



## Some Shocking Stories: The Behavioral Response Of Cancer Mortality To Spikes In Alcohol And Tobacco Consumption

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### ABSTRACT

Tobacco and alcohol have long been acknowledged as carcinogens holding a critical role in the progression of a various cancers. Identifying the degree to which tobacco and alcohol can impact cancer mortality is necessary to developing effective public health strategies and mitigating risks with preventative measures. The goal of this study is to characterize the behavior of cancer mortality in response to shocks in alcohol and tobacco consumption utilizing aggregate U.S. data. This is the first study of its kind to examine the intertemporal relationship between cancer mortality and its determining factors within a dynamic system. Our results indicate cancer mortality displays persistence and its path dependency varies considerably between the shock factors. An unexpected shock to alcohol consumption results in cancer mortality taking about 17 years to return to its pre-shock level, whereas tobacco consumption shocks recover the original cancer mortality level in about 10 years. Alcohol has a more dominant effect on cancer mortality regardless of time dimension. As a result, policies that have been previously emphasized toward mitigating tobacco consumption may prove prudent in addressing alcohol as a public health concern with respect to cancer mortality.

### KEYWORDS

Cancer mortality, tobacco and alcohol consumption, temporal dynamics, long-run relationship, vector error-correction model, impulse response.

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## INTRODUCTION

The World Health Organization indicates alcohol and tobacco to be significant behavioral contributors to cancer risk (WHO, 2018). Alcohol, as classified by the National Cancer Institute, is a carcinogen capable of detrimental health effects including immune susceptibility and fatality (NIH, 2018). Research conducted by the International Agency for Research on Cancer states alcohol is causally linked to a multitude of cancers, including within the breast, pharynx, liver and colon (IARC, 2010). Multiple studies have also identified a dose-response relationship between alcohol and cancer, in which higher volumes of alcohol consumed may increase the risk of cancer (Kirpalani, 2017). Other epidemiological findings introduce the variance of alcohol intake across different factors. Meta-analyses of cancer data and alcohol intake demonstrate that alcohol's immunosuppressive attributes are responsible for increased risk across multiple cancers, and such is mediated by the amount of alcohol consumed (Bagnardi et. al., 2001). The dose-response relationship is easily highlighted across most cancer types, as studies found that increased intake per week is associated with an 18% increase in colon cancer risk and a 19% increase in rectal cancer risk. Whereas, a daily increase of intake is associated to 20% increase in both oral cavity and pharynx cancer risk. The frequency of intake also poses potential risk, in which a positive linear relationship was suggested with intake frequency (of one additional standard drink/day) and breast cancer risk (increase by 2%) (de Menezes, et. al., 2013). Alcohol consumption is therefore a threat in increasing cancer risk and is multi-faceted in its casualties. This is of significant interest for cancer research and is often debated upon for preventative measures in health policies.

As noted by the National Cancer Institute, tobacco is a leading cause of cancer and cancer deaths (NIH, 2017). While tobacco itself

is not considered a carcinogen, smoking releases carcinogenic chemicals capable of promoting disease and cancers. Tobacco-related research suggests that its content may alter/genetically mutate the DNA of bodily cells, hence promoting proliferative or suboptimal performance of cellular function (Desrichard et. al., 2018). This is confirmed with the overexpression of different cancer-associated receptors present on the cells of tobacco smokers which may suggest an increased risk (Peterson et. al., 2020). In regards to long-term risks, tobacco use has caused significant detriments in lung, pharynx, larynx, liver, stomach, pancreas health and so on (American Cancer Society, 2018). Epidemiological studies not only identify the causal relationship between tobacco and lung cancer, but also introduce increased cancer risk for frequent tobacco users, where stronger probabilities of cancer exist for the lung, laryngeal, pharyngeal, oral and upper digestive regions (Gandini et. al., 2008). Tobacco is therefore of interest due to its versatility of detrimental health effects.

It is evident that in both cases of alcohol and tobacco, health effects and mortality depend on its consumption. Research conducted by Jiang et al, published in 2018, identify that altering the consumption of alcohol and tobacco per capita can affect population mortality in alcohol-related diseases such as liver cirrhosis and heart disease, but such form of research has not been conducted heavily in cancer. They aim to address a seeming pattern on cancer mortality, with particular emphasis to its length given tobacco and alcohol use in the Australian population. Their research reveals that a relationship exists between decreasing both population-level drinking and smoking with cancer mortality reduction. This suggests that alterations in health policy regarding tobacco and alcohol use may indeed have an impact on cancer mortality (Jiang et. al., 2018).

The purpose of this paper is to further characterize the impact of aggregate alcohol and tobacco consumption on overall cancer risk present in the U.S. population. While Jiang et. al. identifies a correlatory relationship between aggregate cancer and alcohol/tobacco consumption, the statistical analysis is confined to identifying the strength of correlation. This study will go beyond this by diagnosing the nature of the relationship utilizing a dynamic statistical model as applied to the US. This is of particular importance to suggesting directions in American public health policies and further contribute to a growing body of knowledge surrounding cancer mortality.

The contributions of this paper are as follows: (i) to the author's knowledge, this paper offers a seminal attempt at exploring the temporal behaviour cancer mortality in response to alcohol and tobacco consumption over a long time span in the US; (ii) we identify the nature of these relationships from a dynamic perspective as opposed to simply observing correlations; (iii) we illustrate the persistence of the response over different time horizons; (iv) we conduct simulations to observe the response path of cancer mortality to shocks in these causative factors to isolate the impact of policy instruments to combat increases in the cancer mortality rate.

## METHODOLOGY AND MODELLING

We utilize time series modelling techniques appropriate for using with long time spans. These techniques, while well established in the statistical literature need to be combined with policy inference specific to this area of study. As there is a wealth of literature on the technical side, we provide here a summary of the major features of what these techniques provide.

Engle and Granger 1987, demonstrated that once a number of variables (say,  $x_t$  and  $y_t$ ) are found to be cointegrated or share a long-run stable relationship, there always exists a corresponding error-correction representation which implies that changes in the dependent variable are a function of the level of disequilibrium in the cointegrating relationship (captured by the error-correction term) as well as changes in other explanatory variable(s). This is the bedrock model that also allows us to test for causal interactions amongst the variables we include in the model. To illustrate we simplify and use a two factor system.

If we exploit the idea that there may exist co-movements between factors related to cancer mortality, such as alcohol and tobacco consumption, and possibilities that they will trend together in finding a long-run stable equilibrium, by the Granger representation theorem we may posit the following testing relationships which constitutes our vector error-correction model:

$$\begin{pmatrix} \Delta x_t \\ \Delta y_t \end{pmatrix} = \begin{pmatrix} d_{11}(L) & d_{12}(L) \\ d_{21}(L) & d_{22}(L) \end{pmatrix} \begin{pmatrix} \Delta x_t \\ \Delta y_t \end{pmatrix} + \begin{pmatrix} \delta ECT_{t-1} \\ dECT_{t-1} \end{pmatrix} + \begin{pmatrix} c_1 \\ c_2 \end{pmatrix} + \begin{pmatrix} v_{1t} \\ v_{2t} \end{pmatrix} \Delta \quad (1)$$

where  $\Delta$  is a difference operator, ECT refers to the error-correction term derived from long-run cointegrating relationship via the Johansen method,  $c_1$  and  $c_2$  are constants, and  $v_1$  and  $v_2$  are serially-uncorrelated random error terms with mean zero.

Using the VECM as a base model, we use this to conduct several simulations whereby the factor variables are subject to shocks to its underlying variance. Here, we conduct, using a similar representation, generalized impulse response analysis that provide us with a snapshot of how the response of each of

these variables included in the system behave over time, following a shock. This feature allows us to track the behaviour of a single shock in isolation while allowing for the dynamics of the entire system in terms of its interactions. Further, given that the basis of the shock is embedded as part of dynamic system, the results also allow us to observe the path, dependency and persistence of shocks from an illustrative viewpoint.

## DATA AND ESTIMATION RESULTS

Our data set describing cancer mortality (CAN) is deaths per 100,000; ALC is average per capita alcohol consumption in litres per person per year, TOB is total sales of tobacco per adult, per year; and HLX is per capita health expenditure (a control factor). All data are observed annually for the US and covers the period 1938 to 2018. For all transformations, annual percentage growth rate of GDP at market prices based on constant local currency; aggregates are based on constant 2015 U.S. dollars. All data apart, from health expenditure, are sourced from the “Our World in Data” database (<https://ourworldindata.org/>). Health expenditure is sourced from OECD Data (<https://data.oecd.org/>).

Prior to testing for cointegration, we investigated the integrational properties of each of the variables by applying a battery of unit-root testing procedures. These tests verify to what degree the time series under analysis are integrated. We use two tests based on different null hypotheses. The KPSS test due to Kwiatkowski, Phillips, Schmidt and Shin, 1992 is used for testing that the null hypothesis of the series is stationary around a deterministic linear trend. To provide additional evidence, and as a check on the robustness of these tests we performed supplementary tests. Based on augmented Dickey-Fuller and Phillips-Perron tests which are presented in Appendix: Table A1 [see Dickey and Fuller 1981, Perron 1988, Phillips

and Perron, 1988], we could not find any significant evidence that the variables in the vector  $[CAN_t, ALC_t, TOB_t, HLT_t]$ , were not integrated of order one or  $I(1)$ <sup>1</sup>. These results are not surprising given Nelson and Plosser's 1982 findings that most aggregate time series are difference stationary processes.

Given the common integrational properties of these variables, we next proceeded to test for the presence of cointegration in the vector  $[CAN_t, ALC_t, TOB_t, HLT_t]$ . Results of Johansen's LR and trace tests (see Johansen, 1988 and Johansen and Juselius, 1992) are presented in Appendix Table: A2, and indicate that there exists at most one cointegrating relationship since  $r = 0$  is clearly rejected in favour of  $r = 1$ ; but  $r \leq 1$  cannot be rejected by the 95% critical values. Given that there exists  $(n-r)$  common trends within the system, we can conclude that there exist several common trends within the vector of variables. In addition, we tested the long-run restriction that cancer mortality (CAN) was statistically zero or insignificant. In all variations of these tests we could not find any conclusive evidence to suggest that CAN does not belong in the system. The tests appear in the final column as a chi-square statistic being rejected across all version of the model.

The charts illustrate the response path taken by cancer mortality when we independently subject alcohol and tobacco consumption to a once off positive shock. These are displayed in Figures 1A and 1B, for response of cancer mortality from shocks to alcohol and tobacco consumption; and Figures 2A and 2B for the factor shocks related to the joint behaviour of alcohol and tobacco consumption. We find that cancer mortality displays persistence and

<sup>1</sup>Using the appropriate notation, a series  $x_t$  is said to be integrated of order  $d$ , if it has an invertible ARMA representation after being differenced  $d$  times. For example, a stationary series is indicated by  $I(0)$ , whereas a non-stationary series in levels, but stationary in first differences is indicated by  $I(1)$ .

its path dependency varies considerably between the shock factors of alcohol and tobacco consumption. Note that while shocks are independent, the technique itself allows us to treat the system as simultaneous i.e. While we shock a certain factor, say alcohol consumption, the system assumes that other shocks are not in some way “switched off” allowing the dynamics of the system to react as a function of the individual shock.

## DISCUSSION AND POLICY IMPLICATIONS

In the following, we discuss these results in reference to the econometric interdependence between cancer mortality, alcohol and tobacco consumption, controlling for health expenditures. We provide a summary of the main message the empirical analysis revealed.

Through the impulse response function (see Figure 1A and 1B) the chart shows that, following the initial shock, the return to its pre-shock cancer mortality level was longer in alcohol (approximately 17 years) in comparison to the shock in tobacco consumption (10 years) on cancer overall. All results directly identify the importance of alcohol as a causative factor toward influencing cancer mortality, and the weakness of tobacco can be explained through biological, social and public health factors unseen in alcohol consumption.

Figures 2A and 2B illustrate the joint behaviour of alcohol and tobacco shocks on each other. Analyses of the response of tobacco consumption to a shock in alcohol consumption seem to have a pronounced effect (see Figure 2A). On the other hand, tobacco shocks have only a slight impact on alcohol consumption, yet its impact is purely once off and ever decreasing (See Figure 2B). A shock in alcohol consumption demonstrates a significant positive spike in tobacco consumption before returning to pre-shock level. This further supports our findings on the

dominance of alcohol, the minimal effect tobacco consumption has on both alcohol consumption and overall cancer mortality. Alcohol may synergize the relationship between tobacco and tobacco-specific cancers, in which people are more inclined to smoke with alcohol consumption and may be more at risk for lung and oral cancers as a result.

Why do we observe a more significant impact of alcohol consumption on cancer mortality, than when compared to tobacco consumption using the same model? Numerous explanations can be offered though none are definitive or even exhaustive.

The slower recovery time to pre-shock level in cancer mortality can be explained by the presence of chemical compound, acetaldehyde. Acetaldehyde is an ethanol metabolite and classified carcinogen found in both alcohol and tobacco. The high concentration of this toxic substance can have detrimental effects on human health by progressing cancer in tandem with alcohol consumption (Paton, 2005). Studies indicate acetaldehyde can increase cancer-causing cellular behaviors by actively interfering in the DNA replication and repairment of damaged DNA (Kwo et al., 1998). This can result in the proliferation of damaged cells, and/or increase mutations on genes responsible for antitumor responses. This metabolite, on its own, has various evidence to suggest carcinogenic behavior. The genetic polymorphisms in ethanol-degrading enzymes lead to a higher acetaldehyde presence and may be responsible for individual susceptibilities in a variety of alcohol-related diseases and breast cancer (Guo et. al., 2010; Scoccianti et. al., 2014). The toxic effects of acetaldehyde also increase risks of upper respiratory tract, liver and colon cancers (NIH, 2007).

Acetaldehyde has a very pronounced impact with alcohol by affecting metabolism, organ functionality and general decreases in



immunity. The duration of time in which acetaldehyde (from alcohol ingestion) is present in the body may be detrimental to health functionality and consequently increasing cancer risk (National Institute on Alcohol Abuse and Alcoholism, 2007). Studies have shown that larger amounts of alcohol may result in higher concentrations of acetaldehyde in the body/blood, and the added variability in metabolic processes/enzymatic function can impact the rate at which the carcinogenic substance is expelled from the body (Seitz et al., 2007). Acetaldehyde also exists in tobacco, in which it enters the body through the burning and inhalation of tobacco smoke. Though found in both substances, acetaldehyde has a greater impact on alcohol consumption in comparison to tobacco consumption. Studies demonstrating the impact of alcohol consumption on bodily concentration identified that ethanol concentrations over a range of 53 to 33 mM/acetaldehyde can lead to an average of 42.7  $\mu$ M acetaldehyde in the blood. Whereas, cigarette smoke maintains 1  $\mu$ g of acetaldehyde, and its levels are almost undetectable in the blood at moderate consumption (Korsten et. al., 1975). From a public health standpoint, the impact of acetaldehyde post alcohol consumption can lead to detrimental long term cancerous consequences unseen in tobacco. As a result, a longer rebound period is needed post alcohol shock compared to tobacco shock, indicating a serious effect on cancer mortality.

The buoyancy and recovery of tobacco consumption and cancer mortality is approximately 10 years. Aggregate substance sales over an 88-year period (see Figure 3) indicate tobacco sales have steadily decreased overtime while alcohol consumption increased. The importance of consumer advertisements and policies impact the relative sales of each substance, hence contributing to its consumption.

Adjustments from a public policy perspective can therefore mediate how information gets presented to the American public. During the 1980's, more emphasis was placed on tobacco research and health effects in comparison to alcohol. Public policy measures, such as the Surgeon General's overall negative evaluation for identifying risk factors with tobacco, discontinuing cigarette rationing, and adding smoke-free environments to public facilities may have impacted societal tobacco expenditure. The advertising industry, playing to consumer wants, were restricted from promoting tobacco on television and radio effective 1971 (Stratton et al., 2001). This, in tandem with the promotion of tobacco alternatives, such as nicotine patches and e-cigarettes may have contributed to the overall decline of tobacco sales. This trend however, is not noted with alcohol consumption as shown that as tobacco was decreasing past 1978, alcohol consumption steadily inclined.

Overall, there are less legislation and intervention strategies for alcohol in comparison to tobacco. Aside from altering the national drinking age to 21, little national intervention has been conducted to impact consumption and/or public health measures. This is most noted with the continual promotional freedom alcohol maintains over tobacco, where studies showed that exposure to alcohol promotions is causally related to increased alcohol consumption in young adults (Weerasinghe, 2020). Alcohol is an unregulated industry, as noted by how tobacco sales have had to succumb to public health measures, including noted cautions on labels, banned promotional advertisements and so on. Regulations surrounding alcohol are said to be "self-imposed" and "voluntary at the federal level" despite its known carcinogenic implications (American Addiction Centers, 2020). Given this, the intensity of alcohol's impact on cancer mortality can flourish not just on a biological scale, but on a societal scale as well.

## LIMITATIONS AND CONCLUSIONS

Our findings demonstrate the dominance of alcohol on cancer mortality with an emphasis on its comparison to tobacco consumption. Not only is there relevance toward the biological aspects contributing to increased cancer risk, but there is an evident lack of alcohol-reducing behaviors promoted in media. Tobacco has had much reduction in consumption with public promotion of anti-smoking ads and the release of smoke-alternatives. It is recommended that public policy direct attention toward mediating the dominant alcohol factor. To address alcohol consumption as a health risk will require a multi-faceted strategy. Alcohol is present through various age groups and is utilized in multiple settings from celebratory events to at home. Not only this, but alcohol lacks the pleasurable alternatives that tobacco offers, while also being more accessible/user-friendly. As a result, implementation of appropriate promotion and drinking policies is necessary to mediate and control cancer risk.

This study provides empirical analysis and suggests potential policy implications for a growing public health concern. The results clearly emphasize the intensity of alcohol consumption on cancer mortality by using a unique, econometric approach to public health issues. The analysis is not without its limitations and these include further characterizing cancer mortality in reference to other factors. Our study looks at aggregate cancer data which may impact how tobacco and alcohol consumption affect cancer on a biological scale. For example, certain cancers may be more prone to impact on tobacco (ex. Lung cancer and tobacco smoke) than alcohol unnoted in the study. Furthermore, since alcohol consumption is a suggested indicator of cancer mortality, more studies should be conducted on the impact of alcoholic content strengths and amount on cancer mortality (ex. impact differences between beer and spirits) subject to data availability and quality.

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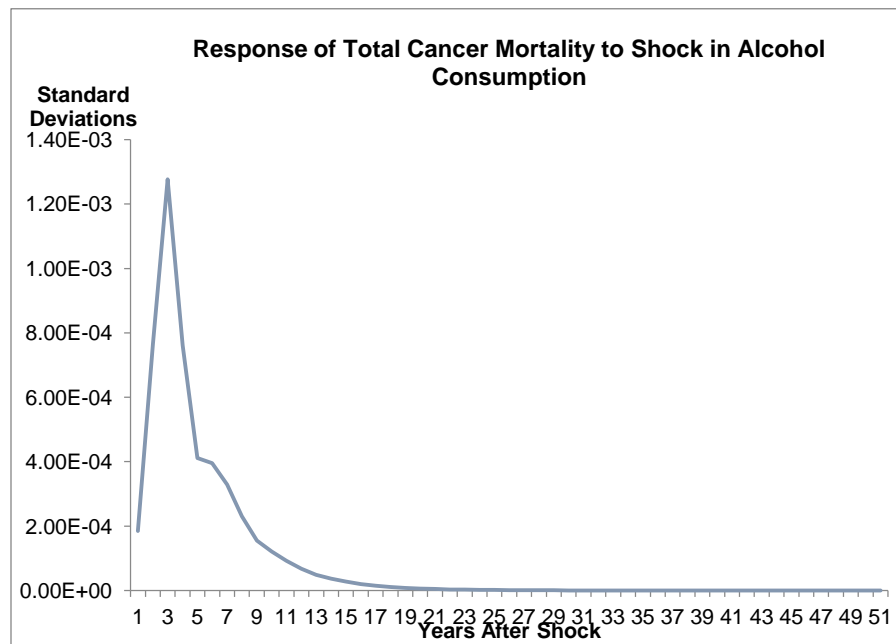
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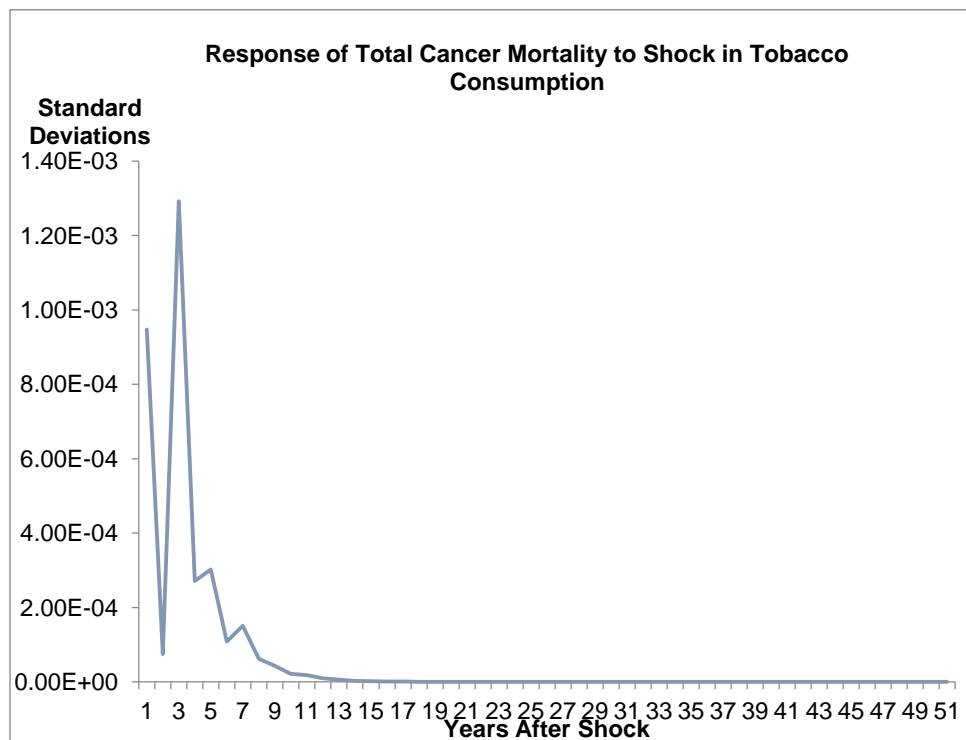


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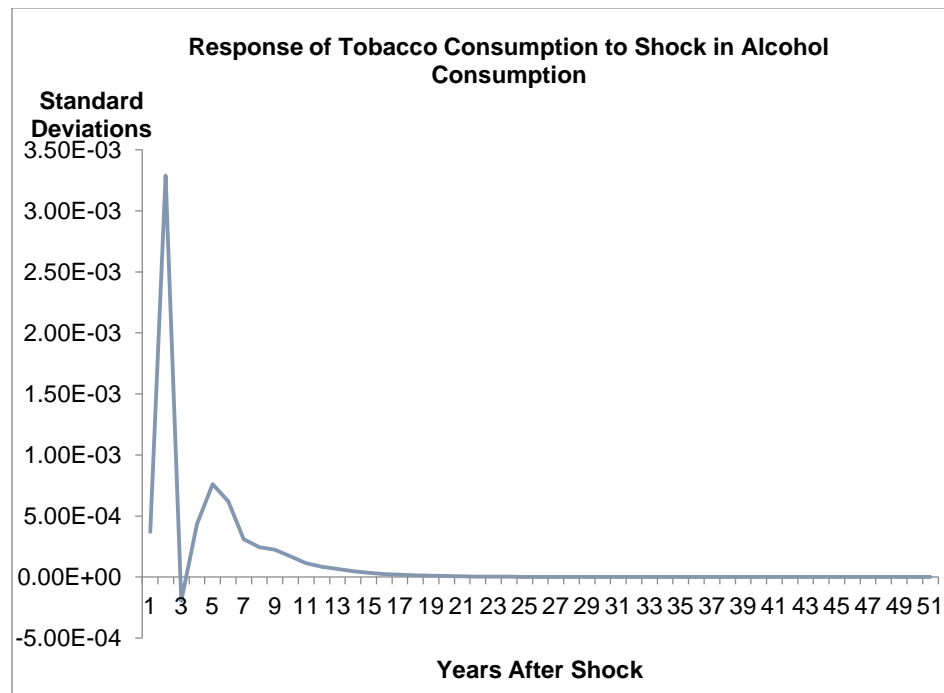
**Figure 1A. Impulse Response of Cancer Mortality to a Shock in Alcohol Consumption**



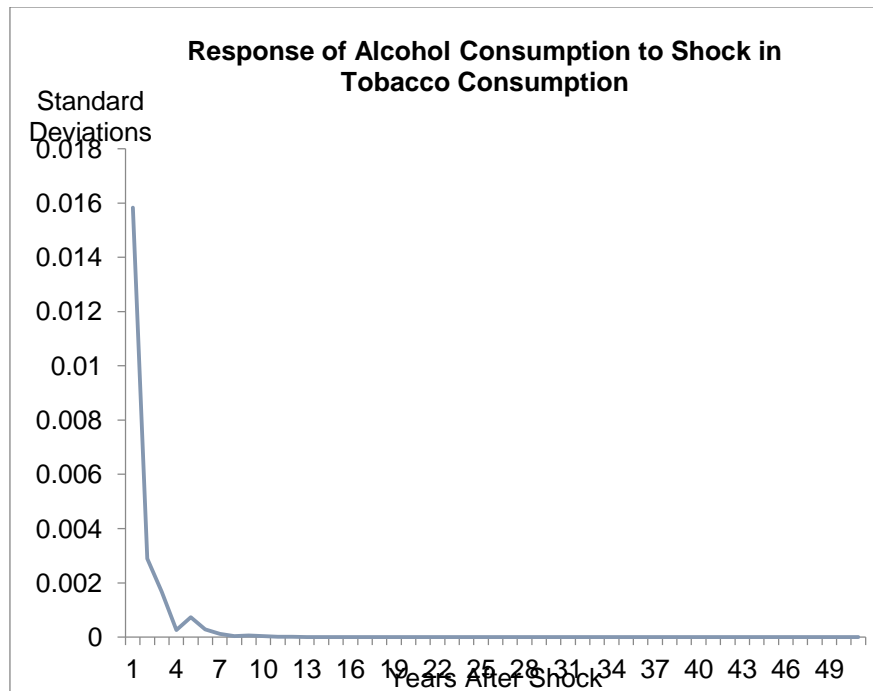
**Figure 1B. Impulse Response of Cancer Mortality to a Shock in Tobacco Consumption**



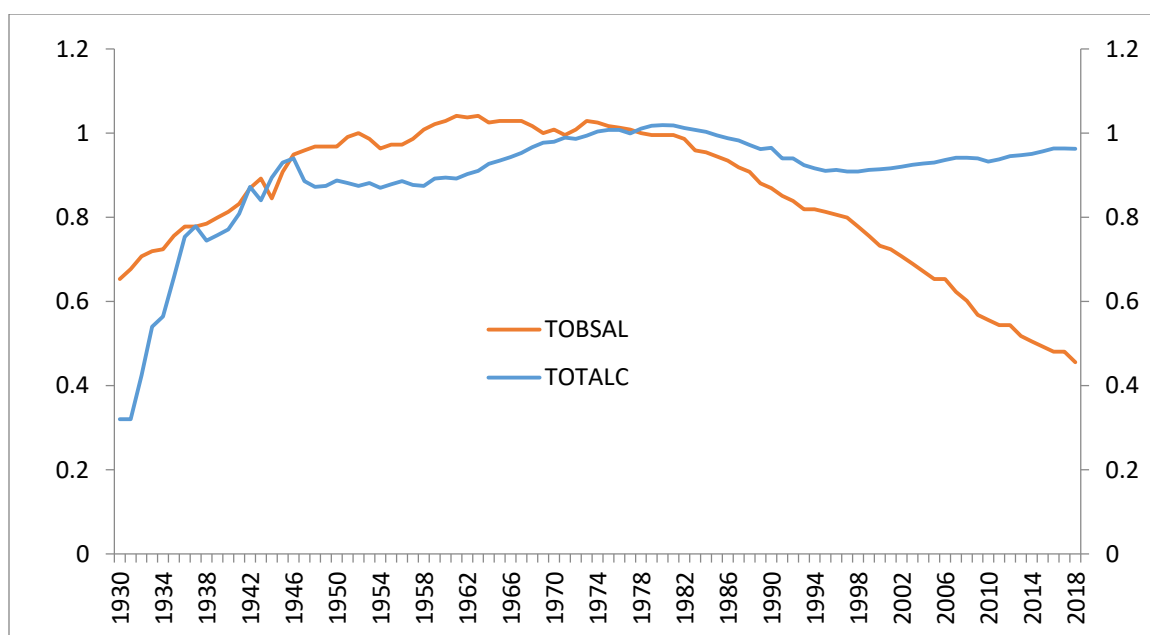
**Figure 2A. Impulse Response of Tobacco Consumption to a Shock in Alcohol Consumption**



**Figure 2B. Impulse Response of Alcohol Consumption to a Shock in Tobacco Consumption**



**Figure 3. Total Alcohol Consumption and Tobacco Sales in the United States: 1930-2018**



Notes: TOBSAL is total tobacco sales; TOTALC is total aggregate alcohol sales. Time plot appears in logs of actual figures observed annually.

**Appendix Table A1. Tests of the Unit Root Hypothesis**

	Aug Dickey-Fuller				Phillips-Perron				
	$\hat{\alpha}_1$	$\hat{\alpha}_2$	$Z(\hat{\alpha})$	$Z(t_{\hat{\alpha}})$	$Z(\hat{\alpha}_1)$	$Z(\hat{\alpha}^*)$	$Z(t_{\hat{\alpha}}^*)$	$Z(\hat{\alpha}_2)$	$Z(\hat{\alpha}_3)$
<i>Levels</i>									
CAN	-1.63	-2.33	-4.14	-2.17	2.07	-5.07	-1.85	4.15*	5.02
ALC	-1.04	-2.79	-2.35	-1.42	2.15	-4.35	-2.29	2.38	3.19
TOB	-1.02	-2.15	-3.77	-0.99	2.79	-7.05**	-1.88	2.13	2.38
HLX	-0.74	-2.06	-0.90	-2.52	2.19	-4.96	-2.63	2.58	4.65
<i>First Differences (<math>\hat{\alpha}</math>)</i>									
$\hat{\alpha}$ CAN	-4.18	-6.54	-8.65	-4.81	6.07	-5.15	-5.09	4.32	5.57
$\hat{\alpha}$ ALC	-3.62	-5.77	-4.47	-3.68	4.54	-6.58	-4.39	3.98	4.02
$\hat{\alpha}$ TOB	-4.53	-5.06	-4.62	-5.69	4.36	-7.25**	-4.20	3.34	4.55
$\hat{\alpha}$ HLX	-3.45	-4.32	-5.95	-4.33	4.34	-8.91	-4.00	3.56	4.12

Notes: The sample consists of logged-annual time-series observations (1938-2019). The optimal lag used for conducting the Augmented Dickey-Fuller test statistic was selected based on an optimal criteria [Akaike's Final Prediction Error (FPE)], using a range of lags. The truncation lag parameter  $l$  used for the Phillips-Perron tests was selected using a window choice of  $w(s, l) = 1 - [s/(l + 1)]$  where the order is the highest significant lag from either the autocorrelation or partial autocorrelation function of the first differenced series. Relevant test equations and related technical descriptions for all unit root testing procedures appear in Appendix: A1. Presented for *levels* tests only: \*\*\*, \*\* and \* indicate significance at the 1%, 5% and 10% levels respectively.



**Appendix Table A2. Multivariate Johansen Tests for Cointegrating Relationships Between Cancer Mortality, Alcohol and Tobacco Consumption and Health Expenditures**

	Optimal Lag	Test Statistic	$\chi^2$ Test of	
$H_0$ and $H_1$	Used in VAR	Max Eigenvalue	Trace	Restriction
No Intercepts; No Trends				
$r = 0 \quad r > 0$	2	40.53**	70.69**	9.27 <sup>†</sup> [12.03]
$r \leq 1 \quad r = 2$		16.44	30.15	
$r = 2 \quad r > 3$		13.42	13.72	
$r \leq 3 \quad r = 4$		0.31	0.29	
Restricted Intercepts; No Trends				
$r = 0 \quad r > 0$	2	45.44**	67.37**	15.13 <sup>†</sup> [11.22]
$r \leq 1 \quad r = 2$		19.14	17.21	
$r = 2 \quad r > 3$		15.64	7.13	
$r \leq 3 \quad r = 4$		4.42	4.47	
Unrestricted Intercepts; No Trends				
$r = 0 \quad r > 0$	1	47.46**	64.55**	11.14 <sup>†</sup> [9.58]
$r \leq 1 \quad r = 2$		10.01	9.01	
$r = 2 \quad r > 3$		9.42	11.37	
$r \leq 3 \quad r = 4$		4.55	4.01	
Unrestricted Intercepts; Restricted Trends				
$r = 0 \quad r > 0$	2	45.16**	62.33**	7.73 <sup>†</sup> [15.64]
$r \leq 1 \quad r = 2$		19.77	3.01	
$r = 2 \quad r > 3$		12.04	14.73	
$r \leq 3 \quad r = 4$		5.25	5.15	
Unrestricted Intercepts; Unrestricted Trends				
$r = 0 \quad r > 0$	1	48.57**	68.78**	13.11 <sup>†</sup> [11.94]

$r \leq 1$	$r = 2$	7.54	11.24
$r = 2$	$r > 3$	15.64	20.37
$r \leq 3$	$r = 4$	5.77	5.25

Notes:  $r$  indicates the number of cointegrating relationships. The optimal lag structure for the VAR was selected by minimising the Akaike's FPE criteria. All estimated coefficients of the cointegrating vectors are available on request. Critical values are sourced from Johansen and Juselius (1990). \*\* indicates rejection at the 95% critical values.  $\dagger$  and  $\ddagger$  indicate significance at the 1 and 5 per cent levels, associated with a chi-square statistic testing the restriction that  $ALC [TOB]$  in the cointegrating vector  $[CAN, ALC, TOB, HLT]$  is equivalent to zero statistically.

**Appendix Table A3. Summary of Diagnostic Tests for Equations used in Vector Error Correction Model**

Country/Eq	Serial Correlation		Heteroskedasticity		Funct Form	Normality
	LM(1)	LM(2)	Het	ARCH	RESET	JB
☐CAN	0.55	1.16	0.16	0.33	4.16 (2,15)	1.13
☐ALC	0.78	1.33	1.36	0.06	0.56 (3,12)	1.17
☐TOB	1.42	1.55	0.77	1.09	3.22 (3,12)	2.14
☐HLX	0.74	2.12	1.27	1.27	4.67 (3,12)	2.55

Notes: Distributional properties of diagnostics are respectively: LM(1) and LM(2) as  $\chi^2(1)$  and  $\chi^2(2)$  testing for the null of no first and no fourth order serial correlation amongst the residuals; Het: a  $\chi^2(1)$  test based on regression of squared residuals on a constant and squares of the fitted values; a  $\chi^2(1)$  test for first-order ARCH effects; Ramsey's REgression Specification Error (F) Test with (n, m) degrees of freedom; and the Jarque-Bera  $\chi^2(2)$  LM test for normality of residuals.