ECONOMIC DEVELOPMENT
AND WELL-BEING
RESEARCH GROUP

EDWRG Working Paper Series
January 2021

The impact of mental health behaviour on tobacco consumption in South Africa

Working Paper Number 02-2021

Nomsa Y. Nkomo, Beatrice D. Simo-Kengne and Mduduzi Biyase

Cite this paper: Nkomo, Y. Nomsa, Beatrice, D. Simo-Kengne., & Biyase, M. (2021). The impact of mental health behaviour on tobacco consumption in South Africa. *EDWRG Working Paper Number 02-2021*.

The impact of mental health behaviour on tobacco consumption in South Africa

Nomsa Y. Nkomo[1], Beatrice D. Simo-Kengne[2] and Mduduzi Biyase[3]

Abstract

The relationship between smoking and mental health behaviours is unclear and the factors that account for their comorbidity have received limited attention. This study aims at clarifying such association in South Africa using the collated five waves of the National Income Dynamic Study. Heckman selection, double-hurdle, and control function approaches were used to account for both selection bias and endogeneity. Empirical results reveal that besides socio-economic factors, mental health behaviours proxied by depression and sleepless significantly influence more the decision to smoke than the smoking intensity. Furthermore, tobacco consumption is found to be significantly higher among males, though the gap seems to be narrowing partly due to ads pursuing the untapped female market. Interestingly, the smoking probability proves to be more prominent in the younger generation and tends to decline with the age, while married individuals have a lower likelihood to smoke and when they do; their smoking frequency is less compared to unmarried individuals.

Keywords: Depression, tobacco consumption, selection bias, endogeneity

JEL Classification: C23, I18, L66

1. Introduction

Tobacco is the single largest preventable cause of death and chronic disease in the world today. Studies have shown that between the 1950s and 1990s, the death rates for smokers within the age range of 35 and 69 were approximately three times as much as those of non-smokers (WHO, 1997), killing up to one in two long-term consumers, many of them before age 65. The World Health Organisation pointed out that at least one billion individuals have died as a result of tobacco-related diseases in the 20th era (WHO, 2015). Cigarettes are commonly used tobacco substances despite its negative impact on the body system (Peto et al., 2006). Its consumption is a cause of non-communicable diseases (NCDs) such as lung cancer and is expected to claim approximately six million lives by 2030 worldwide (Holland, 2015) if the present trend continues. According to the US National Institutes of Health, National cancer institute, Tobacco smoking is a cause of nearly 90% of lung cancers besides other cancers. It is also reported that for people under the age of 65 years, 45% of coronary heart disease cases amongst men, and 40% in women, are attributed to tobacco use (Mathers and Loncar, 2006).

[1]Corresponding author, Email. nomsaynkomo@gmail.com, School of Economics, University of Johannesburg.
[2] Email. bdsimo-kengne@uj.ac.za, PEERC, School of Economics, University of Johannesburg.
[3] Email. mbiyase@uj.ac.za, School of Economics, University of Johannesburg.

While most adults worldwide are conscious of the physical health risks of tobacco consumption, research indicates that smoking also affects people's mental health. Though smoking provides short term benefits such as improved mood and concentration, reduced anger and stress, as well as relaxation of the muscles, these assistances are outweighed by the higher rates of smoking-related physical health problems, such as lung cancer, that are common in people with mental health problems. The motives for smoking vary individually, and understanding the reasons for smoking might assist those who want to stop smoking.

Buckley, (2007) found that social and psychological factors also contribute to keeping smokers smoking. For instance, many teenagers point out that the reason why they start to smoke is to experiment, but other factors encouraging someone to become a regular smoker include having friends or family who smoke and the parents' attitude towards smoking (Ritt-Olson et al., 2005). As teenagers grow into adulthood, they are more likely to become regular smokers if they drink alcohol or drug abuse or live in poverty. Issues like these trigger the likelihood of an individual to encounter stress (Munafo et al., 2008) and eventually to start smoking. Some adults claim that smoking becomes a habit as it helps them to relax and minimize stress (Khantzian.1997). The hypothesis that individuals smoke as the means of relieving' stress is known as 'self-medication'. Thus, stress is one of the manifestations of mental health behaviors, which causes some people to find it difficult to cope with work or pressure and other people to feel a headache, breathlessness or to become easily irritable and anxious. Continuous feeling of stress is also associated with depression, which often causes individuals to seek self-medication including smoking and/or drinking alcohol more than usual.

Although smoking is thought to help people relax, Buckley (2007) has shown that smoking escalates feelings of anxiety and tension. Nicotine makes an instant sense of relaxation and people would believe that it reduces stress and anxiety. This particular feeling of relaxation is short-term as it would soon give way to withdrawal symptoms and continues to increase cravings. Accordingly, Escobedo et al. (1998) the rate of smoking in adults with depression is approximately twice as high compared to adults without depression. The same study also pointed out that individuals suffering from depression experience difficulties when they try to quit smoking. In addition, several studies concluded that most individuals start smoking before showing any signs of depression, so it is uncertain whether smoking causes depression or depression inspires people to smoke. However, a higher rate of depression has been reported in smoking individuals than non-smokers (Breslau et al., 1998). Thus, high depression symptoms are likely to be linked to the progressive use of tobacco.

Despite the established association between smoking and socioeconomic status substantiating the strong belief that smoking can cause depression (Boden et al., 2010), little is known on the relationship between cigarette smoking and mental health behaviours or mood swings. Depression might cause individuals to smoke as self-medication; implying that mental health behaviours (MHBs) can cause individuals to conduct themselves differently or use certain products to try to calm their condition down. However, the symptoms of depression individuals experience through cigarette use may lead to a high risk of further stress and depression. Conversely, infrequent tobacco use may lessen individuals' mood swings while continuous use may worsen it, or it may be triggered by shared risk factors such as hereditary; hence making the association, not causal. Therefore, the relationship between smoking and MHBs (namely depression and stress) remains complex and understanding such association becomes imperative to inform health and social

policy decisions. This is particularly relevant for South Africa where one-third of its population suffers from mental illnesses[4].

While oppression leads to depression, which aggravates mental disorder, South Africa has historically experienced oppression due to apartheid. Although critics have admonished against its adverse ongoing effects on the mental health of South Africans (Dawes et al., 1989; Dawes, 1990), approximately three decades after the abolishment of apartheid, mental health illnesses (depression) are still reflected as one of the main public health problems in South Africa (Tomlinson et al., 2009). Considering that, depressed individuals will initiate regular smoking to self-medicate against depression moods; it is surprising how existing studies on tobacco use in South Africa have focused solely on socioeconomic and demographic motives of smoking. This study investigates the plausibility of mental health conditions to act as a stimulus of smoking in South Africa. It analyses both the smoking decision and smoking intensity using a dynamic survey which is a national representative of the entire population, the outcome of which provides indispensable guidelines for designing and/or tayloring smoking policy for mentally ill individuals.

2. Literature review

The theory of normal consumer choice assumes that every person (or household) is likely to be a consumer of all goods, provided they can afford it. However, this might not be applicable for some commodities such as tobacco, which some people cannot be persuaded to consume, irrespective of the price and disposable income levels. For these goods, non-consumption is not necessarily explained by economic affordability while non-zero consumption may also be attributed to other behavioural factors such as mental conditions. Thus, in the context of tobacco consumption, zero spending echoes either the decision to participate in regular smoking or a normal corner solution where only potential smokers define the parameters of tobacco consumption in the Engle curve (Blaylock and Blisard, 1993). This has resulted in modelling tobacco consumption with discrete random preference systems where smokers are assumed to have a diverse preference structure than non-smokers (Pudney, 1989) including addiction, self-medication, imitation, enjoyment among others. Consequently, smoking behaviour can be understood as an intertemporal decision with instantaneous satisfaction and a long-term health risk (Miura, 2019).

Tobacco smoking can lead to poor mental and physical health. can be a In the empirical literature, tobacco use has been established as the main cause of death arising from cancer, heart disease, stroke lung diseases, diabetes and respiratory diseases (West, 2017). Unlike these smoking led diseases, mental disorders can arise as both a cause and consequence of smoking although most of the existing evidence focuses on smoking as a driver of mental illnesses.

Though inconclusive about the causal direction between smoking and mental disorder, numerous studies affirm a positive link between smoking and mental illnesses. However, some prior studies report a negative or zero relationship between cigarette smoking and depression. Audrain-MCgovern et al. (2006) reported the absence of an association between depression and tobacco consumption. Similarly, Park et al. (2007) investigated the link between smoking and mental illness and found no evidence of link. These findings were consistent with White et al. (2007) who

[4] http://www.sadag.org/index.php?option=com_content&view=article&id=2178:sa-s-sick-state-of-mental-health&catid=74&Itemid=132

also found no association between depression and successive smoking among African and Hispanic Americans.

Contrary to these studies, Munafo et al. (2008) emphasized the use of tobacco as a cause of depression and concluded that the relationship between smoking and MHBs may be clarified by causal inference. Using the logistic regression, Kang and Lee. (2010) conducted a study on the causal connection between depression and tobacco consumption and concluded that smoking instigated depression. Another causal study by Boden et al. (2010) uses longitudinal data on adolescents and proved that smoking is a cause of mental illness such as depression. Besides these studies showing that cigarette smoking is a predictor of depression, some research has established that smoking escalates the dangers of depression (Pasco et al., 2008; Breslau et al., 1998). Fergusson et al. (2003) and Choi et al. (1997) concluded that peer tobacco consumption leads to depression among adolescents.

Other studies separated this relationship on gender lines. Kandel et al. (2007) discovered that smoking trends in men are related to depression in the future while Kandel provided support that the connection between tobacco use and depression is stronger in females as compared to males (Repetto et al., 2005). Upon investigating the risk of depression on smoking women, Milic et al. (2010) concluded that tobacco use is linked to an increased rate of depression in women and has the potential of causing harmful consequences on mental health. In support of the study mentioned above, Pasco et al. (2008) explored the role of tobacco smoking as a cause of major depression among women. This study utilized cross-sectional and longitudinal data and concluded that depression disorders in women result from smoking and that smoking continued to be a predictor of depression after controlling for family and hereditary background.

However, lab-based studies provided evidence that cigarette smoking eliminates mental health disorders or negative moods (Wahl et al., 2005; Kassel et al., 2006). There seems to be a paradox, empirically, in showing that the use of cigarettes may cause mental illness while some smokers say that they smoke to ease their mental health behaviours.

While most of the evidence point to the causality running from smoking to mental illnesses (Choi et al., 1997; Wu and Antony, 1999; Goodman and Capitman, 2000), others suggest a reverse causality of mental illness or depression to tobacco consumption. This is in line with the perception that depressed individuals may acquire a smoking habit to try to self-medicate the negative effects as compared to non-depressed or non-stressed individuals. Consistently, several studies have shown that mental health behaviours such as depression predict the initiative to smoke which leads to regular smoking (Escobedo et al., 1998; Fergusson et al., 2003; Rohde et al., 2004). Investigating the transmission channels, some studies documented that tobacco consumption gives benefits to the person by calming their negatives effects of depression (Swendsen and Merikangas, 2000; Ritt-Olson et al., 2005). Accordingly, individuals who suffer from mental illnesses are more likely to become dependent on substances than individuals who do not have mental disorders (Kessler et al., 1997). Wang et al. (1996) substantiated that depression leads to smoking initiation which progresses to regular smoking. Furthermore, Kassel and Hankin (2006) suggested that depression might cause an individual to be more vulnerable to smoking influences, which in turn encourages tobacco use.

McManus et al., (2010) observed that individuals with mental illness begin smoking at an early age and tend to be more addicted to tobacco use than the general population. Similarly, Whitbeck et al. (2009) reported a positive relationship between smoking and impairment amongst

investigated university students resulting from the increased level in the depression symptom. Weinstein et al. (2008) investigated the unpredictability of mood changes and cigarette smoking escalation among adolescents with the conclusion that negative mood is a risk element that leads to increased smoking in the future and that the mood-stabilizing effects may support and preserve daily cigarette use among the users. This corroborates the findings by Fergusson et al. (2003) that major depression leads to an increased rate of daily smoking.

Generally, limited studies suggested that depression/anxiety was accompanied by some type of future smoking behaviour. Although the literature on the potential relationship between smoking and depression is unpredictable in terms of the causal factor, the conclusion of direct causality from smoking to depression is strongly supported in most studies (Fluharty et al., 2017). However, some researchers are favourable to possible dual effects between smoking and depression; implying that such association is bidirectional. These include Brown et al. (1996), Wang et al. (1996), Windle and Windler (2001), Breslau et al. (1998), and Wiesner and Ittel (2002) who indicated that tobacco consumption and depression reciprocally influence each other. These feedback effects between smoking and depression channel occurs through two main mechanisms (Boden et al., 2010). Firstly the one involving the correlated risk factors and secondly, the direct link, which states that smoking is a strong cause of depression. In line with these studies, Brook et al. (2006, 2008) reported that depression and worry were concomitant with smoking habits. Ritt-Olson et al. (2005) inferred from a cross-sectional study that depressed adolescents that are more exposed to peer cigarette smoking promote more smoking uptake. Finally, Green et al. (2006) and Steuber and Danner (2006) specified a bidirectional association between tobacco consumption and depression that was only detected in females. However, some studies supporting a reciprocal relationship between the smoking and depression pointed to a possible lingering link the two variables (Hu et al., 2006; Munafo et al., 2008).

Other studies state a unique influence, implying that they do not directly influence each other (Wu and Antony., 1999; Wang et al., 1996; Fergusson et al., 2003). More interestingly, Park and Romer (2007) report that the relationship between depression and smoking in adolescence is a bidirectional link although they might be some factors that might cause this relationship to be spurious. Nevertheless, Kassel et al. (2003) state that the association between mental illness and smoking may not have a direct effect, but rather an indirect through an additional variable and their link may not be causal but caused by mutual genetic or environmental factors. Yet, there exists limited evidence on the genetic influences of tobacco use (Lyons et al., 2008; Dierker et al., 2002).

Given the complexity of the causal analysis between depression and smoking behaviour, this study rather focuses on the potential comorbidity between tobacco use and mental health conditions in South Africa. While the existing literature on such relationship is country-specific, their inference is mainly drawn from developed countries experience and therefore cannot be used to inform policy strategies in developing countries. Considering the relatively high rate of depression in South Africa, we provide an empirical assessment of the perception that depressed individuals may acquire a smoking habit to self-medicate the negative feelings as compared to non-depressed or non-stressed individuals. To this end, we exploit the unique feature of the dynamic survey dataset now available in South Africa and implement various empirical setups to account for relevant econometric issues including heterogeneity, endogeneity and selection bias. Unlike previous studies on smoking determinants in South Africa (Jordaan et al., 1999; Panday et al., 2007; Peltzer., 2011; Vellios and van Walbeek., 2016), this subject matter has virtually received no evaluation.

3. Dataset

This study used the National Income Dynamics Study (NIDS) dataset to investigate the impact of MHBs on tobacco consumption. The dataset is a longitudinal survey of South African households. This survey is biennial and is conducted by the Southern Africa Labour and Development Research Unit (SALDRU). This study will use all the five (5) waves that have been released so far to carry out this investigation. The variables incorporated in this study include the key explanatory variable MHB (proxied by depression); the education variable, a categorical variable that is categorized into five divisions, no schooling, primary, secondary, matric and tertiary; urbanization; race; household income; gender; marital status; employment status; and age. All the other variables are either binary or categorical except household income, total consumption, and age. The dependent variables are the regular smokers and the intensity of smoking. The regular smoking variable was drawn from the survey questions, where individuals were asked whether they smoke or not. This variable was categorized to binary (zero if they do not smoke and one if they smoke)

The National Income Dynamic Study (NIDS) provides informative data on South African individuals.

3.1.Estimation strategy

An empirical concern associated with the analysis of mental health-smoking nexus is that some respondents report smoking a fair amount of cigarettes per day or not smoking at all. Our data comprise a fair amount of respondents who are non-smokers. The reason for not smoking may be attributable to unaffordability of cigarettes or simply taste and preferences of the respondent in question. There are many ways (empirically) to account for this truncated distribution of smoking behavior. The most commonly used methods include Tobit model (named after Tobin 1958), Heckman's Selection model or Heckit (named after Heckman 1979) and Cragg's Double Hurdle (named after Cragg 1971). Given the assumption underpinning the Tobit model this study employs the latter approaches that are more flexible and able to cope with challenges associated with analyzing smoking behavior. It adopts Heckit model to account for the sample selectivity bias in their study. Heckit is preferred because it allows for the possibility that variables influencing the probability and the amount of consumption are not the same (which is not the case with the Tobit model—assumes that the variables influencing the probability and the amount of consumption are the equivalent).

The Heckman selection model assumes the existence of the underlying regression association, (Lewis 1974; Heckman 1976) and has been used by several studies in the field of smoking behaviour patterns (Madden, 2008; Moshoeshoe, 2012; Wodjao, 2007). It consists of two single equations, one concentrating on the participation into the sample (sample selection equation), and the other equation connecting the covariates that are important to the outcome (smoking intensity). Specifically,

The two latent response Y_{it}^{*}(smoking frequency), and S_{it}^{*}(regular smoking) the selection equation is displayed as follows:

$$Y_{it}^{*} = X_{1it}\beta 1 + \mu_{it} \quad (1)$$

Secondly, the selection model.

$$S_{it}^{*} = Z_{it}\gamma + \nu_{it} > 0 \quad (2)$$

Where Y_{it}^{*} and S_{it}^{*}represents the unobserved latent variables, X_{it} and Z_{it} are explanatory in both models respectively. Generally, X is presumed to be a subset of Z, in that that all elements predicting smoking frequency (Y/outcome), predict regular smoking as well (S/selection). The symbol μ and ν display the normally distributed error terms, and β and ɣ present the parameters to be estimated. The smoking frequency which is the outcome variable is observed only if regular smoking or selection is above zero, i.e.:

$$S_{it} = \begin{pmatrix} 1 \; if \; S_{it}^{*} > 0 \\ 0 \; if \; S_{it}^{*} \geq 0 \end{pmatrix} \quad (3)$$

The key notion of the Heckman selection model is the fact that it appears theoretically and relatively possible that unobservable factors may have an influence on both smoking frequency and the possibility of smoking regularly (S_{it} or selection). If endogeneity is absent or controlled for, the model is reduced to a two-step selection model, the selection effect gives the outcome that is expected for the observed equation when all the explanatory variables are held constant including the endogenous variable.

Double-hurdle model

Similar to the Heckman selection, the double-hurdle (Cragg, 1971) also makes use of two separate equations. Firstly, the decision to become a regular smoker and secondly, the frequency of smoking per individual. This model accounts for zeros in participation derived from the decision to smoke or and assumes that the two separate hurdles must pass before the positive level of smoking will be observed.

However, double –hurdle model has some limitations. One of those limitations is that it is derived from the theory of bivariate normality of the error terms. If the normality theory is violated, the maximum probability estimates of the model will be biased. This may be mainly applicable when the model is useful to an outcome variable with a skewed distribution, as is regularly the case with survey data on tobacco consumption.

Different from the Tobit model which does not sufficiently present participation decision and actual consumption, the Double Hurdle achieves this by the use of different (independent) underlying variables y_{1it}^{*} and y_{2it}^{*}. The former underlying variable denotes the utility from participation in smoking while the latter represents the utility from tobacco consumption. The model is given below as;

$y_{1it}^{*} = x_{1it}'\beta_1 + u_{it}$ participation hurdle (regular smoker) (8)

Equation (9) is a binary response of an individual who is a regular smoker

$Regular\_smoker_{it} = 1$ when $y_{1it}^{*} > 0$ and $Regular\_smoker_{it} = 0$ when $y_{1it}^{*} \leq 0$

$y_{2it}^{*} = x_{2it}'\beta_2 + \nu_{it}$ tobacco consumption hurdle (frequency of smoking) (9)

This is a truncated regression and can be observed if

$Smoking\_frequency_{it} = y_{2it}^{*}$ when $y_{1it}^{*} > 0$ and $Smoking\_frequency = 0$ when $y_{1it}^{*} \leq 0$

Cragg(1971) formulated the likelihood function displayed in equation (10)

$$L_{Craggit} = \prod P(y_{1it}^{*} \leq 0) \prod P(y_{1it}^{*} > 0) \prod f(y_{2it} | y_{1it}^{*} > 0) \quad (10)$$

and was later clarified by Amemiya (1984) for u_{it} and v_{it} as

$$L_{Craggit} = \prod I(y_{1it}^{*} \leq 0)[1 - \Phi(\frac{x_{1it}'\beta_1}{\sigma_1}) \prod I(y_{1it}^{*} > 0)\, \Phi(\frac{x_{1it}'\beta_1}{\sigma_1}) \frac{1}{\sigma_2} \Phi(\frac{y_{2it} - x_{2it}'\beta_2}{\sigma_2}) \quad (11)$$

Empirically, the first hurdle is believed to be a function of non-economic factors impacting tobacco consumption, so disposable income is omitted from the first hurdle (Newman et al, 2003). The omission of this variable is determined solely by non-economic aspects (Pudney, 1989; Yen, 2005). The two selected models might suffer from some methodological issues such as measurement errors and simultaneity bias that can be corrected by using a control function.

Control function Approach

To address the problem of endogeneity in the two-stage estimation process, Semykina and Woolridge (2010) propose the control function approach. Besides the selection bias, this approach ensures the consistency of estimates in the presence of endogenous controls.

Considering that besides endogeneity tobacco consumption entails selction bias, the following steps are followed to control for both issues. In the initial stage, the participation decision equation (similar to equation 8) is estimated using probit and the inverse mills ratio (IMR) derived from the probit output is used to correct for selection bias. Similarly, the endogeneous variable is regressed on its instrument besides other coviariates and the estimated residuals are used to correct for endogeneity. In the second stage, the IMR and the estimated residuals are included as additional explanatory variables in the consumption equation.

Starting from the endogenity issue, tobacco consumption can be modelled as follows:

$$TC_{it} = \alpha_1 + \beta_1 W_{TCit} + \delta_1 MHBs_{it} + \mu_{it} \quad (12)$$

where W_{TCit} is all other variables that have an impact on tobacco consumption.

$E(W_{TCit}, \mu_{it}) = 0$ might be violated due to endogeneity (13)

Because of the simultaneity effect, MHBs can also be expressed as a function of tobacco consumption; leading to the following equation:

$$MHBs_{it} = \alpha_2 + \beta_2 W_{MHBsit} + \delta_2 TC_{it} + \varepsilon_{it} \quad (14)$$

where W_{MHBsit} is all other variables that have an impact on MHBs including "no exercise or lack of physical activity". In effect, lack of physical activity is considered as an important predictor of MHBs (Hamilton et al., 2003; Kendler et al., 1993 and Frijters et al., 2010) while this variable is unlikely to influence smoking except through MHBs. Therefore zero correlation is asuumed to hold between W_{MHBsit} and the errors term.

$$E(W_{MHBsit}, \varepsilon_{it}) = 0 \quad (15)$$

Following the literature (Garen, 1984; Mwabu, 2009; Wooldridge, 2010; Baye and Fambon, 2010), the endogeneity bias is alleviated by estimated the modified version of equation (12) obtained by including the estimated residuals from the reduced form model of MHBs (Equation 16).

$$TC_{it} = \alpha_1 + \beta_1 W_{TCit} + \theta\hat{\varepsilon}_{it} + \pi(\hat{\varepsilon}_{it} MHBs_{it}) + \mu_{it} \quad (16)$$

From Equation 12 to Equation 16, $\hat{\varepsilon}_{it}$ is residuals derived from the reduced form model of MHBs (Equation 14) while ($\hat{\varepsilon}_{it} MHBs_{it}$) is an interaction of the residuals and MHBs variables (depression or sleepless). The projected error term, $\hat{\varepsilon}_{it}$ serves as a control for unobservable variables that are correlated with MHBs, thus allowing this endogenous covariate to be used as a normal variable during estimation. The interaction term, ($\hat{\varepsilon}_{it} MHBs_{it}$), controls for the effect of neglected non-linear interaction of unobservable variables with the input into tobacco consumption (Heckman and Robb 1985; Mwabu, 2009).

Endogeneity may arise from various sources. In this study, we proxied mental health behaviour by depression and sleepless, but these proxies cannot fully characterize MHBs and this may lead to endogeneity due to measurement errors. The other methodological issue is the omitted variable bias which occurs as a result of using non-randomly selected samples to estimate behavioural association as an ordinary specification. The resulted missing data problem bias will eventually cause endogeneity. The third source of endogeneity is attributed to reverse causality, which might occur when the explanatory variable, in this case, MHBs is conjointly determined with the dependent variable (Tobacco consumption).

Equation (16) is further extended to include the initial decision individuals make to be smoker or non smoker. This smoking decision is pertained to selection bias which can be mitigated using the IMR from the probit model of the smoking participation function represented below.

$$Regular\_smoker_{it} = A_0 + A_1 z_{it} + u_{it} \quad (17)$$

where $Regular\_smoker_{it}$ is a binay smoking decision and z_{it} the set of its determinants.

Thus, the second and last stage of the control function approach consists to improve Equation (16) by including the IRM estimated from Equation (17); allowing to account for both endogeneity and selection bais.

$$TC_{it} = \alpha_1 + \beta_1 W_{TCit} + \theta\hat{\varepsilon}_{it} + \pi(\hat{\varepsilon}_{it} MHBs_{it}) + \delta IMR + \mu_{it} \quad (18)$$

From the equation above, it is possible to test whether $\theta = \pi = 0$ (coefficients of the residuals and the interacted term with the residuals) using the t-test. The existence of endogeneity is confirmed if $B_3 \neq 0 \; and \; \pi \neq 0$.[5]

Descriptive statistics and preliminary analysis

[5] Similarly, it is equally expected that the relevance of selection bias be tested using a t-test on the coeffient δ. Practically, in STATA, the two steps are carried out simultaneously such that the estimates of the second equation are automatically corrected for the selection bias without necessary displaying its coefficient in the output table.

Table 1 below presents a summary of the key features of the sample analysed which is a merged NIDS dataset of the 2008, 2010, 2012, 2014 and 2017 waves. The total sample size is 92460 observations (individuals) with an average age of approximately 25 years and a majority of who are female (54.6%).42, 5% of the sample self-reported that they were depressed, and 13% indicated experiencing signs of sleepless or stress. The statistics also suggest that the maximum number of cigarettes smoking individuals can take per day is five. For all the binary variables, the outcome of zero is treated as a reference variable as compared to an outcome of one. Accordingly, the following reference variables are considered: male, rural, Black, not married (widowed, separated, divorced, never married), no schooling, employed, non-smoker. All the variables were controlled for time-fixed effects and issue specifics attached to different waves.

TABLE 1: DESCRIPTIVE STATISTICS

Variable	Description	Mean	Std.Dev	Min	Max
Regular smoker	1 if a regular smoker, 0 otherwise	0.0344	0.1822	0	1
Smoking frequency	Avg amount of cigarettes consume a day	3	1.144	1	5
Sleepless	1 if sleepless, 0 otherwise	0.1305	0.3368	0	1
Urban	1 if urban, 0 otherwise	0.5429	0.4981	0	1
Female	1 if female, 0 otherwise	0.5466	0.4978	0	1
Married	1 if married, 0 otherwise	0.2603	0.4388	0	1
Own-house	1 if own house, 0 otherwise	0.7902	0.4071	0	1
Depressed	1 if depressed, 0 otherwise	0.423	0.494	0	1
Western Cape	1 if Western cape, 0 otherwise	0.1213	0.3265	0	1
Eastern Cape	1 if Eastern cape, 0 otherwise	0.0697	0.2547	0	1
Northern Cape	1 if Northern Cape, 0 otherwise	0.051	0.22	0	1
Free State	1 if Free State, 0 otherwise	0.2856	0.4517	0	1
KwaZulu Natal	1 if KwaZulu Natal, 0 otherwise	0.065	0.2465	0	1
North West	1 if North West, 0 otherwise	0.1284	0.3345	0	1
Gauteng	1 if Gauteng, 0 otherwise	0.0707	0.2563	0	1
Mpumalanga	1 if Mpumalanga, 0 otherwise	0.0766	0.2659	0	1
Unemployed	1 if unemployed, 0 otherwise	0.3798	0.4853	0	1
Age	Age of each individual	24.83	.19.88	0	107
Age-SQD	Age of each individual squared	101.06	1372.87	0	11449
Coloured	1 if Coloured, 0 otherwise	0.1428	0.3498	0	1
Asian_Indian	1 if Asian or Indian, 0 otherwise	0.0292	0.1683	0	1
White	1 if White, 0 otherwise	0.0806	0.2722	0	1
Primary	1 if primary, 0 otherwise	0.3049	0.4603	0	1
Secondary	1 if secondary, 0 otherwise	0.2584	0.4378	0	1
Matric	1 if matric, 0 otherwise	0.1076	0.3033	0	1
Tertiary	1 if tertiary, 0 otherwise	0.1132	0.3169	0	1
lHHincome	Household total income	8.5331	1.0778	0.677	14.773

Note. Avg refers to average

4. Empirical results and discussion

Table 2 shows the probit regression model results of the determinants of tobacco consumption. In Model 1, only socio-economic factors are controlled for whilst Model 2 and Model 3 include socio-economic factors and account for MHBs proxied by depression and sleepless. 17368 observations in the data set were part of the estimations. The likelihood ratio (LR) χ^2 test, probability value and log-likelihood, display the overall significance of the models. The output for Model 2 and Model 3 shows that the models accounting for MHBs is the best as it has a log-higher likelihood and close to zero. The LR $\chi^2$of 127.48 with the probability value of 0.000 (Model 1), LR $\chi^2$of 124.21 with the probability value of 0.000 (Model 2) and LR $\chi^2$of 122.79 with the probability value of 0.000 (Model 3) and indicates that all these models are statistically significant.

TABLE 2: PROBIT MODEL ESTIMATES OF THE DETERMINANTS OF TOBACCO CONSUMPTION

VARIABLES	Model 1	Model 2	Model 3
Depressed		0.228***	
		(0.056)	
Sleepless			0.374***
			(0.073)
Urban	0.196**	0.196**	0.189**
	(0.082)	(0.082)	(0.082)
Female	-1.030***	-1.039***	-1.047***
	(0.076)	(0.076)	(0.076)
Married	0.026	0.040	0.031
	(0.076)	(0.076)	(0.076)
Own-House	-0.047	-0.041	-0.040
	(0.072)	(0.071)	(0.072)
Western Cape	-0.358***	-0.358***	-0.339**
	(0.137)	(0.137)	(0.137)
Eastern Cape	-0.135	-0.128	-0.119
	(0.127)	(0.127)	(0.126)
Northern Cape	0.124	0.111	0.122
	(0.160)	(0.160)	(0.160)
Free State	-0.602***	-0.601***	-0.597***
	(0.136)	(0.136)	(0.136)
KwaZulu Natal	-0.228	-0.242	-0.235
	(0.163)	(0.163)	(0.163)
North West	-0.102	-0.107	-0.104
	(0.124)	(0.123)	(0.123)
Gauteng	-0.350**	-0.341**	-0.367**
	(0.158)	(0.157)	(0.158)
Mpumalanga	-0.494***	-0.484***	-0.501***
	(0.169)	(0.169)	(0.169)
Unemployed	-0.079	-0.083	-0.091
	(0.067)	(0.067)	(0.067)
Age	0.059***	0.056***	0.057***
	(0.010)	(0.010)	(0.010)
Age-SQD	-0.000***	-0.000***	-0.000***
	(0.000)	(0.000)	(0.000)
Coloured	0.798***	0.815***	0.792***
	(0.120)	(0.120)	(0.120)
Asian_Indian	0.359*	0.377*	0.344
	(0.211)	(0.211)	(0.212)
White	0.780***	0.808***	0.772***
	(0.121)	(0.121)	(0.121)
Primary	0.074	0.063	0.074
	(0.154)	(0.154)	(0.154)
Secondary	-0.117	-0.122	-0.114
	(0.157)	(0.156)	(0.157)
Matric	-0.131	-0.134	-0.126
	(0.170)	(0.169)	(0.169)
Tertiary	-0.108	-0.107	-0.103
	(0.168)	(0.168)	(0.168)
lHHincome	0.003	0.009	0.009
	(0.034)	(0.034)	(0.034)
Constant	-3.304	-3.359	-3.359
	(0.409)	(0.409)	(0.409)
Observation	17,368	17,368	17,368
LR test chi2(01)	127.48	124.21	122,79
P>Chibar2	0.000	0.000	0.000
Log-likelihood	-2081.78	-2073.62	-2069.31

Note: ***, **,* denote statistical significance at 1%, 5% and 10% level of significance respectively. Standard errors are reported in parentheses. Models 2 and 3 control MHBs proxied by depression and sleepless, respectively. There are nine provinces in South Africa: Western Cape, Eastern Cape, Northern Cape, Free State, Kwa-Zulu-Natel, North West, Gauteng Mpulaga and Limpopo. The estimated output uses Limpopo as the reference region.

These results indicate that several socio-economic factors play an important role in explaining the smoking decision. However, when MHBs (depression and sleepless) are controlled for in Model 2 and Model 3 respectively, results also indicate a positive and highly significant relationship between MHBs and tobacco consumption. This suggests that MHBs have a significant influence on individual's decision to start smoking. However, probit estimates of smoking decision might be biased as they do to take into consideration selection bias and possible endogeneity issues. Hence, the probit regression outputs are not reliable for making meaningful inferences. This necessitates the adoption of more robust estimation techniques that address both issues and this paper considers the Heckman selection model, Double- hurdle model and the control function approach.

TABLE 3: HECKMAN MODEL, DOUBLE- HURDLE MODEL AND CONTROL FUNCTION MODELS

	Heckman Selection		*Double Hurdle*		*Control Function*	
	Smoking selection	*Smoking intensity*	*Smoking selection*	*Smoking intensity*	*Smoking selection*	*Smoking intensity*
Depressed	0.169***	-0.0033	0.228***	0.0079	0.135*	-0.0220
	(0.0251)	(0.0350)	(0.0568)	(0.0228)	(0.0742)	(0.0304)
Urban	0.142***	0.0350	0.197**	0.0682**	0.251***	0.0201
	(0.0322)	(0.0383)	(0.0820)	(0.0304)	(0.0907)	(0.0383)
Western Cape	-0.172***	-0.0098	-0.359***	-0.0122	-0.338**	-0.0392
	(0.0551)	(0.0571)	(0.137)	(0.0478)	(0.138)	(0.0574)
Eastern Cape	-0.118**	-0.0327	-0.129	-0.0082	-0.257*	0.0302
	(0.0509)	(0.0473)	(0.127)	(0.0406)	(0.155)	(0.0618)
Northern Cape	0.174**	0.0219	0.111	0.0825	0.319	-0.0752
	(0.0675)	(0.0694)	(0.160)	(0.0589)	(0.206)	(0.0886)
Free State	-0.366***	0.105	-0.600***	0.0757	-0.548***	0.0280
	(0.0539)	(0.0745)	(0.136)	(0.0483)	(0.140)	(0.0593)
KwaZulu_Natal	-0.124*	0.0928	-0.242	0.122*	-0.0481	-0.0641
	(0.0667)	(0.0694)	(0.163)	(0.0625)	(0.205)	(0.0885)
North West	-0.0847	0.0411	-0.105	0.0465	-0.0119	-0.0103
	(0.0539)	(0.0519)	(0.123)	(0.0466)	(0.138)	(0.0608)
Gauteng	-0.141**	0.112*	-0.340**	0.0942*	-0.360**	0.112
	(0.0637)	(0.0650)	(0.157)	(0.0570)	(0.159)	(0.0683)
Mpumalanga	-0.228***	0.0426	-0.483***	0.0169	-0.606***	0.102
	(0.0666)	(0.0750)	(0.169)	(0.0625)	(0.188)	(0.0818)
Female	-1.250***	-0.0419	-1.039***	-0.205***	-0.960***	-0.305***
	(0.0263)	(0.187)	(0.0762)	(0.0286)	(0.0921)	(0.0405)
Married	-0.388***	0.0839	0.0425	0.0212	-0.166	0.164**
	(0.0331)	(0.0637)	(0.0758)	(0.0297)	(0.156)	(0.0646)
Unemployed	-0.0273	0.0115	-0.0886	0.0095	0.0062	-0.0563
	(0.0285)	(0.0287)	(0.0642)	(0.0263)	(0.0886)	(0.0357)
Age	0.103***	0.0205	0.0559***	0.0313***	0.113***	-0.0022
	(0.0043)	(0.0162)	(0.0100)	(0.0042)	(0.0385)	(0.0155)
Age-SQD	-0.0011***	-0.0001	-0.0004***	-0.0002***	-0.0009***	0.0001
	(0.0005)	(0.0001)	(0.0001)	(0.0004)	(0.0003)	(0.0001)
Coloured	1.046***	0.0681	0.818***	0.235***	0.607***	0.409***
	(0.0472)	(0.155)	(0.119)	(0.0409)	(0.185)	(0.0745)
Asian_Indian	0.723***	0.105	0.385*	0.275***	0.156	0.396***
	(0.0810)	(0.133)	(0.209)	(0.0763)	(0.258)	(0.106)
White	1.138***	0.617***	0.818***	0.793***	0.532**	1.023***
	(0.0528)	(0.171)	(0.116)	(0.0469)	(0.230)	(0.0939)
Primary	0.0274	0.0674	0.0646	0.0679	0.0693	0.0791
	(0.0659)	(0.0637)	(0.154)	(0.0592)	(0.154)	(0.0691)
Secondary	-0.179***	0.121*	-0.119	0.0548	-0.201	0.164**
	(0.0665)	(0.0684)	(0.156)	(0.0598)	(0.165)	(0.0726)
Matric	-0.424***	0.137	-0.128	0.0317	-0.273	0.192**
	(0.0722)	(0.0936)	(0.167)	(0.0663)	(0.190)	(0.0848)
Tertiary	-0.440***	0.149	-0.0974	0.0655	-0.347	0.259***
	(0.0726)	(0.0944)	(0.164)	(0.0659)	(0.228)	(0.0981)
lHHincome	-0.0384***	0.0529***		0.0629***	-0.0841	0.104***
	(0.0147)	(0.0158)		(0.0138)	(0.0702)	(0.0281)
Own-House	-0.140***	-0.0026	-0.0383	-0.0319	-0.106	0.0402
	(0.0299)	(0.0360)	(0.0709)	(0.0267)	(0.0840)	(0.0331)
Resid					-1.184	1.115***
					(0.910)	(0.361)
Resid*depressed					-0.434**	-0.164*
					(0.219)	(0.0925)
Constant	-1.865***	0.646	-3.311***	0.321**	-3.922***	0.763***
	(0.166)	(0.488)	(0.296)	(0.155)	(0.567)	(0.229)
Observations	20,247	20,247	17,371	3,357	17,368	3,475
Wald chi2	(24)606.88	(24) 606.88	(23)328.47	(24)898.00	(26)326.77	(26)615.59
P>Chi2	0.000	0.000	0.000	0.000	0.000	0.000

Note: ***, **,* denote statistical significance at 1%, 5% and 10% level of significance, respectively. Standard errors are reported in parentheses. There are nine provinces in South Africa: Western Cape, Eastern Cape, Northern Cape, Free State, Kwa-Zulu-Natel, North West, Gauteng Mpulaga and Limpopo. The estimated output uses Limpopo as the reference region.

TABLE 4: HECKMAN MODEL, DOUBLE- HURDLE MODEL AND CONTROL FUNCTION MODELS

	Heckman Selection		*Double Hurdle*		*Control Function*	
	Smoking selection	*Smoking intensity*	*Smoking selection*	*Smoking intensity*	*Smoking selection*	*Smoking Intensity*
Sleepless	0.153***	0.0571	0.373***	0.0838***	-0.240	-0.189
	(0.0354)	(0.0407)	(0.0737)	(0.0315)	(0.365)	(0.177)
Urban	0.141***	0.0353	0.190**	0.0643**	0.0120	0.0639*
	(0.0321)	(0.0383)	(0.0819)	(0.0304)	(0.0876)	(0.0383)
Western Cape	-0.162***	-0.0148	-0.339**	-0.0124	-0.0416	-0.0395
	(0.0551)	(0.0564)	(0.137)	(0.0477)	(0.146)	(0.0618)
Eastern Cape	-0.119**	-0.0334	-0.120	-0.0053	0.129	-0.0735
	(0.0508)	(0.0471)	(0.126)	(0.0406)	(0.133)	(0.0541)
Northern Cape	0.183***	0.0263	0.122	0.0822	0.0680	0.0725
	(0.0675)	(0.0699)	(0.160)	(0.0588)	(0.159)	(0.0722)
Free State	-0.360***	0.0920	-0.596***	0.0716	-0.597***	0.0593
	(0.0538)	(0.0741)	(0.136)	(0.0482)	(0.135)	(0.0582)
KwaZulu_Natal	-0.117*	0.0877	-0.235	0.119*	-0.468***	0.0802
	(0.0666)	(0.0689)	(0.163)	(0.0624)	(0.169)	(0.0766)
North West	-0.0828	0.0366	-0.102	0.0438	-0.313**	0.0660
	(0.0539)	(0.0516)	(0.123)	(0.0465)	(0.129)	(0.0574)
Gauteng	-0.147**	0.105	-0.366**	0.0882	-0.657***	0.0980
	(0.0637)	(0.0651)	(0.158)	(0.0570)	(0.167)	(0.0713)
Mpumalanga	-0.235***	0.0344	-0.501***	0.0135	-0.545***	0.0098
	(0.0666)	(0.0754)	(0.169)	(0.0624)	(0.168)	(0.0757)
Female	-1.249***	-0.0769	-1.047***	-0.209***	-1.237***	-0.239***
	(0.0263)	(0.189)	(0.0763)	(0.0285)	(0.0867)	(0.0374)
Married	-0.395***	0.0741	0.0328	0.0206	0.133*	0.0023
	(0.0330)	(0.0650)	(0.0757)	(0.0297)	(0.0782)	(0.0356)
Unemployed	-0.0271	0.0081	-0.0962	0.0059	-0.241***	0.0136
	(0.0285)	(0.0286)	(0.0642)	(0.0263)	(0.0726)	(0.0296)
Age	0.105***	0.0231	0.0577***	0.0312***	0.0305***	0.0412***
	(0.0043)	(0.0166)	(0.0100)	(0.0042)	(0.0109)	(0.0052)
AgeSQD	-0.0011***	-0.0001	-0.0004***	-0.0002***	-0.0003***	-0.0003***
	(0.0005)	(0.0001)	(0.0001)	(0.0004)	(0.0001)	(0.0005)
Coloured	1.034***	0.0929	0.795***	0.233***	0.733***	0.244***
	(0.0472)	(0.155)	(0.119)	(0.0409)	(0.119)	(0.0498)
Asian_Indian	0.704***	0.121	0.352*	0.274***	0.237	0.226**
	(0.0811)	(0.131)	(0.209)	(0.0761)	(0.211)	(0.0908)
White	1.120***	0.643***	0.781***	0.788***	0.590***	0.802***
	(0.0528)	(0.171)	(0.115)	(0.0469)	(0.123)	(0.0574)
Primary	0.0318	0.0680	0.0757	0.0669	0.0517	0.0819
	(0.0660)	(0.0635)	(0.154)	(0.0592)	(0.154)	(0.0692)
Secondary	-0.175***	0.119*	-0.111	0.0569	-0.0156	0.104
	(0.0665)	(0.0679)	(0.156)	(0.0597)	(0.157)	(0.0701)
Matric	-0.425***	0.126	-0.121	0.0313	-0.0128	0.0851
	(0.0722)	(0.0938)	(0.168)	(0.0662)	(0.170)	(0.0773)
Tertiary	-0.441***	0.142	-0.0944	0.0685	0.103	0.0738
	(0.0726)	(0.0948)	(0.164)	(0.0658)	(0.172)	(0.0778)
lHHincome	-0.0413***	0.0519***		0.0630***	0.0952**	0.0318**
	(0.0146)	(0.0159)		(0.0138)	(0.0378)	(0.0160)
House_dwelling	-0.139***	-0.0041	-0.0372	-0.0291	0.0208	-0.0088
	(0.0298)	(0.0360)	(0.0710)	(0.0267)	(0.0729)	(0.0290)
Resid					1.548***	-0.0109
					(0.260)	(0.107)
Resid*sleepless					-0.531	-0.233
					(0.327)	(0.156)
Constant	-1.840***	0.706	-3.286***	0.320**	-1.469***	0.286
	(0.165)	(0.490)	(0.295)	(0.155)	(0.498)	(0.215)
Observations	20,247	20,247	17,371	3,357	17,368	3,475
Wald chi2	(24)613.88	(24)613.88	(24)332.19	(24)906.66	(26)348.97	(26)608.82
P>Chi2	0.000	0.000	0.000	0.000	0.000	0.000

Note: ***, **,* denote statistical significance at 1%, 5% and 10% level of significance respectively. Standard errors are reported in parentheses. There are nine provinces in South Africa: Western Cape, Eastern Cape, Northern Cape, Free State, Kwa-Zulu-Natel, North West, Gauteng Mpulaga and Limpopo. The estimated output uses Limpopo as the reference region.

Table 3 displays the results of Heckman model, Double- hurdle model and Control function models. The log-likelihood information provided indicates the fitness of the models. Specifically, the log-likelihood, Wald test and the likelihood ratio (LR) tests score mall probability values; ensuring the overall goodness of fit of all the models considered.

Across models, it appears that some factors have an influence on the participation equation (smoking selection) not in the outcome: equation (smoking intensity).

The smoking selection estimates in the Heckman selection use a standard probit function that ignores the outcome equation. When selection bias is controlled for, Heckman selection (column 1) indicates that mental health behaviours proxied by depression in table 3 and sleepless in table 4 and control variables such as urban areas, gender, age, age squared, race (black being the reference

category), married, Secondary, Matric, Tertiary, household income, owning house, as well as geographical location have significant effects on the selection equation; indicating that they are important drivers of the smoking participation. Whereas the estimates for the intensity equation (colum 2) portray a different conclusion from the selection model. The coefficients become insignificant except race (white population), secondary and household income. This simply shows that the determinants of smoking are not the same as the ones that control the intensity of tobacco consumption. The double hurdle model indicates that MHBs, urban, female, age, age squared, race and provinces are vital factors in determining the frequency of smoking.

In the double hurdle model, the smoking selection results indicate that most of these variables have affected smoking participation owing to their statistical significance. These include depressed, sleepless, urban location, gender, age, age squared, race (black being the reference category), and provincial location (Western Cape, Free State, Gauteng, and Mpumalanga). Marital and employment status, residing in Eastern Cape, KwaZulu Natal and Northwest, educational status as well as house ownership do not significantly predict tobacco use (participation). Consistent with the Heckman selection, the first hurdle estimates suggest that individuals that suffer from MHBs are more likely to smoke and females are less likely to smoke than males. In addition, being in the urban area also leads to a higher probability of an individual to become a regular smoker. This might be attributed to the exposure to all the publicity and image portrayed about "coolness" of tobacco smoking in the cities through television and peers. Furthermore, age and age squared are significant in participation and consumption equations. The coefficient of age is positive and highly significant, while age squared is significant with a negative sign. This means that the younger generation is more likely to be regular smokers as compared to the older cohort, simply because older individuals have smoked more in their lives and better understand the health risks attached to smoking, in line with lifecycle pattern in smoking behaviour (Kerr, D.,2004; Aristei et al., 2005).

While the selection bias can be mitigated in the Heckman selection and double hurdle models, the estimated effects of MHBs (displayed in table 3 and 4 respectively) on smoking might suffer from endogeneity. The control function estimates address the potential endogeneity of the hypothesized relationship between MHBs and tobacco use. The results from the control function frequency estimates (column 6) indicate that being a female individual, race, being a Gauteng resident and all the educational variables except primary education and income level are significant determinants of smoking intensity. The levels of education (secondary, matric and tertiary) in table 3 are significant, as opposed to both the Heckman selection and double hurdle results before the controlling for endogeneity. The coefficients of these variables signal that people that attain these levels of education are more likely to smoke more. This might be because of the addictive nature of tobacco use since even though individuals gain more knowledge and awareness knowledge on the risks of smoking as they further their studies, they continue to smoke. The significance of the coefficients of age and age squared variable is in support of previous studies such as Adda and Lechene (2001) that show the possibility of an inverted-U shape relation between age and tobacco consumption. An individual's trade-off decision to smoke is likely derived from the present utility of smoking and death risk increase by age. Hence the coefficient of younger age is higher than the old age smokers.

However, the endogeneity bias appears rather marginal for both smoking selection and smoking intensity when MHBs is proxied by sleepless and only for smoking intensity when depressed is used to measure MHBs; owing to the fact that extra variables (resididuals and interactedresiduals with MBHs) display statistically insignificant or weakly significant (10% significance level) coefficients. This simply indicates the limited bias in the estimates of the first two models (Heckman selection and double hurdle) and hence the validity of their results to infer policy recommendation.

5. Conclusion and policy recommendations

This study investigates the relationship between MHBs and tobacco consumption using the Heckman selection, Double-hurdle and control function models. Firstly, the main explanatory variable mental health behaviours showed positive and highly significant coefficients, implying that people tend to smoke when they have mental health challenges. MHBs appear to be a significant driver of the smoking decision (according to the Heckman selection model) as well as smoking intensity based on Double hurdle model. In terms of the residential area, results illustrate that leaving in urban areas does contribute significantly to the decision to smoke or not (participation equation) but not in the intensity or the amount of tobacco smoked. Age was also a significant factor in determining tobacco use with results showing that as people get older, so does their smoking, which could largely be explained by the fact that human health status deteriorates as we

grow older. In terms of the race, blacks were least likely to be regular smokers, possibly signifying a protective factor(s) against tobacco use in the black community, or the connotations associated with tobacco smoking within the black culture and possibly high levels of unemployment population that almost makes it impossible to support the habit. The estimates for coloured and white are positive with highly statistically significant coefficients as compared to their counterparts (black race). These findings are in support of studies conducted prior (Rodriguez et al., 1999) with evidence showing the association between being unemployed and in use of any substance but with dissimilarities across population groups in terms of socio-demographic factors.

Results also indicate that the proportion of females that are regular smokers are less than that of males. This implies that females are less likely to smoke as compared to their male counterparts, this is consistent with the analysis conducted by Boden et al. (2010) and (McGue et al., 2000). However, there is an evidence that marital status is very important in explaining the frequency of smoking, as shown by the positive relationship between being married and tobacco consumption. Specifically, in comparison to unmarried counterparts, married individuals smoke more according to the control function model. These results are likely to mirror the stereotypical norms of males concerning smoking. Likewise, people living in urban areas are more likely to smoke compared to rural dwellers; non-African communities have a higher probability of smoking and men have a higher smoking frequency rate than females.

Furthermore, the findings report a negative and significant relationship between tobacco consumption and education levels (namely secondary, matric and tertiary) but only in the Heckman selection model (column 1) but a positive relationship in all the other models. The results from these variables show that people that attain these levels of education are more likely to smoke, meaning that even when individuals further their studies, and more knowledge on the risks of smoking and they continue to smoke. Thus, the higher the level of education, the more likely an individual smokes more cigarettes. This might reflect the addictive nature of tobacco in that even those that seemingly are more aware of the risks of tobacco smoking, actually smoke more or the affordability of tobacco among those who are well educated. The contrasting results of the association between education and smoking is however consistent with the previous literature. Some researchers found that education in its different levels is positively related to tobacco use (Hu and Tsai, 2000; Yu and Abler 2007) while others have reported the negative effect (Decker and Schwartz et al, 2000; Yen 2005).

Over the years, there have been several policies implemented concerning tobacco consumption. In 2019 alone, 136 countries initiated at least one policy that aims to lessen smoking (WHO, 2019). These policies were applied on a national level rather than targeting the affected group. This study provides positive insights into the smoking behaviour of people with MHBs people in South Africa. Particularly, people suffering from mental health behaviour smoke more than those who are not affected. These findings advocate that socio-demographic and mental health features influence smoking behaviours. The outcomes from this study raise important concerns on the trends of tobacco use in South Africa and the accompanying factors leading to its use. Policies and behavioural intervention are recommended to reduce smoking to improve the lives of people suffering from mental health behaviours among South Africans. The available tobacco control policies directed to the general population may not have worked as effectively for people with mental illness. The main focus of the smoke-free policy and tobacco ban should be tailored for to individuals suffering from MHBs as well.

References

Adda, J. and Lechene, V. (2001). Smoking and endogenous mortality: Does heterogeneity in life expectancy explain differences in smoking behavior? SSRN 285273

Aristei, D. and Pieroni, L. (2005). A double-hurdle approach to modeling tobacco consumption in Italy. *Applied Economics, 40*(19):2463-2476.

Baye, F.M. and Fambon, S. (2010), "Parental Literacy and Child Health Production in Cameroon," *African Journal of Economic Policy*, 17(2):99-130.

Blaylock, J.R. and Blisard, W.N. (1993). Women and the demand for alcohol: Estimating participation and consumption. *Journal of Consumer Affairs, 27*(2):319-334.

Boden, J.M., Fergusson, D.M. and Horwood, L.J. (2010). Cigarette smoking and depression: tests of causal linkage using a longitudinal birth cohort. *British Journal of Psychiatry*, 196: 440–46.

Breslau, N., Kilbey, M.M. and Andreski, P. (1993). Nicotine dependence and major depression. New evidence from a prospective investigation. *Archives of General Psychiatry*, 50:31–5.

Breslau, N., Peterson, E., Schultz, L., Chilcoat, H. and Andreski, P. (1998). Major depression and stages of smoking: A longitudinal investigation. *Archives of General Psychiatry*, 55:165–166.

Brown, R.A., Lewinsohn, P.M., Seeley, J.R. and Wagner, E.F. (1996). Cigarette smoking, major depression, and other psychiatric disorders among adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*,35:1602–1610.

Buckley, P.F. (2007). Dual diagnosis of substance abuse and severe mental illness: the scope of the problem. Journal of *Dual Diagnosis*, 3(2):59-62

Challier, B., Chau, N., Predine, R., Choquet, M. and Legras, B. (2000). Associations of family environment and individual factors with tobacco, alcohol and illicit drug use in adolescents. *European Journal of epidemiology, 16*(1):33-42.

Chen, E., Matthews, K.A. and Boyce, W.T. (2002). Socioeconomic differences in children's health: how and why do these relationships change with age? *Psychological bulletin, 128*(2):295.

Choi, W.S., Patten, C.A., Gillin, J.C., Kaplan, R.M. and Pierce, J.P. (1997). Cigarette smoking predicts the development of depressive symptoms among U.S. adolescents. *Annals of Behavioral Medicine*, 19:42–50.

Connell, A.M., Dishion, T.J. and Deater-Deckard, K. (2006).Variable and person-centered approaches to the analysis of early adolescent substance use: Linking peer, family, and intervention effects with developmental trajectories. *Merrill-Palmer Quarterly* (1982):421-448.

Cragg, J. (1971). Some statistical models for limited dependent variables with application to the demand for durable goods, *Econometrica*, 39:829-844.

Dierker, L.C., Avenevoli, S., Stolar, M. and Merikangas, K.R. (2002). Smoking and depression: an examination of mechanisms of comorbidity. *American Journal of Psychiatry*,159:947–53.

Douglas, S. (1998). The duration of the smoking habit. *Economic Inquiry*, 36(1)*:*49-64.

Escobedo, L.G., Reddy, M. and Giovino, G.A. (1998). The relationship between depressive symptoms and cigarette smoking in US adolescents. *Addiction.* 93:433–40.

Fergusson, D.M., Goodwin, R.D. and Horwood, L.J., (2003). Major depression and cigarette smoking: results of a 21-year longitudinal study. *Psychological Medicine, 33*(8):1357-1367.

Fluharty, M., Taylor, A., Grabski, M. and Munafò, M. (2017). The Association of Cigarette Smoking With Depression and Anxiety: A Systematic Review, *Nicotine & Tobacco Research*, 19(1): 3–13,

Frijters, P., Johnston, D.W. and Shields, M.A. (2010). Mental health and labor market participation: Evidence from IV panel data models. IZA Discussion Paper No. 4883.

Garcia, J. and Labeaga, J.M. (1996) Alternative Approaches to Modelling Zero Expenditure: An Application to Spanish Demand for Tobacco. *Oxford Bulletin of Economics and Statistics*, 58: 489-506.

Garen, J. (1984). "The Returns to Schooling: A Selectivity Bias Approach with a Continuous Choice Variable," *Econometrica*, 52(5), 1199-1218.

Goodman, E. and Capitman, J. (2000). Depressive symptoms and cigarette smoking among teens. *Pediatrics*. 106:748–55.

Hagquist, C. (2002). Socioeconomic differences in smoking behavior among adolescents: the role of academic orientation. *Childhood, 7*(4): 467-478.

Hamilton, B.H. and Nickerson, J.A. (2003). Correcting for endogeneity in strategic management research. *Strategic Organization* 1(1): 51–78.

Heckman, J. (1979) Sample selection bias as a specification error, *Econometrica*, 47: 153-161.

Holland, A.C. (2015). Smoking Is a Women's Health Issue Across the Life Cycle. *Nursing for Women's Health, 19*(2):189-193.

Hou, L., Jiang, J., Liu, B., Han, W., Wu, Y., Zou, X., Nasca, P.C., Xue, F., Chen, Y., Zhang, B. and Pang, H. (2015). Smoking and adult glioma: a population-based case-control study in China. *Neuro-oncology*, 18(1):105-113.

Hu, T.W. and Tsai, Y.W. (2000). Cigarette consumption in rural China: survey results from 3 provinces. *American Journal of Public Health*, 90(11):1785.

Jordaan, E.R., Ehrlich, R.I. and Potter, P. (1999). Environmental tobacco smoke exposure in children: household and community determinants. *Archives of Environmental Health: An International Journal, 54*(5): 319-327.

Kang, E.J. and Lee, J.H. (2010). A longitudinal study on the causal association between smoking and depression. *Journal of Preventive Medicine and Public Health, 43*(3):193-204.

Kaplan, S., Nielsen, T.A.S. and Prato, C.G. (2016). Walking, cycling and the urban form: a Heckman selection model of active travel mode and distance by young adolescents. *Transportation Research part D: Transport and Environment*, 44:55-65.

Kassel, J.D., Stroud, L.R. and Paronis, C.A. (2003). Smoking, stress, and negative affect: correlation, causation, and context across stages of smoking. *Psychological Bulletin*, 129:270–304.

Kassel, J.D. and Hankin, B.L. (2006). Smoking and depression. In: Steptoe, A., editor. Depression and Physical Illness. Cambridge University Press; New York, NY, 321-347.

Kendler, K.S., Gardner, C.O. and Prescott, C.A. (2005). Toward a comprehensive developmental model for major depression in women. *Focus*, 3: 83–97.

Kendler, K.S., Neale, M.C. and Maclean, C.J. (1993). Smoking and major depression. A causal analysis. *Archives of General Psychiatry*, 50:36–43.

Kerr, D. (2004). Family transformations and the well-being of children: Recent evidence from Canadian longitudinal data, *Journal of Comparative family studies*, 35(1): 73-90.

Kessler, R.C., Crum, R., Warner, L.A. and Nelson, C.B. (1997). Lifetime occurrence of DSM-III-R alcohol abuse and dependence with other psychiatric disorders in the National Comorbidity Survey. *Archives of General Psychiatry*, 54(4): 313-321.

Khantzian, E.J. (1997). The self-medication hypothesis of substance use disorders: a reconsideration and recent applications. *Harvard Review of Psychiatry*, 4:231–44.

Korhonen, T.1., Broms, U., Varjonen, J., Romanov, K., Koskenvuo, M., Kinnunen, T. and Kaprio, J. (2007). Smoking behavior as a predictor of depression among Finnish men and women: a prospective cohort study of adult twins. *Psychological Medicine, 37(5):705-15.*

Lewis, H.G. (1974). Comments on selectivity biases in wage comparisons. *Journal of Political Economy*, 82 (6):1145-1155.

Lyons, M., Hitsman, B. and Xian, H. (2008). A twin study of smoking, nicotine dependence, and major depression in men. *Nicotine & Tobacco Research*, 10:97–108.

Madden, D. (2008). Sample selection versus two-part models revisited: The case of female smoking and drinking. *Journal of health economics*, *27*(2):300-307.

Mathers, C.D. and Loncar, D. (2006). Projections of global mortality and burden of disease from 2002 to 2030. *PLOS Medicine, 3(*11):e442.

McGue, M., Elkins, L. and Lacono, W.G. (2000). Genetic and environmental influences on adolescent substance use and abuse. *American journal of medical genetic*, 96:671-677.

Milic, M., Gazibara, T., Pekmezovic, T., Kisic, D., Tepavcevic, D., Maric, G. and Popovic, A. (2010) Tobacco smoking and health-related quality of life among university students: Mediating effect of depression. *PLoS ONE* 15(1): e0227042.

Miura, T. (2019). Does time preferences affect smoking behavior? A dynamic panel analysis. *Journal of Behavioral and Experimental Economics*, 78:170-180.

Moshoeshoe, R. (2012). Consumer demand for alcoholic beverages and tobacco in Lesotho: A double-hurdle approach. *Economic Research Southern Africa (ERSA), Working paper*, (315).

Munafo, M.R., Hitsman, B., Rende, R., Metcalfe, C. and Niaura, R. (2008). Effects of progression to cigarette smoking on depressed mood in adolescents: evidence from the National Longitudinal Study of Adolescent Health. *Addiction*, 103:162–71.

Mwabu, G. (2009). The Production of Child Health in Kenya: A Structural Model of Birth Weight, *Journal of African Economies*, 18(2): 212-260.

Newman, C., Henchion, M. and Matthews, A. (2003). A double-hurdle model of Irish household expenditure on prepared meals, *Applied Economics*, 35:1053-1061.

Panday, S., Reddy, S.P., Ruiter, R.A., Bergström, E. and De Vries, H. (2007). Determinants of smoking among adolescents in the Southern Cape-Karoo region, South Africa. *Health promotion international, 22*(3):207-217.

Park, S. and Romer, D. (2007). Associations between smoking and depression in adolescence: an integrative review. *Journal of Korean Academy of Nursing, 37*(2):227-241.

Pasco, J.A., Williams, L.J., Jacka, F.N., Ng, F., Henry, M.J., Nicholson, G.C., Kotowicz, M.A. and Berk, M. (2008). Tobacco smoking as a risk factor for major depressive disorder: a population-based study. *The British Journal of Psychiatry, 193*(4):322-326.

Peltzer, K. (2011). Determinants of exposure to second-hand tobacco smoke (SHS) among current non-smoking in-school adolescents (aged 11–18 years) in South Africa: results from the 2008 GYTS study. *International journal of environmental research and public health, 8*(9):3553-3561.

Peto, R., Lopez, A.D., Boreham, J., Thun, M. and Heath, J.R. C. (2006). Mortality from Smoking in Developed Countries 1950-2000. Oxford University Press, Oxford.

Pfeifer, C. (2012). A note on smoking behavior and health risks taking. *Nordic Journal of Health Economics, 1(2):135-151*.

Pomerleau, J., Gilmore, A., McKee, M., Rose, R. and Haerpfer, C.W. (2004). Determinants of smoking in eight countries of the former Soviet Union: results from the living conditions, lifestyles and health study. *Addiction, 99 (*12):1577-1585.

Pudney, S. (1989) Modelling Individual Choice: The Econometrics of Corners, Kinks and Holes, New York, *Basil Blackwell*.

Ritt-Olson, A., Unger, J. and Valente, T. (2005). Exploring peers as a mediator of the association between depression and smoking in young adolescents. *Substance Use & Misuse,* 77–98.

Rivers, D. and Vuong, Q.H. (1988). Limited Information Estimators and Exogeneity Tests for Simultaneous Probit. *Journal of Econometrics,* 39: 347-366.

Rodriguez, L. A. G. and Ruigomez, A. (1999). Increased risk of irritable bowel syndrome after bacteria gastroenteritis: cohort study. *The BMJ, 318 (*7183):.565-566.

Rohde, P., Kahler, C.W., Lewinsohn, P.M. and Brown, R.A. (2004). Psychiatric disorders, familial factors, and cigarette smoking: II. Associations with progression to daily smoking. *Nicotine & Tobacco Research,* 6:119–32.

Schoretsaniti, S., Filiippidis, F.T., Vardavas, C.I., Dimitrakaki, C., Behrakis, P., Connolly, G. N. and Tountas, Y. (2014). 5-Year trends in the intention to quit smoking amidst the economic crisis and after recently implemented tobacco control measures in Greece. *Addictive behaviors, 39(1):*140-145.

Schwartz, J., Timonen, K.L. and Pekkanen, J. (2000). Respiratory effects of environmental tobacco smoke in a panel study of asthmatic and symptomatic children. *American Journal of respiratory and critical care medicine, 161(3):* 802-806.

Schwiebert, J. (2015). A detailed decomposition for nonlinear econometrics models. *The Journal of Economic Inequality, 13(1):*53-67

Semykina, A. and Wooldridge, J.M. (2010). Estimating Panel Data Models in the Presence of Endogeneity and Selection. *Journal of Econometrics,* 157:375-380.

Sreeramareddy, C.T. and Pradhan, P.M.S. (2015). Prevalence and social determinants of smoking in 15 countries from North Africa, Central and Western Asia, Latin America and Caribbean: secondary data analyses of demographic and health surveys. *PLoS One* 10(7): e0130104.

Swendsen, J.D. and Merikangas, K.R. (2000). The comorbidity of depression and substance use disorders. *Clinical Psychology Review, 20*(2): 173-189.

Tauras, J.A. (2006). Smoke-free air laws, cigarette prices, and adult cigarette demand. *Economic lnquiry, 44,(2):*333-342.

Van Walbeek, C. (2005). The economics of tobacco control in South Africa. PhD Thesis. University of Cape Town

Van Walbeek, C.P (2000). Impact of the Recent Tobacco Excise Tax Increases on Future Government Revenue Potential, Research Release No. 1. Cape Town, South Africa: Applied Fiscal Research Centre, University of Cape Town.

Vellios, N. and van Walbeek, C. (2016). Determinants of regular smoking onset in South Africa using duration analysis. *BMJ open*, *6*(7): e011076.

Wahl, S.K., Turner, L.R., Mermelstein, R.J. and Flay, B.R. (2005). Adolescents' smoking expectancies: psychometric properties and prediction of behavior change. *Nicotine & Tobacco Research,* 7:613–23.

Wang, M.Q., Fitzhugh, E.C., Turner, L.F.U.Q. and Westerfield R.C. (1996). Association of depressive symptoms and school adolescents' smoking: a cross-lagged analysis. *Psychological Reports,* 79:127–30.

Weinstein, S.M., Mermelstein, R., Shiffman, S. and Flay, B. (2008). Mood variability and cigarette smoking escalation among adolescents. *Psychology of Addictive Behaviors* 22:504–13.

West, R. (2017). Tobacco smoking: Health impact, prevalence, correlates and interventions. *Psychology and Health*, 32(8): 1018-1036.

Whitbeck, L.B., Yu, M., McChargue, D.E. and Crawford, D.M. (2009). Depressive symptoms, gender, and growth in cigarette smoking among indigenous adolescents. *Addictive Behaviors,* 34:421–426.

Windle, M. and Windle, R.C. (2001). Depressive symptoms and cigarette smoking among middle adolescents: prospective associations and intrapersonal and interpersonal influences. *The Journal of Consulting and Clinical Psychology*, 69:215–26.

Wodjao, T.B. (2007). A Double-Hurdle Model of Computer and Internet Use in American Households.

Wooldridge, J.M. (2010). Econometric Analysis of Cross Section and Panel Data. *MIT Press: Cambridge, MA.*

World Health Organization. (1997). Tobacco or health: A global status report. Geneva: World Health Organization.

World Health Organization. (2015). WHO report on the global tobacco epidemic 2015: raising taxes on tobacco. World Health Organization. *ISBN* 978-92-4-150912-1.

Wu, L.T. and Anthony, J.C. (1999). Tobacco smoking and depressed mood in late childhood and early adolescence.

Xu, X., Wong, S.C., Zhu, F., Pei, X., Huang, H. and Liu, Y. (2017). A Heckman selection model for the safety analysis of signalized intersections. *PLoS ONE,* 12(7):e0181544-e0181544.

Yen, S.T. (2005). Zero observations and gender differences in cigarette consumption, *Applied Economics*, 37:1839–1849

Yu, X. and Abler, D. (2008). Family structure, education and cigarette smoking of the adults in China:A double-hurdle model. *Penn State University, University Park,PA.*