ECONOMIC EVALUATION OF INTERVENTIONS FOR PROBLEM DRINKING AND ALCOHOL DEPENDENCE: DO WITHIN-FAMILY EXTERNAL EFFECTS MAKE A DIFFERENCE?

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Abstract — Aims: To propose methods for the inclusion of within-family external effects in clinical and economic evaluations. To demonstrate the extent of bias due to the exclusion of within-family external effects when measuring the relative performance of interventions for problem drinking and alcohol dependence. Methods: The timing and magnitude of treatment effects are modified to accommodate the external health-related quality of life impact of having a problem or dependent drinker in the family home. Results: The inclusion of within-family external effects reduces cost per QALY estimates of interventions for problem drinking and alcohol dependence thereby improving the performance of all evaluated interventions. In addition, the inclusion of within-family external effects improves the relative performance of interventions targeted at those with moderate-to-severe alcohol dependence as compared to interventions targeted at less severe alcohol problems. Conclusions: Failure to take account of external effects in clinical and economic evaluations results in an uneven playing field. Interventions with readily quantifiable health benefits (where social costs and benefits are predominantly comprised of private costs and benefits) are at a distinct advantage when competing for public funding against interventions with quantitatively important external effects.

INTRODUCTION

Even within a relatively narrow set of interventions, failure to take account of all costs and consequences is likely to bias performance measures and may result in an inefficient allocation of resources. In relation to interventions for problem drinking and alcohol dependence, the incidence of alcohol-related violence (Graham and West, 2001), the incidence of alcohol-related road-trauma (ATSB, 2002) and numerous findings as to the educational and psychological effects on the children of heavy drinkers (e.g. Drake and Vaillant, 1988; Bush et al., 1995; Velleman and Orford, 1999; Christoffersen and Soothill, 2003) suggest that a major part of the disease burden is borne by family members and the wider community. To date, attempts to calculate the burden of disease (BoD) attributable to alcohol use have not separated out the share of disability-adjusted life years or quality-adjusted life-years foregone due to external effects. The global BoD attributable to alcohol use calculated by the WHO did, however, include a number of disease categories with non-negligible external effects including conditions arising during the perinatal period, unintentional injuries such as motor vehicle accidents, and intentional injuries such as alcohol-related violence (see Ezzati et al., 2004; Rehm et al., 2004). While the burden attributable to such conditions (30% of all alcohol-attributable DALYs) has a private as well as an external component, it should be emphasized that the WHO study failed to include the full range of external effects and that the BoD calculations provide a relatively poor guide of the avoidable burden due, for example, to a transition from problem drinking or alcohol dependence to safe consumption patterns.

Estimating the total (private or ‘first-person’ plus external) health gain associated with moderation of problem drinking or recovery from alcohol dependence (the avoidable BoD attributable to problem drinking or alcohol dependence rather than to alcohol use more generally) requires evaluation over a much larger set of ‘soft’ outcomes including family disharmony, educational outcomes, and psychological distress. The complexity and data requirements of ‘first-best’ and ‘second-best’ approaches to calculating the avoidable burden have inevitably led to the routine exclusion of external effects from clinical and economic evaluations. The aim of the current study is therefore to propose a simple, direct, and transparent approach to provide an indication of the likely importance of external effects for interventions for difficult social problems such as alcohol and substance misuse. Issues surrounding the interpretation of relative performance indices are then discussed by comparing base case cost per QALY estimates against cost per QALY estimates after the inclusion of within-family external effects.

EXTERNAL EFFECTS

The nature of the relationship between an intervention (A) and health outcomes (G) is illustrated in Fig. 1 below. Intervention A is designed to modify lifestyle D. Any change in lifestyle may potentially influence health in several ways:

(i) Directly—For instance when a person feels better when they moderate their drinking behaviour through improvement in cognitive function and activities of daily living (such impacts will be contemporaneous with the intervention);

(ii) Indirectly and over time through disease pathways (F)—Usually mediated through changes in observable clinical parameters (E). An example is moderation of drinking behaviours resulting in a reduced risk of liver cirrhosis, pancreatitis, and certain types of cancer including liver cancer, breast cancer, and colon cancer (Grønbæk, 2001) thereby lowering the risk of disability and specific mortality, or via secondary prevention, where cessation of alcohol consumption in persons with existing liver...
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Fig. 1. Relationship between intervention, lifestyle, mortality and quality of life.

cirrhosis, pancreatitis, or cancer reduces the risk of complications;

(iii) *Inversely*—Observed behaviour may be influenced by a change in health status which in turn results in a further deterioration in health status. For instance, an exogenous decline in health status (such as loss of mobility or increased pain) may lead to a change in lifestyle (such as increased alcohol consumption) and a second-order decline in health status;

(iv) *Via externalities*—Health impacts from the behaviour change of an individual may accrue to others. For example, alcohol misuse (or adoption of safe drinking behaviours) can have major consequences for family members and others in the community. Such impacts may be direct and contemporaneous (as in foetal alcohol syndrome, domestic violence, and road trauma), or mediated through disease (as in the effect of alcoholic dementia on family).

The availability of data describing the direct, indirect, and inverse inter-relationships between alcohol consumption and health status of the individual problem drinker makes the inclusion of these effects in clinical and economic evaluations a relatively straightforward matter. The inclusion of external effects, on the other hand, is problematic because of a lack of data and the complexity of the relationship between problem drinking and the health and well-being of others. The exclusion of external effects is reasonable in situations where they are not quantitatively important, relative to the health impact on the individual. However, in relation to interventions for problem drinking and alcohol dependence, a focus entirely on the individual is inappropriate because the evidence suggests that a major part of the disease burden is borne by family members and the wider community.

The importance of within-family external effects has been extensively documented in the literature. The children of alcoholics are subject to an increased risk of hyperactivity, psychomotor delays, short attention, and ‘acting out’ (Aronson et al., 1985). An increased incidence of child abuse and neglect has been observed in families where there is evidence of alcohol abuse (Reich et al., 1988). Velleman and Orford (1999) found that the children of problem drinkers were significantly more likely than a comparison group to have ‘experienced disharmony, often involving domestic violence, in their families of upbringing’ (Velleman and Templeton, 2003 p. 105). Velleman and Orford (1999) suggested that because of family disharmony, the ‘children of parents with a drinking problem are at significant risk of a range of emotional, conduct, and learning problems whilst they are living at home and in contact with the problem drinking parent’ (Velleman and Templeton, 2003 p. 105). Similar findings have been reported elsewhere in the literature (e.g. Drake and Vaillant, 1988; Bush et al., 1995; Christoffersen and Soothill, 2003). More generally, ‘family members of all age groups (children, partners, siblings, parents and other close relatives) are often negatively affected. The result is that family members commonly develop problems in their own right, often developing high levels of physical and psychological symptoms’ (Velleman and Templeton, 2003 p. 108).

External effects beyond the family unit may also be quantitatively important when calculating the burden attributable to problem drinking and alcohol dependence, and the burden avoided due to intervention. External effects beyond the family unit are manifest in indicators such as the incidence of alcohol-related violence and road-trauma. For example, alcohol and/or drugs were a major factor in an estimated 30% of fatal road-crashes between 1988 and 1998 on Australian roads (ATSB, 2002). Thirteen percent of drivers and motorcycle riders involved in fatal road-crashes on Australian roads in 1998 had a blood-alcohol concentration (BAC) ≥0.050 gm/100 ml (ATSB, 1998) and ~6% of drivers and motorcycle riders involved in pedestrian deaths on Australian roads in 1992 had a BAC ≥0.050 gm/100 ml (FORS, 1997). Given the availability of administrative data from police reports and insurance claims, it is a relatively simple matter to then calculate the burden of alcohol-related road trauma attributable to alcohol use as compared to abstinence. However, identifying the burden of alcohol-related road-trauma that would be avoided due to intervention is a much more complex task. Whereas the risk of ICD-10 dependence is increasing in both the level of annual consumption and the frequency of heavy (>5 drinks/day) drinking, the risk of drunk driving is substantial even at an average consumption of less than one drink per day (Midanik et al., 1996). Moreover, the risk of drunk driving is invariant to increases in the frequency of heavy drinking from less than once per week (<50 days/year) to nearly every day (>300 days/year) of the year (Midanik et al., 1996). It is therefore less clear that the risk of alcohol-related trauma is elevated for dependent drinkers as compared to those drinking within national guidelines because, for many people, it is possible to drink heavily on a weekly basis and yet still remain within national guidelines.

Likewise, the risk of alcohol-related violence depends on a range of situational factors as well as on the level and pattern of alcohol consumption (Parker, 1993). Much of the burden of alcohol-related violence is borne by young, single, males with a highly variable consumption pattern who are more likely to be weekend binge-drinkers than physically dependent...
This is confirmed by Australian data with regard to the context of alcohol-related violence. Teece and Williams (2000) found that 68% of victims of alcohol-related physical assaults in the 1998 Australian National Drug Strategy Household Survey (NDSHS) reported that the assault occurred at a location outside the family home (such as licensed premises, a public thoroughfare, place of employment, or educational institution). Moreover, about 80% of all types of alcohol-related violence (including verbal assault, physical assault, and being ‘put in fear’ by an alcohol-affected person) as reported in the 1998 NDSHS were perpetrated by a workmate or schoolmate (5.9%), friend or acquaintance (17.6%), or a person unknown to the victim at the time of the incident (56.1%). Male victims of alcohol-related violence were significantly more likely than female victims to identify a location outside the family home (χ² = 32.14, P < 0.000) and the perpetrator as a workmate or schoolmate (7.7% vs 4.0%, P < 0.01) or person unknown to them (64.0% vs 47.1%, P < 0.000). While the burden of alcohol-related violence in the wider community is no less important than within-family external effects, estimating the share of that burden that is avoidable through interventions for problem drinking and alcohol dependence is difficult where situational factors dominate.

METHODS

A time-dependent state-transition model was used to estimate QALYs gained per person for interventions for problem drinking and alcohol dependence as compared to usual care. The characteristics of the interventions, the structure of the model, and results from the base case analysis are detailed in a companion paper (Mortimer and Segal, 2005). Briefly, the modelled cost-utility analysis for base-case analysis was based on a common structure, adapted to reflect the characteristics of the target population for each intervention. The following assumptions were common across all evaluated interventions:

- **Time-dependent state-transition model.**
- **Cycle length = 6 months** (except naltrexone model where cycle length = 3 months).
- **Modelled out to full life-expectancy.**
- **Health-related Quality of Life (HRQoL) gain directly attributable to behaviour change varies depending on severity of alcohol problems as per disability-weights from Stouthard et al. (1997) such that returning problem and dependent drinkers to a ‘safe consumption’ pattern is assumed to imply annual QALY-gains of 0.110 and 0.330, respectively.**
- **Mortality differential based on Rehm et al. (2001).**
- **Exclude downstream cost-offsets in the base-case analysis.**
- **Annual discount rate of 5% applied to both costs and health gains.**

Building on this common-structure, a number of intervention-specific assumptions were made to reflect differences in the magnitude and persistence of treatment effect for each intervention and to specify important characteristics of the relevant target population (e.g. with respect to age and severity of alcohol misuse). For example, a time-dependent state-transition model with seven non-absorbing and one absorbing state (dead) was used to estimate QALYs gained per person for the brief interventions evaluated by Wilk et al. (1997). The seven non-absorbing states comprised a tunnel sequence of three problem drinking states (problem1, problem2, and problem3), a tunnel sequence of three moderate drinking states (moderate1, moderate2, and moderate3) and a single ‘dependence’ state to capture the differential rate at which problem drinkers might progress to dependence in the absence of intervention or where brief intervention is ineffective. There is no ‘abstinent’ state, because all subjects in the pooled sample from the Wilk et al. (1997) meta-analysis were problem drinkers on entry to the trials and because the only outcome measure is a moderation of consumption rather than abstinence. Because brief interventions are ineffective in treating physical dependence on alcohol, the model structure does not permit recovery from ‘dependence’ to either ‘problem’ or ‘moderate’. Due to gender differences in pooled estimates of treatment effect and the availability of supporting data by age/sex band, the model was run for men and women separately.

In contrast, the model used to estimate QALYs gained per person for the naltrexone plus counselling intervention included just six non-absorbing and one absorbing state (dead). The six non-absorbing states comprised a tunnel sequence of three dependence states (dependence1, dependence2, and dependence3) and a tunnel sequence of three recovered states (recovered1, recovered2, and recovered3). There is no ‘abstinent’ state because relapse was defined as a return to consumption of five or more drinks in a day for males and four or more drinks in a day for females rather than a lapse in abstinence. Neither ‘moderate’ nor ‘problem’ states appear in the model because all subjects in the pooled sample from the Streeton and Whelan (2001) meta-analysis were dependent drinkers on entry to the trials. All persons in the model population therefore commence in the ‘recovered1’ state. The risk of death is elevated for persons characterized as ‘recovered’ (but lower than for ‘dependence’) and a return to either ‘abstinence’, ‘moderate’, or ‘problem’ state is not permitted because it is difficult to completely undo the damage done during dependence (Rehm et al., 2001).

This paper further modifies the common structure of the model to accommodate the external HRQoL impact of having a problem or dependent drinker in the family home. The cost of alcohol-related trauma on those outside the family unit would ideally also have been included to more accurately reflect the benefits of moving from problem drinking or alcohol dependence to safe consumption patterns. For reasons outlined above (i.e. difficulties in estimating the avoidable burden of alcohol-related violence in the wider community and alcohol-related road-trauma) we limit consideration to within-family external effects as a first step.

External effects within the family unit are calculated for the average number of persons per household in the target population. While the average number of persons per household might plausibly be expected to vary according to alcohol consumption, evidence from the 1995 Australian National Nutrition Survey (ABS, 1995) identified only trivial differences in the average family size of abstinent, moderate, and heavy drinkers. The average family unit is therefore assumed to comprise the treated individual plus an average of two other
persons with a maximum possible annual HRQoL-weight of 3.0 in the event of full health for all three individuals. This is consistent with assumptions made elsewhere in the literature when estimating the social impact of problem drinking and alcohol dependence (e.g. Velleman and Templeton, 2003 p. 103).

While it is clear that problem drinking and alcohol dependence is associated with substantial deleterious effects on family members, the size of this effect cannot as yet be established from the literature. In the absence of quantitative estimates, within-family external effects are routinely excluded and the implicit assumption is made that living with a problem or dependent drinker has no impact on family members. We contend that falling back on this implicit assumption of a null effect is not appropriate and that second-best and third-best approaches to incorporating within-family external effects are preferable.

The second-best approach would entail mapping HRQoL-weights to various categories of physical assault/psychological distress and to combine this mapping with the relative risk of each category of physical assault/psychological distress for persons living with a problem or dependent drinker as compared to controls. However, the HRQoL impact of living with a problem or dependent drinker is multi-faceted and would be difficult to capture by combining the HRQoL effects of proximate events such as abuse, neglect, and educational outcomes. Moreover, the potential importance of contextual factors and individual characteristics in moderating both immediate, as well as more distal, consequences complicates attempts to estimate the avoidable burden. Finally, the data requirements of performing a mapping over relevant events and health states are such that the second-best approach would entail a series of assumptions to arrive at a final estimate of HRQoL-weights.

Given that the additional complexity of the second-best approach would entail parameter assumptions or expert-opinion to fill evidence gaps, we opt for a more direct and transparent approach to capture within-family external effects. The third-best approach relies on plausible assumptions with regards to the magnitude, timing, and persistence of within-family HRQoL effects. With regards to the magnitude of within-family HRQoL effects, we assume that the HRQoL weight applicable to the problem drinker is also applicable to each person in the family unit (Stouthard et al., 1997). In other words, the HRQoL impact of alcohol use on the individual is used as a proxy for the external HRQoL effects within the family unit. For example, the target population in the Wilk et al. (1997) trials is limited to ‘less severely affected drinkers who exhibit little or no alcohol dependence’ (p. 5). The external HRQoL effects are therefore likely to be relatively mild in comparison to the external HRQoL effects in individuals exhibiting alcohol dependence. Applying a disability weight of 0.110 reported by Stouthard et al. (1997) for problem drinking (equivalent to a quality weight of 0.890) to all persons in the family unit would give us 2.67 QALYs per family per year spent in the problem drinker state and an annual QALY gain of 0.33 = 3.0 – 2.67 for every problem drinker who successfully moderates his/her consumption.

In contrast, nearly all subjects in the Sellman et al. (2001) sample were mildly to moderately dependent drinkers on entry to the trials such that treatment response is operationalized as a move from ‘dependence’ to ‘recovered’. In the absence of supporting data, we make the conservative assumption that the HRQoL weight for the ‘recovered’ state is approximately equal to the HRQoL weight for problem drinkers. Applying the disability weight of 0.330 for alcohol dependence (equivalent to a quality weight of 0.670) being the simple average of disability weights for manifest alcoholism (0.550) and problem drinking (0.110) reported by Stouthard et al. (1997) to all persons in the family unit would give us 2.01 QALYs per family per year spent in the dependence state and an annual QALY gain of 0.66 = 2.67 – 2.01 for every recovered drinker. These assumptions imply that the HRQoL impact on the family increases in line with the HRQoL impact on the problem or dependent drinker.

With regard to the timing and persistence of within-family HRQoL effects, we make the conservative assumption that recovery or moderation of drinking behaviours results in an immediate improvement in first-person HRQoL but a lagged improvement in the HRQoL of family members. Likewise, we err on the side of caution and make the conservative assumption that within-family external effects persist for a relatively short duration that differs depending on the average age of the target population for each of the interventions. For example, the average age of the target population for the Moderation-oriented Cue Exposure (MOCE) intervention was 41 years such that within-family HRQoL effects persist for just 4 years until the cohort reaches 45 years of age. In contrast, the average age of the target population for the Motivational Enhancement Therapy (MET) intervention was 35 years such that within-family external effects persist for 10 years until the cohort reaches 45 years of age. In other words, modelling assumptions with regards the persistence of within-family external effects reflect the fact that the HRQoL impact on the children of problem and dependent drinkers is often naturally curtailed by a change in life-stage. Velleman and Orford (1999), for example, argue that ‘the adulthood risks run by offspring of parents with drinking problems have been over-emphasised in the past, and the resilience of the majority of such offspring overlooked’ (Velleman and Templeton, 2003 p. 106). While the HRQoL effects on the spouse or partner of problem or dependent drinkers are less likely to be naturally curtailed by a change in life-stage, the risk of separation/divorce is elevated for families with high levels of alcohol consumption [Evidence from a US time-series suggests that a 1 litre per capita increase in alcohol consumption increases divorce rates by roughly 20% after controlling for the secular trend in divorce rates (Caces et al., 1999)] (Halford and Osgarby, 1993) and break-up of the family unit might produce a similar effect. It is difficult, however, to isolate a causal link and it may simply be that marital disharmony leads to elevated alcohol consumption (rather than vice versa) but recent studies have attempted to disentangle the feedback loops and lag structure in the relationship between alcohol problems and separation/divorce (Perreira and Sloan, 2001). Nonetheless, extending even a fraction of the external HRQoL impact out to full life-expectancy is likely to result in an over-estimate of the importance of within-family external effects.

The assumptions described above were incorporated in a manner consistent with the characteristics of the relevant
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Brief interventions for problem drinking

<table>
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<tr>
<th>Intervention (Rx) vs comparator</th>
<th>Assumptions to incorporate within-family external effects</th>
<th>Study</th>
<th>S/QALY (range from sensitivity analysis)</th>
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<tr>
<td>Brief vs nil</td>
<td>HRQoL gain of 0.11 per person per year for alcprob3 → moderate2 External HRQoL effect persists from age = 30 years to age = 45 years</td>
<td>Wilk et al., 1997</td>
<td>&gt;671 (245–10 549) ≥251 (93–3448)</td>
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<td>Simple vs nil</td>
<td>HRQoL gain of 0.11 per person per year for alcprob3 → moderate2</td>
<td>Saunders et al., 1991</td>
<td>&gt;82 (30–760) ≤42 (15–262)</td>
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<td>Brief vs nil</td>
<td>External HRQoL effect persists from age = 40 years to age = 45 years</td>
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<td>≤118 (47–1104) ≥60 (24–377)</td>
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<td>Extended vs nil</td>
<td>External HRQoL effect persists from age = 35 years to age = 45 years</td>
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<td>≤282 (121–2654) ≤142 (61–900)</td>
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Psychotherapy for mild to moderate dependence

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<td>MOCE vs BSCT</td>
<td>HRQoL gain of 0.22 per person per year for dependence3 → recovered2 External HRQoL effect persists from age = 41 years to age = 45 years</td>
<td>Heather et al., 2000</td>
<td>2145 (599–∞) 1020 (285–∞)</td>
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<td>MET vs NFC</td>
<td>HRQoL gain of 0.22 per person per year for dependence2 → recovered2 External HRQoL effect persists from age = 35 years to age = 45 years</td>
<td>Sellman et al., 2001</td>
<td>3366 (679–∞) 1359 (274–∞)</td>
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<td>NDRL vs NFC</td>
<td>External HRQoL effect persists from age = 41 years to age = 45 years</td>
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<td>∞ (1001–∞) ∞ (404–∞)</td>
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Drug-therapy for detoxified patients with a history of severe physical dependence

<table>
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<th>Intervention (Rx) vs comparator</th>
<th>Assumptions to incorporate within-family external effects</th>
<th>Study</th>
<th>S/QALY (range from sensitivity analysis)</th>
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<tr>
<td>Naltrexone+ vs placebo+</td>
<td>HRQoL loss of 0.22 per person per year for recovered3 → dependence2 External HRQoL impact persists from age = 41 years to age = 45 years</td>
<td>Streton and Whelan, 2001</td>
<td>12 966 (3725–∞) 5191 (1468–∞)</td>
</tr>
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S/QALY. Discounted at 5% per annum; S, 2003 Australian Dollars; QALY, Quality-adjusted life year; MOCE, Moderation-oriented cue exposure; BSCT, Behavioural self-control training; MET, Motivational enhancement therapy; NDRL, Non-directive reflective listening; NFC, No further counseling; Naltrexone+, naltrexone plus counseling; Placebo+, placebo plus counseling; Bs, Brief interventions.

Alcprob3 denotes the third and final state in the problem drinker tunnel sequence. Moderate2 denotes the second state in the moderate or ‘safe’ drinker tunnel sequence. Similarly, dependence3 denotes the third state in the alcohol dependence tunnel sequence and recovered2 denotes the second state in the recovered tunnel sequence.

RESULTS

The evaluated interventions were selected to provide an example of the choices facing policy-makers when allocating funding for the treatment and prevention of problem drinking and alcohol dependence. Three types of interventions were considered: (i) brief interventions for problem drinking (Saunders et al., 1991; Wilk et al., 1997), (ii) psychotherapy for mild to moderate dependence (Heather et al., 2000; Sellman et al., 2001), and (iii) drug-therapy adjuvant to counselling for detoxified patients with a history of severe physical dependence. A description of each of the evaluated interventions, comparators, and target populations is provided in a companion paper (Mortimer and Segal, 2005).

Based on the modelled cost-utility analysis, the brief interventions evaluated by Wilk et al. (1997) are estimated to deliver 0.243 QALYs gained per treated male if within-family external effects are included as compared to 0.091 QALYs gained in the base case. Similarly, the inclusion of within-family external effects increases QALY gains to 0.330 per treated female from 0.125 per treated female in the base case. The average incremental cost of the brief interventions as compared to no alcohol-related treatment is unchanged at 60.98 AUD per treated person. The cost per QALY gained is therefore estimated at 251 AUD in men and 185 AUD in women.

At the lowest level of intensity, brief intervention of simple advice alone is estimated to deliver up to 0.421 QALYs gained per treated person after the inclusion of within-family external effects at an incremental cost of 14.91 AUD per treated person as compared to no alcohol-related treatment. More intensive intervention produced additional QALY gains, with the potential to deliver up to 0.757 QALYs gained per treated person after the inclusion of within-family external effects at an incremental cost of 90.03 AUD per treated person. Estimates of cost per QALY gained for initiation or escalation of the brief interventions are therefore approximately halved after the inclusion of within-family external effects as compared to the base case.

The inclusion of within-family external effects has a slightly greater impact on cost per QALY estimates obtained for psychotherapeutic interventions for mild to moderate dependence. MOCE is estimated to deliver 0.244 QALYs gained per completer after the inclusion of within-family external effects at an incremental cost of 249 AUD per completer as compared to Behavioural Self-Control Training (BSCT). The cost per QALY gained in a predominantly target population (e.g. with respect to age and severity of alcohol misuse) for each intervention. Table 1 summarizes the assumptions used to incorporate within-family external effects for each of the evaluated interventions for problem drinking and alcohol dependence.
male population with moderate dependence is estimated at 1020 AUD after the inclusion of within-family external effects as compared to 2145 AUD in the base case.

In a population with age, gender balance, and severity similar to that in the MOCE vs BSCT model, MET is estimated to deliver 0.287 QALYs gained per completer after the inclusion of within-family external effects. The incremental cost is unchanged at 389 AUD per completer as compared to usual care implying a cost per QALY gained of 1359 AUD after the inclusion of within-family external effects as compared to 3366 AUD in the base case.

Finally, naltrexone plus counselling in detoxified patients with a history of severe physical dependence is estimated to deliver 0.132 QALYs gained per completer as compared to placebo plus counselling, after the inclusion of within-family external effects. The incremental cost per completer is unchanged at 685 AUD as compared to placebo plus counselling. The cost per QALY gained for the naltrexone vs placebo comparison is therefore estimated at 5191 AUD after the inclusion of within-family external effects as compared to 12966 AUD in the base case.

Table 1 summarizes cost per QALY ratios before and after the inclusion of within-family external effects. The ratio of family to base case cost per QALY estimate for each intervention decreases slightly as the severity of the target population increases. Whereas the ratio is approximately 51/100 for the brief interventions evaluated in the Saunders et al. (1991) meta-analysis, the ratio decreases to 48/100 for the MOCE vs BSCT comparison in mild to moderately dependent drinkers and to 40/100 for the naltrexone vs placebo comparison in detoxified drinkers with a history of severe physical dependence. The Wilk et al. (1997) and Sellman et al. (2001) interventions deviate from the inverse relationship between the family: base case ratio and severity due to a lower age at commencement (e.g. Wilk et al., 1997: age = 30 years, Saunders et al., 1991: age = 40 years).

In short, the inclusion of within-family external effects improves the performance of all interventions, with the adjustment slightly greater for interventions targeted at those with moderate to severe alcohol dependence as compared to interventions targeted at less severe alcohol problems when the age of the target population is held constant.

CONCLUSIONS

This paper demonstrates a third-best approach for the inclusion of within-family external effects in clinical and economic evaluations. The substantial divergence between base case cost per QALY estimates and cost per QALY estimates after the inclusion of within-family external effects confirms that failure to take account of external effects in clinical and economic evaluations results in an uneven playing field that may distort resource allocation. Specifically, the failure to capture all relevant dimensions of health benefit in clinical and economic evaluations places interventions with readily quantifiable benefits at a distinct advantage when competing for public subsidy against interventions with quantitatively important (but difficult to quantify) external effects.

The third-best approach adopted here has the advantage of being simple, direct, and transparent. While we recognize the lack of a quantitative evidence-base for some of the assumptions underpinning the analysis, our aim is to provoke debate by highlighting the potential importance of the routine exclusion of external effects from clinical and economic evaluations. Moreover, the potential for substantial improvement in the relative performance of interventions with quantitatively important external effects should provide an incentive for researchers to fill the data gaps that preclude ready application of second-best and first-best approaches.

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