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# THE SHEFFIELD ALCOHOL POLICY MODEL – A MATHEMATICAL DESCRIPTION

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#### **SUMMARY**

This methodology paper sets out a mathematical description of the Sheffield Alcohol Policy Model version 2.0, a model to evaluate public health strategies for alcohol harm reduction in the UK. Policies that can be appraised include a minimum price per unit of alcohol, restrictions on price discounting, and broader public health measures. The model estimates the impact on consumers, health services, crime, employers, retailers and government tax revenues. The synthesis of public and commercial data sources to inform the model structure is described. A detailed algebraic description of the model is provided. This involves quantifying baseline levels of alcohol purchasing and consumption by age and gender subgroups, estimating the impact of policies on consumption, for example, using evidence on price elasticities of demand for alcohol, quantification of risk functions relating alcohol consumption to harms including 47 health conditions, crimes, absenteeism and unemployment, and finally monetary valuation of the consequences. The results framework, shown for a minimum price per unit of alcohol, has been used to provide policy appraisals for the UK government policy-makers. In discussion and online appendix, we explore issues around valuation and scope, limitations of evidence/data, how the framework can be adapted to other countries and decisions, and ongoing plans for further development. © 2014 The Authors. *Health Economics* published by John Wiley & Sons Ltd.

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#### 1. INTRODUCTION

Alcohol can cause health and social harms (WHO, 2011), and policies can influence its affordability, availability and use (Babor *et al.*, 2010). Existing alcohol policy modelling literature examines policies' impact on consumption and/or harms (Chisholm *et al.*, 2004; Gunningschepers, 1989; Hollingworth *et al.*, 2006). These population cohort models have discrete 'drinking states', for example, abstention, moderate consumption and heavy consumption (which is problematic because econometric analyses of price elasticities assume a continuous distribution of drinkers' consumption), and use either a birth-cohort approach or an age-cohort approach (Holder and Blose, 1987) together with mathematical risk functions of harm given a level of alcohol consumption to calculate the 'potential impact fraction' (PIF = ratio of weighted average revised risk given a policy to the weighted average baseline risk given current consumption levels).

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This paper sets out a mathematical description of the Sheffield Alcohol Policy Model version 2.0. UK policy-makers (Department of Health in England (DH), then National Institute for Health and Clinical Excellence (NICE) and later other governments and agencies) commissioned systematic evidence reviews (Booth *et al.*, 2008) and data gathering leading to model version 1.1 (Brennan *et al.*, 2008) examining potential pricing and promotion policies, and version 2.0 (Purshouse *et al.*, 2009a) focussed on (i) minimum unit pricing and discounting restrictions; (ii) regulating 'alcohol outlet density' and licensing hours; (iii) advertising controls; and (iv) alcohol screening and brief intervention strategies. For a more detailed discussion of the existing international literature and of the strengths, limitations and implications of the Sheffield Alcohol Policy Model, see the online appendix (web reference). In the results section, here, we present exemplars of the results framework with reference to published reports and journal articles on policy applications. The central purpose of this paper is to set out the data used and the full algebraic model description as follows.

#### 2. METHOD

## 2.1. Data and evidence

The model development process included iterative discussions with policy-makers and experts, and systematic searching for evidence and data sources (Brennan *et al.*, 2013). An overview of inputs and outputs is shown in Figure 1.

Here, we set out the main data/evidence sources. (See online appendix web reference for deeper discussion, and also Purshouse *et al.*, 2009a, for full details for data inputs in NICE report: pp. 19–94, Tables 2.1–2.16, Figures 2.1–2.22.)

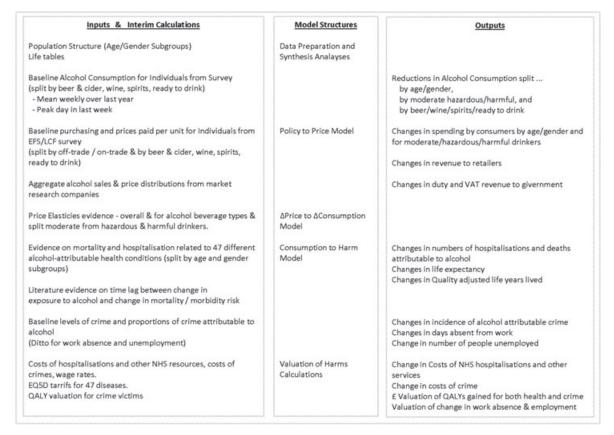


Figure 1. Overview of model inputs, structures and outputs

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The General Household Survey (GHS) collects mean weekly alcohol consumption and peak day (in the last 7 days) consumption. We split into four beverage categories: beers, wines, spirits and 'ready-to-drinks' (RTDs or 'alcopops'), and we defined 54 population subgroups split by age, gender and level of alcohol consumption (moderate/hazardous/harmful). The sample was 14 289 individuals for England excluding outliers in 2006.

The Expenditure and Food Survey (EFS) uses a 14-day diary to record household purchases including the type of alcohol, the place of purchase and, crucially, the price paid per volume in litres of product, converted to alcohol units, using percentage alcohol by volume (%ABV) (Office for National Statistics and Department for Environment, 2007). We classified 16 defined beverage types: beers, wines, spirits and alcopops, split further into on-trade and off-trade, and split again into a lower-priced and higher-priced band. We analysed 'transaction' level data for 69 618 individuals (44 150 purchased alcohol) over 5 years 2001/2 to 2005/6.

Market research company data were obtained on aggregate sales of beers, wines, spirits and alcopops in off-trade and on-trade by different price bands as well as the extent of price discounting. Combining this with EFS data, we quantified price distributions by beverage category for each of the 54 population subgroups (see Table 1, Purshouse *et al.*, 2010b).

Evidence for the effects of policies came from systematic evidence reviews and new data analyses. Our 243-page review for DH (Booth *et al.*, 2008) identified two recent meta-analyses of international price elasticities (Gallet, 2007; Wagenaar *et al.*, 2009), whilst the NICE systematic reviews (Jackson *et al.*, 2009a; Jackson *et al.*, 2009b) identified evidence on screening and brief interventions. For the base-case model, price elasticities were estimated from the adjusted EFS dataset using an iterative three-stage least squares regression relating consumption to price for the 16 beverage categories and to consumption of other non-durable goods (online appendix in Purshouse *et al.*, 2010a) adjusting for gender, age group, ethnicity, education, geographical region, household composition, household size, income and employment status. Estimates converged for subgroups (i) moderate drinkers and (ii) hazardous and harmful drinkers combined, producing two 16 × 16 elasticity matrices each with own price and cross-price elasticity estimates.

Health harms attributable to alcohol were quantified by age/gender subgroup for risk of mortality and risk of hospitalisation in 47 different alcohol-attributable health conditions (Jones *et al.*, 2008). Published literature provided risk function curves relating mean weekly alcohol consumption to individual's mortality or disease prevalence (Corrao *et al.*, 2004; Gutjahr *et al.*, 2001; Hamajima *et al.*, 2002; Rehm *et al.*, 2004; Rehm *et al.*, 2010) and evidence about the time lag between change in exposure to alcohol and change in risk (Norstrom and Skog, 2001).

Similar risk functions were estimated for 20 classifications of crime related to peak day consumption. Data on total crimes reported was adjusted for under-reporting (Cabinet Office/Strategy Unit, 2003; Health Improvement Analytical Team, 2008; Home Office Research, 2011), apportioned to age/gender subgroups using data on offenders (Office for National Statistics, 2005), and then estimated as attributable to alcohol (Offending Crime and Justice Survey (OCJS) 2005). Similarly, work absence is related to peak day consumption, using two-part linear relative risk (RR) functions fitted for each age/gender group, using data from the Labour Force Survey 2006 (Office for National Statistics, 2011), and attributable proportions from literature evidence (Roche *et al.*, 2008). Finally, unemployment was related to mean weekly consumption amongst harmful drinkers using Health Survey for England data showing that men aged 22 to 64 years categorised as 'problem drinkers' had a lower probability of being in work (MacDonald and Shields, 2004).

# 2.2. Mathematical framework

2.2.1. Modelling baseline consumption and purchasing patterns. The model is fundamentally based upon what happens to an individual's mean weekly consumption of units of alcohol (variables denoted c, 1 unit = 10 ml ethanol). Mean weekly consumption  $c_{ijk}$  is the mean (over the previous year) weekly consumption by individual i, who is a member of population subgroup j, of beverage category k. We denote  $c_{ij}$  as the mean weekly consumption by individual i, who is a member of population subgroup j, summed across the K types of beverage, that is,

$$c_{ij} = \sum_{k=1}^{K} \left( c_{ijk} \right) \tag{1}$$

Some harms attributable to alcohol are related to the extent of intoxication, that is, an individual drinking beyond certain levels or 'bingeing'. We denote  $b_{ijk}$  as the consumption undertaken of beverage category k on the highest consuming day during the last week by individual i, who is in population subgroup j. Similarly, we denote  $b_{ij}$  as the peak day consumption by individual i, in population subgroup j, summed across the K=16 types of beverage, that is,

$$b_{ij} = \sum_{k=1}^{K} \left( b_{ijk} \right) \tag{2}$$

Fifty-four population subgroups are denoted by index j, based on separate age groups (×9, i.e. 11–15, 16–17, 18–24, 25–34, 35–44, 45–54, 55–64, 65–74 and 75+), gender (×2) and baseline mean weekly consumption in units for the individual (×3, i.e. moderate including zero consumption/hazardous/harmful).

$$i \in \{Moderate\}$$
 if  $0 \le c_{ij} \le moderateguideline$  (3)

$$i \in \{Hazardous\}$$
 if moderateguideline  $< c_{ij} \le harmfulguideline$  (4)

$$i \in \{Harmful\}$$
 if  $c_{ij} > harmfulguideline$  (5)

where *moderateguideline* = 21 units per week for men and 14 for women, and *harmfulguideline* = 50 units per week for men and 35 for women.

The second key dimension of the modelling concerns the prices paid for different beverages (variables denoted p). Data on prices paid by individuals within their EFS 2-week diary window are available at transaction level. For each transaction, we used information on %ABV to calculate the number of alcohol units and hence derive a price per unit of alcohol paid. Prices from different years were adjusted using Office for National Statistics (ONS) alcohol-specific inflation indices. We denote the price per unit of alcohol paid for transaction l of a beverage in category k by individual i who is in subgroup j as  $p_{ijkl}^{EFS}$ . We denote the quantity of units of ethanol purchased in the transaction as  $Q_{ijkl}$ , and the sample weight for the household as  $W_{ij}$ .

For each beverage category k and within each subgroup j, the transaction level data form an empirical distribution of prices paid per unit of alcohol that we denote by the variable  $\mathbf{p}_{jk}^{EFS}$ . For example, if there are 1000 transactions for cheaper off-trade beer for men aged 45 to 54 years who purchased at levels within the moderate threshold per week within the EFS, then  $\mathbf{p}_{men45-54,cheaperofftradebeer,moderate}^{EFS}$  is the list of the 1000 prices per unit paid. We denote the number of EFS transactions occurring for beverage category k within subgroup k by subgroup k by subgroup k is therefore

$$\overline{p}_{jk}^{EFS} = \frac{\sum_{l=1}^{L_{jk}} \left( p_{ijkl}^{EFS} \times W_{ijkl} \right)}{\sum_{l=1}^{L_{jk}} W_{ijkl}}$$
(6)

where  $W_{ijkl} = W_{ij} \times Q_{ijkl}$ .

Similarly, percentiles of the weighted price per unit distribution from the EFS can be calculated for each subgroup or more broadly for each beverage type. For example, we denote the  $x^{th}$  percentile of the weighted price distribution for all transactions buying beverage type k as

$$p_k^{EFS} x^{th} percentile = p_k^{EFS} (x\%)$$
 (7)

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We found discrepancies in purchase price distributions between EFS data and the aggregate sales data obtained from market research companies of beers, wines, spirits and alcopops in the off-trade (AC Nielsen) and ontrade (CGA Strategy). EFS data reported marginally lower mean prices (i.e. a higher proportion of cheaper alcohol) than the actual sales data. We therefore used linear interpolation to adjust the individual level EFS price data so that the adjusted cumulative price distribution matched the actual sales data price distribution at 10 specified price points. For example, in the off-trade, we obtained data from Nielsen, which provided, for the four categories off-trade beers, wines, spirits and RTDs, a price distribution. The price distribution for each beverage type is summarised as the 'sales volume' of units of alcohol sold within specified price points in terms of price per unit.  $\mathbf{SVol}_k$  is the vector of the sales volume of beverage k sold within each of the 10 specified price per unit points (<15p, 20p, 25p, 30p, 35p, 40p, 50p, 60p, 70p, 70p+). The analogous vector of sales value  $\mathbf{SVal}_k$  is also available. We denote the sales volume of units of alcohol between price points  $p_g$  and  $p_h$  as to  $SVol_k^{g,h}$ . Thus,

$$\mathbf{SVol}_k = \left(SVol_k^{0,15p}, SVol_k^{15p,20p} \dots SVol_k^{70p,\infty}\right) \tag{8}$$

and from these, we calculate the cumulative percentiles of the sales volume distribution at each price point

$$\%\mathbf{cumSVol}_{k} = \left(\%SVol_{k}^{0,15p}, \%SVol_{k}^{0,20p} \dots \%SVol_{k}^{0,70p}, \%SVol_{k}^{0,\infty}\right)$$
(9)

From this, using equation (7), we can generate the prices from the EFS price distribution, which match to each of the corresponding percentiles in equation (9). This gives a vector of

**Matched p**<sub>k</sub><sup>EFS</sup> = 
$$\left(p_k^{EFS}\left(\%SVol_k^{0,15p}\right), p_k^{EFS}\left(\%SVol_k^{0,20p}\right), \dots p_k^{EFS}\left(\%SVol_k^{0,\infty}\right)\right)$$
 (10)

To understand the interpolation process, consider the Nielsen range of [20p, 25p]. The equivalent range in the EFS is given by  $\left[p_k^{EFS}\left(\%SVol_k^{0,20p}\right), p_k^{EFS}\left(\%SVol_k^{0,25p}\right)\right]$ . Then, for each EFS transaction within this latter band, we adjust the price paid as follows:

$$\forall p_{ijkl}^{EFS} \in \left[ p_k^{EFS} \left( \%SVol_k^{0,20p} \right), p_k^{EFS} \left( \%SVol_k^{0,25p} \right) \right]$$

$$p_{ijkl}^{Revised} = 20 + (25 - 20) \times \left(\frac{p_{ijkl}^{EFS} - p_k^{EFS} \left(\% S Vol_k^{0,20p}\right)}{p_k^{EFS} \left(\% S Vol_k^{0,25p}\right) - p_k^{EFS} \left(\% S Vol_k^{0,20p}\right)}\right)$$
(11)

These adjusted transaction prices  $p_{ijkl}^{Revised}$  are the ones used throughout the modelling, and hereafter, we simply denote them as  $p_{ijkl}$ .

The key calculation for many aspects of the modelling is then to compute the mean (revised) price paid for beverage category k by subgroup j after introduction of the policy:

$$\overline{p}_{jk} = \frac{\sum_{l=1}^{L_{jk}} \left( p_{ijkl} \times W_{ijkl} \right)}{\sum_{l=1}^{L_{jk}} W_{ijkl}} = mean(\mathbf{p}_{jk})$$
(12)

Similarly, percentiles of the weighted price per unit distribution can be calculated for each subgroup.

If the numbers of transactions for a beverage within a subgroup were small, then subgroups were combined (e.g. 'cheaper off-trade alcopops' combining moderately purchasing men 65 to 74 years and moderately purchasing men 75+ years until the sample size is  $L_{jcombined,k} > 5$ ) to ensure reasonable estimation of means and percentiles of price distributions.

2.2.2. Policy to price modelling. The two main pricing policies examined by the model to date are as follows: (i) minimum unit pricing – whereby products are not allowed to be sold at a price per unit of alcohol less than  $P_{min}$  pence per unit, and (ii) restrictions on price-based discounting, whereby, for example, products are not allowed to be sold for a price lower than D% below their usual price.

To model the effects of minimum unit pricing on the price distribution of beverages, we made two simple assumptions. (i) Any transactions within the empirical distribution  $\mathbf{p}_{jk}$ , which occurred with a price per unit lower than  $P_{min}$  prior to the policy, would in future be priced at exactly  $P_{min}$  after policy implementation. (ii) Products sold in transactions at prices higher than  $P_{min}$  per unit would be completely unaffected. The model framework can include more complex models of the pricing responses of manufacturers and retailers, but in the absence of evidence on supply side responses to a minimum price policy, these simple assumptions have been used. We denote  $p_{ijkl}^*$  as the future post-policy implementation price of the product, which was actually purchased within the transaction data at  $p_{ijkl}$ . Then, for a minimum price policy, the simple price change rule is

If 
$$\left\{ p_{ijkl} < P_{min} \right\}$$
, then  $\left\{ p_{ijkl}^* = P_{min} \right\}$  (13)

If 
$$\left\{p_{ijkl} \ge P_{min}\right\}$$
, then  $\left\{p_{ijkl}^* = p_{ijkl}\right\}$  (14)

To model restrictions in off-trade price discounting, we used evidence on current discounting behaviours. This came in the form of two discounting matrices for each beverage category k. The first matrix ( $D_k(a,u)$ ) below) gives the proportion of the product sales volume, which has an actual sold price in the band, say 30p–35p, which has a usual (i.e. when undiscounted) price in different bands, for example, percentage with usual price at 35p–40p, 40p–45p etc. This discounting matrix has 10 rows reflecting the actual price per unit bands and 10 columns reflecting the 'usual' price, so that each row adds up to 100%. The second matrix ( $(M_k(a,u))$  below) sets out the average magnitude of promotional discount. Each element of this matrix is a positive percentage, which quantifies current average level of discounting; for example, products that have an actual price of 30p–35p per unit, which are usually sold at 35p–40p (i.e. are discounted downwards in price by one price band), have an average discount in price of 12.1% (see Brennan et al., 2008, Table 10, p. 45). The usual price is therefore given by  $p_{ijkl}/(1-m_{ka,u})$ .

$$D_{k}(a,u) = \begin{pmatrix} d_{k1,1}\% & d_{k1,2}\% & \dots & d_{k1,10}\% \\ 0\% & d_{k2,2}\% & \dots & d_{k2,10}\% \\ \vdots & & & & \\ 0\% & 0\% & \dots & d_{k10,10}\% \end{pmatrix}$$
(15)

$$M_{k}(a,u) = \begin{pmatrix} m_{k1,1}\% & m_{k1,2}\% & \dots & m_{k1,10}\% \\ 0\% & m_{k2,2}\% & \dots & m_{k2,10}\% \\ \vdots & & & & \\ 0\% & 0\% & \dots & m_{k10,10}\% \end{pmatrix}$$
(16)

To estimate the effects of a ban on discounting, we assumed that currently discounted prices would uplift to their 'usual' price level. A total ban is implemented in the model by each transaction at price  $p_{ijkl}$  being cloned 10 times and weights ( $0 \le weight \le 1$  and summing to 1) being given to each based on the matrix elements  $D_k(a,u)$ , and then uplifted by a factor based on the relevant element of matrix  $M_k(a,u)$ . More complex discounting policy restrictions can be modelled; for example, ban all discounts greater than a specified level such as  $D_{max} = 20\%$ . To implement these, we again used the transaction cloning process with weights based on matrix  $D_k(a,u)$  but assumed that if the actual mean discount level between price bands in matrix  $M_k(a,u)$  was a greater discount than the  $D_{max}$  level, then prices would be shifted upwards. If, on the other hand, the current level of discounting was smaller than the proposed  $D_{max}$  level, then prices for those products would be

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left unchanged.

If 
$$\{m_{ka,u} \ge D_{max}\}$$
, then  $\{p*_{ijkl} = p_{ijkl} \times \frac{1}{(1 - m_{ka,u})} \times (1 - D_{max})\}$  (17)

If 
$$\{m_{ka,u} < D_{max}\}$$
, then  $\{p*_{ijkl} = p_{ijkl}\}$  (18)

Similar methods were implemented for on-trade discounting.

2.2.3. Price to consumption modelling. The modelling of changes in consumption as a consequence of changes in price is undertaken in an aggregate way at the subgroup/beverage level. After the policy, the baseline empirical price per unit distribution  $\mathbf{p}_{jk}$  becomes  $\mathbf{p}_{jk}^*$ , and we can compute the pre-policy and post-policy mean price per unit and calculate the percentage increase in mean price for each subgroup j and beverage k, that is,

$$\%\Delta\overline{p}_{jk} = \frac{\overline{p^*}_{jk} - \overline{p}_{jk}}{\overline{p}_{jk}} \tag{19}$$

By multiplying this percentage change in mean price by an estimated own price elasticity for subgroup j (which we denote as  $e_{jk,k}$ ), one can calculate the change in expected purchasing of beverage type  $k_1$ , if only the price of beverage type  $k_1$  changes, as

$$\%\Delta \overline{SVol}_{jk_1} = \%\Delta \overline{p}_{jk_1} \times e_{jk_1,k_1}$$
(20)

$$\Rightarrow \overline{SVol}_{jk_1}^* = \overline{SVol}_{jk_1} \times \left(1 + \%\Delta \overline{p}_{jk_1} \times e_{jk_1, k_1}\right) \tag{21}$$

The Sheffield Alcohol Policy Model version 2 is set up to use as input a matrix of elasticities  $E_j$  for the 16 beverages for different subgroups j:

$$E_{j} = \begin{pmatrix} e_{j1,1} & e_{j1,2} & \dots & e_{j1,16} \\ e_{j2,1} & e_{j2,2} & \dots & e_{j2,16} \\ \vdots & & & & \\ e_{j16,1} & e_{j16,2} & \dots & e_{j16,16} \end{pmatrix}$$
(22)

The own price elasticities for beverage k are on the diagonal, that is,  $e_{jkk}$ . The off-diagonal elements of the matrix contain cross-price elasticities between different beverage types. The effects of a change in price of beverage type  $k_1$  on the purchasing of beverage  $k_2$  are given by these elements. Thus, if only the price of beverage  $k_1$  were changing, the effect on purchasing of  $k_2$  would be given by

$$\%\Delta \overline{SVol}_{jk_2} = \%\Delta \overline{p}_{jk_1} \times e_{jk_1,k_2}$$
(23)

$$\Rightarrow \overline{SVol}_{jk_2}^* = \overline{SVol}_{jk_2} \times \left(1 + \%\Delta \overline{p}_{jk_1} \times e_{jk_1,k_2}\right)$$
 (24)

We compute the combined effect of own and cross-price elasticities on expected changes in consumption of each beverage k within each subgroup j using the following process. We begin with  $c_{ijk}$  as the baseline consumption of alcohol units of beverage k by individual i in subgroup j.

We already have the baseline mean price paid for each type of beverage k by subgroup j from equation (12), which we can also denote as a 16-element vector

$$\overline{\mathbf{p}}_{i} = \left[\overline{p}_{ik}\right] = \left[\overline{p}_{i1}, \overline{p}_{i2}, \dots, \overline{p}_{i16}\right] \tag{25}$$

Using the price changes from  $p_{ijkl}$  to  $p_{ijkl}^*$  for the policy modelled (from equations (13), (14), (17) and (18)), we obtain the vector of new mean prices and the associated relative percentage changes in mean price paid for each type of beverage k by subgroup j, that is,

$$\overline{\mathbf{p}}_{i}^{*} = \left[ \overline{p}_{i1}^{*}, \overline{p}_{i2}^{*}, \dots, \overline{p}_{i16}^{*} \right]$$
 (26)

$$\%\Delta \overline{\mathbf{p}}_{i}^{*} = \left[\%\Delta \overline{p}_{i1}^{*}, \%\Delta \overline{p}_{i2}^{*}, \dots, \%\Delta \overline{p}_{i16}^{*}\right]$$
(27)

The resulting new level of mean consumption is calculated by applying the elasticity matrix to the relative price changes. This results in an expected total mean weekly consumption of alcohol units for individual *i* as being

$$c_{ijk}^* = c_{ijk} \times \left(1 + \% \Delta \overline{p}_{jk} \times e_{jk,k}\right) \times \left(1 + \sum_{k_i \neq k} \left(\% \Delta \overline{p}_{jk_i} \times e_{jk_i,k}\right)\right)$$
(28)

This change in mean weekly consumption for individuals, from  $c_{ij}$  under no policy change to  $c_{ij}^*$ , drives the estimation of differences for many harms in the model. However, for harms related to intoxication, we further need to estimate the change in the peak day maximum consumption  $b_{ij}$  for each individual i. We considered simply using a pro rata change in binge behaviour based on the relative change in mean weekly consumption. However, on investigating the relationship between mean weekly consumption and peak day maximum consumption within the GHS dataset, we found a simple regression model that enables us to adjust for age and gender. The resulting regression equation took the form:

$$E(b_{ii}) = const + \beta_1 c_{ii} + \beta_2 agecategory + \beta_3 gender category$$
 (29)

To estimate the change in  $b_{ij}$ , we simply compute the newly expected peak day consumption level as  $E\left(b_{ij}^*\right) = const + \beta_1 c_{ij}^* + \beta_2 agecategory + \beta_3 gender category$ , and we use the relative change in expected level to adjust the actual baseline peak day maximum for the individual, that is,

$$b_{ij}^* = b_{ij} \times \frac{E\left(b_{ij}^*\right)}{E(b_{ij})} \tag{30}$$

# 2.3. Consumption to expected harms modelling

2.3.1. Principles in modelling harms. Following epidemiological literature (Jones et al., 2008), we distinguish between wholly and partially attributable harms, and also 'chronic harms' which depend on long-term drinking levels and 'acute harms', which depend upon intoxication at a particular time. We require evidence on the baseline level of harm occurring. We denote the absolute level of harm h occurring within a year in a population subgroup s at baseline as  $H_s^h$  and the mean annual risk of harm per person within that subgroup therefore as  $\overline{R}_s^h$ . That is,

$$H_s^h/PopEngland_s = \overline{R}_s^h$$
 (31)

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For partially attributable conditions, the absolute level of harm  $H_s^h$  includes cases caused by alcohol and cases due to other causes. The model estimates incremental change for individuals i in subgroup j by considering how risk of each harm  $\overline{R}_s^h$  changes over time, that is,  $\overline{R}_s^h(t)$ . The key to this approach is essentially the relative change in risk over time, known as the potential impact fraction (PIF) (Gunningschepers, 1989), and thus, we model the mean annual absolute risk over time as

$$\overline{R}_s^h(t) = \overline{R}_s^h(t=0) \times PIF_s^h(t)$$
(32)

We denote the annual absolute risk of incurring harm h for an individual i (and later, by aggregating, for a subgroup j) as a function of consumption for that individual:

$$R_{ij}^{h} = R_{ij}^{h} \left( c_{ij}, b_{ij} \right) \tag{33}$$

Hence, the formula for the PIF is

$$PIF_s^h(t) = \frac{\sum_{i \in s} R_{ij}^h(t)}{\sum_{i \in s} R_{ij}^h(t = 0)}$$
(34)

where  $R_{ij}^h(t) = R_{ij}^h(c_{ij}(t), b_{ij}(t))$ . Note, when we apply this functionality with individuals in the GHS, we utilised the survey weights, for example,  $w_i$ , not shown in equations here. In equation (34), the risk functions  $R_{ij}^h$  are the absolute annual risk of the harm h occurring. Where evidence is available on relative risks rather than absolute risks, we take a similar approach. If  $rr_{ij}(c_{ij})$  is the relative risk of harm compared with abstainers (i.e. rr = 1 for abstainers and values greater than 1 for drinkers), then the annual absolute risk can be written:

$$R_{ij}^{h}\left(c_{ij},b_{ij}\right) = \overline{R}_{s}^{h}(t=0) \times rr_{ij}^{h}\left(c_{ij},b_{ij}\right) \times Constant \tag{35}$$

where the *Constant* adjusts for the fact that the rr is 1 for abstainers rather than for the average of the population. The PIF at time t is then given by equation (35) evaluated at t, divided by equation (35) evaluated at baseline time t = 0. The first factor  $\overline{R}_s^h(t = 0)$  and the *Constant* cancel out, giving

$$PIF_s^h(t) = \frac{\sum_{i \in s} \left( rr_{ij}^h(t) \right)}{\sum_{i \in s} \left( rr_{ij}^h(t=0) \right)}$$
(36)

The model risk function represents either the annual risk of a harm occurring, for example, risk of mortality occurring in the year, or the annual risk of being in a harmed state, for example, risk of suffering from a defined condition/disease such as hypertension within the year. In version 2, 'chronic harms' are a function exclusively of mean weekly drinking  $c_{ij}$ , and 'acute harms' a function exclusively of peak day maximum drinking  $b_{ij}$ .

For partially attributable harms, we either used risk curves directly from literature to quantify  $rr_{ij}^h$  or calibrated our own risk functions based on aggregate evidence available. To do calibration, we utilise the concept of the alcohol-attributable fraction (AAF). The AAF for a particular harm h, within a subgroup s, is defined as the proportion of cases caused by alcohol. This is calculated by comparing the total risk given current consumption, that is,  $\sum_{i \in s} R_{ij}^h \left( c_{ij}, b_{ij} \right)$ , with the counter-factual risk if nobody in the population were exposed to alcohol, that is,  $\sum_{i \in s} R_{ij}^h \left( c_{ij} = 0, b_{ij} = 0 \right)$ . A relative risk framework has a similar form; thus,

$$AAF_{j}^{h} = \frac{\sum_{i \in s} R_{ij}^{h} (c_{ij}, b_{ij}) - \sum_{i \in s} R_{ij}^{h} (c_{ij} = 0, b_{ij} = 0)}{\sum_{i \in s} R_{ij}^{h} (c_{ij}, b_{ij})}$$
(37)

$$AAF_{j}^{h} = \frac{\sum_{i \in s} rr_{ij}^{h} (c_{ij}, b_{ij}) - \sum_{i \in s} rr_{ij}^{h} (c_{ij} = 0, b_{ij} = 0)}{\sum_{i \in s} rr_{ij}^{h} (c_{ij}, b_{ij})}$$
(38)

All calibrated risk functions in version 2 have used the relative risk form specified in equation (38).

2.3.2. Risk over time. In most model applications, we assume a steady state of current consumption given current policies, and we compare this with an instantaneously achieved new steady-state level of consumption if the proposed policy were implemented. The model does, however, have the functionality for subgroups' consumption to change over time and thus to model changes in risk over time.

We denote time using both time points and time periods. That is, t=0 is the start time of the model. At t=0, we denote each individual's consumption without the policy  $\left(c_{ij}^0,b_{ij}^0\right)$  and consumption with the policy as  $\left(c_{ij}^{*0},b_{ij}^{*0}\right)$ . t=1 is 1 year after policy implementation, and  $t=2,3,\ldots$  the future time points. We denote the consumption at time point t as  $c_{ij}^t,b_{ij}^t$ . Consumption over time can therefore be denoted as vectors of consumption values, that is,

$$\mathbf{c}_{ij} = (c_{ij}^0, c_{ij}^1, c_{ij}^2, \dots), \qquad \mathbf{b}_{ij} = (b_{ij}^0, b_{ij}^1, b_{ij}^2, \dots)$$
(39)

$$\mathbf{c}_{ij}^* = \left(c_{ij}^{*0}, c_{ij}^{*1}, c_{ij}^{*2}, \ldots\right), \qquad \mathbf{b}_{ij}^* = \left(b_{ij}^{*0}, b_{ij}^{*1}, b_{ij}^{*2}, \ldots\right) \tag{40}$$

We model the risk during a particular time period as related to the consumption level at the start of that period. Thus, the risk during the first year of the model is  $R_{ij}^h\left(c_{ij}^0,b_{ij}^0\right)$  without the policy and  $R_{ij}^h\left(c_{ij}^{*0},b_{ij}^{*0}\right)$  with the policy. The risk during period t to t+1 is given by

$$R_{ij}^{h,(t,t+1)} = R_{ij}^{h} \left( c_{ij}^{t}, b_{ij}^{t} \right) \tag{41}$$

and the average risk for the number of people in subgroup j who are still alive at time t (denoted by  $A_j^t$ ) is given by

$$\overline{R}_{j}^{h,(t,t+1)} = \frac{1}{A_{j}^{t}} \sum_{i} R_{ij}^{h} \left( c_{ij}^{t}, b_{ij}^{t} \right)$$
(42)

and similarly, the PIF over time can be calculated using

$$PIF_{j}^{h}(t,t+1) = \frac{\sum_{i} R_{ij}^{h} \left(c_{ij}^{t}, b_{ij}^{t}\right)}{\sum_{i} R_{ij}^{h} \left(c_{ij}^{0}, b_{ij}^{0}\right)}$$
(43)

or 
$$PIF_{j}^{h}(t,t+1) = \frac{\sum_{i} rr_{ij}^{h} \left(c_{ij}^{t}, b_{ij}^{t}\right)}{\sum_{i} rr_{ij}^{h} \left(c_{ij}^{0}, b_{ij}^{0}\right)}$$
 (44)

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2.3.3. Consumption to mortality in health harms. We model the annual risk of death for each alcohol-related disease h. We also model annual mortality due to 'other causes' by especially constructing a life table with alcohol-related conditions excluded, using single-year age bands by gender ranging from  $(11, \ldots, 99)$ . The number of individuals alive in age/gender group g at time t is denoted  $A_g^t$ . We model annual time cycles as a Markov model. The number of deaths in period t to t+1 from harm h is denoted  $D_g^{h,(t,t+1)}$ . We also separate three drinker subgroups at this stage, splitting group g into moderate, hazardous and harmful drinkers. The number dying during period (t, t+1) is given by

$$D_g^{h,(t,t+1)} = A_g^t \times \overline{R}_{j|g \in j}^{h,(t,t+1)}$$

$$\tag{45}$$

$$= A_g^t \times \overline{R}_{j|g \in j}^h(t=0) \times PIF_j^h(t,t+1)$$

$$\tag{46}$$

Then, the number of people alive at time t + 1 is

$$A_g^{t+1} = A_g^t - \sum_{h \in \{H_m\}} D_g^{h,(t,t+1)} - \sum_{h \in \{Oth_m\}} D_g^{h,(t,t+1)}$$

$$\tag{47}$$

where  $\{H_m\}$  is the set of health harms for alcohol-related mortality and  $\{Oth_m\}$  is the aggregate 'other cause mortality'. The number of people in broader age bands (e.g. men 45–54 years) at time t+1 is simply the sum of those alive in single-year bands, that is,

$$A_j^{t+1} = \sum_{g \in j} A_g^{t+1} \tag{48}$$

2.3.4. Risk of disease prevalence. We define the set of harms that cause increases in disease prevalence or incidence  $\{H_{prev}\}$ . As for mortality, the prevalence harm occurring by subgroup is modelled using risk functions, and equations (33) to (42) apply. The number of people in the subgroup suffering harm  $h \in \{H_{prev}\}$  during period (t, t+1) is denoted  $H_j^{h,(t,t+1)}$  and is given by

$$H_j^{h \in \{H_{prev}\},(t,t+1)} = A_j^t \times \overline{R}_j^{h \in \{H_{prev}\},(t,t+1)}$$
(49)

2.3.5. Estimating absolute and relative risk as a function of consumption from available evidence. A considerable evidence base exists on the relative rather than absolute risk of harm caused by alcohol. In terms of evidence available, we found two common scenarios. The first scenario applies to many 'chronic' partially attributable conditions. Here, relative risk functions exist from international literature for harm h (i.e. we know the second factor of equation (35)), and we have local evidence on the mean annualised risk of harm for England (i.e. we know the first factor of equation (35)). Thus, we can directly calculate  $R_{ij}^h$  and, in turn, calculate the AAF via equation (37). In the second scenario, we do not have published relative risk functions but instead have the local mean annualised risk of harm (first factor of equation (35)) alongside the local estimates of the attributable fraction  $AAF_j^h$ . Here, we assumed a functional form for the relative risk functions; for example, relative risk equals a linear function of peak day maximum consumption,  $rr_{ij}^h(c_{ij}, b_{ij}) = \beta_j^{1h} + \beta_j^{2h} \times b_{ij}$ . Having made this assumption, we calibrate the value of the relative risk function parameter  $\beta_j^{2h}$  until the AAF calculated in equation (38) equals the observed AAF evidence we have available. A similar process is undertaken for the wholly attributable risk functions using absolute levels of risk as in equation (37).

2.3.6. Risk functions: chronic/acute and wholly/partially attributable diseases. We categorised 47 International Classification of Diseases (ICD) diagnosis-defined conditions into four types: chronic harms related to mean alcohol consumption and acute harms related to peak day consumption, with a further split into conditions wholly or partially attributable to alcohol.

For chronic illnesses partially attributable to alcohol (e.g. oesophageal cancer), published literature provided continuous risk curves relating mean weekly alcohol consumption to an individual's relative risk  $\left(rr_{ij}^{h}(c_{ij})\right)$  of mortality or disease prevalence differentiated by gender when available (Corrao *et al.*, 2004; Gutjahr *et al.*, 2001; Hamajima *et al.*, 2002; Rehm *et al.*, 2004; Rehm *et al.*, 2010). We assumed identical relative risk curves for each age group, but using absolute risk levels for each age/sex group at baseline from published data (e.g. annual mortality rate for oesophageal cancer for men aged 45–54 years), we had enough information to compute the change in the subgroup risk for a policy by multiplying the baseline absolute risk by the potential impact fraction from equation (36).

For the other three types of harm, published risk function curves were unavailable. We developed two-part linear risk functions whereby the risk is flat from zero consumption up to a particular threshold and then rises linearly as consumption increases. For acute harms partially attributable to alcohol (e.g. fatal road traffic accidents), published evidence existed on the AAFs (e.g. 37% of fatal road traffic accidents for men aged 25–34 years are attributable to alcohol (Jones *et al.*, 2008)), and we use equation (35) to define absolute risks in terms of relative risks and then define

$$rr_{ij}^{h}(b_{ij}) = 1$$
 if  $b_{ij} < threshold$  (50)

$$rr_{ij}^{h}(b_{ij}) = 1 + \beta^{h} \times (b_{ij} - threshold)$$
 if  $b_{ij} \ge threshold$  (51)

where threshold = 3 units for women and 4 units for men. If we denote  $\widehat{AAF}_{j}^{h}$  as the observed estimate of the AAF from published data for subgroup j, then we estimate  $\widehat{\beta}^{h}$  as the  $\beta^{h}$  that is the solution to equation (38):

$$\widehat{AAF}_{j}^{h} = \frac{\sum_{i} R_{ij}^{h} (b_{ij}) - \sum_{i} R_{ij}^{h} (b_{ij} = 0)}{\sum_{i} R_{ij}^{h} (b_{ij})} = \frac{\sum_{i} rr_{ij}^{h} (b_{ij}) - \sum_{i} rr_{ij}^{h} (b_{ij} = 0)}{\sum_{i} rr_{ij}^{h} (b_{ij})}$$
(52)

which we estimated using the 'Solver' algorithm in MS Excel. For acute harms wholly attributable to alcohol, the AAF = 1 by definition and the two-part linear absolute risk function approach is fitted to the total observed rate per population of incidents  $\hat{H}_j$  for each age/sex subgroup (e.g. annual mortality rate for accidental poisoning by exposure to alcohol for men aged 25–34 years) using the same thresholds as in equations (50) and (51). That is, we estimate  $\hat{\beta}^h$  as the  $\beta^h$  that is the solution to

$$\widehat{H}_{j} = \frac{1}{N_{j}} \sum_{i} R_{ij}^{h} \left( b_{ij} \right) \quad where, \tag{53}$$

$$R_{ij}^{h}\left(b_{ij}\right) = 0 \quad if \quad b_{ij} < threshold$$
 (54)

$$R_{ij}^{h}\left(b_{ij}\right) = \beta^{h} \times (b_{ij} - threshold) \quad if \quad b_{ij} \ge threshold$$
 (55)

For chronic diseases wholly attributable to alcohol, we used the same two-part linear approach set out in equations (53) to (55) except that the chronic disease risk functions are related to mean weekly consumption  $c_{ij}$  with an assumed *threshold* for the start of rising risk of 2 units per day for women and 3 for men. Finally, for the chronic illnesses, debate exists about the time lag between change in exposure to alcohol and change

in risk (Norstrom and Skog, 2001). We made an assumption, consistent with average estimates from literature, that it would take 10 years for a change in consumption levels for a subgroup to achieve its full effect in terms of reduced risk. For intervening years, we assume a linear trend towards full effect at year 10, so that at year 1 the risk reduction is 1/10 of full effect, at year 2 it is 2/10 etc. Thus, following the notation of equation (53), if we denote the risk 10 years after an instantaneous change in consumption from  $c_{ij}$  without the policy to  $c_{ij}^*$  following the policy as  $R_{ij}^{h,(9,10)}$ , then the risk in an intervening period (t,t+1) is given by

$$R_{ij}^{h,(t,t+1)}\left(c_{ij}^{*}\right) = \frac{t+1}{10}R_{ij}^{h}\left(c_{ij}^{*}\right) \tag{56}$$

or equivalently,

$$PIF_{j}^{h,(t,t+1)} = \frac{t+1}{10} PIF_{j \text{ fulleffect}}^{h}$$
(57)

- 2.3.7. Consumption to crime harms. Crime was assumed partially attributable to peak alcohol consumption. Again, two-part linear RR functions were fitted (as per equations (51) and (52)). We derived AAFs for 20 classifications of crimes (in six broad areas: violent disorder, wounding, assault without injury, vehicle-related thefts, burglary/robbery/other theft and criminal damage), using OCJS England and Wales 2005 data, and questions that ask convicted offenders whether they undertook the offence because they were drunk (Home Office Research, 2011). Reported crime rates were uplifted for under-reporting and apportioned into population subgroups using separate data on offenders found guilty or cautioned (2005). RR functions were estimated for men and women and for two age groups, under 16 years and between 16 and 25 years separately, and we assumed relative risks for over 25 s to be the same as those for 16–25 s.
- 2.3.8. Consumption to employment and work absence harms. The government had analysed work absence and unemployment due to alcohol (Health Improvement Analytical Team, 2008). We extended this, assuming unemployment was only a risk for people who drink at harmful levels, and that there is no time delay between change in prevalence of consumption and change in risk of not working. We used the two-part linear approach to fit unemployment RR functions (using thresholds defining harmful drinkers). The AAF for unemployment was estimated based on a study (MacDonald et al., 2004), which examined Health Survey for England data for men aged 22 to 64 years and found that being a 'problem drinker' (defined using psychological/physical symptoms or quantity/frequency of consumption) reduced the probability of being in work by 6.9%. We assumed the same figure for women but adjusted taking account of differential work participation rates. Total observed absolute unemployment figures were taken from the Labour Force Survey 2006. If we denote  $\widehat{AAF}_j^h$  as our estimate of the observed unemployment AAF for subgroup j, then we estimate  $\widehat{\beta}^h$  as the  $\beta^h$  that is the solution to the adapted equation (37)

$$\widehat{AAF}_{j}^{h} = \frac{\sum_{i} rr_{ij}^{h} \left(c_{ij}\right) - \sum_{i} rr_{ij}^{h} \left(c_{ij} = 0\right)}{\sum_{i} rr_{ij}^{h} \left(c_{ij}\right)}$$

$$(58)$$

$$rr_{ij}^{h}\left(c_{ij}\right) = 1$$
 if  $c_{ij} < threshold$  (59)

$$rr_{ij}^{h}\left(c_{ij}\right) = 1 + \beta^{h} \times (c_{ij} - threshold)$$
 if  $c_{ij} \geq threshold$  (60)

where threshold = 5 units per day for women and 7.1 units per day for men.

Work absence, like crime, was assumed partially attributable to alcohol and related to peak alcohol consumption. The two-part linear approach fitted relative risk functions for each age/gender group (equations (50) to (52)). AAFs for absenteeism were derived from an Australian study (Roche *et al.*, 2008), the only one identified, which examined the causal relationship between alcohol and absence from work. Baseline absent days for each age/gender subgroup were obtained from Labour Force Survey 2006.

2.3.9. Valuation. We estimate the direct financial effects of policies on consumers' spending, retailer and government revenues. We quantify the overall change in the volume of units of alcohol sold, that is,  $c_{ijk}^*$  from equation (28) for each modelled individual, and the associated mean price paid for the beverage category by that individual's drinker/age/gender subgroup  $\overline{\mathbf{p}}_{i}^{*}$  from equation (26). Finally, we multiply up to the England population to quantify the associated value of sales (in £s). To assess changes to government revenues, the sales value for each beverage type is apportioned into money to the retailer, alcohol duty to government and valueadded tax (VAT). Monetary valuation of health harms includes the direct cost incurred in providing treatment or services, calculated by applying annual National Health Service (NHS) cost of treating diseases attributable to alcohol (Health Improvement Analytical Team, 2008) to the number of hospital admissions for each disease (from Jones et al., 2008). Monetary valuation of the population quality-adjusted life years was undertaken based on utilities extracted from the Health Outcomes Repository database (Health Outcomes Data Repository, 2011), which measures the EQ-5D by condition around 6 weeks after hospital discharge. In our work for the Department of Health, and under their instruction, a quality-adjusted life year was valued at £50 000 and discounted at 1.5%, while health care costs were discounted at 3.5%. In the work for NICE, quality adjusted life years (QALYs) were valued at £20 000 with discount rates of 3.5% for both costs and QALYs. Monetary valuation of crime harms used unit costs of crime (Brand and Price, 2000; Dubourg et al., 2005). The harm to the victim of a crime was also taken into account through the impact on quality of life (Dolan et al., 2005), assuming the financial value of a crime victim QALY in this case to be £81 000 (Carthy et al., 1998). The valuation of workplace absence due to sickness and unemployment was quantified based on average earnings in each age/gender group.

### 3. RESULTS FRAMEWORK

Model results for a 50p minimum price per unit policy versus current pricing can be shown for all England and separately for moderate, hazardous and harmful drinker subgroups (Table I). Estimated changes in mean consumption are shown, also split by beverage type, as requested by policy-makers. Mean change in purchase spending per year is shown, both on-trade and off-trade, alongside changes in government (duty and VAT) and retailer revenues. For health, crime and workplace harms, the model results detail the volume of annual incidents, for example, reduction in violent crimes or reduction in deaths due to road accidents, and these are summarised more broadly, for example, acute/chronic disease mortality and hospitalisation rates. Monetary costs to the health service and criminal justice system are shown, as are QALYs gained, both health and crime related. Workplace harms and their monetary valuations are also shown. Policy-makers can therefore examine effects by outcome and by drinker subgroup. In a move towards a partial cost-benefit analysis approach, the monetary valuation of the harm reduction across health, crime and workplace harm is combined, allowing policy-makers an indication of the relative effects across these three sectors and a summary total.

Table II compares the summary results for 18 different policies including general price rises, price rises targeted only at low-priced products, minimum prices per unit, discounting restrictions or bans, and whatif effects of advertising, outlet density or licensing hours restrictions. Policy-makers can compare effects on consumption, purchasing, revenues and harm reductions, to see, for example, that a low threshold minimum price of 15p or 25p per unit has small effects, that the effects of a minimum price threshold accelerate as it is increased because a greater proportion of the market is affected, that banning discounts over 50% (buy one get one free offers) has minimal effects, and that all of these policies affect harmful drinkers differentially from moderate drinkers (see Meier *et al.*, 2010; Purshouse *et al.*, 2009a; and Purshouse *et al.*, 2010b for detailed results and implications for policy).

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Table I. Model results for a 50p minimum price per unit policy scenario (see Purshouse et al., 2009a)

			Hazardous 18–24 years	Moderate All ages	Hazardous All ages	Harmful All ages	England total
Consumption							
Mean consumption per person per w	eek		-0.70	-0.18	-1.49	-7.05	-0.84
Mean consumption per drinker per w			-0.70	-0.22	-1.49	-7.05	-1.05
% Change in mean consumption			-2.6%	-3.8%	-5.4%	-10.1%	-6.7%
Baseline mean weekly consumption	(units per	person)	27.10	4.67	27.35	69.70	12.63
Change in volume of consumption	On-trade	Beer	-43.80	-4.34	-54.49	-278.47	-37.80
(Units per drinker per year)	on trade	Wine	-16.37	-7.20	-27.18	-82.98	-17.60
(Cinis per drimer per year)		Spirit	-30.73	-4.78	-35.12	-114.11	-20.33
		RTD	0.09	0.00	0.00	-0.40	-0.03
	Off-trade	Beer	49.21	4.77	37.81	106.05	20.28
	OII trade	Wine	0.46	0.00	0.56	0.56	0.17
		Spirit	3.15	0.22	0.60	1.06	0.37
		RTD	1.32	0.07	0.21	0.73	0.15
Purchasing							
Baseline value of purchasing (£ per of	drinker per	year)	£1538.59	£275.66	£1070.38	£2447.79	£633.77
	•		612.21	06.60	025 (2	067.27	617.07
Change in value of purchasing		Off-trade	£13.21	£6.60	£35.62	£67.37	£17.87
(£ per drinker per year)		On-trade	£69.88	£6.46	£47.21	£127.92	£25.30
		Total	£83.09	£13.06	£82.82	£195.29	£43.17
Total change in retailer revenue		Off-trade	£17.92	£157.84	£319.58	£305.15	£784.33
(£m after duty and VAT)		On-trade	£37.01	£93.30	£232.59	£226.39	£553.13
		Total	£54.93	£251.14	£552.18	£531.54	£1,337.47
Total change in VAT and duty receiv	ed (£m)		£3.97	£1.15	-£2.97	-£65.17	-£66.93
Health conditions and health service	es.						
Deaths due to alcohol-related		Chronic	-1	-27	-967	-1759	-2754
diseases (per annum at full effect)		Acute	-1	-89	-140	-76	-306
Alcohol-related hospital admissions		Chronic	-98	-9202	-21 795	-52 017	-83 025
(per annum at full effect)		Acute	-157	-4562	-6419	-3611	-14633
Monetary costs (£m per annum at fu	ll effect)		-1	-57	-100	-144	-302
QALYs gained by policy (per annum	at full eff	ect)	82	4615	7664	10 563	22 859
Alcohol-related crime							
Change in the number of offences (p	er annum)		-4068	-3694	-17381	-17 799	-42 523
Costs (£m)	<i>'</i>		-6	-6	-20	-21	-49
QALYs gained by policy			79	87	308	350	774
Unemployment and absences							
Unemployment (per annum)		Volume	0	0	0	-25905	-25905
r - 7 (r		Cost (£m)	0	Ö	0	-631	-631
Work absence (per annum)		Volume	-26 622	-89 813	171 150	-179 457	-442 273
<i>u</i> ,		Cost (£m)	-2	-9	-16	-18	-44
Total cumulative discounted costs (y	ears 1 to 1	0)					
Total change in health care costs + v			-20	-746	-1222	-1579	-3550
Total change in crime costs + value of	of QALYs	(£m)	-61	-62	-215	-236	-537
Total value of changes to employment	nt (£m)		-13	-74	-136	-5398	-5608
Total (£m)			-94	-881	-1573	-7213	-9695

Table II. A comparison of modelled outcomes for a range of policies

	Change	Change consumption (%)	(%	Change	Change in purchasing (%)	(9	Change in purchasing (£m)	chasing (£m)	Change	Change in harm (per year/after 10 years)	fter 10 years)	Change in ha	Change in harm spending (£m per year/after	per year/after
	England Total	Moderate	Homeful	England Total	Moderate	Homeful	Datailar	Dute t VAT	Danthe	Haolib OAIVe	Crima OALV.	Hanlih	Crima	Employment
Policy scenario	Dilgiand 10tal	drinkers	drinkers	England total	drinkers	drinkers	revenue	revenue	Dealis	nearini CALLS	Cillie CALIS	пеаш	CIIIIe	Emproyment
General price +10%	-4.2	-3.5	-4.5	5.7	6.7	5.2	£1051.70	£4.54	77.7121—	-12 366.65	-1621.66	-£161.65	-£98.15	-£337.73
General price +25%	-10.9	-8.8	-11.7	12.2	15.4	10.8	£2337.36	-£54.58	-3785.61	-30732.92	-4143.21	-£401.87	-£250.43	-£853.33
Low-priced off-trade products +25%	-0.9	-0.5	-1.4	1.6	1.0	2.0	£284.91	£8.40	-524.80	-3812.94	5.79	-£49.72	-£0.77	T£77.97
Low-priced on-trade products +25%	-0.6	9.0—	-0.4	1.4	1.6	1.5	£249.35	£17.30	-133.71	-1370.61	-718.04	-£19.72	-£48.75	-£13.87
All low-priced products + 10%	-0.6	-0.5	-0.7	1.4	1.1	1.6	£239.14	£14.57	-265.30	-2092.39	-288.34	-£27.94	-£20.03	-£37.33
Minimum price 15p (off-trade and on-trade)	0.0	0.0	0.0	0.1	0.1	0.2	£21.47	£3.54	-0.88	-5.18	21.09	-£0.30	£1.11	-£1.53
Minimum price 25p (off-trade and on-trade)	-0.1	0.0	-0.5	0.8	0.4	1.2	£137.12	£19.79	-47.36	-330.03	64.40	-£5.36	£3.28	-£36.74
Minimum price 50p (off-trade and on-trade)	-6.7	-3.8	-10.1	8.9	4.7	8.0	£1337.47	-£66.93	-3060.23	-22858.74	-773.53	-£301.50	-£49.01	-£674.35
Minimum price 70p (off-trade and on-trade)	-17.5	-11.5	-22.7	10.2	8.7	10.6	£2295.23	-£387.66	-7262.95	-55083.73	-2333.82	-£734.57	-£144.62	-£1338.66
Minimum price 40p off and 100p on Trac	-3.4	-2.0	-5.5	6.4	8.4	7.6	£1142.37	£54.97	-1568.06	-12081.34	-898.44	-£162.34	-£56.11	-£380.73
30p minimum price beers only	-0.2	0.1	6.0—	1.0	0.5	1.3	£168.53	£25.98	T2.77	-349.84	10.34	-£6.11	£0.77	-£83.64
Ban off-trade discounting if > 50%	0.0	0.0	0.0	0.0	0.0	0.0	£0.98	-£0.05	-2.34	-17.55	-0.02	-£0.23	-£0.01	-£0.29
Ban off-trade discounting if > 20%	-0.8	-0.5	8.0—	9.0	0.5	9.0	£116.37	-£11.10	-334.69	-2563.34	-95.93	-02.73	-£5.91	-£56.75
Total ban off-trade discounting	-2.7	-1.9	-3.0	2.0	1.6	2.2	£411.86	-£43.76	-1163.53	-8932.99	-401.54	-£115.34	-£24.40	-£210.58
Ban off-trade discount if reg price <30p	0.0	0.0	-0.1	0.1	0.1	0.2	£18.91	£2.38	-10.75	-80.44	-0.83	-£1.15	-£0.06	-£7.51
Total advertising ban	-26.9	-26.9	-26.9	-26.9	-26.9	-26.9	-£3316.38	-£1708.74	-8233.53	-69 551.49	-10345.33	-£926.52	-£620.79	-£1828.96
10% reduction in outlet density	13.2	11.5	14.1	8.6	6.7	12.4	£1114.85	£715.02	5219.57	42 560.96	4454.37	£559.03	£256.34	£1217.50
10% reduction in licensing hours	-12	-12	-12	2	- 1	- 1	-£146.49	-£75.48	-417.42	-3463.39	-466.20	-£45.32	96 LC3-	-£04.07

Sensitivity analyses undertaken were mostly on parameter values, for example, changing the time lag assumption for chronic health harms from a 10-year base-case to 5 or 15 years (Purshouse et al., 2009a). Our most complex sensitivity analyses relate to price elasticities. These included three analyses adjusting the basecase elasticity matrices for the two groups (moderate drinkers, and hazardous/harmful drinkers combined): (i) adjusting for age/gender by weighting cross-price elasticities differentially given each age/gender subgroup's buying preferences for different beverage types; (ii) adjusting purchasing data to account for observed differences between some age/sex subgroups purchasing data (EFS) and their consumption (GHS), for example, beer bought by women but consumed by men; and (iii) a what-if analysis in which heavy drinkers are assumed to be one-third less responsive to price changes than moderate drinkers (Chisholm et al., 2004). We also examined using long-run UK elasticity estimates based on high-level time series data (Huang, 2003), in which both own-price and cross-price elasticities are greater than those derived from EFS. Finally, we undertook probabilistic sensitivity analysis to explore uncertainty in the coefficients of the base-case elasticities and showed that this uncertainty is much less significant than the structural assumptions described earlier. The results of these sensitivity analyses demonstrated that the main themes of the model findings, for example, that higher thresholds for a minimum unit price have accelerated effects on harmful drinker consumption and harm reductions, were robust to the methods of estimating price elasticities (full details are in pp. 135-139 of Purshouse et al., 2009a).

Some model outputs were able to be compared against external data or other studies. Firstly, we modelled a scenario of zero alcohol consumption in the population and compared against government reports (Department of Health, 2008; Jones *et al.*, 2008), and results showed the same order of magnitude for annual for alcohol-attributable mortalities (SAPM 11 641 versus Jones *et al.* 2008 11 169<sup>1</sup>), NHS costs per annum (SAPM £3.1bn versus DH report £2.8bn), financial value of work absence (SAPM £1.4bn versus DH report £1.4 to £2.0bn), and unemployment (SAPM £2.6bn versus DH report £2.0–£2.5bn). There were differences in crime financial valuations between SAPM and government reports, explainable by our updating evidence on lower AAFs and our exclusion of lost output due to premature deaths for homicide. Secondly, we cross-checked the most important emergent model parameter against published literature estimates. The overall price elasticity from recent meta-analyses found a median price elasticity of demand for alcohol of -0.535 (Gallet, 2007) and a mean price elasticity for alcohol of -0.51 (Wagenaar *et al.*, 2009). This is compared with an overall effect within our model for a 1% general price rise of around -0.42, showing a very similar magnitude to external data.

#### 4. DISCUSSION

In the UK, the model has been influential in policy development, analysis and debate. In England, having considered evidence including our 2008 report, the Chief Medical Officer called for a 50p minimum unit price. The UK government has since proposed and consulted on minimum unit pricing (HM Government, 2012) and is now planning to implement a policy of no sales of alcohol below the costs of duty plus VAT – a policy with an order of magnitude smaller effect than a 45p minimum unit price (Meng *et al.*, 2013a; Meng *et al.*, 2013b). In Scotland, a policy to restrict price discounts was approved in 2010 (Scottish Government, 2010), and since the 2011 election in Scotland, a minimum price bill has been passed (Scottish Government, 2012). The model was used during this decision-making process, and the adaptation to Scotland required the use of different but very similarly structured datasets on baseline consumption, mortality, hospitalisations and crime (Purshouse *et al.*, 2009c; Meng *et al.*, 2010; Meng *et al.*, 2012). The proposal for a 50p per unit minimum price in Scotland is now being contested in European courts where 'proportionality' of the pricing policies as defined under EU law will be an issue; that is, is the degree of interference in the market defensible given the potential benefit to public health? The narrative of these policy debates has focused on impacts on subgroups of interest

<sup>&</sup>lt;sup>1</sup>14 982 additional deaths caused by alcohol on p. 13 of Jones *et al.*, 2008, minus the 3813 reduced mortalities saved by positive effects of alcohol on p. 26 of Jones *et al.*, 2008.

(e.g. low-income drinkers, responsible drinkers, heavy drinkers and young drinkers), and our detailed framework provides an integrated assessment of the estimated health, crime, workplace and spending outcomes for these subgroups.

Since the original framework was developed, we have engaged in adaptations to other interventions and other countries. Within England, the framework can be fairly easily applied to any intervention where there is an effect on alcohol consumption levels; for example, we have been able to adapt the model to produce estimates of the cost-effectiveness of screening and brief interventions for alcohol in a variety of different settings (Purshouse *et al.*, 2009b). Country adaptations beyond England and Scotland have also been undertaken. The model was adapted to model the minimum pricing policies in two provinces in Canada (Hill-McManus *et al.*, 2012), and the screening and brief intervention modelling to the Netherlands, Italy (Angus *et al.*, 2014) and Poland, all of which have proven feasible, with the key issue being each country's existing datasets around consumption, mortality, hospitalisations, crime and employment.

There remain limitations in the evidence base to inform the model. Firstly, an ideal dataset comprising both price and consumption data collected longitudinally on individuals is not available in the UK. This meant that the econometric modelling used within SAPM 2.0 followed a relatively simple approach. We have recently developed a 'pseudo-panel' approach, and future policy analyses will use new elasticities (Meng *et al.*, 2014). Secondly, the GHS consumption dataset measures peak consumption by asking respondents about the heaviest drinking day in the last week, and we recognise that evidence that combines both frequency and consumption on heavier drinking occasions would be useful (Meier *et al.*, 2013; Stockwell *et al.*, 2004). Thirdly, there are several limitations in the harms evidence including whether different illnesses may have different time lags after consumption changes, how each type of harm is related to consumption (e.g. suicide or heart disease may be caused by a combination of mean and peak consumption), what the threshold is above which risk begins to increase for acute harms, and what the proportions are of crime and workplace outcomes that are attributable to alcohol. A deeper discussion of alternative and potential developments to our model framework (e.g. individual level simulation of disease incidence), of the several monetary valuation issues, and of the limitations of data and evidence is given in online appendix web reference.

#### 5. CONCLUSION

In conclusion, we have developed a general framework for alcohol policy modelling, which utilises baseline alcohol consumption from large-scale surveys, econometric modelling for drinker population subgroups, and quantified relationships between levels of (mean and peak) alcohol consumption and attributable harms in three domains: health, crime and workplace. A broad valuation of harms analysis, applying financial costs to each type of harm, provides estimates of total financial value of the harms avoided by different policies. The framework will develop further as research progresses and, we hope, continue to be useful for evaluating strategies to reduce alcohol-related harm.

# CONFLICT OF INTEREST

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#### ORIGINAL PUBLICATION

Aspects of this work have been previously published or submitted to journals either in the form of reports to government agencies or as peer-reviewed journal articles. All of these are referenced within the text. The key new work here is the clear mathematical description of the methods – this has not been shown or submitted elsewhere.

#### **ETHICS**

The study is based mostly upon synthesis of published literature and available data. Formal ethical approval via NHS ethics committees was not needed. Where required for each data source, approvals were obtained, and confidentiality contracts agreed.

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