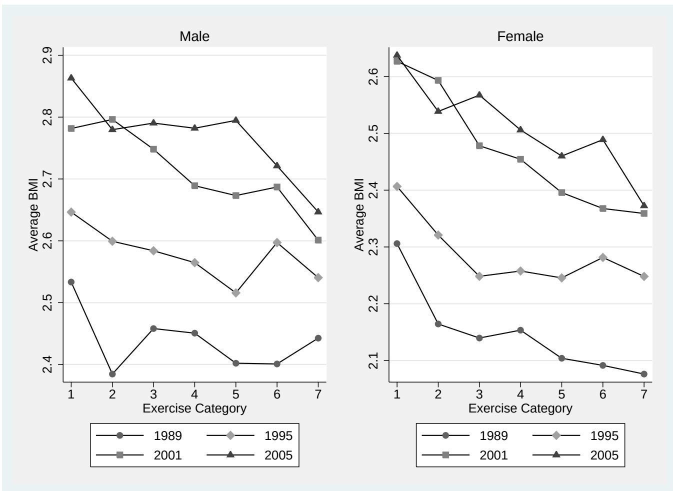
Gender Specific

Education Categories:- 1: PG and Bachelor, 2: Diploma, 3: No Non-school/still at school

Figure 4: Exercise and BMI



Full Sample



Gender Specific

Table 1: Selected Descriptive Statistics

Year → **Variable ↓**	**2005**	**2001**	**1995**	**1989**
Highest Education: PG	0.0574	0.1723	0.0923	0.0960
Highest Education: Bachelor	0.4709	0.3470	0.1452	0.3597
Highest Education: School	0.4607	0.4548	0.0027	0.5307
Married	0.5339	0.5790	0.6313	0.6997
Full Time Work	0.1808	0.1701	0.0397	0.6157
Part Time Work	0.0236	0.0318	0.3234	0.0420
Not in labour Force	0.3585	0.3585		
Migrant	0.2704	0.2781	0.2850	0.2829
Male	0.4710	0.4670	0.4980	0.4929
Age 25 - 34 Yrs	0.1800	0.2026	0.2268	0.2465
Age 35 - 44 Yrs	0.2115	0.2343	0.2305	0.2228
Age 45 - 54 Yrs	0.1852	0.1842	0.1796	0.1508
Age 55 - 64 Yrs	0.1560	0.1306	0.1159	0.1246
Age 65 and Above	0.1974	0.1826	0.1453	0.1520
First Income Decile	0.0552	0.0735	0.0726	0.0615
Second Income Decile	0.0746	0.1017	0.0738	0.0692
Third Income Decile	0.1179	0.0885	0.0799	0.0911
Fourth Income Decile	0.0961	0.0715	0.0795	0.0946
Fifth Income Decile	0.0921	0.0698	0.0802	0.0942
Sixth Income Decile	0.0888	0.0722	0.0845	0.0913
Seventh Income Decile	0.0760	0.0774	0.0889	0.1013
Eighth Income Decile	0.0940	0.0819	0.0919	0.1040
Ninth Income Decile	0.1138	0.0929	0.1016	0.1119
Tenth Income Decile	0.0933	0.0970	0.1059	0.1189
Current Smoker	0.2141	0.2442	0.2382	0.2814
Non Regular Smoker	0.2944	0.3212		
Smoke Indoor	0.0770			
Serves of Fruit	0.5435	0.5323		
Serves of Vegetables	0.8089	0.7864		
Whether has Skim Milk	0.5186	0.4956		
Whether has Private Health Insurance	0.4222			
Exercise Illness	0.0603	0.0137	0.0055	0.1247
Average BMI	2.7085	2.6409	2.4887	2.3672
Average BMI (Males)	2.8161	2.7409	2.6139	2.4913
Average BMI (Females)	2.6126	2.5533	2.3644	2.2466
Proportion Doing Any Exercise***	0.4011	0.4198	0.4061	0.3757
Proportion Doing Any Exercise*** (Males)	0.4391	0.4682	0.4462	0.4153

Continued ...

Table 1: (continued)

Year → **Variable ↓**	**2005**	**2001**	**1995**	**1989**
Proportion Doing Any Exercise*** (Females)	0.3672	0.3774	0.3664	0.3373
Duration of Exercise	5.9098	6.1384	6.7473	6.7377
Duration of Exercise (Males)	6.7829	7.0549	7.8862	7.8066
Duration of Exercise (Females)	4.9803	5.1421	5.3714	5.4588
Sample Size	17271	15935	34235	35980

* * *: Exercise Duration Computed for those that do any exercise.

Table 2: BMI Category by Gender and Year

Year → **Variable ↓**	**2005**	**2001**	**1995**	**1989**
Full Sample				
Under weight	2.47	2.82	8.94	11.33
Normal	43.23	46.82	45.53	49.9
Over weight	35.27	33.81	33.26	29.51
Obese	19.03	16.55	12.27	9.27
Males				
Under weight	0.8	0.95	3.89	5.6
Normal	36.32	40.14	42.79	48.4
Over weight	43.34	42.77	41.34	37.26
Obese	19.53	16.14	11.97	8.74
Females				
Under weight	3.96	4.46	13.95	16.89
Normal	49.39	52.67	48.24	51.35
Over weight	28.08	25.95	25.24	21.97
Obese	18.57	16.92	12.57	9.79

Table 3: Exercise Category by Gender and Year

Year → Variable ↓	2005	2001	1995	1989
Full Sample				
No Exercise	59.89	58.02	59.39	62.43
Exercise 0 - 1 Hours	5.65	6.6	5.95	5.67
Exercise 1 - 2 Hours	7.34	7.39	6.78	5.97
Exercise 2 - 4 Hours	9.46	9.55	8.98	8.4
Exercise 4 - 8 Hours	9.55	9.67	9.46	8.59
Exercise 8 - 14 Hours	4.88	4.98	5.16	5.01
Exercise > 14 Hours	3.23	3.79	4.27	3.94
Males				
No Exercise	56.09	53.18	55.38	58.47
Exercise 0 - 1 Hours	5.38	6.45	5.61	5.4
Exercise 1 - 2 Hours	6.64	6.81	6.45	5.64
Exercise 2 - 4 Hours	10.07	10.12	9.3	8.52
Exercise 4 - 8 Hours	10.89	11.57	10.53	9.66
Exercise 8 - 14 Hours	6.18	6.25	6.57	6.71
Exercise > 14 Hours	4.74	5.62	6.15	5.61
Females				
No Exercise	63.28	62.26	63.36	66.27
Exercise 0 - 1 Hours	5.89	6.72	6.29	5.93
Exercise 1 - 2 Hours	7.97	7.89	7.1	6.29
Exercise 2 - 4 Hours	8.91	9.05	8.68	8.28
Exercise 4 - 8 Hours	8.36	8.01	8.4	7.55
Exercise 8 - 14 Hours	3.71	3.87	3.76	3.36
Exercise > 14 Hours	1.88	2.19	2.4	2.32

Table 4: Effect of Exercise on BMI

Year → **Model Specification ↓**	**2005**	**2001**	**1995**	**1989**
Exercise Variable: Categorical				
Ordered Probit	-0.025^{***}	-0.034^{***}	-0.014^{***}	-0.015^{***}
(Exogenous)	(0.005)	(0.005)	(0.003)	(0.003)
2SLS	0.028	0.023	0.311	0.272^{***}
	(0.032)	(0.030)	(0.284)	(0.082)
Exercise Variable: Continuous[a]				
Ordered Probit	-0.012^{***}	-0.024^{***}	-0.012^{***}	-0.012^{***}
(Exogenous)	(0.001)	(0.001)	(0.001)	(0.001)
Ordered Probit	0.046	0.120	6.600	0.360^{***}
(Endogenous)	(0.046)	(0.120)	(5.400)	(0.120)
Sample Size	17271	15935	34235	35980

a: Unit of exercise: 120 mins (2 hrs) per fortnight.

Standard errors are in parentheses.

Regressions control for other covariates including socioeconomic characteristics.

$***$:significant at 1% level.

$**$:significant at 5% level.

$*$:significant at 1% level.

Table 5: Effect of Exercise on the probability of being Overweight or Obese

Year →	**2005**	**2001**	**1995**	**1989**
Model Specification ↓				
Exercise Variable: Categorical				
Probit	-0.027^{***}	-0.036^{***}	0.003	-0.035^{***}
(Exogenous)	(0.007)	(0.006)	(0.004)	(0.004)
2SLS	0.009	-0.004	0.104	0.110^{***}
	(0.020)	(0.019)	(0.143)	(0.051)
Exercise Variable: Continuous^{a}				
Probit	-0.012^{***}	-0.012^{***}	-0.012^{***}	-0.012^{***}
(Exogenous)	(0.001)	(0.001)	(0.001)	(0.001)
Probit	0.019	0.009	4.56	0.240^{***}
(Endogenous)	(0.048)	(0.056)	(6.720)	(0.120)
Sample Size	16844	15485	31174	31905

Sample includes only those individuals with BMI $>$ 18.5

a: Unit of exercise: 120 mins (2 hrs) per fortnight.

Regressions control for other covariates including socioeconomic characteristics.

Standard errors are in parentheses.

$***$:significant at 1% level.

$**$:significant at 5% level.

$*$:significant at 1% level.

Table 6: Table: Effect of Exercise on the possibility of being Obese

Year → **Model Specification ↓**	**2005**	**2001**	**1995**	**1989**
Exercise Variable: Categorical				
Probit	-0.058^{***}	-0.064^{***}	-0.058^{***}	-0.053^{***}
(Exogenous)	(0.008)	(0.008)	(0.006)	(0.006)
2SLS	-0.017	-0.006	-0.008	0.025
	(0.016)	(0.015)	(0.091)	(0.029)
Exercise Variable: Continuous[a]				
Probit	-0.036^{***}	-0.036^{***}	-0.024^{***}	-0.024^{***}
(Exogenous)	(0.001)	(0.001)	(0.001)	(0.001)
Probit	-0.058	-0.12	-0.240	0.120
(Endogenous)	(0.063)	(0.012)	(8.520)	(0.120)
Sample Size	16844	15485	31174	31905

Sample includes only those individuals with BMI $>$ 18.5

[a]: Unit of exercise: 120 mins (2 hrs) per fortnight.

Regressions control for other covariates including socioeconomic characteristics.

Standard errors are in parentheses.

$***$: significant at 1% level.

$**$: significant at 5% level.

$*$: significant at 1% level.

Table 7: Table: Effect of Exercise on the possibility of being Overweight

Year → **Model Specification ↓**	**2005**	**2001**	**1995**	**1989**
Exercise Variable: Categorical				
Probit	-0.0091	-0.0175***	-0.0112**	-0.0244***
Exogenous	(0.0078)	(0.0074)	(0.0051)	(0.0045)
2SLS	0.019	0.008	0.132	0.100**
	(0.021)	(0.019)	(0.153)	(0.051)
Exercise Variable: Continuous[a]				
Probit	-0.0000	-0.0001**	-0.0000	-0.0001**
Exogenous	(0.0001)	(0.0000)	(0.0000)	(0.0000)
Probit	0.036	0.012	6.120	0.240**
Endogenous	(0.600)	(0.060)	(7.320)	(0.120)
Sample Size	13558	12847	26972	28570

Sample includes only those individuals with BMI $>$ 18.5 and BMI $\leq$ 30

[a]: Unit of exercise: 120 mins (2 hrs) per fortnight.

Regressions control for other covariates including socioeconomic characteristics.

Standard errors are in parentheses.

$***$: significant at 1% level.

$**$: significant at 5% level.

$*$: significant at 1% level.

Table 8: The Effect of Exercise on BMI: All Weight Categories by Gender

Year →	**2005**	**2001**	**1995**	**1989**	**2005**	**2001**	**1995**	**1989**
Model Specification ↓	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
		Males				***Females***		
Exercise Variable: Categorical								
Ordered Probit	-0.021	−0.020***	−0.013***	−0.009***	−0.034***	−0.052***	−0.020***	−0.028***
(Exogenous)	(0.008)	(0.007)	(0.005)	(0.004)	(0.008)	(0.008)	(0.005)	(0.005)
2SLS	-0.026	0.020	0.364	0.298***	0.117**	0.021	0.056	0.211
	(0.039)	(0.034)	(0.279)	(0.078)	(0.054)	(0.058)	(0.999)	(0.187)
Exercise Variable: Continuous[a]								
Ordered Probit	−0.012***	−0.012***	-0.003	-0.001	−0.018***	−0.024***	−0.012***	−0.012***
(Exogenous)	(0.004)	(0.003)	(0.002)	(0.001)	(0.008)	(0.006)	(0.003)	(0.003)
Ordered Probit	-0.025	0.020	-0.600	0.408***	0.180**	0.082	0.062	0.480
(Endogenous)	(0.055)	(0.048)	(0.360)	(0.084)	(0.084)	(0.120)	(0.360)	(0.240)
Sample Size	8135	7442	17048	17733	9136	8493	17187	18247

a: Unit of exercise: 120 mins per fortnight.

Regressions control for other covariates including socioeconomic characteristics.

Standard errors are in parentheses.

∗ ∗ ∗:significant at 1% level.

∗∗:significant at 5% level.

∗:significant at 10% level.

Table 9: The Effect of Exercise on the Probability of Being Obese by Gender

Year →	**2005**	**2001**	**1995**	**1989**	**2005**	**2001**	**1995**	**1989**
Model Specification ↓	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
		Males				***Females***		
Exercise Variable: Categorical								
Probit	−0.061***	−0.055**	0.058***	−0.049***	−0.055***	−0.081***	−0.060***	−0.060***
(Exogenous)	(0.011)	(0.011)	(0.008)	(0.008)	(0.012)	(0.012)	(0.009)	(0.009)
2SLS	0.029	0.026	0.078	0.038	-0.001	−0.073**	-0.270	-0.008
	(0.021)	(0.017)	(0.100)	(0.027)	(0.025)	(0.030)	(0.432)	(0.074)
Exercise Variable: Continuous[a]								
Probit	−0.036***	−0.032***	−0.020***	−0.020***	−0.045***	−0.048***	−0.044***	−0.033***
(Exogenous)	(0.008)	(0.008)	(0.005)	(0.006)	(0.011)	(0.012)	(0.008)	(0.008)
Probit	0.087	0.072	-0.480	0.192	-0.001	-0.312	-0.516	0.948
(Endogenous)	(0.073)	(0.060)	(0.600)	(0.132)	(0.114)	(0.156)	(0.600)	(0.600)
Sample Size	8070	7371	16384	16740	8774	8114	14790	15165

Sample includes only those individuals with BMI > 18.5

a: Unit of exercise: 120 mins per fortnight.

Regressions control for other covariates including socioeconomic characteristics.

Standard errors are in parentheses.

∗ ∗ ∗:significant at 1% level.

∗∗:significant at 5% level.

∗:significant at 10% level.

Table 10: The Effect of Exercise on the Probability of Being Obese by Gender

Year →	**2005**	**2001**	**1995**	**1989**	**2005**	**2001**	**1995**	**1989**
Model Specification ↓	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Males				***Females***			
Exercise Variable: Categorical								
Probit	-0.011	-0.019^{**}	-0.016^{***}	-0.028^{***}	-0.050^{***}	-0.065^{***}	-0.043^{***}	0.041^{***}
(Exogenous)	(0.009)	(0.009)	(0.006)	(0.005)	(0.010)	(0.010)	(0.007)	(0.006)
2SLS	-0.014	-0.007	0.191	0.154^{**}	0.044	-0.001	-0.184	-0.025
	(0.025)	(0.022)	(0.169)	(0.053)	(0.033)	(0.037)	(0.436)	(0.110)
Exercise Variable: Continuous[a]								
Probit	-0.012^{*}	-0.014^{***}	-0.003	-0.006^{**}	-0.027^{***}	-0.037^{***}	-0.021^{***}	-0.025^{***}
(Exogenous)	(0.006)	(0.005)	(0.003)	(0.002)	(0.012)	(0.008)	(0.005)	(0.005)
Probit	-0.028	-0.012	-0.600	0.324^{***}	0.110	-0.004	-0.192	-0.024
(Endogenous)	(0.066)	(0.012)	(0.600)	(0.096)	(0.096)	(0.054)	(0.048)	(0.360)
Sample Size	8070	7371	16384	16740	8774	8114	14790	15165

Sample includes only those individuals with BMI > 18.5

a: Unit of exercise: 120 mins per fortnight.

Regressions control for other covariates including socioeconomic characteristics.

Standard errors are in parentheses.

$***$:significant at 1% level.

$**$:significant at 5% level.

$*$:significant at 10% level.

Table 11: The Effect of Exercise on the Probability of Being Overweight by Gender

Year →	**2005**	**2001**	**1995**	**1989**	**2005**	**2001**	**1995**	**1989**
Model Specification ↓	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
		Males				***Females***		
Exercise Variable: Categorical								
Probit	0.028***	0.014	0.011*	−0.009*	-0.017	−0.018*	−0.013*	−0.025***
(Exogenous)	(0.009)	(0.009)	(0.006)	(0.005)	(0.011)	(0.010)	(0.007)	(0.007)
2SLS	-0.001	-0.017	0.181	0.141***	0.050	0.051	-0.060	-0.047
	(0.027)	(0.024)	(0.175)	(0.052)	(0.035)	(0.036)	(0.385)	(0.119)
Exercise Variable: Continuous[a]								
Probit	0.009*	0.002	0.005**	3.24E-05	-0.004	-0.012	-0.003	−0.012**
(Exogenous)	(0.006)	(0.005)	(0.003)	(0.002)	(0.012)	(0.008)	(0.006)	(0.005)
Probit	-0.008	-0.036	-0.600	0.312***	0.132	0.240*	-0.050	-0.112
(Endogenous)	(0.072)	(0.060)	(0.480)	(0.096)	(0.108)	(0.144)	(0.480)	(0.516)
Sample Size	8070	7371	16384	16740	8774	8114	14790	15165

Sample includes only those individuals with BMI > 18.5 and BMI ≤ 30

a: Unit of exercise: 120 mins per fortnight.

Regressions control for other covariates including socioeconomic characteristics.

Standard errors are in parentheses.

∗ ∗ ∗:significant at 1% level.

∗∗:significant at 5% level.

∗:significant at 10% level.

Table 12: The Effect of Covariates on BMI

Year → Covariates ↓	1989	1995	2001	2005
Income deciles				
Whole Sample	Inv. U shaped	Positive at lower quartiles	Inv. U shaped	Positive
Obese	Positive at lower quartiles	Positive at lower quartiles	Positive at lower quartiles	Positive at lower quartiles
Overweight	Inv. U shaped	Positive at lower quartiles	Inv. U shaped	Positive
Age deciles				
Whole Sample	Positive	Not Significant	Positive	Positive
Obese	Positive	Not Significant	Positive	Positive
Overweight	Positive	Not significant	Positive	Positive
Education (Dummy: Base Case: Still at school, Not Applicable)				
Whole Sample	Negative for PG	Not significant	Negative for PG	Negative for PG
Obese	Negative for PG	Not significant	Not significant	Not significant
Overweight	Negative for PG	Not significant	Not significant	Negative for PG

References

AE (2006). The economic costs of obesity. Technical report, Access Economics, Canberra.

Averett, S. and S. Korenman (1999). Black-white differences in social and economic consequences of obesity. *International Journal of Obesity 23*(2), 166 – 173.

Baum, C. and W. Ford (2004). The wage effects of obesity: a longitudinal study. *Health Economics 13*, 885 – 899.

Cawley, J. (1999). *Obesity and Addiction.* Ph. D. thesis, Ph. D. Dissertation, Department of Economics, The University of Chicago.

Cawley, J. (2004). The impact of obesity on wages. *Journal of Human Resources 39*, 451 – 474.

Cawley, J. and T. Philipson (1999). An Empirical Examination of Information Barriers to Trade in Insurance. *The American Economic Review 89*(4), 827–846.

Chiappori, P. and B. Salanie (2000). Testing for Asymmetric Information in Insurance Markets. *Journal of Political Economy 108*(1), 56.

Chou, S.-Y., M. Grossman, and H. Saffer (2004). An economic analysis of adult obesity: Results from the behavioral risk factor surveillance system. *Journal of Health Economics 23*, 565 – 587.

Cutler, D. M., E. L. Glaeser, and J. M. Shapiro (2003). Why have americans become more obese? *Journal of Economic Perspectives 17*(3), 93 – 118.

Doiron, D., G. Jones, and E. Savage (2007). Healthy, wealthy and insured? The role of self-assessed health in the demand for private health insurance. *Health Economics, Forthcoming*.

Finkelstein, E. A., I. C. Fiebelkorn, and G. Wang (2003). National medical spending attributable to overweight and obesity: How much and who's paying. *Health Affairs*.

Hamermesh, D. S. and J. E. Biddle (1994). Beauty and the labour market. *American Economic Review 84*(5), 1174 – 1194.

Lakdawalla, D. and T. Philipson (2002a). The growth of obesity and technological change. Technical Report 8946, National Bureau of Economic Research, Working Paper.

Lakdawalla, D. and T. Philipson (2002b). The Growth of Obesity and Technological Change: A Theoretical and Empirical Examination.

Lakdawalla, D. N. and T. J. Phillipson (2006). Elgar Companion to Health Economics. Chapter Economics of Obesity, pp. 72–82. Cambridge: Edward Elgar.

Morris, S. (2006). Body mass index and occupational attainment. *Journal of Health Economics 25*(2), 347 – 364.

Philipson, T. (2002). The world-wide growth in obesity: An economic research agenda. *Health Economics 10*(1), 1 – 7.

Philipson, T. and R. Posner (2003). The Long-Run Growth in Obesity as a Function of Technological Change. *Perspectives in Biology and Medicine 46*(3), S87–S107.

Rashad, I. (2006). Structural estimation of caloric intake, exercise, smoking and obesity. *The Quarterly Review of Economics and Finance 46*, 268 – 283.

Rivers, D. and Q. H. Vuong (1988). Limited information estimators and exogeneity tests for simultaneous probit models. *Journal of Econometrics 39*, 347 – 366.

Savage, E. and D. Wright (2003). Moral hazard and adverse selection in Australian private hospitals: 1989-1990. *Journal of Health Economics 22*(3), 331–59.

WHO (2004). Obesity and underweight. Technical report, World Health Organization.