A Response to Two Papers Critiquing the Total Consumption Model by Kari Poikolainen Response

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Population Alcohol Consumption as a Predictor of Alcohol-Specific Deaths: A Time-Series Analysis of Aggregate Data

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ABSTRACT

Aims: The study examines whether the number of alcohol-specific deaths can be predicted by population total and/or beverage-specific alcohol consumption and if, how precisely. The data are annual series of spirits, wine, beer and total consumption and alcohol-specific deaths in Finland in the years 1969-2015.

Methods: We specify a ARDL (Auto Regressive Distributed Lags) model with cointegrated variables, to be used in prediction. In our model the number of alcohol specific deaths is the response variable, and log of spirits consumption and log of non-spirits consumption, are the explanatory variables. The response variable has one added annual lag and the explanatory variables have both four annual added lags in the model.

Results: In our data alcohol-specific deaths, log of spirits and log of non-spirits consumption are significantly cointegrated. The precision of the estimated model is good. The prediction results include prediction of the 2008 downturn in alcohol deaths, using the data from the years 1969 – 2004, forecasting the as yet unknown 2016 alcohol deaths on the basis of known values of alcohol consumption up to 2016, and forecasts of future (2017-2020) alcohol deaths from 2016 on. Forecasted effects of a proposed Finnish alcohol policy change, leading to six percent total consumption increase, are estimated.

Conclusions: The number of alcohol-specific deaths can be predicted with an appropriate time-series regression model on the basis of population consumption. It is important to consider also beverage type because of the improved predictive power. The model is useful in an evaluation of proposed alcohol policy changes.

INTRODUCTION

It is commonly thought that total consumption of alcohol in a country determines how much harm is caused by drinking alcoholic beverages. Inspection of graphs showing per capita alcohol consumption and various harms over calendar time by the naked eye may suggest correspondence. This can be misleading because successive observations are not independent and trends and other forms of non-stationarity may lead to apparent but spurious associations. Special time-series methods are needed to control for autocorrelation and non-stationarity.

In addition to total alcohol consumption, type of beverage may also play a role. In a pooled cross-sectional time-series analysis, spirits consumption was found to associate as strongly as total alcohol consumption with cirrhosis mortality in five countries, Australia, Canada, New Zealand, the United Kingdom and the United States in 1953-1993, while wine and beer were not significant (Kerr et al., 2000). Here and later in our article, spirits denote distilled, that is hard, liquor. In another cross-sectional time-series analysis, higher spirits consumption was found to associate more strongly than other beverages with higher cirrhosis, head and neck cancer and ischemic heart disease (IHD) mortality in 48 states of USA in 1957-2002, while higher beer and wine consumption were found to associate with lower ischemic heart disease mortality (Kerr and Ye, 2011). Alcohol-related disease mortality declined by 7.0% after a 1990 tax increase for spirits and beer. On the other hand, a spirits-only tax increase was not significantly associated with mortality, and small tax decreases on beer between 1996 and 2006 had no measurable effect on mortality (Delcher et al., 2012). Thus, beverage type may influence the number of alcohol-related deaths.

The above studies have not examined cointegration among the response and explanatory series. In this article, we model the relation between total and beverage-specific alcohol consumption and alcohol-specific mortality with a number of lagged variables. We show that the presence of cointegration gives us increased predictive precision.

The data pertains to Finland from the years 1969-2015/2016. Alcohol-specific mortality contains causes of death with a mention of alcohol in the diagnosis name.

MATERIALS AND METHODS

Data

We studied alcohol-specific deaths, defined as cause-of-death group 41 in Finland. The series consisted of 46 consecutive years, 1969-2015. From 1969 to 1986 these deaths comprised ICD-8 codes 291, 303, 5710, 577(only males) and E860. From 1987 to 1995, respectively ICD-9 codes 291, 303, 3050, 3575, 4255,5353, 5710-5713,5770D-5770F,5771C-5771D,7607A,7795A and E851. From 1996 and later the ICD-10 codes were F10, G312, G4051, G621, G721, I426, K292, K70, K860, K852, 0354, P043, Q860 and X45. These are underlying causes of deaths, that is disease or injury that initiated the train of morbid events. Contributory causes of death are not included, because their causal role is unclear. Data on the aforementioned deaths were extracted from the registers of Statistics Finland (Suomen virallinen tilasto 2016) and consumption in absolute (100%) alcohol, consumed as various beverages, were obtained from the register in the National Institute for Health and Welfare (www.thl.fi) in Finland. Alcohol-specific mortality showed a clear increasing trend in Finland from 1969 to 1990. The rate of increase became higher in 1987 when the ICD-9 cause-of-death classification, containing more codes with alcohol etiology than the former one,

was first applied. The peak was reached in 2007. The ICD-10 cause-of-death classification, containing again more codes with alcohol etiology than the former one, was first applied in 1995, the year when Finland joined the European Union.

The following variables were studied (variable names in parentheses): number of alcohol-specific deaths (*alcdeath*) per 100 000 person-years in population aged 15 years or older, total annual alcohol consumption per capita in liters of absolute alcohol (*totalcons*), distilled spirits, beer and wine consumption per capita in liters of absolute alcohol in population aged 15 years or older (*spirits, beers, wines*) and beers and wines together (*non-spirits*). A derived variable, the ratio of spirits and total consumption in percent *(spiritspct)* was also explored. All these variables were also examined in natural logs. Figure 1 gives an overview of the annual development of the main variables over the period 1969-2015.

***********Insert Figure 1 about here***********************

Methods

A visual examination shows that none of the series are trend-stationary; the type of trend varies from series to series. It is also obvious that fixed time trends are not an appropriate description of the non-stationarity (both because fixed trends lack substantial credibility and because they would require a higher degree polynomial representation). The remaining possibility is that the trends are stochastic. Thus, to set up a regression model we need to eliminate the spurious effects of stochastic trends in the data, to avoid 'spurious regression' (Newbold and Granger, 1974).

We tested all the series (also in logs) for unit roots, i.e. the hypothesis that the trends could be due to random walks. We first applied augmented Dickey-Fuller unit root tests to the series in

levels (also in logs), at their non-differenced, original values. The tests showed that unit root hypotheses could not be rejected for any series, either with or without the assumption of a linear trend component in the data. Further unit root tests showed that the unit root hypothesis *could* be rejected for the once differenced versions of the series. With further examination of the auto-correlation structures, we inferred that the series could be assumed to be stationary, I(0), in differenced form and thus I(1) in levels.

Cointegration relationships

Informally, we can say that non-stationary time series are cointegrated when they move 'together in time'. Cointegrated series are bound together by an error correcting feedback mechanism, in contrast to a spurious regression where the series which, while actually independent, merely *seem* to move together for some while. More formally cointegration means here that there exist linear combinations of our I(1) series that are I(0). We tested for the cointegration relationships among *alcdeath* and the consumption variables using the Bounds test (Pesaran et al., 2001) and the Johansen trace test (Johansen, 1995).

We found that the three-variable vector {*alcdeath,* log(*spirits*)*,* log(*non-spirits*)} was cointegrated. Indeed, the null hypothesis of no long-run relationship was clearly rejected, with a high significance level (Bounds F-statistic = 9.19, I(1) bound at the 0.01 limit = 5.0, $p<0.01$).

Several other combinations vectors were tested. To name but a few, the vectors {*alcdeath,* log(*spirits*)*,* log(*totalcons*)} and {*alcdeath, totalcons, spiritspct*} were cointegrated. Neither the pairs {*alcdeath,* log(*spirits*)} nor {*alcdeath,* log(*totalcons*)} nor any other pair formed with *alcdeath* or log(*alcdeath*) were cointegrated.

It should be obvious, by substantial reasoning, that the explanatory direction is from consumption variables to alcohol deaths. We furthermore tested the direction by applying Wald tests of Granger-causality to the key variables and came to the same conclusion.

The tests and all estimations in the paper were performed either with EViews (9.5) or STATA (10.0) software.

1st difference model

As an introductory application, we proceed by estimating the concurrent effects of consumption variables on alcohol deaths in first-difference form with ordinary least squares (OLS). The basic linear first difference regression model is of the form

$$\Delta alcdeath_t = \beta_0 + \beta_1 \Delta spirits_t + \beta_2 \Delta totalcons_t + \cdots + \varepsilon_t \qquad (1)$$

with possible additional explanatory variables. The difference operator Δ is defined as $\Delta alcdeath_t = alcdeath_t - alcdeath_{t-1}$ where t refers to an observation year and the error term ε_t is assumed (and later tested) to meet the usual OLS assumptions. The model thus explains year-to-year changes in alcohol deaths by concurrent year-to-year changes in consumption variables. We used also the differences in natural logs, e.g. $\Delta \log(spirits)_t$, instead of differences like $\Delta spirits_t$. Note that we have included a constant in the equation (equivalent to a linear trend in levels). We applied the usual regression diagnostics to the residuals, tested the significance of explanatory variables and assessed the goodness of fit.

The ARDL model – lagged explanatory variables.

It is well known that there is a considerable time lag between heavy consumption and death from alcohol. We thus needed to consider lagged explanatory variables. Given the cointe-

gration of variables and direction of (Granger) causality, we use the ARDL (standing for Auto Regressive Distributed Lags) methodology for our modeling. As indicated by the name, the ARDL models may include lagged values (lags) of both the dependent and the independent variables. In particular, with an underlying ARDL and cointegrated I(1) variables, we can estimate a cointegration or error correcting form for the short run (or year-to year) changes *and* a long-run or equilibrium form of the relationship between the variables.

The basic underlying form of an ARDL model with a response variable *y* and just one explanatory variable *x,* is in levels

$$y_t = \beta_0 + \beta_1 y_{t-1} + \beta_2 y_{t-2} + \cdots + \beta_p y_{t-p} + \alpha_0 x_{t-0} + \alpha_1 x_{t-1} + \cdots + \alpha_q x_{t-q} + \varepsilon_t.$$

The model above is denoted as ARDL(*p,q*) with *p* the number of lags in the response variable and *q* the number of lags in the explanatory variable. A model with two or more explanatory variables is analogous.

Specification of an ARDL -model requires many decisions and judgements but the basic difficulty is in the specification of lag lengths. Using the Bayes Schwartz information criteria for lag lengths and many other specification checks and tools we arrived at an ARDL(*1,4,4*) -model specified in the basic form as

$$y_t = \alpha_0 + \alpha_1 y_{t-1} + \beta_0 x_{1t} + \beta_1 x_{1t-1} + \beta_2 x_{t-2} + \beta_3 x_{1t-3} + \beta_4 x_{1t-4}$$

$$+\gamma_0 x_{2t} + \gamma_1 x_{2t-1} + \gamma_2 x_{2t-2} + \gamma_3 x_{2t-3} + \gamma_4 x_{2t-4} + \varepsilon_t, \qquad (2)$$

where *y* = *alcdeath* with added lag 1 variable, x_1 = log(*spirits*) with lags 0 – 4, and x_2 = log(*non-spirits*), with lags 0 – 4 and $\alpha_i, \beta_j, \gamma_k, \varepsilon_t$ designating the regression coefficients and the error term, respectively.

Some of the choices made in model specification need to be spelled out. First, we decided to use a basic model where the response variable was expressed in levels and the explanatory variables in logs (the level-log specification). For the response variable the choice of the non-logged form was based on the observation that the logged form caused heteroskedasticity in the residuals. Apart from heteroskedasticity there was no essential difference in model performance. For the explanatory variables logging was preferable for at least two reasons: easy interpretation of the coefficients (percent change) and cointegration of spirits and non-spirits with alcohol deaths when in log form. Further, total consumption as an explanatory variable leads to interpretational difficulties once any of its subcomponents (e.g. *spirits*) are also in the model and total consumption alone is an inefficient predictor. This is why we decided to leave total consumption out, given that spirits and non-spirits together cover the information in total consumption.

It is in practice more interesting to examine the error correcting or cointegration form of (2). This representation of the ARDL(1,4,4) model is in our case of the form

$$\Delta y_t = \delta_0 \Delta x_{1t} + \delta_1 \Delta x_{1t-1} + \delta_2 \Delta x_{t-2} + \delta_3 \Delta x_{1t-3} + \theta_0 \Delta x_{2t} + \theta_1 \Delta x_{2t-1} + \theta_2 \Delta x_{2t-2} +$$

$$\theta_3 \Delta x_{2t-3} + \alpha EC_{t-1} + \varepsilon_t \qquad\qquad (3)$$

with y, x_1 and x_2 as defined in (2), δ_i, θ_j being regression coefficients, EC_t the error correction term, (or, the cointegration relationship), α the 'speed' coefficient for the error correction term and ε_t the usual error term. Here the constant is assumed to be 'restricted' to the error correction term. We will not go into further details of the fairly complicated modelling, testing or estimation issues, see (Pesaran et al., 2001)

RESULTS

Estimation and specification results from the concurrent 1st difference models

The basic model of type (1) gives rise to only two feasible regression equations, given in Table1 with estimation results. The models are presented in a transformed semi-logarithmic model, where $\Delta alcdeath$ is replaced by $\Delta \log(alcdeath)$ and $\Delta spirits$ with $\Delta\log(spirits)$. Essentially the same results were obtained without the logarithmic transformation but logarithms are used because of ease of interpretation and uniformity with the ARDL -model. From Table 1 we can see that both spirits and total consumption (in separate models) are significant explanatory variables. Changes in (the log of) spirits predict concurrent changes in alcohol deaths as well as or better than changes in (the log of) total consumption. The residual diagnostics showed no deviation from OLS assumptions. For instance, the Box-Ljung Q-tests for autocorrelated residuals were not significant for any lag up to 24. No other available consumption variable (such as *beers*, *wines, nonspirits, spiritspct*) reached significance or improved explanatory power, either alone or with other variables. Using both spirits and total consumption in the same model as in (1) neither improved fit nor reached significance for either variable. It should be noted that the Root MSE's, estimates of residual standard deviation, are fairly high, 2.13 and 2.19, respectively. This means that predictions/forecasts from the models have fairly large prediction limits.

***************Insert Table 1 about here*********************

To sum up, the models of type (1) establish a statistically significant but weak predictive relationship between concurrent alcohol deaths and either spirits or total consumption.

Estimation results from the ARDL model

The form (2) of the ARDL(1,4,4) model represents the short term annual *changes* in alcohol deaths. Table 2 shows the estimated coefficients of model (2), i.e. the coefficients of lagged $\Delta\log(spirits)$ and $\Delta\log(nonspirits)$ variables, of the lagged error correction variable EC, and the coefficients of the equilibrium equation, together with their standard errors and significance

*********Insert Table 2 about here*****************

It is noteworthy that the error correction term EC_{t-1} is negative, fairly large in absolute value (-0.63) and statistically highly significant. The error correction term EC_{t-1} is the difference be-tween the observed $aldeath_{t-1}$ and its long run (see below) predicted value. EC_{t-1} works as a negative feedback, adjusting shocks in alcohol deaths rapidly (-0.63 meaning in about a year and a half) back towards equilibrium, in turn given by the long run equation

$$alcdeath = -104.35 + 17.44 * \log(spirits) + 67.74 * \log(nonspirits). \qquad (4)$$

The equation (4) describing the long run relationship between the response and the ex-planatory variables, shows that for a given non-spirits consumption, a one percent increase in per capita spirits consumption tends in the long run equilibrium towards an approximate increase of alcohol deaths by 0.174 deaths per 100 000. Similarly, for a given per capita spirits consumption a one percent increase in non-spirits percentage tends to increase alcohol deaths by 0.67 deaths per 100 000. The equilibrium states are not observed but remain theoretical constructs.

Figures 2 and 3 show the fit (observed and predicted values) of the ARDL(1,4,4) model for the levels (Figure 2) and annual changes (Figure 3). The model is clearly and measurably superior to the basic models (1.1 and 1.2) in terms of fit (approximate $R^2 = 0.75, RootMSE = 1.35$).

************* Insert Figures 2 & 3 about here. *********************

Forecasts from the ARDL model

An *ex ante* forecast from 2005 on

Apart from the *ex post* predictions (fits) in Figures 2 and 3, we demonstrate *ex ante* alcohol death forecasts. First, we remind the reader that alcohol deaths increased until about 2007 and have decreased ever since. To test the forecasting capability of the model in *ex ante* forecasting we re-estimated the ARDL(1,4,4) model using only the years 1969 – 2004, and forecast the alcohol deaths for the years 2005 – 2015, using only the 1969-2004 model and the available alcohol consumption numbers for 2005-2015. The result is presented graphically in Figure 4. It can be seen that the model is able to forecast the peak and the decrease following 2007 well.

************** Insert Figure 4 about here ***********************************

A one-step and a policy change forecast for 2016

At the time of the writing the 2016 consumption figures are already available but the number of deaths is yet to be released. Now, using the full model for 1969-2015, we get a forecast of 36.95 deaths per 100.000 (with prediction limits 33.5 - 40.3) for the year 2016 (Table 3 below).

It has been proposed that a future change in alcohol policy in Finland would increase the total consumption by up to 6% by an increased non-spirits consumption (Mäkelä and Österberg, 2016). Let us now assume, counterfactually, a 6% increase having happened in 2016, which implies that non-spirits consumption would have increased from the observed 6.61 litres to 7.11 litres per capita. The model forecast is now 36.90 deaths per 100.000 (with prediction limits 33.4 - 40.4), see Table 3. Above we have seen that the no-policy change no-increase forecast was 36.95. Thus, a one year 6% shock would not matter very much and certainly would not be statistically significant.

Effects of a 6% increase in the near future

Forecasting the future is always subject to great imprecision but the following exercise demonstrates further the effects of proposed changes in consumption. We compare the forecasts of the future five years, 2016 – 2020 under two different assumptions. First, we forecast the future consumption of spirits and non-spirits per capita for the years 2017 – 2020, using standard ARIMA forecasting (technical details not presented). Second, we assume, counterfactually, a permanent level shift of 6% of the total consumption from the level forecast by the ARIMA models, the increases taking place entirely in non-spirits consumption. Finally, we compare the forecasts of alcohol deaths from the ARDL model in the two series. The assumption of a permanent shift is obviously a rather extreme choice, given that temporary shocks are more realistic and tend to fade out, but this is only for comparison. The results are given in Table 3. A note of warning is that the prediction intervals for the years 2017 – 2020 are certainly too narrow. They are calculated conditional on the ARIMA forecasts of spirits and non-spirits, the forecast error in these not being accounted for. As seen from Table 3, the assumed consumption shift tends to increase the number of alcohol deaths, but very slowly in the first years. The increases are not likely to be statistically significant, but the trend is clear.

*********Insert Table 3 about here******************

DISCUSSION

Strengths. Earlier studies of aggregate alcohol consumption have applied differencing and Box-Jenkins (ARIMA) modeling. We showed that cointegration analysis and autoregressive distributed lags models are useful in alcohol studies. In particular, a good fit to the data (or relatively precise *ex post* point predictions) were obtained by the ARDL(1,4,4) model. The existence of clear and significant cointegrating relations is in itself important, and enhances the credibility of a stable association between consumption, beverage type and alcohol deaths.

In forecasting, we were able to show that the model is able to predict *ex ante* the downward turn in alcohol deaths in 2007. Some short-term forecasts were even slightly surprising. A 6% increase in non-spirits consumption did not predict any increase in alcohol deaths, albeit in the context of a particular year (2016). However, a counterfactual experiment of a permanent 6% increase in non-spirits consumption showed a clear, slow increase in alcohol deaths.

Limitations. One should note that our data basis for time-series analysis is annual aggregate data and that therefore the number of observations available (N=47) is limited. Much of the inference would improve on the one hand on having longer series than ours – although ours is as long as that in the earlier studies mentioned in the introduction - on the other hand 47 years can be a (too) long period to be stable from an epidemiological or societal point of view.

We did not include unrecorded consumption in our data for two reasons. First, estimates on unrecorded alcohol consumption are less reliable than recoded consumption. The latter is based on the sales statistics while the former is compiled from several sources, and is based

mainly on telephone interviews. Secondly, annual estimates on unrecorded consumption showed a slower increasing trend than recorded consumption in 1965-1995 and no clear trend change after that (Yearbook of alcohol and drug statistics 2015, figure 2). Thirdly, we did not have data on the consumption of industrial alcohol products, not aimed for human consumption, and containing over 90 % of alcohol. These substances were popular in the 1960's and 1970's in Finland and frequently detected in alcohol-specific deaths at that time (Poikolainen, 1977). Thus, the importance of spirits seems to be somewhat underestimated in our data. Fourthly, we did not have annual data on the number of alcoholics. Mortality, and especially that due to alcohol, is high among alcoholics while non-alcoholic drinkers have considerably lower death rates (Dawson, 2000; Lundin et al., 2015). Therefore, no direct causal conclusions can be made about the effect of alcohol consumption in a population on the alcohol-related mortality, based on the present data. However, our findings suggest a useful way to make near future forecasts.

Alcohol-specific deaths are a category containing etiologic diagnoses, that is, alcohol is mentioned in the disease name. Therefore, alcohol is a necessary cause. Not a sufficient cause, since death is caused by many factors. Alcohol may, or may not, be the decisive factor. The accuracy in ascertaining these deaths depends on the judgement of the cause-of-death determination which remains to be unknown. Attributions to alcohol may be under- or overestimated. Each revision of the ICD has provided more diagnoses with alcohol etiology which may have increased the number of alcohol-specific deaths. We did not study alcohol-attributable fractions of other deaths. If these were included the associations might be weaker, because alcohol intake has been found to decrease the risk of death for some of these other diseases.

It is unknown how much these results can be generalized. The possible effects are likely to be time and country specific, rather than universal. The effects may vary by country and time, depending on many factors, such as drinking habits, relative price, availability, beverage preferences and other ingredients than alcohol in the beverages.

To sum up, ARDL models with cointegrated variables offer better forecasting precision over mere differencing and/or various Box-Jenkins methods that have been more common in alcohol research. However, the better precision is due to the cointegration relationship present in our data, explicitly taken into account in the model. Nevertheless, future improvements in the precision of forecasts would be welcome. In studying the relationship between alcohol consumption and alcohol-specific deaths it is important to consider, not only total consumption, but also beverage types.

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Tables

Table1. Regression models of change in alcohol deaths on change in spirits and change in total consumption 1969-2015

	Coefficient of	Estimate (std.error)	t-value ($p > t$)	R^2	Root MSE
Model 1.1					
				0.159	2.13
	$\Delta\log(spirits)$	13.71 (4.74)	2.89 (0.006)		
	$constant$	0.58 (0.31)	1.86 (0.069)		
Model 1.2					
				0.116	2.19
	$\Delta\log(totalcons)$	20.35 (8.47)	2.40(0.027)		
	$constant$	0.36 (0.33)	1.09 (0.278)		

Regression estimates from the 1st differences models, for years 1969 - 2015, adjusted N=46.

Model 1.1 expresses changes in alcohol deaths per 100 000 explained by changes in the logarithm of spirits per capita, given by the linear equation $\Delta alcdeath_t = \alpha_0 + \alpha_1 \Delta\log(spirits)_t$. Model 1.2 gives changes in alcohol deaths per 100 000 explained by changes in the logarithm of total consumption per capita, given by the equation $\Delta alcdeath_t = \beta_0 + \beta_1 \Delta\log(totalcons)_t$

Table 2. Results from the ARDL(1,4,4) model in cointegration form

Cointegrating Form. Dependent variable: $\Delta alcdeath_t$

Variable	Coefficient	Std. Error	t-Statistic	Prob.
$\Delta\log(spirits)_t$	23.80	4.74	5.02	0.000
$\Delta\log(spirits)_{t-1}$	15.63	4.80	3.26	0.003
$\Delta\log(spirits)_{t-2}$	-5.10	5.24	-0.97	0.337
$\Delta\log(spirits)_{t-3}$	10.29	4.54	2.26	0.030
$\Delta\log(nonspirits)_t$	0.79	7.76	0.10	0.919
$\Delta\log(nonpirits)_{t-1}$	-22.72	9.06	-2.51	0.017
$\Delta\log(nonspirits)_{t-2}$	-12.14	9.42	-1.29	0.206
$\Delta\log(nonspirits)_{t-3}$	-25.86	8.95	-2.89	0.007
EC_{t-1}	-0.63	0.10	-6.23	0.000

$EC_t = alcdeath_t -$
$[17.44 * \log(spirits)_t + 67.74 * \log(nonspirits)_t - 104.35]$

Long Run Coefficients

Variable	Coefficient	Std. Error	t-Statistic	Prob.
$\log(spirits)$	17.44	3.65	4.77	0.000
$\log(nonspirits)$	67.74	3.15	21.49	0.000
$constant$	-104.35	8.70	-11.99	0.000

The estimated constrained error correcting form and the long-run form of the ARDL(*1,4,4*) model given in formula (3). Shown are the estimated coefficients of the lagged $\Delta\log(spirits)$ and $\Delta\log(nonspirits)$ variables, of the lagged error correction variable EC_{t-1}, and the coefficients of the equilibrium equation and their standard errors and t-tests for coefficient significance.

Table 3. Forecasts of alcohol deaths for years 2016 - 2020

		YEAR				
MODEL		2016	2017	2018	2019	2020
ARDL(1,4,4) 1969 – 2015. No policy change in 2016	Alcohol deaths forecast per 100.000	**36.95**	**35.17**	**34.70**	**35.21**	**34.28**
	.95 prediction limits of forecast	33.5 - 40.3	31.6 - 38.7	31.1 - 38.2	31.5 - 38.9	30.6 - 37.9
ARDL(1,4,4) 1969 -2015. 6% consumption increase from 2016 on.	Alcohol deaths forecast per 100.000	**36.90**	**36.63**	**37.66**	**37.14**	**38.12**
	.95 prediction limits of forecast	33.4 - 40.4	33.0 - 40.3	34.0 - 41.3	33.5 - 40.8	34.5 - 41.8

Forecasts, for the years 2016 – 2020, of alcohol deaths derived from the ARDL(1,4,4) model, formula (3) of the years 1969-2015. The underlying explanatory values of $\Delta\log(spirits)$ and $\Delta\log(nonspirits)$ for the years 2017 -2020 are obtained by standard ARIMA forecasting. The first row of forecasts is estimated without additional assumptions, the second row by assuming a 6% increase in total consumption, entirely in the non-spirits. For the years 2017-2020 the 0.95 prediction limits are conditional on the predicted explanatory variables, and are thus too narrow in reality.

Figure Legends

Figure 1.

Time series of the main variables 1969 -2015. All figures are annual and relate to the population over 15 years of age in Finland. The alcohol consumption figures (the left axis) are in litres absolute alcohol per capita (15 years +). The alcohol deaths (the right hand axis) are per 100.000 of population (15 years +).

Figure 2.

Observed and predicted values in levels with .95 prediction limits from the estimated ARDL(1,4,4) model. The model coefficients are given (in cointegration form) in Table 2.

Figure 3.

Observed and predicted values in first differences from the estimated ARDL(1,4,4) model. The model coefficients are given (in cointegration form) in Table 2.

Figure 4.

Observed alcohol deaths and forecast values from a ARDL(1,4,4) model estimated on the 1969 – 2004 data. The *ex ante* forecasts with prediction limits are for the years 2005 – 2015.