Per capita alcohol consumption and sickness absence in Norway

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Background: There is only one previous study addressing the relationship between population drinking and sickness absence. That study, based on Swedish time-series data, showed a statistically significant relationship between per capita alcohol consumption and the male sickness absence rate. Estimates suggested that a 1-l increase in consumption was associated with a 13% increase in sickness absence among men. In the present study, we aim at replicating and expanding the Swedish study on the basis of data for Norway. Methods: The outcome measure comprised annual data for Norway on registered sickness absence for manual employees covering the period 1957–2001. The unemployment rate was included as a control, as this factor may be correlated with alcohol as well as sickness absence. Alcohol consumption was gauged by sales of alcohol (total and beverage specific by beer, spirits and wine) per inhabitant 15 years and above. The data were analysed using the Box–Jenkins method for time-series analysis. Results: The results suggested that a 1-l increase in total consumption was associated with a 13% increase in sickness absence among men (P < 0.05). This corresponds to an elasticity coefficient equal to 0.62. The alcohol effect was not significant for women. Unemployment was negatively associated with the outcome for men as well as for women (P < 0.05). In the beverage-specific analyses, spirits were statistically significant for men (P < 0.05), but not beer and wine. Conclusion: The present findings strengthen the conclusion from the Swedish study, that sickness absence may be added to the list of indicators of alcohol-related harm.

Keywords: alcohol consumption, Norway, sickness absence, time-series analysis, unemployment.

Introduction

Considering the established relationships between alcohol consumption and a number of ill-health outcomes, it would seem plausible that there exists a link between alcohol consumption and sickness absence. First, there are acute consequences of excessive alcohol intake, such as accidents, not to mention hangovers that would elevate the risk of sickness absence (legitimate or not). Second, chronic heavy alcohol consumption may cause a number of psychiatric (e.g. depression) and somatic ailments (e.g. cancer and liver cirrhosis) that would qualify for sickness absence. This assumption is supported by several studies. A recent Finnish study,¹ based on individual-level data, reports a significant relationship between alcohol consumption and sickness absence. In an Australian survey,² 3.5% of the respondents who were employed and current drinkers reported having missed at least 1 day from work in the preceding 3 months due to their own drinking. The estimated number of alcohol-related sick-leave days and total number of sick-leave days, respectively, imply that the former comprise about 6% of the total number of sick-leave days in Australia. In two Norwegian studies,³,⁴ it was estimated that between 14% and 50% (depending on assumptions) of the total short-term sick leave (1–3 days) could be attributed to alcohol. The fraction of alcohol-related sick leave was highest among those who had a high consumption of alcohol. Similarly, several studies report an elevated risk for sickness absence among heavy drinkers.⁵–⁷ However, the picture is blurred by the common finding that also abstainers are at an increased risk; that is, a U-shaped risk function is typically found with moderate drinkers showing the lowest risk.⁸–¹¹ The U-shaped risk function may be interpreted in more than one way. In one study,¹ it is regarded as a consequence of the putative protective effects of moderate consumption on the risk of, inter alia, the common cold and other forms of infections. Another interpretation is that the elevated risk for abstainers is due to self-selection (that people with poor health abstain from alcohol), but also that the increased risk for heavy drinkers may at least partly be due to contaminating factors. There is also the possibility of reversed causation, that is, that sickness absence is a risk factor for excessive drinking. There are, thus, several methodological problems that are potential threats to valid inferences in the present context. In fact, a comprehensive review¹² suggested that the existing literature did not provide evidence that was reliable enough for establishing a relationship between alcohol and sickness absence. The main reason for this was methodological limitations including poor measurements (of exposure, outcome or both), selection bias and high attrition rates. This state of affairs speaks to a supplementary methodological approach, e.g. analyses of aggregate time-series data. One advantage of this method is that it overcomes the problems that seem to be particularly acute in the present context, namely those of self-selection and reversed causation.¹³ This approach has been applied to estimate the population-level relationship between alcohol consumption and a large number of outcomes, e.g. cirrhosis mortality, accident mortality, suicide, all-cause mortality and violence. Generally, all of these outcomes have proved to be associated with overall consumption, although the magnitude of the estimated association varies across outcomes and cultural settings (see Norström and Ramstedt¹⁴ for a review). However, the study by Norström¹⁴ is the only one that has addressed the link between alcohol and sickness absence at the aggregate level. On the basis of Swedish time-series data for the period 1935–2002, a statistically significant relationship between per capita alcohol consumption and the male sickness absence rate was found. It was estimated that a 1-l increase in consumption...
was associated with a 13% increase in sickness absence among men. But as this is the only study thus far, it would seem urgent to test whether that finding can be replicated using other data. In the present study, we thus perform a similar analysis using Norwegian time-series data. However, our analyses extend beyond a mere replication, and take some further steps by addressing two additional issues, the first being that of beverage-specific effects: generally, spirits consumption seems to have a stronger link to adverse consequences than what is the case for beer and wine. For instance, rates of cirrhosis mortality have been found to be more closely related to aggregate consumption of spirits than to intake of other alcoholic beverages. In the context of sickness absence, Vahtera et al. found a U-shaped risk curve for beer and wine, but a linear one for spirits. A plausible interpretation of these patterns is that spirits intake is a marker for a risky drinking pattern. Different sources of data pertaining to Norway indeed suggest that spirits and to some degree beer play a major role in heavy chronic drinking as well as in binge drinking. Because these forms of drinking seem to be plausible mechanisms in the link between alcohol and sickness absence, it can be hypothesized that the latter would be more strongly related to beer and spirits than to wine. The second issue concerns the explanation of trends in sickness absence: provided that a significant alcohol effect is found, it is of interest to investigate to what degree temporal trends and shifts in sickness absence can be accounted for by changes in per capita alcohol consumption.

**Methods**

Sickness absence was measured as the percentage of reported days of sickness absence of all working days, separately for men and women (source: Confederation of Norwegian Enterprise; http://www.nho.no/english/). It should be noted that these data do not pertain to the whole workforce, but only to manual workers (whose share of the workforce varied between 59% and 73% during the study period). Data for non-manual workers were not available. In 2002, there was an influential change in the recording practice, and the study period was therefore confined to the period 1957–2001. The proxy for alcohol consumption, expressed in litres of per capita alcohol consumption.

Unemployment was included as a control variable because of its documented relationship with sickness absence, and its possible link with alcohol consumption. Unemployment was measured as the unemployed percentage of the labour force according to the labour force surveys (source: Statistics Norway). During the study period, there were changes in the recording practice in 1967 and 1971. Further, in 1978, three waiting days were eliminated, implying that after this change employees were compensated from the first day of a sick-leave period. The possible effects of these changes were gauged by dummy variables that are coded zero before the change and one otherwise.

Separate models were estimated for men and women. As already mentioned, unemployment was included as a control variable; however, there may well exist additional potential confounders. As an alternative precaution against omitted variable risk, we estimated a model for men in which female sickness absence was included among the explanatory variables together with alcohol consumption. The rationale for such a specification is that there is a large number of etiological factors that have similar effects on male and female sickness absence; the latter is then regarded as a proxy for such common factors. An analogous model specification has been used when estimating the relationship between aggregate alcohol consumption and male all-cause mortality. In these studies, female mortality was included as a control variable, the argument being that it functions as an indicator of causal factors that are common to male and female mortality, such as advancements in medical technology and environmental factors in a broad sense. This specification is a more flexible alternative to using excess male mortality as the outcome. Inspection of the scattergram between unemployment and sickness absence (logged) suggested that a quadratic transformation of the unemployment indicator linearized the relationship and thus improved the fit to the data.

The data were analysed using the technique for time-series analysis that has been developed by Box and Jenkins, often referred to as Autoregressive Integrated Moving Average (ARIMA) models. The presence of strong time trends (figures 1 and 2) in the data necessitates a filtering to achieve the stationarity required for the ARIMA modelling. In this case, a simple differencing was sufficient to remove non-stationary trends; that is, rather than using raw series, the yearly changes were analysed. Differencing greatly reduces the risk of obtaining spurious correlations, as an omitted variable is more likely to be correlated with the explanatory variable as a result of common trends than as a result of synchronization in the yearly changes. Further, the noise term, which includes explanatory variables not considered in the model, is allowed to have a temporal structure that is modelled and estimated in terms of autoregressive (AR) or moving average (MA) parameters. The model residuals should not have any temporal structure, that is, they should not differ from white noise; this was tested using the Box–Ljung statistics . A -value with an associated -value exceeding 0.05 indicates that the residuals are white noise.

The likely presence of long-term effects of alcohol on sickness absence in addition to the acute effects necessitates the incorporation of a lag structure into the model. We adopted a lag scheme that has proved feasible in modelling the alcohol effect on all-cause mortality as well as sickness absence. In these analyses, a weighted alcohol series with geometrically declining lag weights proved to be feasible. The lag parameter equalled 0.7. There was a truncation at lag five; further, the lag weights were rescaled to sum to unity. The following semi-log model was used to estimate the effects on sickness absence of the various explanatory variables:

\[
\ln S_t = \beta + \gamma_A W_t + \sum c_i \cdot C_{it} + \epsilon_t
\]

Sickness absence is indicated by , alcohol consumption (weighted) by . is a set of control variables that differs between model specifications. signifies the noise term, which includes other etiological factors. The alcohol effect is expressed by the parameter . The percentage increase in sickness absence associated with a 1-l increase in consumption is given by the expression: .

**Results**

As can be seen in figure 1, there was a steady increase in sickness absence during the first part of the study period; during the first three decades, the rate for men and women increased by about 50%. From the mid-1980s, there was a decade of marked decline, followed by an equally long steady increase. The rates for men and women are strongly synchronized, not only in the trends , but in the yearly changes as well . This corroborates our assumption that the sickness absence rates for men and women
are largely driven by common factors. Trends in per capita consumption (figure 2) are fairly similar to those for sickness absence; the correlation between alcohol consumption and male sickness absence is 0.83. We now turn to the more rigorous test of the relationship provided by the model estimations (table 1). Both of the model specifications yield statistically significant alcohol effects on male sickness absence; according to Model 1 (controlling for female sickness absence), a 1-l increase in consumption is associated with a 13% increase in male sickness absence. The corresponding figure for Model 2 (controlling for unemployment) is 14% [calculating the elasticity on the basis of the mean consumption during the study period (4.7 l) and the effect estimate of 13%/l yields a coefficient equal to 0.62; i.e. a 10% increase in alcohol consumption would be associated with a 6.2% increase in male sickness absence].

It is further noticed that the alcohol effect was not significant in the model for women (Model 3). The unemployment indicator was significant for men as well for women, and with the expected sign, i.e. inversely related to sickness absence. None of the models required any AR or MA parameters to obtain a white noise structure of the residuals. With regard to the beverage-specific effects, only the spirits indicator proved to be statistically significant in the model for men (Model 4). A corresponding model for women (not shown) did not reveal any significant effect of any of the beverage-specific indicators.

Unemployment was included because it was assumed to be linked to per capita alcohol consumption (as well as to sickness absence). To test the validity of this assumption, we estimated a model with alcohol consumption (logged) as the dependent variable, and unemployment as the explanatory

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Figure 1 Trends in sickness absence

Figure 2 Trends in per capita alcohol consumption and unemployment
variable. We obtained a statistically significant estimate equal to −0.035 (SE = 0.012, P = 0.004). An increase in unemployment by one percentage point is thus associated with a decrease in alcohol consumption by about 3.5%. Further, we estimated Models 1 and 2 using contemporaneous consumption as the explanatory variable rather than the weighted construct. For Model 1, we obtained a significant alcohol effect estimate equal to 0.072 (SE = 0.022, P = 0.002). The result for Model 2 was practically the same. This means that a good half of the total impact of alcohol on sickness absence is instantaneous (occurs within 1 year), while the remaining part is distributed over a longer period of time.

The results thus suggest that changes in alcohol consumption as well as unemployment tend to be associated with changes in sickness absence. Another question is the magnitude of these relationships; are they strong or weak? One way of assessing this issue is to check to what degree the temporal pattern in these two determinants can account for the major shifts and trends in sickness absence. The graphs in figure 3 give an answer to that question. In addition to the observed rate of sickness absence, the figure presents two predicted rates: one that is based on the trajectory in consumption (and the estimated alcohol effect), and another where also the estimated impact of trends in unemployment is incorporated (note that the levels of the predicted series are arbitrary; further, in view of the insignificant alcohol effect for women, these projections are only made for men). There are three major shifts in the rate of sickness absence; the first one is a 50% increase starting in the mid-1960s. About one-third of this increase is accounted for by the change in the alcohol indicator, while changes in unemployment had no additional explanatory power. The second big shift is the 35% decrease starting in the mid-1980s. The two determinants can jointly explain about half of this decrease, but this time it is the unemployment indicator that does most of the job. That is also the case when we come to the last shift, the increase by 40% that began in 1994. A good half of this increase would be due to our two explanatory variables. The explanatory power of

**Table 1** Estimated effects of alcohol consumption on sickness absence

<table>
<thead>
<tr>
<th></th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
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<tr>
<td></td>
<td>Estimate</td>
<td>SE</td>
<td>Estimate</td>
<td>SE</td>
</tr>
<tr>
<td>Alcohol</td>
<td>0.125**</td>
<td>0.042</td>
<td>0.134*</td>
<td>0.060</td>
</tr>
<tr>
<td>Sq Unemployment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ln female SAR</td>
<td>0.533***</td>
<td>0.081</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beer</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Spirits</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Wine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dummy 67</td>
<td>0.046</td>
<td>0.033</td>
<td>0.074</td>
<td>0.045</td>
</tr>
<tr>
<td>Dummy 71</td>
<td>−0.078*</td>
<td>0.034</td>
<td>−0.075</td>
<td>0.046</td>
</tr>
<tr>
<td>Dummy 78</td>
<td>0.048</td>
<td>0.047</td>
<td>0.096</td>
<td>0.062</td>
</tr>
<tr>
<td>Diagnostics</td>
<td>5.26 (P &gt; 0.38)</td>
<td>5.72 (P &gt; 0.20)</td>
<td>3.73 (P &gt; 0.58)</td>
<td>4.94 (P &gt; 0.42)</td>
</tr>
</tbody>
</table>

Semi-logarithmic ARIMA models estimated on differenced Norwegian data, 1957–2001
a: Box–Ljung test for autocorrelated residuals
*P < 0.10; **P < 0.05; ***P < 0.01; ****P < 0.001

**Figure 3** Observed and predicted trends in male sickness absence
the two determinants is, thus, far from non-trivial, although their relative importance varies over time.

Discussion

A large body of research indicates that overall alcohol consumption is related to numerous harm indicators, but its relationship to sickness absence has only been established in one previous study based on Swedish time-series data. In the present study, we have broadened the empirical base by replicating the analyses using Norwegian time-series data. At this stage, it is warranted to point out the limitations of the present study. First, it is well known that a non-negligible fraction of the total alcohol consumption is unrecorded and thus not covered by the sales data. This may bias the estimated alcohol effect upwards (if there is a positive correlation between recorded and unrecorded consumption) as well as downwards (negative correlation between recorded and unrecorded consumption). There is hardly any information on which to base an assessment of which, if any, of these forms of bias is most likely. Second, the sickness absence data pertain to manual workers only, which limits the generalizability of the findings. Lastly, there is the possibility that some omitted variable, related to alcohol as well as sickness absence, has biased the outcome in spite of our various devices intended to minimize this risk. However, what gives us some confidence in the results is that the estimated alcohol effect, 13%/l alcohol, was conspicuously close to what was found for Sweden, a country similar to Norway with respect to drinking culture as well as the feature of the sickness absence system. It can further be noted that this estimate is on a par with the alcohol effect that has been obtained for various other alcohol-related outcomes, including fatal accidents, suicide and assaults. At the same time, the estimated effect is markedly lower than those pertaining to indicators that are alcohol induced by definition, e.g. alcoholic liver cirrhosis. The findings are thus consistent internally (across the two studies pertaining to Sweden and Norway) as well as externally, that is compared with findings for other outcomes.

One may ask why the alcohol effect is not statistically significant for women. The most important reason for this is in all probability that women drink much less; their consumption is about 40% of that for men. This means that the expected alcohol effect for women is less than half of that for men. In addition, there are differences in drinking patterns that work in the same direction: compared with men, women have less preference for spirits and are less inclined to binge drink. Thus, the expected alcohol effect for women is probably too small to reach statistical significance considering the fairly wide confidence intervals that are present.

It has been shown that a large fraction of the estimated alcohol-attributable costs to society are borne by workplaces. For Norway, this cost has been estimated at 1.7 billion Norwegian kroner (NOK) for 2001 (0.2 billion Euro). Although this kind of estimate is crude and based on a number of assumptions, the obvious policy implications of our findings are that a lowered level of population drinking would reduce not only the individual human cost due to sickness absence, but in addition the economic costs for industry and society.

Finally, a remark as to future research in this area. For the purpose of testing whether the finding for Sweden could be replicated, the choice of Norway, which has a similar drinking culture, would seem to be feasible. However, it would also be of interest to probe the link between alcohol and sickness absence in data for countries with quite different drinking patterns.

Acknowledgements

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Key points

- A previous study based on Swedish time-series data found a statistically significant relationship between per capita alcohol consumption and male sickness absence. This finding was replicated in the present study, based on Norwegian time-series data.
- The beverage-specific analysis suggested that consumption of spirits (but not of wine and beer) was associated with male sickness absence. A plausible interpretation of this is that spirits consumption is a marker of a risky drinking pattern.
- One policy implication of our findings is that lowered per capita alcohol consumption would reduce not only the individual human cost due to sickness absence, but in addition the economic costs for industry and society.

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