

**FAMILY HISTORY OF CHRONIC DISEASE AND
PARTICIPATION IN HEALTHY BEHAVIOURS**

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Abstract

This paper presents an investigation into whether individuals incorporate information on their family genetic (health) endowment into decision making regarding participation in behaviours related to the production of health capital. Competing theoretical perspectives exist as to how genetic endowment should affect participation in health-related behaviours. There is also a growing consensus that the availability of genetic information holds important consequences for the individual in terms of investment in human capital, the pricing and availability of health insurance, and labour market opportunity. Therefore the question of how individuals use the greater availability of genetic data is important. Our empirical results indicate that poor genetic endowment tends to be associated with a lower probability of participation in alcohol consumption and cigarette smoking, but also a lower probability of participation in regular exercise.

Keywords: chronic disease genetic endowment health-related behaviour

Introduction

Genetic endowment plays a significant role in the production of health. The link between genes and susceptibility to a number of common diseases (including various types of cancer, cardiovascular disease, Alzheimer's and cystic fibrosis) is well established (Osann, 1991; Schwartz, et al., 1995; Tabarrok, 1994). Therefore, an individual's ability to produce a certain stock of health capital (or a particular health outcome) is determined in large part by the genes she inherited. Several dozen studies have documented the immediate and lifetime costs associated with morbidity due to these common diseases (e.g., Manning, et al., 1991; Chodick, et al., 2005; Payne, et al., 2002, Ory, et al., 2005; Lee, et al., 2004). However, the connection between genetic endowment and health outcome is not straightforward, mainly because the risk of developing many diseases, including diabetes, stroke and high blood pressure, depends as well on environmental factors (Tabarrok, 1994). The risk of developing these diseases can be modified by participation in certain healthy behaviours (for example, maintaining a certain diet and regular physical activity) or by avoiding certain unhealthy behaviours (for example, smoking and excessive alcohol consumption).

The marketing (e.g. television commercials) of healthy behaviours (avoidance of unhealthy behaviours) has already begun to emphasize the importance of genetic aspects of some diseases. Health economists also advocate the use of genetic data to evaluate a candidate for health insurance based on the higher insurance risk posed by individuals with certain genetic mutations (Lemaire, et al., 2000). Plus there is the possibility of discrimination in access to health insurance (Armstrong et al. 2003). Yet it is not clear how individuals use the information on their genetic endowment. On the one hand poor

genetic endowment might lead to behaviours that reduce the risk of developing a chronic disease. That is, the individual might substitute healthy behaviours for inherited health risks (see, for example Dicke and Gerking, 1997). On the other hand, other individuals may choose unhealthy behaviours because, given their endowment, the number of (and amount of each) healthy behaviours required to modify health risks is more than the individual wishes to implement (more than the optimal amount for the individual) or because the individual finds healthy behaviours distasteful.¹ Alternatively, some individuals prefer to substitute medication for healthy behaviours in order to achieve or maintain a certain stock of health capital (Chen, et al., 2002) despite evidence suggesting the marginal impact of health care on health is low, at least in developed countries (Birch and Stoddart, 1991).

Not only is it important to understand the relationship between family endowment and health-related behaviours from a micro (or individual level) perspective, it is of importance at the macro (organizational) level as well. In the past few decades, employers have experienced a dramatic increase in health care expenditures and have begun to develop and implement strategies to ensure cost containment occurs (e.g., Chiappetta, 2005; Coulter, 2006; Thorpe, 2005). Further, some employers are beginning to assess family endowment in an effort to determine appropriate deductibles or coverage extended to employees, or potential workplace problems (e.g., Lemaire, et al., 2000; Brown, et al., 2005). Organizations may be reluctant to employ individuals with family endowments which could adversely affect the individual, and ultimately the organizational, performance due to increased absences/use of sick days, potential

¹ For example, anecdotal evidence based upon numerous conversations with colleagues and students suggest the vast majority, particularly females, and even those who currently exercise, express strong dislike for exercise.

accidents leading to worker's compensation claims, reduced morale, etc. Organizations are also initiating wellness programs in order to encourage employees and their dependents to engage in healthy behaviours. As such, the present study has significant workplace implications and should prove insightful from policy formulation and health care cost containment perspectives.

The early economic theory of health production was developed by Becker (1965) and Grossman (1972). In this tradition individuals maximize well-being subject to constraints such as income and prices. Well-being is derived from the consumption of market goods and goods (such as health) produced by the individual. The individual solves this constrained optimization problem to determine the well-being maximizing stock of health capital, which in turn is produced by combining market inputs (such as medication), behaviours (such as regular physical activity) with the individual's health endowment, which is predetermined by genetic inheritance. Production of health depends upon the health production function, which determines the efficiency with which inputs are converted to health and the rate of depreciation of the stock of health capital produced. An important implication of the Becker-Grossman theoretical perspective is that individuals will compensate for poor genetic endowment by selecting healthy behaviours. That is, in producing health, behaviours and endowment are substitutes.

More recent theoretical work has suggested a more complex relationship between endowment and healthy behaviours. Birch and Stoddart (1991) showed that given the resources available to the individual (for example genetic endowment) unhealthy behaviours might be an optimal choice. For example, the individual's endowment might be such that there is little reason to believe that future benefits of healthy behaviours will

be realized. In their words “[M]arginal benefits of health-related behaviour would also be affected if the future value of the returns to the behaviour differed across individuals. In other words incentives to adopt healthy lifestyles may differ.” (pp. 182).

To give a concrete example, for an individual with a family history of obesity the marginal cost of regular physical activity might exceed the present marginal benefit or any expected future benefit. Furthermore, if an individual faces multiple or severe health risks due to poor endowment the consequences of unhealthy behaviours may seem relatively minor. That is, the incentive to invest in health production depends on the individual’s perception of the chance of survival (Dow, et al., 1999).²

This line of reasoning suggests some individuals complement poor genetic endowment with unhealthy behaviours, such as smoking and excessive alcohol consumption, or fail to choose healthy behaviours, such as regular exercise.

This paper makes a contribution to the literature on the relationship between family genetic endowment and health-related behaviours on several levels. To date we are aware of few studies (Ganz, 2001; Dickie and Gerking, 1997) which test the relationship between family health endowment and choice of health-related behaviours by adults using directly observable exogenous measures of health endowment. Second, while Ganz considered the effect of family history of cancer on smoking and alcohol consumption we are able to consider family history of a number of diseases (cancer, diabetes, heart disease, high blood pressure, high cholesterol and stroke). This gives us the additional advantage of being able to allow different endowment effects on different

² To give a concrete example, for a soldier on the battlefield the risk to health of smoking a cigarette (to be realized at some future date) is infinitesimally small compared to current risks.

behaviours. We consider the effect of family health endowment on three behaviours (alcohol consumption, regular exercise and smoking).

Theoretical Model

We assume individuals maximize utility subject to the standard economic constraints.

Simply put, utility is a function of goods purchased in the market (M) and health produced by the individual (H).

$$\text{Max } U = U(M, H)$$

The production of health is subject to a health production technology whose inputs include the individual's genetic endowment (E), endogenous health behaviours (V) and exogenous characteristics (X)

$$H = H(E, V, X).$$

Finally, the individual faces the budget constraint

$$P_V V + M = Y,$$

where P_V are prices for V and Y is income. We assume $H'(V) > 0$ for healthy behaviours, that is, the marginal product of healthy behaviours is positive.

Our main interest in this paper is whether genetic endowment and behaviours are complements or substitutes. That is, whether individuals with poor genetic endowment seek to reduce the risk their endowment pose to their health by adopting behaviours that reduce such risk or whether individuals with poor endowments increase the risk of developing the disease with unhealthy behaviours. There are several diseases that have come to be termed "lifestyle" diseases because the probability of developing those diseases is modifiable by participating in a mix of healthy behaviours. So our empirical

questions are whether V is a function of E and what is the sign of $V'(E)$. Both $V'(E) < 0$ (Becker-Grossman) and $V'(E) > 0$ (Birch-Dow) are theoretical possibilities.

Furthermore, since an individual has many genetic components and there are many health-related behaviours we suggest $V'(E)$ could be negative with respect to V_j and E_i but positive with respect to V_k and E_l for any endowment component/health-related behaviour pair.

Data and Methods

The data used in this study were collected in 2001 by the Caribbean Food and Nutrition Institute (CFNI) in Jamaica. The target population was adults aged 18 to 64 years. The survey was designed as a stratified multi-staged sample. The first stage is the selection of census Enumeration Districts (EDs). Enumeration districts are fully contained within each administrative region (parishes). The EDs are selected with probability proportional to their size (measured by the number of dwellings in each ED). The second stage is the selection of dwelling units within each ED. From each ED an equal number of dwellings were selected using systematic sampling with a random start. At the household level one person was selected for interview by trained professionals. That individual must be between 18 and 64 years of age and should be the person who last celebrated a birthday.

Interviewers told potential respondents they were from a research company in Kingston (including the name of the company), conducting a survey about dietary and exercise behaviour on behalf of CFNI. Once a respondent from the survey household was selected to be interviewed, he or she was told the interview could take up to 45 minutes and that their cooperation was appreciated. A total of 1513 respondents participated.

Dependent Variables

The purpose of this study is to investigate the extent to which family genetic endowment affects choice of health-related behaviours. The three behaviours of interest are alcohol consumption, cigarette smoking and physical activity.

Alcohol consumption

The survey contains three questions related to alcohol consumption. The first question is a simple participation question (Do you drink alcohol?). The possible responses were yes, no and occasionally. The second question asked what type of alcoholic beverages are consumed (beer, wine, rum and stout). And finally respondents were asked the quantity consumed per week. For the purpose of this paper we focus on the first (participation) question. We created a binary variable (yes = 1, no and occasionally = 0) to model alcohol consumption.

Cigarette smoking

The survey respondents were asked if they were ever a smoker and if they are currently a smoker. We took full advantage of the presence of quitters by creating a multiple response smoking variable (never = 0, quitters = 1, current smokers = 2). Smoking is of interest because it has been identified as a risk factor for several diseases including some types of cancer, and stroke (Osann, 1991; Oglobin and Brock, 2003).

Physical activity

The health and healthcare benefits of exercise in reducing the risk of coronary heart disease, cerebrovascular disease (stroke) and diabetes mellitus have been shown to be substantial (Nicholl, et al., 1994). Light physical activity has also been shown to lead to modest reductions in the number of doctor's visits by older individuals (Lee and

Kobayashi, 2001). Physical exercise has also been linked to reductions in neuroendocrine stress hormones and depressive symptoms (Chanudda, et al. 2005).

Respondents were asked which of five statements best describe their current exercise habit. The statements ranged from “At present I do not exercise and I do not intend to start in the next six months” to “At present I exercise regularly and have been doing so for longer than six months.” Respondents were told that by regular exercise the interviewer means planned exercise for at least one-half hour for three or more days a week. We created a binary variable equal to one if the respondent is a regular participant in an exercise program.

Independent variables

Family Health Endowments

The data set contains two questions on knowledge of family health endowment: whether a family member has been diagnosed with one of six chronic diseases (cancer, diabetes, heart disease, high blood pressure, high cholesterol and stroke) and whether a family member has died from one of those diseases. We created six binary variables for each of the diseases with which family members have been diagnosed (yes = 1, no = 0). In addition, we created a discrete variable measuring the number of chronic diseases diagnosed in the family and a binary variable equal to one if at least one disease has been diagnosed in a family, that is, if the former variable is non-zero.

In a similar vein we created six binary variables, one for each of the diseases from which a family member has died (yes = 1, no = 0). A count variable was also created to indicate the number of diseases from which family members had died. Finally, a binary

variable was created to indicate whether a family member had died from at least one disease. About 10 percent of respondents indicated they were not aware of any family member who had died from one of the diseases listed above (responses of “don’t know”). For each of the binary variables these individuals were given a zero response and a separate variable was created to identify these individuals. Hence we have a seventh binary variable which includes all the “don’t knows.” One benefit of having this group in the data is that if health-related behaviours are unrelated to genetic endowment then such behaviours should also be neutral with respect to not knowing one’s endowment.

Other independent variables

We use a set of fairly standard independent variables. Variables include education, marital status, age, race, gender, area of residence (urban versus rural) and employment status (employed versus unemployed). We created two binary education variables, one for individuals whose highest education is high school completion and another for individuals who have completed college education. For marital status we have a single variable (single versus not single). Age is entered as a continuous variable measured in years. Race is entered as a binary variable (black = 1, all others = 0). The major data weakness is a lack of income data. It is typical for surveys in Jamaica (except for the living standards measurement survey) to not ask questions about income because of the complexity of obtaining a full measure of income. While this is a data weakness, it is not clear that it is serious. Studies of health-related behaviour have produced at best inconsistent results of the relationship between income level and healthy behaviour.

Estimation Strategy

We estimated three equations, one for each of the three health-related behaviours: alcohol consumption, cigarette smoking and exercise. The alcohol consumption and participation in physical exercise variables are binary variables. We use the following standard simple logistic regression model to estimate coefficients for participation in alcohol consumption ($j=1$) and physical activity ($j=2$):

$$\Pr(P_{ij} = 1) = \Lambda(\beta_j' x_i) \quad j = 1, 2$$

where $P_{i1} = 1$ if an individual consumes alcohol, and $P_{i2} = 1$ if an individual exercises regularly, $\Lambda(\cdot)$ is the logistic cumulative distribution function, β_j is a vector of coefficients to be estimated for participation in behaviour j and x_i is a vector of individual i 's characteristics. For participation in physical activity separate equations were estimated for women and men under the assumption that women and men have different motivation for participation in physical activity and in light of our anecdotal evidence that females express stronger distaste for exercise than males.

Participation in cigarette smoking has three outcomes; never smoked ($j=0$), smoked and quit ($j=1$) and currently smoking ($j=2$). We estimated the equation using multinomial logistic regression. Therefore, the model of cigarette smoking participation is

$$\Pr(S_i = j) = \frac{\ell^{\beta_j' x_i}}{\sum_{k=0}^2 \ell^{\beta_k' x_i}} .$$

where $S_i = 0, 1$ or 2 according to whether the individual has never smoked cigarettes, has smoked cigarettes and quit, or currently smokes cigarettes, respectively. We estimated

coefficients (and odd-ratios) for quitters and current smokers, with “never smoked” as the omitted category.

Results

Descriptive statistics are presented in Table 1. Over 21 per cent of the respondents have smoked cigarettes, eight per cent have smoked and quit while nearly 14 per cent smoke currently. Just over one in five individuals consume alcohol frequently, with an average weekly consumption of eight drinks per week. Men are twice as likely to participate in exercise as women (41 per cent vs. 21 per cent). In terms of family history of chronic disease the most frequent disease is high blood pressure (44 per cent) followed by diabetes (34 percent). Over 60 per cent of respondents have a family member who has been diagnosed with one of the six chronic diseases, with an average of one chronic disease per family. Thirty percent of respondents have had a family member die of one of these diseases, while ten per cent of respondents said they did not know of a family member who had died from any of these diseases.

Table 1 about here

Table 2 presents the results of the estimation of the alcohol consumption equation. There is a clear relationship between family history of chronic disease and alcohol consumption. Individuals who have a family member who had died from at least one chronic disease are significantly less likely to consume alcohol regularly (OR = 0.68). This suggests that individuals who have a family history of chronic disease are selecting

to avoid alcohol in health production. It is also important to note that individuals who are not aware of their family history are significantly more likely to be consuming alcohol than individuals whose family members had not died from these diseases (OR = 1.61). It is hard to over-emphasize the importance of this result. If knowledge of family history of disease was unrelated to health-related behaviour then we should expect neutrality between lack of knowledge and behaviour. As we shall see as well with respect to smoking this is clearly not the case.

Table 2 about here

The results of the smoking equation estimation are presented in Table 3. The upper panel of the table presents the results for quitters while the lower panel presents results for current smokers. Individuals whose family members have died from a chronic disease are less likely to be current smokers (OR = 0.80) and more likely to be among the quitters (OR = 1.66). However, the variable is statistically significant only among quitters. As with alcohol consumption, there is a significant relationship between not knowing one's family history and unhealthy behaviour. These individuals are likely to be current smokers (OR = 1.72) or have smoked and quit (OR = 3.22). These results, taken together, suggests that at least some amount of quitting smoking is related to the death of a family member from an disease whose risk is modifiable by avoiding smoking.

Table 3 about here

Tables 4 and 5 present the results of estimating the equations for exercise, with separate results for women and men, respectively. In the case of female participation in

physical activity, this is the only instance in which we find a significant relationship between the diagnosis of (as opposed to death from) a chronic disease within one's family and health-related behaviour. Women whose family members have been diagnosed with a chronic disease are less likely to participate in regular exercise (OR = 0.71). Male participation in exercise only shows sensitivity to having a family member who has died from heart disease (OR = 0.44).

To interpret these results recall the suggestion by Birch and Stoddart (1991) that participation in health-related behaviour depends on both the marginal product of the activity in health production and the direct marginal utility of the activity itself. For many individuals physical activity produces direct disutility (plus there are potentially other costs associated with regular exercise, including the possibility of injury). The health benefit of exercise is the reduction in risk of developing a disease whose risk is modifiable through exercise. Participation in exercise requires the expected health benefit to outweigh the disutility of the activity. We have shown that individuals with a family history of chronic disease are less likely to smoke and consume alcohol. One could argue that participation in some healthy behaviours (avoiding cigarettes and alcohol) reduces the marginal benefit of additional healthy behaviour (exercise). Finally, since most individuals are not completely risk averse, which would mean the individual wishes to completely insure against all risks, given some participation in healthy behaviours, individuals might be willing to accept whatever risks remain thereafter.

Finally, a comment on the difference in female/male participation in exercise is warranted. While we expected higher participation in exercise by males, the difference in participation (21 per cent versus 41 per cent) is quite large. It should be noted that 83 per

cent of the survey participants are black, which closely resembles the racial makeup of the population (85 per cent black). Note also that in Table 4 the coefficient on the black female variable is negative, with an associated odds-ratio of 0.66. We can offer two potential explanations for these results. First it is possible that in the marriage market of black populations there is weight placed on women having certain physical traits (e.g., roundness or curviness). The second is there might be different standards for perception of healthiness for male and female in black populations. If being petite is viewed as a sign of poor health there would be little incentive to women to exercise.

Summary and Conclusion

This paper presented an investigation into whether individuals are more likely to participate in healthy behaviours or avoid unhealthy behaviours if their family has a proven history of diagnosis with, or death from chronic illnesses whose risks are modifiable through participation in (avoidance of) certain behaviours. The major findings are as follows. First, the six chronic illnesses identified are prevalent in the study population. Sixty percent of respondents have a family history of diagnosis with at least one disease, while 30 percent have family members who have died from at least one disease. Second, family history of disease reduces the probability of participation in alcohol consumption and cigarette smoking, but also reduces the probability of participation in regular exercise. Third, women are significantly less likely to participate in regular exercise than men.

Our data provide support for both theoretical perspectives. Individuals appear to substitute away from certain unhealthy behaviours if their family health endowment is

poor as the Becker-Grossman hypothesis suggests. Furthermore, individuals who are not aware of their family health endowment appear, on average, to be more likely to participate in some types of unhealthy behaviours. However, specifically concerning participation in exercise, the expected benefit of exercise does not seem to compensate for the disutility often associated with exercise.

Given the prevalence of diseases in the population it seems that there are significant health benefits to be gained from a healthy lifestyle. Not to mention the possibility of limiting health care costs. It seems particularly important to devise incentives to promote regular exercise among women.

References

- Armstrong, K., Weber, B., FitzGerald, G., Hershey, J.C., Pauly, M.V., Lemaire, J., Subramanian, K., Asch, D.A. Life insurance and breast cancer risk assessment: adverse selection, genetic testing decisions, and discrimination. *Am J Med Gen* 2003; **120A**: 359-364.
- Becker, G.S. (1965), 'A theory of the allocation of time', *Economic Journal*, **75**: 493-517.
- Birch S, Stoddart G. (1991), 'Incentives to be healthy: an economic model of health-related behaviour', in Lopez-Casasnovas G (ed.). *Incentives in Health Services*. Barcelona: Springer-Verlag
- Brown, H.S., III, Pagan, J.A., Bastida, E. (2005), 'The impact of diabetes on employment: genetic IVs in a bivariate probit', *Health Economics*, **14**: 537-544.
- Nabkasorn, C., Miyai, N., Sootmongkol., N, Junprasert, S., Yamamoto, H., Arita, M., Miyashita, K. (2005), 'Effects of physical exercise on depression, neuroendocrine stress hormones, and physiological fitness in adolescent females with depressive symptoms', *European Journal of Public Health*, **16**: 179-184.
- Chen, S, et al. (2002), 'Prices and health: identifying the effects of nutrition, exercise, and medication choices on blood pressure', *American Journal of Agricultural Economics*, **84**: 990-1002.
- Chiappetta, T.O. (2005), 'Managing healthcare costs', *Public Personnel Management*, **34**: 313-320.
- Chodick G, Heymann A.D., Wood F, Kokia E. (2005), 'The direct medical cost of diabetics in Israel', *European Journal of Health Economics*, **6**: 166-171.
- Coulter C.H. (2006), 'The employer's case for health management', *Benefits Quarterly*, **22**: 23-33.
- Dickie M, Gerking S. (1997), 'Genetic risk factors and offsetting behaviour: the case of skin cancer', *Journal of Risk Uncertainty*, **15**: 81-97.
- Dow W.H., Holmes J, Philipson T, Sala-i-Martin X. (1999), 'Longevity complementarities under competing risks', *American Economic Review*, **89**(5): 1358-1371.
- Ganz M.L. (2001), 'Family health effects: complements or substitutes', *Health Economics*, **10**: 699-714.

- Grossman M. (1972), 'On the concept of health capital and the demand for health', *Journal of Political Economy*, **80**(2): 223-255.
- Lee M, Kobayashi S. (2001), 'Proportional treatment effects for count response panel data: effects of binary exercise on health care demand', *Health Economics*, **10**: 411-428.
- Lee, J.M., Turin, M, Botteman, M.F., Stephens, J.M., Pashos, C.L. (2004), 'Economic burden of head and neck cancer: a literature review', *European Journal of Health Economics*, **5**: 70-80.
- Lemaire J, et al. (2000), 'Pricing term insurance in the presence of a family history of breast or ovarian cancer', *North American Actuarial Journal*, **4**(2): 75-87.
- Manning WG, Keller EB, Newhouse JP, Sloss EM, Wasserman J. (1991), *The Costs of Poor Health Habits*. Cambridge, MA: Harvard University Press
- Nicholl, J.P., Coleman, P. Brazier, J.E., (1994), 'Health and healthcare costs and benefits of exercise', *PharmacoEconomics*, **5**(2): 109-122.
- Ogloblin, C., Brock, G. (2003), 'Smoking in Russia: the 'Marlboro Man' rides but without 'Virginia Slims' for now', *Comparative Economic Studies*, **45**: 87-103.
- Ory, C., Vanderplas, A., Dezii, C., Chang, E. (2005), 'Congestive heart failure: attributable costs within the managed care setting', *Journal of Pharm Fin, Economics & Policy*, **14**(2): 87-97.
- Osann, K.E. (1991), 'Lung cancer in women: the importance of smoking, family history of cancer, and medical history of respiratory disease', *Cancer Research*, **51**: 4893-4897.
- Payne KA, et al. (2002), 'Long term cost-of-illness in stroke: an international review', *PharmacoEconomics*, **20**; 813-825.
- Schwartz, A.G., Yang, P., Swanson, G.M. (1996), 'Familial risk of lung cancer among nonsmokers and their relatives', *American Journal of Epidemiology*, **144**(6): 554-562.
- Tabarrok, A. (1994), 'Genetic testing: an economic and contractarian analysis', *Journal of Health Economics*, **13**: 75-91.
- Thorpe, K.E. (2005), 'The rise in health care spending and what to do about it?', *Health Affairs*, **24**: 1436-1445.

Table 1: Descriptive Statistics

Variable	N	Mean	Std. Deviation
Dependent Variables			
Ever a smoker	1513	.2148	.41082
Smoked but quit	1513	0.078	.26825
Currently a Smoker	1513	.1368	.34376
Drinker ³	1513	.2062	.40472
Participate in regular exercise - Women	973	.2076	.40580
Participate in regular exercise - Men	540	.4130	.49252
Independent Variables			
Satisfied with health	1513	.7284	.44496
Currently working	1513	.6259	.48405
Urban Resident	1513	.6098	.48795
Age	1507	34.78	12.166
Females	1513	.6431	.47925
Complete Sec ed	1513	.4501	.49767
College ed	1513	.1474	.35461
Black	1513	.8301	.37563
Single	1513	.4997	.50017
Single female	1513	.3219	.46735
Family diagnosed with diabetes	1513	.3351	.47218
Family diagnosed with heart disease	1513	.0925	.28987
Family diagnosed with blood pressure	1513	.4408	.49665
Family diagnosed with stroke	1513	.0991	.29895
Family diagnosed with cancer	1513	.1355	.34236
Family diagnosed with cholesterol	1513	.0330	.17882
Number of diseases diagnosed	1513	1.1362	1.18689
Family diagnosed with at least one disease	1513	.6127	.48730
Family died of at least one disease	1513	.3007	.45873
Don't know if family died from disease	1513	.1038	.30506
Are you satisfied with your health	1513	.7284	.44496

³ The average “drinker” consumes 8.14 alcoholic drinks per week. Occasional drinkers consume 0.03 alcoholic drinks per week. Occasional drinkers were therefore grouped with non-drinkers.

Table 2 Logistic regression results, Dependent variable: Do you drink, yes = 1

Variables	B	S.E.	Wald	Sig.	Odds-ratio
black	-.023	.191	.015	.903	.977
single	-.163	.156	1.099	.294	.850
female	-1.919	.147	170.328	.000	.147
age	-.009	.007	1.582	.209	.992
employed	.346	.161	4.613	.032	1.414
secondary ed	-.281	.161	3.065	.080	.755
college ed	-.689	.236	8.542	.003	.502
don't know family history	.478	.214	4.995	.025	1.612
fam died of disease	-.385	.169	5.208	.022	.680
area	.219	.147	2.220	.136	1.245
Constant	-.072	.391	.034	.853	.930
Model					
Diagnostics					
Chi-Square	245.4	p-value = 0.000			
Log-likelihood	1283.6				
Cox & Snell R ²	.151				
Nagelkerke R ²	.234				

Table 3 Logistic regression results: Smoking status (Quitters = 1, Smokers = 2, Omitted category: never a smoker = 0)

Variable	B	Std. Error	Wald	Sig.	Odds-ratio
Quitters					
Intercept	-4.504	.636	50.077	.000	
black	-.877	.250	12.357	.000	.416
single	.054	.243	.049	.825	1.055
female	-1.031	.219	22.139	.000	.357
age	.081	.010	63.409	.000	1.085
employed	.420	.244	2.970	.085	1.522
secondary ed	-.440	.259	2.878	.090	.644
college ed	-.708	.370	3.652	.056	.493
don't know family history	1.168	.308	14.355	.000	3.217
Fam died of disease	.505	.236	4.569	.033	1.657
area	-.133	.217	.376	.540	.875
Smokers					
Intercept	-1.914	.447	18.368	.000	
black	-.369	.213	3.000	.083	.691
single	-.204	.180	1.292	.256	.815
female	-1.532	.170	81.317	.000	.216
age	.035	.008	21.042	.000	1.035
employed	.368	.189	3.775	.052	1.444
secondary ed	-.658	.189	12.050	.001	.518
college ed	-.596	.257	5.386	.020	.551
don't know family history	.542	.249	4.715	.030	1.719
Fam died of disease	-.229	.194	1.404	.236	.795
area	.353	.174	4.133	.042	1.423
Diagnostics					
Chi-square	316.1	p-value = 0.000			
Log-likelihood	1548.5				
Cox & Snell R ²	.19				
Nagelkerke R ²	.26				

Table 4 Logistic regression results: Female Participation in Regular Exercise

Variable	B	S.E.	Wald	Sig.	Odds-ratio
Black	-.419	.202	4.309	.038	.657
Secondary ed	.560	.204	7.534	.006	1.750
College ed	1.205	.249	23.457	.000	3.337
employed	.162	.172	.882	.348	1.176
age	.016	.008	3.648	.056	1.016
single	.292	.177	2.725	.099	1.339
Fam diag with disease	-.347	.170	4.149	.042	.707
area	-.135	.171	.624	.430	.874
Satisfied with health	.695	.196	12.612	.000	2.004
Constant	-2.464	.478	26.538	.000	.085
Diagnostics					
Chi-square	53.2	p-value	=	0.000	
Log-likelihood	935.3				
Cox & Snell R ²	.054				
Nagelkerke R ²	.084				

Table 5 Logistic regression results: Male Participation in Regular Exercise

Variables	B	S.E.	Wald	Sig.	Odds-ratio
secondary ed	.628	.206	9.298	.002	1.873
college ed	.971	.279	12.123	.000	2.641
Satisfied with health	.824	.250	10.822	.001	2.279
under25	.458	.237	3.737	.053	1.581
under35	.714	.213	11.191	.001	2.042
Fam died of heart disease	-.823	.433	3.618	.057	.439
Constant	-1.754	.288	37.052	.000	.173
Diagnostics					
Chi-square	46.8	p-value = 0.000			
Log-likelihood	685.3				
Cox & Snell R ²	.083				
Nagelkerke R ²	.112				