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IZA DP No. 18201

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ABSTRACT

Minimum Unit Pricing, Alcohol Consumption and Infant Health: Evidence from the Alice Springs Liquor Supply Plan*

Restricting purchases or establishing a minimum (floor) price for low-cost, high-strength alcoholic beverages is an increasingly popular policy used to address problem drinking and alcohol-related harm. We study the consumption and short-term health-at-birth impacts of the 2006 Alice Springs Liquor Supply Plan (LSP) which effectively doubled the minimum per-unit price of alcohol by prohibiting sales of large containers of wine. Net alcohol consumption per person dropped by 12%. Both price and consumption remained unaffected in control regions. We estimated a price elasticity of demand for the cheapest drink in the market as low as -0.2. While this change decreased the total volume of pure alcohol consumed, it did not achieve a key policy objective to improve infant health outcomes among babies most at-risk for alcohol-related harm. We discuss mechanisms and potential policy conclusions.

JEL Classification: I12, I140, L51

Keywords: alcohol regulation, health behaviours, birth outcomes, difference-in-difference estimation

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1. Introduction

Governments use a range of tools to regulate access to alcohol, including complete prohibition, taxation, temporal controls and demographic restrictions (Marcus & Siedler, 2015). Such regulations are typically justified by the need to reduce alcohol-related harm, including that experienced by the youngest and most vulnerable members of a community (Elder et al. 2010). Most negative health externalities of alcohol consumption are caused by heavy ("problem" or "binge") drinkers so the most effective government interventions to reduce harm may be those that target this population (Cook and Moore 2000, Thompson 2013, Pogue and Sgontz 1989).

Low-cost, high-strength alcohol beverages are often seen as an important source of problem drinking (Lewer et. al 2016). A minimum unit price ("MUP" or "floor price") on alcohol products is one regulation that primarily targets binge drinkers in low-socioeconomic groups who disproportionately consume the strongest and least expensive products (Ludbrook, 2009) (Craven, et al., 2013) (Vandenberg & Sharma, 2016) (Calcott, 2019) (Griffith, et al., 2020). In 2018, minimum unit pricing was implemented, both nationally in Scotland (Robinson, et al., 2021) and regionally in Australia's Northern Territory (Clifford, et al., 2021). It has existed in Saskatchewan, a Candian province, since 2010 (Stockwell, et al., 2012a) (Stockwell, et al., 2012b). Research on the impact of a MUP is limited but existing studies uniformly suggest that a floor price has the potential to reduce alcohol consumption (O'Donnell, et al., 2019) (Stockwell, et al., 2012a) (Stockwell, et al., 2012b) (Taylor, et al., 2021), but evidence on the impact of MUP on the externalities produced by problem drinking, such as alcohol-attributable hospital admissions (Stockwell, et al., 2013), deaths (Zhao, et al., 2013) criminal offences (Stockwell, et al., 2015) and traffic deaths (Francesconi & James, 2022), is mixed.

In this study, we fill an important knowledge gap in this literature by studying the effectiveness of MUP to reduce consumption and the externalities created for children through in-utero exposure in a highly disadvantaged region of Australia's Northern Territory (NT). We evaluate a defacto minimum unit pricing experiment in Australia that emerged through the Alice Springs Liquor Supply Plan (LSP). The LSP was introduced in October 2006 in response to high levels of alcohol-related harm in Alice Springs, the third largest town in the NT. By banning sales of large containers of wine (cask and fortified) which was by far the cheapest form of alcohol in the NT, the LSP effectively doubled the standard unit price of the cheapest available drink from A$.25 to A$.50.

Our empirical strategy takes advantage of this sudden increase to the floor price of alcohol in Alice Springs leaving other similar communities unaffected. Specifically, we use a standard difference-in-differences (DiD) approach to identify the causal impacts of the policy on alcohol consumption and infant health using sales and price data along with population birth records. We compare the differences in outcomes before and after the policy was rolled out in the Alice Springs region with the differences in outcomes between the same time periods in the Darwin and Palmerston region where no such policy was implemented by the end of 2006.

Studying the effectiveness of alcohol restrictions in the Northern Territory is of paramount policy relevance. Across Australia, the Northern Territory has the highest proportion of First Nations people (to whom we will refer to as Aboriginal[1]) among its population—an estimated 31% (78,600 people) in 2020 (ABS, 2019). Similar to the fate of First Nations peoples in other countries (Anderson, et al., 2016), they are Australia's most vulnerable population, experiencing socially determined inequalities of health (Bond & Singh, 2020). The Northern Territory is of particular international interest as it has one of the highest per capita alcohol consumption levels in the world, with high proportions of problem drinking among both Aboriginal and non-Aboriginal peoples (Skov, et al., 2010).

The Northern Territory also has a disproportionately high number of pregnant women drinking heavily during pregnancy, where harm is expected to be greatest on the unborn child. Heavy drinking during pregnancy can lead to birth defects and developmental disabilities ("Fetal Alcohol Spectrum Disorders" or FASD)[2] as well as an increased risk for miscarriage, stillbirth, preterm birth and low birth weights (Sokol, et al., 2003) (Chen, 2012) (Mamluk, et al., 2020). Substantial evidence exists on the negative longer-term consequences of excessive alcohol exposure in utero

[1] For the purpose of this study, we will use the term Aboriginal peoples, although we would like to highlight that in many places the term First Nations is preferred, Indigenous, or as in the case of Australia, Aboriginal and Torres Strait Islander people (Peters & Mika, 2017).

[2] Diagnosing FASD is a difficult task and estimates of its prevalence vary, but a meta-analysis of the literature has found that FASD affects almost eight children per 1,000 population worldwide, with prevalence rates reaching up to 11% in some countries such as South Africa (see Lange, Probst et al. 2017) and up to 12% in remote Aboriginal communities in Western Australia. Children with FASD experience poor long-term outcomes, facing increased risk of family breakdown, disrupted schooling, unemployment and alcohol and drug misuse, and a significantly higher likelihood of contact with the criminal justice system (reviewed by Popova, Lange and Bekmuradov, et al. 2011). FASD prevalence rates in correction facilities range between 11-23% in Canada, and one in three in youth detention centres in Western Australia (Bower, Watkins et al., 2018). The lifetime consequences of FASD impose a large financial burden on society. Available estimates from Canada suggest an estimated burden of FASD of $1.8 billion per annum (Popova, et al., 2016).

and after birth, including experience of neglect (Laslett, et al., 2015), lower levels of education (Mangiavacchi & Piccoli, 2017) (Hinke Kessler Scholder, et al., 2014) and earnings (Balsa, 2008) (Nilsson, 2017).

The high levels of alcohol consumption among Aboriginal women in the Northern Territory is not unique. Similar concerns have been raised for and among First Nations communities in the United States, Canada and Australia overall. A review in 2017, in Canada, found almost one-in-five First Nations women engage in binge drinking while pregnant (Popova et. al 2017). A 2002 survey of women in remote communities in Western Australia reported more than half drinking at excessive levels (Fitzpatrick et al. 2015). What is unique in a global context is the high degree of paternalism and racial discrimination with which the Northern Territory and federal governments have attempted to address the problem (Clifford, et al., 2021) (Bond & Singh, 2020). Since 1979, a great variety of alcohol restriction policies have been implemented, some locally, some jurisdiction wide, leading to a highly unstable and controversial policy landscape. Many of the policies have directly targeted Aboriginal communities (e.g. local 'dry areas', 'income quarantining') (Cobb-Clark, et al., 2023) (Doyle, et al., 2022) and their problem drinkers (e.g. 'alcohol mandatory treatment'), including pregnant women (Clifford, et al., 2021). Some have even called for the criminalisation of alcohol consumption during pregnancy, with the aim to protect the rights of an unborn child, among them the NT's former Attorney-General John Elferink.[3] Although policies, which reduce heavy drinking among pregnant women, have the potential to greatly improve infant health and other outcomes, they have to be considered in light of this controversial policy environment and the potential unintended consequences they may cause through under-mining local decision-making power and generating hard-to-monitor substitution effects (d'Abbs & Burlayn, 2019).

It is within this complicated web of policies, paternalism, and racial discrimination that the MUP effects have to be considered. In line with international findings on MUP, we find robust evidence that the elimination of the least expensive products per-unit of pure alcohol (large containers of wine) decreased the total amount of alcohol consumed in the affected area. Following these LSP restrictions, full strength beer became the leading low-cost alternative to cask wine.Despite the

[3] See ABC News Lateline, 14 March 2014. Accessed on 18 Nov 2021. https://www.abc.net.au/news/2014-03-14/nt-government-considering-rights-of-unborn-child/5320016

reduction in alcohol consumption, we do not find evidence of improvements in health outcomes for babies who were in utero during the policy rollout. In fact, the policy caused a substantial short term *decrease* in birthweight for babies of Aboriginal mothers. Birthweights fell by approximately 160g, equivalent to a 0.30 SD ($p<0.05$), leading to a significant increase in the risk of low birthweight (<2500 grams). The surprising negative impact of the MUP on birthweight is supported by tentative evidence that the policy might have shifted pregnant mothers away from consuming alcohol during pregnancy to smoking during pregnancy, a result found for Aboriginal mothers (noting that these effects were estimated with great levels of uncertainty in our study).

This observed increase in smoking is potentially an important mechanism behind the negative impact on infant health outcomes, since cigarette smoking during pregnancy is associated with pre-term birth and decreased birthweight (see Stock and Bauld 2020 for a review). Increased smoking during pregnancy may have reduced hunger and therefore the need for adequate food intake. If women substituted away from alcohol to cigarettes, then it is also possible that they substituted to other, potentially illicit substances. Other potential mechanisms behind the increased risk of low birthweight include negative health impacts from a decrease in nutrition if the LSP caused a greater proportion of the household budgets to be spent on alcohol (leaving less income available for food). However, we are not able to shed much light on the role of each of these potential mechanisms in this study.

Our results contribute to a large body of research investigating the relationship between alcohol policies, consumption and infant health.[4] Prior evaluations of public policies that are successful in reducing consumption (e.g. minimum drinking age, taxation) predominately find that lower levels of alcohol consumption during pregnancy lead to better outcomes at birth (Fertig & Watson, 2009) (Zhang 2010, Wüst 2010). We advance this literature in a few ways. First, we provide a cohesive theoretical framework that extends the Pogue and Sgontz (1989) model of alcohol taxation in the presence of abuse costs to analyse the predicted effects of a MUP for alcohol on total social welfare. Second, we add to the evidence base documenting the impacts of the policy on alcohol consumption. To date, quasi-experimental methods have only been used to document changes in consumption in Canada (see Stockwell, Auld and Zhao 2012, Stockwell, et al. 2013, Zhao, et al.

[4] See Albertsen et al. (2004); Berkowitz et al. (1982); Jaddoe et al. (2007); Kesmodel et al. (2000); McDonald, et al. (1992); Mills et al. (1984); Shu et al. (1995); Whitehead and Lipscomb (2003); Windham et al. (1995).

2013) and Scotland (Griffith et al. 2020), while most other existing studies use interrupted time-series approches which cannot isolate the causal impact. Finally, we are the first to address whether a minimum-unit pricing policy can help alleviate the health-at-birth consequences of problem drinking. Our results offer novel evidence in this regard that should help to inform policy makers currently considering similar policies.

Evidence of the impact of the minimum unit price increase in Alice Springs is particularly informative in the Northern Territory given the 2018 universal introduction of the MUP policy. Prominent community groups strongly advocated for the prohibition of the large containers of wine (People's Alcohol Action Coalition, 2017), but the 2018 implementation generated considerable backlash from the community (Smee, 2018). The Northern Territory's MUP was removed in 2025. A report prepared for the Northern Territory Department of Health found evidence of a reduction in alcohol-related harms after the introduction of the policy, but could not attribute this to the MUP itself due to a suite of interventions introduced at the same time (Frontier Economics and Yarning, 2022). Our findings highlight that policies that substantially increase the cost of low-cost, high-strength alcohol products may have important unintended consequences.

The remainder of the paper proceeds as follows. In Section 2, we review theoretical justifications on government intervention in the market for consumption goods that produce negative externalities and present a theoretical model to analyse the impact of minimum unit pricing on social welfare. In Section 3, we review the empirical evidence on the effectiveness of minimum unit pricing and provide the institutional background of alcohol policies in the Northern Territory, focusing on the Alice Springs Liquor Supply Plan introduced in 2006. In Section 4, we describe the empirical framework, identification strategy and data to test model predictions. In Sections 5, we present estimates on the impact of the LSP on alcohol sales, the price elasticity of demand, and birth outcomes. In Section 6, we present findings on mechanisms. The findings are discussed and concluded in Section 7. Supplementary material is provided in an Appendix.

2. Theoretical framework

Excessive alcohol consumption imposes non-monetary costs ("abuse costs") which include private health costs to the user as well as negative externalities. Drinkers are often not fully aware of these costs (Carpenter & Dobkin, 2011). This lack of individual awareness is often used to justify government interventions that aim to make problem drinkers internalise abuse costs and moderate their consumption (Cook & Moore, 2000).[5] Policy interventions in the alcohol market typically increase the total cost of consumption either directly by changing retail prices or indirectly by imposing barriers to access. The change in demand for alcohol under a restriction depends on the sensitivity of consumers to changes in cost following a policy intervention.

The economic literature has approached the role of prices in determining demand for risky substances in the framework of Becker and Murphy's rational addiction model (1988). According to this model, a person's current consumption decisions depend on the expected future consumption costs of the addictive good. As a result, increasing the monetary or non-monetary cost of the addictive good will decrease consumption, proportionate to the change in expected costs. Bernheim and Rangel (2004) introduce time-inconsistency into this model, suggesting that environmental cues may trigger addicts to enter a 'hot' decision-making state in which they continue to consume a substance beyond the point where marginal surplus is negative. If this is the case then addicts will not respond to changes in consumption costs in a fully rational manner, so their demand for alcohol will be less elastic than that of a rational consumer.

A first-best tax would penalise consumers at different rates depending on the total amount of alcohol consumed by each drinker, reflecting the increasing marginal cost of each unit of alcohol consumed (Diamond, 1973).[6] However, such a tax is hard to implement where consumers are heterogeneous in their preferences and marginal externalities exist (Griffith, et al., 2017). A proposed solution to this problem is minimum unit pricing (MUP), a regulated floor price at which one standard drink of alcohol can be sold. As opposed to taxes which impact the price of all alcoholic beverages, minimum unit pricing affects only the price of alcohol sold below the chosen MUP. We analyse the welfare effect of minimum unit pricing through two simple models. First, we extend the Pogue and Sgontz (1989) model of alcohol taxation in the presence of abuse costs

[5] Medical literature has established that the health effects of alcohol follow a J-shaped curve: low to moderate levels of consumption are not harmful and may even have cardiovascular benefits but health consequences increase sharply once consumption reaches a tipping point (Thompson, 2013). Analogously, the negative externalities caused by alcohol are negligible at low levels of consumption but grow quickly once drinking becomes heavy (reviewed by Cook and Moore 2000).

[6] Alternatively, a government could use volumetric or ad-valorem taxes on alcohol, which are easier to implement, however these fail to account for the fact that the marginal cost of each standard drink increases with consumption (Chaloupka, Saffer and Grossman 1993; Griffith, O'Connell and Smith 2017).

to analyse the effect of a price increase on total social welfare. Second, we use the standard microeconomic model of constrained utility maximisation across alcohol and a bundle of consumer goods to discuss the implication of MUP for overall consumption.

Imagine a market for pure alcohol in which consumers choose the type and amount of alcohol to consume based on preferences over quality and price. We model a monopolistically competitive alcohol retail industry comprised of takeaway retailers (selling alcohol for consumption off-premises) and pubs and clubs (selling alcohol for consumption on-premises). In Australia, as in many other countries, entry into the alcohol retail industry is controlled by the liquor licensing authority.[7] Competition between alcohol retailers is primarily based on price, location and service, justifying a model of monopolistic competition.[8]

Minimum unit pricing will only affect consumers who originally purchase alcohol below the chosen MUP, P_{Min}. We focus on this subset of consumers and make the simplifying assumption that these consumers originally purchase pure alcohol at the lowest price per unit, P. Ceteris paribus, the consumption decision of those unaffected by the MUP will be unchanged.

Consumers originally purchasing alcohol at price P are divided into two groups with different demand schedules (see Figure 1): non-abusers (NA) and abusers (A). Abusers' demand curve D_A is located to the right of the non-abusers' demand curve D_{NA}. This means that an abuser will consume more pure alcohol at any price. The demand curve for abusers is assumed to be steeper reflecting a smaller price elasticity of demand. Over the long run, the lowest price P will be constant and equal to the average cost of production. The total cost of consumption is given by the price plus abuse costs/externality E: $P + E$, where the non-linearly, upward-sloping curve E measures the marginal external abuse cost. For non-abusers, E=0, while for abusers, E>0. The externality grows exponentially with every additional unit of alcohol consumed. The steeper E is, the greater the social cost of abusers' alcohol consumption. If drinkers ignore abuse costs in their consumption decision, then equilibrium consumption will impose a welfare loss given by area C.

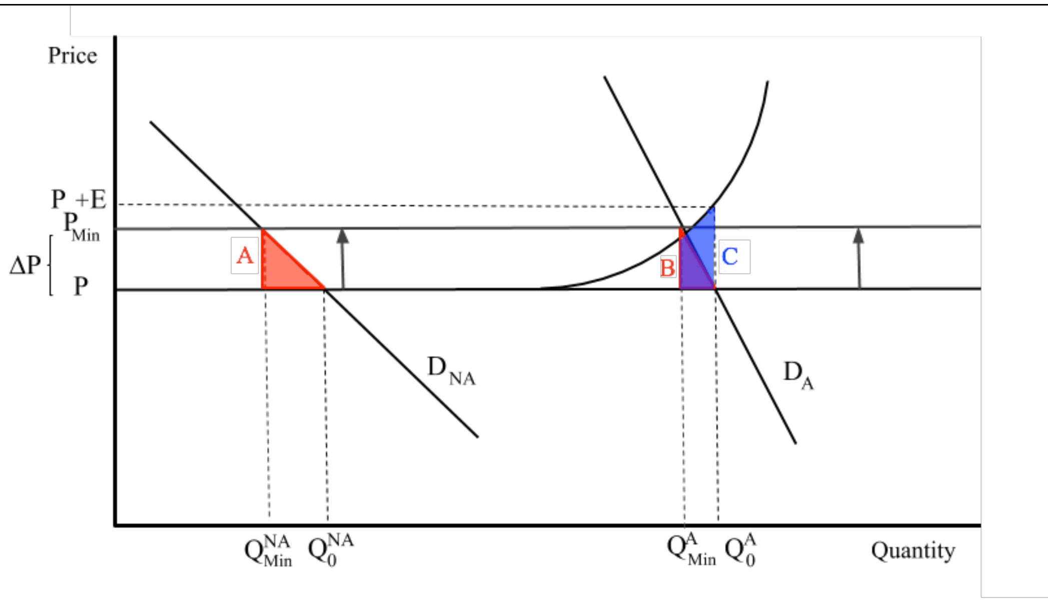
An increase in the MUP of pure alcohol $\Delta P = P_{Min} - P$ will decrease consumption by non-abusers from ${Q_0}^{NA}$ to ${Q_{Min}}^{NA}$, creating consumer surplus loss A for each non-abuser. Consumption

[7] In 2017, there were 515 active liquor licenses operating in the Northern Territory (Northern Territory Government 2017)

[8] Location is a particularly important source of differentiation in the Northern Territory given long distances between regional centres.

by abusers will decrease from ${Q_0}^A$ to ${Q_{Min}}^A$, creating consumer surplus loss B for each abuser. Abuse costs will decrease by the area C for each abuser. Following the original model, we now let E denote the marginal external abuse cost averaged over the change in abusive consumption from ${Q_0}^A$ to ${Q_{Min}}^A$.

Figure 1 Theoretical Impact of an Increase in the Price of Pure Alcohol



Letting N^{NA} denote the number of non-abusers, and N^A denote the number of abusers, the total change in welfare (W) from a change in the MUP (ΔP) can be written as $W = C * N^A - B * N^A - A * N^{NA}$, or:

$$W = E(\Delta Q^A)N^A - \frac{1}{2}\Delta P(\Delta Q^A)N^A - \frac{1}{2}\Delta P(\Delta Q^{NA})N^{NA} \qquad (1)$$

The first term in equation (1) is the decrease in abuse costs; the second and third terms are the decreases in the consumer surplus of abusers and non-abusers, respectively.

We let ϵ_A and ϵ_{NA} denote the own-price elasticities of demand for alcohol for abusers and non-abusers respectively. We assume that (a) $\epsilon_A < 0$ and $\epsilon_{NA} < 0$ i.e. all consumers are responsive to changes in price, and (b) $\epsilon_{NA} < \epsilon_A$ i.e. alcohol abusers are less responsive to changes in price than non-abusers. These assumptions are based on empirical evidence (see Wagenaar, Salois and Komro 2009 for a review).

Using this notation, we can rewrite the changes in quantities demanded as follows:

$$\Delta Q^{A} = \epsilon_{A}(\Delta P)\frac{{Q_0}^{A}}{P} \tag{2}$$

$$\Delta Q^{NA} = \epsilon_{NA}(\Delta P)\frac{{Q_0}^{NA}}{P} \tag{3}$$

Substituting (2) and (3) into (1) and rearranging terms gives:

$$W = E\left(\epsilon_{A}(\Delta P)\frac{{Q_0}^{A}}{P}\right)N^{A} - \frac{1}{2}\frac{\Delta P^{2}}{P}\left[\epsilon_{A}\left({Q_0}^{A}\right)N^{A} + \epsilon_{NA}\left({Q_0}^{NA}\right)N^{NA}\right] \tag{4}$$

We can then take the first derivative with respect to ΔP (the magnitude of the minimum price increase) to show that the impact of a change in the MUP on social welfare is a function of:

$$\frac{dW}{d\Delta P} = E\frac{{Q_0}^{A}}{P}(\epsilon_{A})N^{A} - \frac{\Delta P}{P}\left[\epsilon_{A}\left({Q_0}^{A}\right)N^{A} + \epsilon_{NA}\left({Q_0}^{NA}\right)N^{NA}\right] \tag{5}$$

It follows that:

$$\frac{dW}{d\Delta P} > 0 \text{ iff } (E - \Delta P)\epsilon_{A}\left({Q_0}^{A}\right)N^{A} > (\Delta P)\epsilon_{NA}\left({Q_0}^{NA}\right)N^{NA} \tag{6}$$

From equation (6), we have two necessary conditions for an increase in the MUP to increase total social welfare:

Condition 1. $E > \Delta P$: the average marginal external abuse cost per alcohol abuser is greater than the MUP increase itself (necessary, but not sufficient).

Condition 2. $E(\epsilon_{A})\left({Q_0}^{A}\right)N^{A} > \Delta P(\epsilon_{A}\left({Q_0}^{A}\right)N^{A} + \epsilon_{NA}\left({Q_0}^{NA}\right)N^{NA})$: the total reduction in abuse costs is greater than the total reduction in consumer surplus (necessary and sufficient).

Hence, a MUP which increases alcohol prices of the cheapest alcoholic beverage will improve social welfare if the additional social cost due to alcohol abuse is greater than the actual price increase and if, and only if, the total reduction in the externality produced by problem drinking is greater than the loss in consumer welfare due to the price increase. Condition 2 demonstrates that if abusers are not price elastic ($\epsilon_A = 0$), then the policy cannot yield social welfare gains. Condition 2 also demonstrates that the policy is only effective if it reduces the externalities caused by problem drinking by a sufficiently large amount to offset consumer welfare losses that are induced by price increases (which might affect consumption opportunities of other goods). Therefore, if the policy does not lead to a reduction in externalities at the margin (E=0), then it cannot yield social welfare gains either.

Apart from having zero positive impact on social welfare, a MUP could also lead to unintended consequences, worsening social welfare. These unintended consequences can be demonstrated in a standard microeconomic model of consumer choice. Under a model of constrained utility maximisation, consumers with a fixed budget set choose between pure alcohol and a bundle of other consumption goods. An increase in the minimum price per standard drink will have an income and a substitution effect for consumers previously purchasing the cheapest alcohol. The net effect of a price increase will therefore be a decrease in alcohol consumption and an ambiguous change in consumption of other goods, depending on the consumer's preferences. If the price elasticity of demand for alcohol is inelastic, then consumption of other goods may decrease because consumption of the same quantity of alcohol is now more expensive leaving less income for the purchase of other goods. According to this model, it is possible that the consumption of necessary goods such as food, clothing, and child-centred goods could decrease after the introduction of minimum unit pricing. This could have a negative welfare effect. Moreover, a consumer may substitute towards other addictive goods, such as tobacco or drugs, which could also have a negative welfare effect. The impact of MUP on total social welfare, including consumption of necessary goods or alternative addictive goods, is an important consideration in evaluations of such policies.

Guided by these two theoretical frameworks, we conclude that it is first key to understand the empirical magnitude of the price elasticity of alcohol demand and then to evaluate the change in externalities when the MUP policy was introduced. We will present these estimates in our empirical application.

3. Literature review and institutional background

3.1 What do we know about the impact of minimum unit pricing (MUP)?

Minimum unit pricing continues to be proposed as a policy to address problem drinking. MUP was introduced nationally for the first time in 2018 in Scotland, and in 2024, the Scottish Parliament voted to continue this policy and increase the floor price on alcohol from 50p to 65p. Several provinces in Canada have also historically used MUP to regulate consumption and Australia's Northern Territory expanded the Alice Springs MUP to the entire Territory in October 2018. The empirical evidence base on the effectiveness of MUP is limited, but existing studies predict that MUP will reduce consumption of heavy drinkers. Perhaps the most rigorous analysis on MUP is a series of public health studies on MUP in British Columbia, Canada, which find that periodic increases in MUP led to reduced alcohol consumption (Stockwell, et al., 2012a) and lower rates of alcohol-attributable hospital admissions (Stockwell, et al., 2013) and alcohol-related deaths (Zhao, et al., 2013).[9]

Evidence on the impact of MUP in the Northern Territory is mixed, with considerable debate on its impact both while the policy was in place and after its removal in 2025 (e.g., Taylor and Wright, 2025). Frontier Economics and Yarning (2022) found that the MUP reduced cask wine consumption and that alcohol-related harms decreased following its introduction, but could not attribute the reduction in harm to MUP itself due to a suite of policy interventions introduced at the same time. O'Brien et al (2021) use a linear mixed model to analyse wastewater samples and conclude that alcohol consumption in the Northern Territory dropped significantly after the introduction of the MUP, but that consumption gradually returned to pre-MUP levels over the next 15 months. Taylor et al (2021) observe a 51% reduction in per capita consumption of cask wine in the NT following the introduction of MUP, and overall reductions in per capita alcohol consumption outside of Darwin and Palmerston. The authors note media reports that drinkers may have substituted non-liquor alcohol substances such as methylated spirits. Overall, the effect of MUP in the Northern Territory on both alcohol consumption and health outcomes is difficult to estimate due to the suite of policy interventions introduced at the same time and the impact of COVID-19 (Taylor, 2023).

[9] To our knowledge, this is the only existing quasi-experimental evidence of the impact of minimum unit pricing.

Several studies modelled the impact of MUP in the UK prior to its introduction in Scotland, known as the Sheffield Alcohol Policy Model (Brennan, Meier, et al. 2014). This model indicates that MUP increases are effective in reducing consumption, health care costs and health-related quality of life losses (Purshouse et al. 2010, Brennan et al. 2014) and that the effects of a minimum price threshold amplify as the MUP rises since a greater proportion of the market is affected (Brennan, et al., 2014). However, Snowdon (2015) argues that these models overestimate the effectiveness of MUP by using own-price elasticities higher than current empirical estimates and imposing cross-price elasticities that are not supported by existing literature. A synthesis of evidence on the effect of MUP in Scotland prepared by Public Health Scotland found evidence that MUP reduced deaths directly caused by alcohol consumption (2023).

Studies using Australian and UK household consumption data have found the heaviest-drinking households tend to purchase the cheapest alcohol, suggesting MUP will impact heavy drinkers almost exclusively (Vandenberg and Sharma 2016, Ludbrook, et al. 2012). Holmes et al (2014) use the Sheffield Alcohol Policy Model to estimate that MUP would have the greatest effect on harmful drinkers on low incomes, but suggest this would coincide with substantial gains to this group in terms of morbidity and mortality related to alcohol consumption. Little is known about the price elasticity of demand when only the minimum price of alcohol increases.

The impact of MUP depends on the price elasticity of demand, which means to what degree do people adjust consumption when the minimum price of the good is lifted. A large body of literature has found that alcohol demand is inelastic but downward sloping for both moderate and heavy drinkers (Cook 1981, Cook and Tauchen 1982, Coate and Grossman 1988; reviewed by Wagenaar, Tobler and Komro 2010). Higher prices have been found to reduce drink-driving offences (Kenkel, 1993), motor-vehicle fatalities (Saffer and Grossman 1987, Sen and Campbell 2010), domestic violence (Markowitz, 2000) and child abuse (Markowitz & Grossman, 2000). A number of studies have established a negative relationship between access costs proxied by retailer density and alcohol-related harms (Scribner, MacKinnon and Dwyer 1995, Anderson, Crost and Rees 2018).

Heavy drinkers have been found to be significantly less price elastic than moderate drinkers (Manning, et al., 1995). In an extensive meta-analysis Wagenaar, Salois and Komro (2009) conclude that heavy drinkers have a price elasticity of -0.28, lower than whole-population elasticities of -0.46 for beer, -0.69 for wine and -0.80 for spirits. Peer influence also plays an important role in heavy drinking (Yakovlev, 2018) which may mean that price changes are less effective in environments with high rates of alcohol abuse.

3.2. Can alcohol restrictions improve children's health?

Many insights about welfare effects and unintended consequences of alcohol restrictions come from the prohibition era and policies that target First Nations communities. Miron and Zwiebel (1991) found that the hard alcohol bans from the 1930s in the USA led only to modest reductions in alcohol consumption, which suggests that alcohol was consumed illegally. However, some argue that these modest reductions did improve infant health by reducing mortality (Jacks et. al 2021). While alcohol bans targeting First Nations communities in Alaska have been found to decrease alcohol-related outpatient visits (Chiu et al 1997) and injury mortality rates (Landen et al 1997a), others found that wet reservations have lower rates of cirrhosis mortality, suicide and homicide than dry reservations (May 1976). Landen (1997b) finds a slight *increase* in alcohol-related mortality when reservations become dry. Similarly, Gallaher et al (1992) find evidence of higher pedestrian and hypothermia mortality on dry reservations, possibly caused by residents walking long distances to acquire alcohol.

While blanket alcohol restrictions might have negative side effects, targeted alcohol restrictions have been successful in a range of contexts. Minimum age drinking laws have been found to reduce youth drinking (Carpenter 2004a, 2007, 2011, Dee 1999, Yörük 2011) and related harms including suicide (Carpenter 2004b), crime (Carpenter and Dobkin 2015), risky sexual behaviour (Carpenter 2005), mortality (Carpenter and Dobkin 2009) and low birth weight (Fertig 2009). The threat of punishment is also important: drink-driving laws have been found to reduce motor-vehicle fatalities, provided sanctions are sufficiently harsh (Chaloupka et al 1993, Kenkel 1993). Interventions targeted at heavy drinking may also be effective: Bhattacharya et al (2013) find that the Gorbachev Anti-Alcohol Campaign in Russia which increased prices and restricted retail sales decreased crude death rates, however these increased again sharply at the end of the campaign.

Cil (2017) exploits variation in the introduction of alcohol warning signs (AWS) across the US to establish a link between reduced maternal alcohol consumption and improved birth outcomes (lower rates of very low birth weight and very premature births). Similarly, Fertig and Watson (2009) exploit changes in minimum drinking age laws and find that increased prenatal alcohol exposure associated with a minimum age of 18 is associated with higher rates of low birth weight and premature births. We contribute to this literature by analyzing the impact of another alcohol-related policy, minimum unit pricing, on maternal alcohol consumption and birth outcomes.

3.3. Policy background in the Northern Territory and the Alice Springs Liquor Supply Plan

Alcohol abuse is widely recognised as both a major contributor and outcome of the high levels of disadvantage observed in the Northern Territory. Alcohol consumption in the Northern Territory is amongst the highest in the world at almost 14 litres per capita (Skov, et al., 2010) – roughly equivalent to consumption in Russia before Gorbachev's anti-alcohol campaign (Bhattacharya, et al., 2013). Media attention tends to focus on problem drinking in Aboriginal communities. Aboriginal people in Australia face an alcohol-attributable mortality rate ten times as high as the non-Aboriginal population (Skov, et al., 2010). However, problem drinking is also an issue for non-Aboriginal people in the Northern Territory, who drink 40% more than average Australians and face alcohol-related mortality rates double the national average (Skov, et al., 2010). The estimated social cost of alcohol in the Northern Territory in 2004-05 was quadruple the national estimate at $4,197 per adult (Whetton, et al., 2009).

Alcohol policy in the Northern Territory is a controversial issue. Restrictions on alcohol have historically been discriminatory towards the Aboriginal population (d'Abbs, 2017). One justification for such policies has been concern over high prevalence rates of FASD in some Aboriginal communities in Australia (Fitzpatrick, et al., 2015). However, alcohol problems in the Northern Territory are by no means restricted to Aboriginal drinkers (Riley, et al., 2017).

We contribute to this literature by studying the impact of the prohibition of large containers of wine under the Alice Springs Liquor Supply Plan (2006) that effectively increased the minimum unit price (MUP) of alcohol. This policy was implemented in response to perceptions of high levels of alcohol-related harm (Senior, et al. 2009, Symonds, et al. 2012). The Alice Springs Liquor Supply Plan (LSP) was announced on 7 September 2006 as part of a broader Alcohol Management Plan (AMP) and implemented on 1 October 2006. It was announced in response to concern about alcohol abuse and related harms in the town (Senior, et al., 2009). In particular, the LSP sought to address 'the alarming levels of cask wine consumption' within the town, as well as the 'generally held perception that alcohol related crime and antisocial behaviour increased in Alice Springs over the past few years' (Northern Territory Licensing Commission , 2007). The policy was introduced in the context of a series of trials, such as a ban on cask wine in 2002 (Symons, et al., 2012).[10]

The LSP prohibited the sale of all cask wine in containers over two litres and fortified wine over one litre. Daily takeaway purchases were restricted to one cask of wine or bottle of fortified wine per person which were only available in the last three hours of trading each day.[11] Four and five litre cask wine and two litre fortified wine were previously the cheapest alcoholic beverage available by pure alcohol content. Thus, the ban effectively doubled the minimum price of a standard drink from A$0.25 to A$0.50 (Symons, et al., 2012).

The LSP was introduced before a broader range of smaller, non-invasive interventions occurred in the following year (2007) and larger, invasive interventions occurred in 2008.[12] There were bans on long neck beer in pubs and bars from 1 June 2007 and drinking in public places within 2 km of a licensed premise (1 August 2007). In June 2008, Alice Springs furthermore implemented Alcohol Takeaway Identification cards (June 2008), which required an identity care to purchase alcohol from a takeaway premise.

The Northern Territory Emergency Response (NTER), implemented on 1 July 2007 as a territory-wide package of reforms including alcohol and welfare-payment restrictions, effectively banned alcohol consumption in town camps from 1 December 2007 onward. However, the cornerstone legislation of the NTER, the so-called Income Management policy, affected Aboriginal communities in both the treatment and control groups, although at slightly different timings. Income quarantining was implemented on 7 January 2008 in Alice Springs Town camps and on 25 February 2008 in Darwin Town camps, with negative consequences on children's welfare (see Cobb-Clark et al. 2023, Doyle et al. 2022, Doyle et al. 2025).

[10]Alice Springs is a regional centre and tourist hub located in the Central Northern Territory region. At the time of the restrictions, the town had a permanent population of 27,000 people with a further 9,000 living in the surrounding area. The town has a large Aboriginal population: in 2006, 20.4% of the town's population was Aboriginal, many of whom lived in town camps (Senior, et al., 2009). Alcohol abuse has historically been a major problem in Alice Springs, typically attributed to Aboriginal problem drinking; however, patterns of heavy drinking are typical to both the Aboriginal and non-Aboriginal population (Senior, et al., 2009).

[11] Fortified wine was not a popular form of alcohol however this was included in the ban to prevent switching from cask to fortified wine. Trial cask wine bans in Alice Springs in 2002 led to a 1000% increase in fortified wine sales (Hogan, et al., 2006).

[12] See Senior et al (2009), Table 2.1 for a detailed timeline of interventions in Alice Springs between 2002 and 2009.

Senior et al. (2009) studied the impact of the LSP on wholesale alcohol purchases and crime, comparing unadjusted outcomes in the four quarters a year after the introduction of the policy (December 2007 - September 2008) to unadjusted outcomes in the four quarters before the introduction of the policy (December 2005 - September 2006). The study found that overall alcohol consumption decreased by 18%. Despite a significant reduction in alcohol consumption over time, the authors find no downward trend in the severity of assaults. One limitation of this evaluation is that it focuses on the full 2006 to 2008 period, which is problematic because the period includes the potentially confounding effects by the NTER intervention and other measures accompanying the LSP, discussed above. Symonds et al. (2012) conducted a detailed analysis of cross-correlations between price and alcohol consumption in the Central NT region, finding a significant decline in consumption at the time of the LSP which they attribute to the resultant switch to full strength beer.

Our analysis extends the previous literature by estimating the immediate impact of the LSP on alcohol sales, heavy drinking and birth outcomes using quasi-experimental methods and rich administrative data on alcohol sales, maternal drinking behaviour during pregnancy and birth outcomes.

4. Empirical framework

4.1. Data

We conduct our analysis with three sources of data. To demonstrate the validity of the natural experiment and to calculate price elasticities of demand, we use Licensing NT administrative data on the volume and type of alcohol purchased by Northern Territory-based retailers, available on a quarterly basis from the start of 2003 to end of 2010.[13] We also use publicly available monthly wholesale price data obtained from Symons et al. (2012).

To estimate the impact of the policy on health outcomes of children at birth, we use population-level birth records from the NT Data Linkage Study (NT-DLS) containing outcomes for all

[13] We obtained permission to use this data from the Director-General of Licensing Cindy Bravos on 25 May 2018. Ethics approval to use this data was obtained through the Human Research Ethics Committee of the Northern Territory Department of Health and Menzies School of Health Research on 11 September 2018.

children born in the Northern Territory since 1994 (see Silburn et al 2018 for an overview).[14] The NT-DLS was funded through a Partnership Project between the National Health and Medical Research Council (NHMRC) and the NT Governments, while the data linkage was managed by SA NT DataLink. We use the NT perinatal records (custodian: NT Chief Health Officer), which include demographic variables, and information on maternal health, health behaviour during the pregnancy, labour, birth and perinatal outcomes. These files contain information on 74,425 children who were born in the NT between 1994 and 2013, 40 percent of them are Aboriginal.

4.2. Measures for alcohol sales and consumption

To calculate average wholesale price per litre of pure alcohol, we use proxy price estimates based on a large sample of newspaper advertisements and CPI adjusted data on wholesale prices provided by the Licensing Commission constructed by Symons et al. (2012). Alcohol consumption is proxied with data on wholesale alcohol purchased by retailers, available in the Licensing NT administrative data. We convert total litres of each alcoholic beverage purchased into pure alcohol litres using conversion metrics provided by Licensing NT.[15]

4.3. Measure of infant health outcomes at birth

To proxy for the negative externalities of alcohol abuse, we use birth outcome measures for – birthweight in grams and gestation in weeks -- which are routinely collected in the perinatal data records. Birthweight and gestation are universally recognised indicators of infant wellbeing that are strongly linked to maternal behaviours during pregnancy (e.g. Almond and Mazumder 2011, Aizer and Currie 2014), and especially so in the Northern Territory, where birth weights are significantly lower than in the rest of Australia (Doyle et al. 2022).

Birth weight may be affected by two channels: duration of gestation and size for gestational

[14] Permission to use this data was granted through the Human Research Ethics Committee of the Northern Territory Department of Health and Menzies School of Health Research on 11 September 2018.

[15] A key limitation of this data set is that alcohol purchased by retailers may lead or lag alcohol consumption in the area. Retailers may purchase more or less alcohol in anticipation of policy changes or events in the following quarter, an issue we discuss in more detail below. Higher purchases in one quarter may also reflect unanticipated demand changes in the previous quarter leading to low levels of stock. We also do not observe interstate purchases of alcohol or sales between retailers. This should not affect our analysis as the Liquor Supply Plan only impacted the type of alcohol that retailers were able to sell, so we do not expect sales between retailers in different regions or from interstate to have changed discontinuously upon the implementation/removal of these restrictions.

age. We thus consider measures of low birth (<2500 grams), prematurity (<37 weeks of gestation), and high birth weight (>4000 gram, referred to as macrosomia). Intrauterine growth restrictions (small for gestational age) are generally affected by maternal nutrition (Kramer, 1987). Epidemiological literature has established that maternal binge and heavy drinking increases the risk of both preterm and small-for-gestational-age babies (Whitehead and Lipscomb 2003, Kesmodel, Olsen and Secher 2000, Truong et al. 2013). Other factors influencing birth weight include exposure to harmful environmental factors (e.g. stress, violence, smoking, drug use) and lack of access to medical care (Aizer & Currie, 2014). Further, gestational diabetes is linked to higher birth weight which in turn influences infant, childhood and adult health outcomes (Makgoba, et al., 2012). As such, LSP could impact birth weight in either direction. Birth outcomes may improve if mothers drink less, have more income available to spend on food or experience less stress from alcohol-related disturbances. Birth outcomes could deteriorate if mothers drink more as a reaction against the restriction or are more stressed, or if maternal nutrition decreases in response to more expensive alcohol, which might leave less income available for food.

4.4. Measures of maternal health behaviours during pregnancy

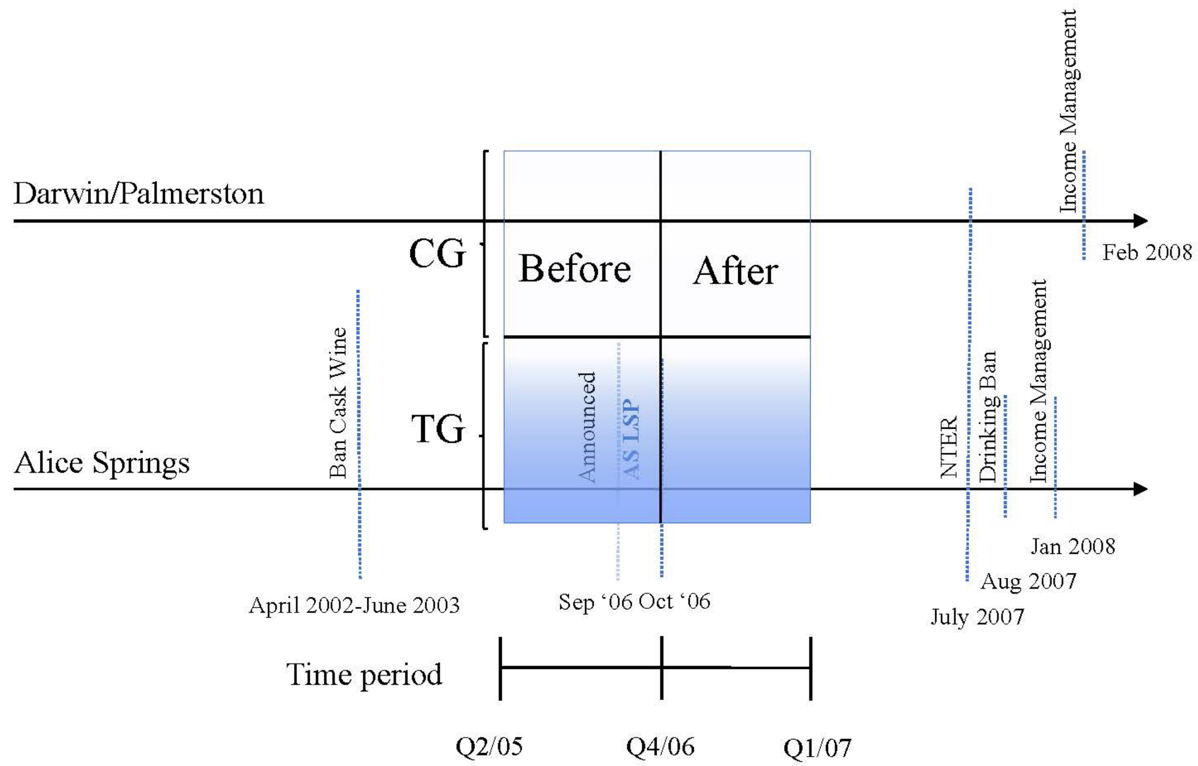
To study the factors that may explain potential MUP policy effects on birth outcomes, we use additional data extracted from the perinatal files. Most importantly, we study maternal health behaviours during pregnancy, such as alcohol and cigarette consumption. Risky health behaviour measures were recorded by a nurse at the first antenatal visit (typically at ~8 weeks gestation). Across the population, alcohol intake in the first trimester is significantly higher than in the later stages of pregnancy (commonly reported in Week 36), due for example to unplanned pregnancies (Nykjaer, Alwan, et al. 2014, Cameron, et al. 2013). As such, we interpret alcohol consumption at the first antenatal visit as reflective of overall levels of female alcohol consumption. Data is calibrated to the approximate date of the 8-week visit based on gestational age in weeks and date of birth. The perinatal files also include a binary measure of whether the mother drinks alcohol at the 36-week antenatal check-up. To be able to use this variable, a proxy for heavy and/or problem drinking, in the analysis, we would have to exclude babies born before 36 weeks. Hence, the variable is missing for all mothers who gave birth before week 36.

Maternal alcohol consumption during pregnancy is an indicator of child wellbeing. It is well established that within-utero alcohol exposure significantly increases risk of Fetal Alcohol Spectrum Disorder (FASD) and other developmental complications, especially if drinking occurs in the first trimester of the pregnancy (Sokol, Delaney-Black and Nordstrom 2003, Hinke Kessler Scholder et al 2014, Nilsson 2017). A reduction in the prevalence of maternal alcohol consumption during pregnancy should therefore improve child wellbeing. A key limitation of our maternal alcohol consumption variable is that we are only able to measure whether or not a mother had been consuming alcohol, not the quantity, frequency, or type of alcohol consumed. We are therefore restricted to analysing the impact of the LSP at the extensive margin of alcohol consumption during pregnancy.

4.5 Identification strategy

To identify the causal impact of the Alice Springs LSP, we use a standard 2 × 2 difference-in-differences (DiD) model, in which we compare differences in outcomes in Alice Springs before and after the introduction of the LSP with differences in outcomes over the same time-period for comparable regions which did not introduce a LSP (or other policies). As a control group, we consider individuals in Darwin and Palmerston, the two largest town centres in the Northern Territory located in the north by the Timor Sea. Figure 2 illustrates the identification strategy, a standard comparison of outcomes across two periods (before versus after) between a treatment and control region.

Figure 2 Identification strategy: Before and after comparison between treatment (Alice Springs) and control regions (Darwin / Palmerston)



Note: CG and TG refere to Control Group and Treatment Group respectively.

To adjust for differences in mean outcomes between Darwin/Palmerston and Alice Springs, we control flexibly for location fixed effects. To adjust for territory-wide time trends in outcomes, we include time fixed effects (quarter-by-year for alcohol sales and month-by-year for birth outcomes). This model is appropriate when interventions are as good as random, conditional on time and location fixed effects (see Abadie and Cattaneo 2018 for a recent overview of program evaluation methods including difference in difference).

The DiD estimator is valid under the assumption that trends in the outcome of interest in the treatment and control groups would have been parallel in the absence of the policy, and that there were no unobserved shocks affecting the outcome of interest in either group. We carefully assess this assumption in subsequent sections. As Figure 2 highlights, we can only estimate the short-term effects of the LSP, up until the end of 2007, when Alice Springs started experiencing a series of additional interventions that were part of the NTER. To ensure our results are not confounded by earlier or later alcohol bans, we restrict our main analysis to the policy period from April 2005 (Quarter 2) through March 2007 (end of Quarter 1).

Figure 3 (a) depicts average alcohol consumption in litres and wholesale prices for treatment and control regions from third quarter 2000 until third quarter 2008. The red vertical dashed line indicates the introduction of the Alice Springs LSP in October 2006. Clearly, the Alice Springs region experienced a significant price shock following the introduction of the LSP, while the control regions did not. Average wholesale prices hiked from \$67.6 per pure alcohol litre to \$87.9, an increase of 30% within a single quarter.

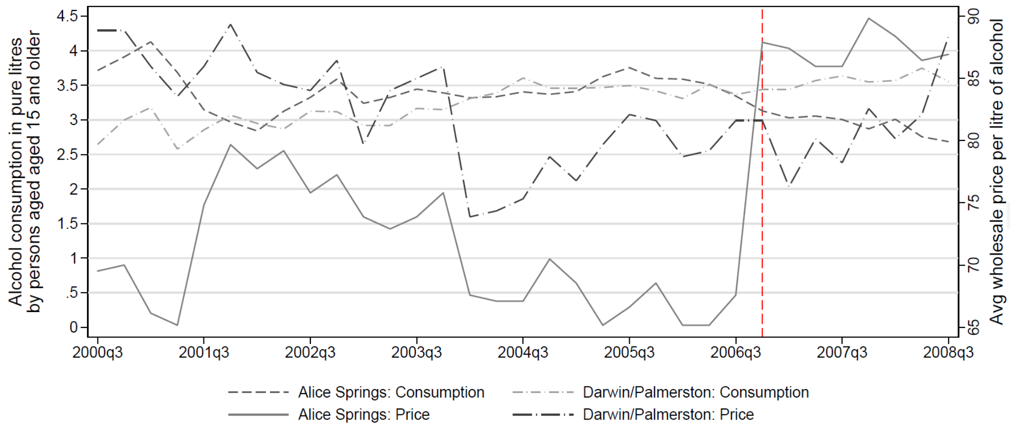
Consumption data over the same time reveals that consumption remained relatively constant in the treatment group between the beginning of 2003 up until quarter 3 of 2006. Total consumption then declined from 3.5 litres per adult person to 3 litres, following the introduction of the LSP, a drop of 14%. Again, no such changes were observed in the control region in Darwin / Palmerston, where alcohol consumption remained constant around 3.5 litre in pure alcohol consumption before and after the LSP introduction.

Figure 3 (b) presents a breakdown of the alcohol consumption by alcohol type in the treatment region. It shows that consumption dropped only for cask and fortified wine and increased for beer, bottled wine and cider. No change is observed for spirits. Figure 3(c) demonstrates that no changes in alcohol consumption occurred for any of the three alcohol types in the control group.

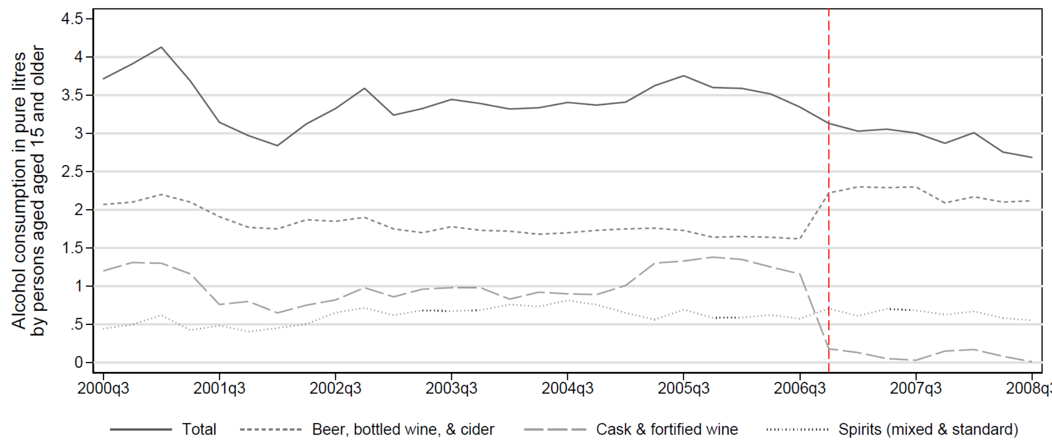
These patterns confirm that a price shock and consumption changes were observed only in the affected region following the introduction of the Alice Springs LSP in October 2006 and support our strategy to estimate the causal impacts of the LSP on alcohol consumption and birth outcomes.

Figure 3 Evolution of consumption and price over time, by treatment and control group

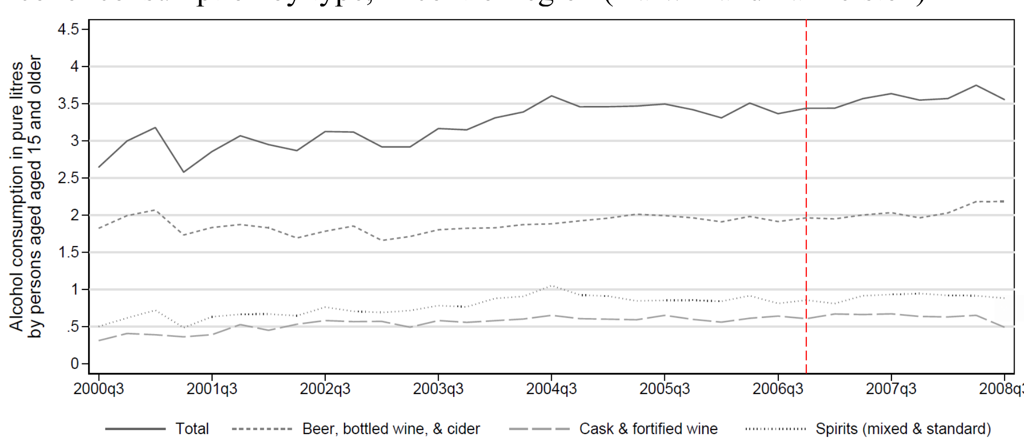
a) Alcohol consumption and price in litre, by treatment and control region



b) Alcohol consumption by type, in treatment region (Alice Springs)



c) Alcohol consumption by type, in control region (Darwin and Palmerston)



Notes: All figures show time series of mean wholesale alcohol process in the treatment (Alice Springs) and groups (Darwin and Palmerston). The red vertical dashed line indicatesv the introduction of the Alice Springs Liquor Supply Plan in quarter 4 of 2006. The analyses use publicly available wholesale data (quantity and price) obtained from Symons et al. (2012) for both treatment and control regions over the period 2003 and 2010.

4.6. Estimation Models

To estimate the impact of the LSP policy on alcohol sales by volume and type, we rely on the following DiD model:

$$Y_{rt} = \beta_0 + \beta_1 D_r + \beta_2 Post_t + \beta_3 D_r \times Post_t + \epsilon_{rt} \quad (1)$$

where the dependent variable (Y_{rt}) represents sales of alcohol to retailer *r* in quarter *t* (total and by type). The effect of the policy is measured with the coefficient β_3 on the interaction an indicator for the treatment region D_{rt} (=1 if Alice Springs, =0 if Darwin / Palmerston) and the post-LSP reform period $Post_t$ (=1 if October 2006 or after; =0 if before October 2006). In extensions to the simple model, we include alcohol retailer fixed effects (δ_r) and quarter-by-year fixed effects (θ_t).[16] Standard errors are clustered by retailer.

To estimate the impact of the LSP on birth outcomes, we rely on the following DiD model:

$$Y_{ilt} = \beta_0 + \beta_1 D_r + \beta_2 Post_t + \beta_3 D_i \times Post_t + \epsilon_{ilt} \quad (2)$$

where the outcome Y_{ilt} measures child $i's$ birth outcome in local area *l* at time *t*. Exposure to the Alice Springs LSP in utero is defined as being exposed to the policy for the full third trimester of pregnancy or earlier. We able to construct the exact date of conception through knowledge of the exact birth date (day, month, year) and the exact number of days in gestation.[17] This definition is based on previous studies that sought to identify the impact of public policy on babies' birth outcomes in the Northern Territory (Doyle et al. 2022, Doyle et al 2025) or babies exposed to the food stamp program in the United States (Almond, et al., 2011). Partially treated infants (those exposed in the third trimester at the time of the policy change) are part of the control group, but we conduct robustness checks where we exclude these infants or add them to the treatment group. We expect babies exposed to the policy from the start of the pregnancy as those who would theoretically most benefit from alcohol price increases.[18]

[16] In these extensions the specifications include non-linear time trends through quarter and year dummies and area-specific time trends (wholesale alcohol data) or fully interacted month and year dummies.
[17] End of trimester one estimated at 12 weeks from conception.
[18] Medical evidence suggests that foetal development is most sensitive to maternal alcohol consumption during the first trimester (Nykjaer, et al., 2014).

In this model, standard errors are clustered by SLA.[19] In extensions to the basic model, we control for infant and maternal demographic characteristics (infant gender, a quadratic in maternal age at the time of birth, the number of prior births), local area fixed effects and year-by-month fixed effects. Local areas are defined by the statistical local areas (SLA).[20]

5. Estimation results

We first present results from estimation of Eq. (1) to quantify the changes in alcohol sales resulting from the implementation of the Alice Springs LSP and then results from estimation of Eq. (2) to measure the impact on birth outcomes of children and maternal health behaviours.

5.1. Did the Alice Springs LSP reduce alcohol sales?

Table 1 reports the estimated treatment effect of the Alice Springs LSP on total sales volume in litres (column (1)) and by alcohol type (columns (2)-(7)). We find that the LSP reduced alcohol sales volume by -309.42 litres per retailer (SE 131.7), an estimate that is statistically significant ($p<0.05$). This point estimates indicates a modest effect, which equates to approximately 33 standard drinks per resident each quarter (not accounting for purchases of alcohol by tourists / non-residents)[21] – roughly the alcohol contained in one four-litre casks of white wine.[22]

[19] In both equations (1) and (2) we use data over a decade so that retailer and area fixed effects are identified more efficiently. However, we flag the policy period, for which the estimates of β_1, β_2, β_3 are estimated, similar to how other papers dealt with small sample sizes but where location fixed effects are important (See Doyle et al. 2022).

[20] Local areas are defined based on 2006 ABS Statistical Local Area (SLA) in perinatal data. Alice Springs town comprises 5 SLAs with 10 in the surrounding Central NT region. In wholesale alcohol data, regions are defined from 2010 ABS Region. While we use different geographical levels depending on the narrowest available location in each data set, these are consistent across regions and years (i.e. Aboriginal location maps to Suburb). Changes in geographical borders for treatment and control groups from 2006 to 2010 are minimal and affect only a small area of the remote Central NT region.

[21] Taking coefficient of 309 litres in our preferred specification and multiplying by the 36 retailers that purchased wholesale alcohol in the previous year indicates the total decline in pure alcohol purchases in Alice Springs town was approximately 42,951 litres per quarter. Dividing by the 2006 population of 26,700 residents (from ABS census data), this equates to 0.42 litre of pure alcohol per capita per quarter. This is equivalent to 33 standard drinks per resident (based on pure alcohol density of 789.24 g/L and Australian standard drink definition of 10g pure alcohol).

[22] Based on government estimates available at http://www.alcohol.gov.au/internet/alcohol/publishing.nsf/content/drinksguide-cnt#wine. A four litres cask of white wine contains 36 standard drinks.

Our estimates are driven by a large reduction in sale volume of cask and fortified wine (491.7 litres, SE 188.3, p<0.05). Sales of full-strength beer and wine increased by 174.2 litres (SE 83.3, p<0.05) and 39.1 litres (SE 21.7, p<0.10). We find no statistically significant change in sales volume of other alcohol types. Robustness checks demonstrate that the results are insensitive to adding control variables (see Appendix Table A 2).

The estimates are causal under the assumption that potential alcohol sales would have evolved in the same way in both treatment and control regions in the absence of the Alice Springs LSP. To test this assumption, we provide event study graphs in Figure 4 for the four significant estimates. Importantly, there are no obvious and significant pre-treatment trends in the short-time period considered (relative to sales in Quarter 2 in 2005), although we observe modest seasonal variation differences in the pre-treatment period. A test of joint insignificance of pre-trend interaction effects is not rejected for total pure alcohol, albeit marginally (p=0.066), cask and fortified wine (p=0.936), and regular wine (p=0.321). However, we reject the test of joint insignificance of the pre-policy interactions for high-strength beer (p<0.01).

These results allow calculation of the elasticity of demand in response to a change in the minimum price. The mean volume of pure alcohol purchased by retailers in the previous year across the two regions was 1,574 litres per quarter.[23] A 309 litres decrease in pure alcohol upon implementation of the LSP therefore represents a 19.6% reduction in pure alcohol purchased by each retailer. Symons et al (2012) report that the minimum price increased by 100% under the LSP. Assuming that wholesale alcohol purchases are a reasonable proxy for actual consumption, this leads to an estimated elasticity of demand to the minimum price per standard drink of -0.20.

In auxiliary analyses, we present direct estimates of the price elasticity of demand using publicly available wholesale data (quantity and price) obtained from Symons et al. (2012) for both treatment and control regions over the period 2003 and 2010. The average price elasticity of demand in Alice Springs is estimated as -0.47 (see Table A 3, Appendix). Our numbers are comparable to estimates provided in other countries. For instance, (Wagenaar, et al., 2009) report an average price elasticity of demand for alcohol of -0.5, and for heavy drinkers of -0.3. Although not zero, we interpret this as inelastic demand for alcohol consumption among problem drinkers.

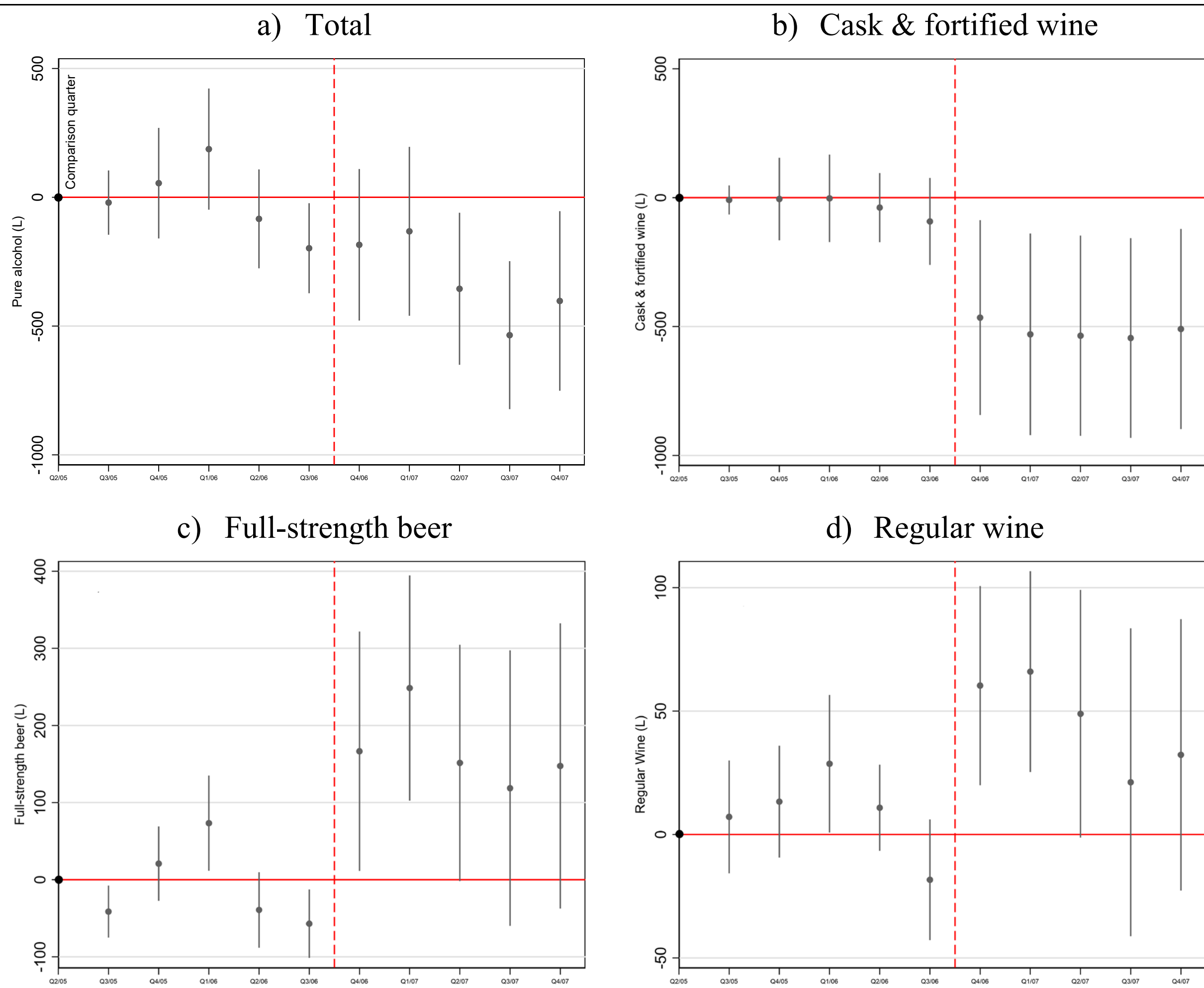
[23] Mean retailer purchases of alcohol over four quarters prior to start Q4 2006 (October 2006) from wholesale alcohol data.

Table 1 Impact of LSP on pure alcohol purchase by quarter (in Litres)

	(1) Total volume of pure alcohol	(2) Cask & fortified wine	(3) Full strength beer	(4) Low strength beer	(5) Regular wine	(6) Cider & other drinks	(7) Spirits and Spirit mixed drinks
LSP Region	-5076.45^{***}	403.41^{**}	-2024.72^{***}	-580.13^{***}	-1172.66^{***}	-690.12^{***}	-1664.65^{***}
	(127.85)	(187.92)	(82.97)	(9.10)	(21.43)	(16.94)	(20.04)
Post period	60.88	47.91^{**}	14.26	14.53^{*}	7.56	0.39	-25.87
	(51.70)	(22.97)	(22.95)	(7.49)	(8.05)	(16.73)	(16.38)
LSP × Post	-309.42^{**}	-491.70^{***}	174.22^{**}	-16.05	39.06^{*}	-25.54	-16.63
	(131.73)	(188.36)	(83.28)	(12.18)	(21.66)	(21.95)	(23.07)
Constant	4976.02^{***}	49.59^{***}	1742.49^{***}	546.14^{***}	1082.46^{***}	587.23^{***}	1515.83^{***}
	(66.45)	(17.22)	(22.54)	(12.15)	(9.35)	(28.12)	(26.35)
Observations	3661	3661	3661	3661	3661	3661	3661
# Retailers	139	139	139	139	139	139	139

Note: This tables presents estimation results of Equation (1). Each model uses data from Quarter 2 2005 up until Quarter 3 2007. The treatment group includes Alice Springs city and region, the control groups include Darwin / Palmerston city and region. Control variables include retailer fixed effects and year by quarter fixed effects. The DiD estimate of the treatment effect of interest is reported as LSP × Post. Clustered standard errors (by retailer) in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Figure 4 Event study - Retailer sales of pure alcohol, total and by category



Note: Reported are estimates and their 95% confidence intervals. Standard errors are clustered at the retailer level. In this difference-in-difference regression model the outcome is alcohol wholesales. The treatment region dummy variable is interacted with quarter of year dummy-variables (omitted category: Q2/2005). We control for retailer fixed effects and quarter by year time-trends. Full estimation results are presented in Table A 2. A test of join insignificance of pre-trend interaction effects is for total pure alcohol: p=0.066, Cask and fortified wine: p=0.936, Regular wine: p=0.321, but we reject the test of join insignificance of the pre-policy interactions for high strength beer: p<0.01.

5.2. Did the Alice Springs LSP improve birth outcomes of babies affected in utero?

Table 2 reports the estimated treatment effect of the policy on birthweight (Panel A), the risk of low birthweight (Panel B), the risk of high birth weight (Panel C), and the risk of premature birth (Panel D). The estimates are reported for a pooled model and separately for non-Aboriginal and Aboriginal mothers. Standard errors, clustered at the local area level, are reported in parentheses. Summary statistics of outcomes are reported in the Appendix (Table A 4). The sample sizes overall are 4,829 children for Aboriginal mothers and 25,555 children for non-Aboriginal mothers born between 2000 and 2009. During the relevant policy period, the numbers in the control and treatment regions are 491 and 240 respectively for Aboriginal mothers, and 2,890 and 570 for non-Aboriginal mothers. Results are presented by Aboriginal status because many of the Northern Territory alcohol policies were targeted at Aboriginal communities.

The LSP policy had no detectable effect on the birth outcomes of non-Aboriginal babies. The impact of the policy on birthweight is positive (18 grams, SE 44.9), but statistically and economically insignificant. The estimated policy effect is zero for all other birth outcomes.

In stark contrast, the LSP significantly worsened birth outcomes of babies born to Aboriginal mothers. In our preferred specification, birthweights reduced by 158 grams (SE 70.8, $p<0.05$) or around 0.25 SD relative to the pre-treatment standard deviation in birth outcomes. The risk of low birth weight is increased by 7.7 ppt (SE 0.032, $p<0.05$). Relative to the pre-treatment risk of low birth weight (0.078), this implies a risk increase of 98.7%. There are no significant effects of the policy on the risk of microsomia or premature birth (Panel C and Panel D).

We conduct several robustness checks to demonstrate the reliability of model assumptions (common trend assumption) and that our results are not sensitive to model specifications. The most important findings are:

The parallel trends assumption holds (See Figure 5). The treatment and control group Aboriginal babies have no different birth outcome trends pre-reform (the comparison quarter was Q2 2005). Aboriginal babies conceived in Q2 2006, who were immediately affected by the policy, have significantly lower birth weights and greater risk of low birth weight.[24]

[24] It is unclear why there are variations in the treatment effect post policy. The treatment effect is only statistically significant for babies affected either in the third or the first quarter of the policy. The data limitations do not allow us to further explore this finding.

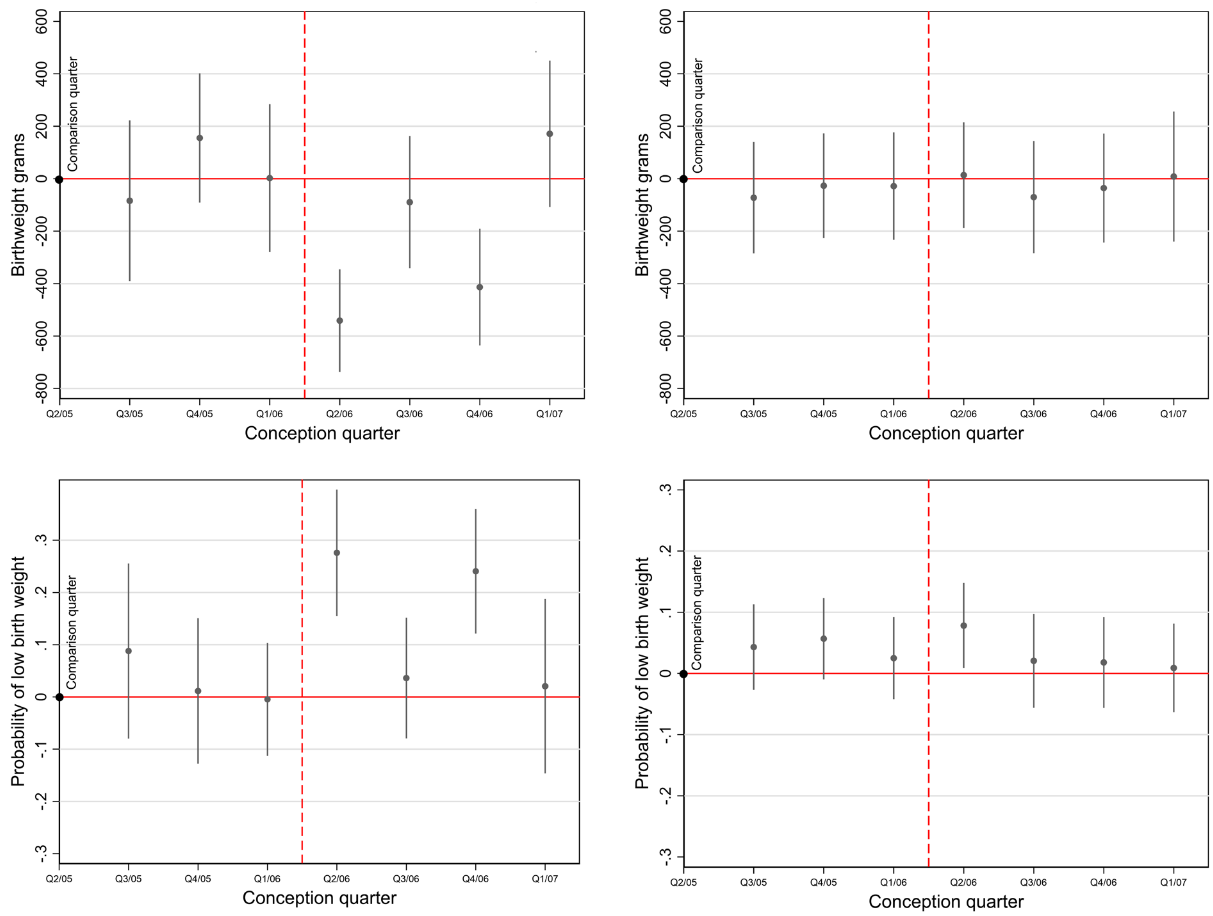
Table 2 Impact of LSP policy on birth outcomes, by Aboriginal status

	(1) All	(2) Aboriginal	(3) Non-Aboriginal
Panel A: Birthweight in grams			
Alice Springs LSP	-27.503	-158.190**	18.047
	(41.286)	(70.786)	(44.934)
Mean dependent variable	3414.020	3320.052	3452.267
Panel B: Probability of low birthweight (<2500 grams)			
Alice Springs LSP	0.016	0.077**	-0.004
	(0.013)	(0.032)	(0.016)
Mean dependent variable	0.062	0.078	0.056
Panel C: Probability of high birthweight – macrosomia (>4000 grams)			
Alice Springs LSP	0.013	-0.004	0.021
	(0.015)	(0.028)	(0.014)
Mean dependent variable	0.132	0.129	0.133
Panel D: Probability of pre-term (gestation < 37 weeks)			
Alice Springs LSP	0.017	0.048	-0.000
	(0.021)	(0.032)	(0.028)
Mean dependent variable	0.085	0.095	0.081
Obs. control group[a]	3381	491	2890
Obs. treatment group[b]	810	240	570
Obs. full sample	30405	4829	25555
Number location clusters	46	43	40
Demographics control	✓	✓	✓
Location fixed effects	✓	✓	✓
Moth-year fixed effect	✓	✓	✓

Note: Reported are difference-in-difference estimation model results of the treatment effect of the Alice Springs Liquor Supply Plan (introduced in Q4 2006) on birthweight measured in grams (Panel A), the probability of being born with low birthweight (Panel B), the probability of fetal macrosomia (birth weight>4000 grams) (Panel C), and the probability of premature birth (<37 weeks gestation) (Panel D). Mean dependent variable refers to the mean in the control group in the pre-treatment period. The treatment region is Alice Springs (City + region), the control region is Darwin and Palmerston (City + region). Sample includes all babies born in these areas between 2000 and 2009, but the treatment effect is the estimate for babies born within the policy rollout period: babies conceived between Q2 2005 and Q1 2007 ([a, b]). Treated babies were exposed to the policy in utero no later than the start of trimester 3 of the pregnancy. Demographic controls include: maternal age and age square, number of pregnancies, and sex of the child. All regressions included location fixed effects and quarter by year fixed effects. Clustered standard errors by location in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Figure 5 Event study graph for birthweight and gestation age

(a) Aboriginal mothers (b) Non-Aboriginal mothers



Note: Reported are difference-in-difference estimates and their 95% confidence intervals. The model specification is the same as in Table 1, Columns (2) and (3). Red dashed line indicates policy timing. Estimates to the right of the red-dashed line are affected by the policy, estimates to the left are pre-policy. All estimates are relative to babies conceived in Q2 2005, one year before the first cohort of babies is affected by the policy (concevived in Q2 2006).

Estimates are insensitive to the type of control variables included (Appendix Table A 5). The estimates on birth weight for babies of Aboriginal mothers range between -171.8 grams (SE 89.4) (no controls) and -109.5 grams (SE 89.2) (interactions between location fixed effects and time trend) and for the risk of low birthweight between 0.081 (SE 0.036) and 0.090 (SE 0.041).

Estimates are insensitive to changing the window of treatment exposure in utero or policy period (Appendix Table A 6). Including babies who were conceived slightly earlier increases the treatment effect to range between -202.9 grams and – 223.6 grams. Shortening the policy period to include babies conceived before the start of Q2 2007 also increases the treatment effect on birthweight, ranging between -202.7 grams and -282.6 grams. Similar results are obtained for the impact of the policy on the risk of low birth weight for Aboriginal babies.

A placebo test reveals no significant treatment effect when shifting the policy experiment to two years prior, the only available window for such test (Appendix Table A 7).

6. Mechanisms

So far, we have found that the MUP produced by the Alice Springs LSP resulted in a modest reduction in pure alcohol sales via reduction in sales of cask and fortified wine that was not fully offset by an increase in the sales of full-strength beer. However, we have also found that birth outcomes remained unaffected overall and for non-Aboriginal babies but worsened for Aboriginal babies. Why? Several potential mechanisms could explain the decline in birthweight among Aboriginal babies following the Alice Springs LSP. First, we test whether the mothers responded to the policy by drinking and smoking (a complement to drinking) less during pregnancy. We find that the risk of drinking alcohol at first and last antenatal visit is reduced by the policy for Aboriginal mothers, albeit the estimate is not statistically significant (Table 3, Panel A), with a reduction of risk of around 7 ppt (SE 7 ppt). However, the risk of smoking by Week 36 is significantly increased by 14.4 ppt (SE 6.8 ppt, $p<0.05$) for Aboriginal mothers. Relative to the pretreatment risk of smoking by Week 36 (42.9%), this implies an increase in risk of 33.6%.

For non-Aboriginal mothers the policy reduced the risk of any alcohol use recorded at the first antenatal visit by 3.3 ppt (SE 1.5 ppt, $p<0.05$). Relative to a baseline risk of 12.9% this implies a reduction in risk of alcohol by 25.6% (Table 3, Panel B).

Thus, while the policy reduced birth weights of babies born to Aboriginal mothers when exposed to the policy in utero, it did not affect the birthweights of babies born to non-Aboriginal mothers, even though their risk of alcohol use was slightly reduced. For Aboriginal mothers the policy also reduced alcohol use but not in statistically significant ways. Risk of smoking increased significantly, which might explain the negative impact of the policy on birthweights.

Table 3 Mechanism - Maternal health behaviours during pregnancy

	(1) Smoking 1st week	(2) Smoking 36 weeks	(3) Alcohol 1st week	(4) Alcohol 36 weeks
Panel A: Aboriginal Mothers				
LSP × Post	0.063	0.144**	-0.073	-0.065
	(0.065)	(0.068)	(0.067)	(0.074)
Mean pre-treatment	0.491	0.429	0.288	0.206
N Pre-treatment	415	349	385	343
N Post treatment	219	203	214	201
N all	2683	2362	2765	2451
Panel B: Non-Aboriginal Mothers				
LSP × Post	0.018	-0.003	-0.033**	-0.017
	(0.020)	(0.016)	(0.015)	(0.013)
Mean pre-treatment	0.218	0.206	0.129	0.073
N Pre-treatment	2739	2454	2648	2398
N Post treatment	533	518	518	501
N all	14869	13489	15354	13978

Note: Treatment for the following outcome variables is defined as being exposed from conception to the policy: Smoking and drinking in 1st antenatal visit. This is necessary because the policy can only affect these outcomes when the policy came into effect just before the pregnancy or in trimester 1. Model controls for full set of control variables, as defined in Column (4) Table 1. Clustered standard errors in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

7. Discussion and Conclusion

Our results indicate that consumers substituted the next cheapest form of alcohol, full strength beer, following the 2006 restrictions on sales of large containers of cask and fortified wine. This suggests that preferences for cask and fortified wine are primarily based on price rather than type of alcohol which is consistent with the disproportionate consumption of the low-cost, high-strength alcohol products among those most affected by the minimum-unit price policy—binge drinkers of low-socioeconomic status. If the key benefit of cask and fortified wine is the low price point, then a direct increase in the MUP should similarly shift consumption from cask and fortified wine to the next-cheapest form of alcohol, full-strength beer.[25]

In analysing the impacts of the policy on birth outcomes of children and maternal health behaviours, we find that the policy affected Aboriginal and non-Aboriginal families in different ways. The risk of teenage pregnancy declined for non-Aboriginal mothers. Teen pregnancy is associated with risky health behaviours of adolescents and poorer outcomes for babies born to teenage mothers. Thus, the policy benefitted both society and children in reducing the negative consequences of risky alcohol consumption.

The policy reduced birthweights of babies of Aboriginal mothers and increased their risk of low birthweight (although did not affect gestational length). This could be interpreted as a negative impact of the policy. One possible explanation for a decline in infant health under the LSP is the presence of an income effect, in which the increased floor price on alcohol led to an increase in expenditure on alcohol and a decrease in expenditure on necessary goods, particularly given our estimate of inelastic demand for alcohol ranging between -0.2 and -0.5. For budget constrained households this most likely meant that alcohol consumption did not change and therefore took up a larger proportion of their available budget.

Observed declines in birthweight may also be driven by increased prenatal maternal stress under the introduction of the policy, which has been linked with reduced gestational age (Torche, 2011). We also find that smoking during pregnancy increased because of the policy, which may reflect a shift away from more expensive alcohol to cigarettes, for which prices remained constant.

A key consideration in interpreting our results is the very high proportion of heavy drinkers in Alice Springs. Pure alcohol consumption per capita in Alice Springs was 16 litres per capita in 2006, one of the highest in the world. Thus, the implied decrease in alcohol consumption at the intensive margin still means very high levels of alcohol consumption, which is an important consideration in extrapolating results to other regions.

[25] This result also implies that the chosen minimum unit price should be set with careful consideration of the price of beer. This has been demonstrated in the introduction of minimum unit pricing in the Northern Territory. A floor price of $1.30 was chosen to be lower than the cost per drink in a standard case of beer, however an increase in the price of cheap generic beer (recently available in 1L bottles) has caused considerable backlash.

There are two key limitations of our data analysis. The data and policy experiment only allow the identification of the causal impact for a very narrow time window. This implies that our analysis is underpowered, which is evidence by large standard errors which bring uncertainty in our statistical inference. Quarterly analysis of birth outcomes in the event study analyses demonstrate a great level of volatility in the treatment effect for babies exposed to the policy by different trimesters.

Another limitation is that our data do not allow to measure the impact of the policy on other measures of social welfare, such as crime, violence, domestic abuse, alcohol related health issues and unplanned pregnancies, which may be more sensitive to the observed change in alcohol consumption at the intensive margin. Furthermore, it would be important to directly study alcohol consumption of youth, who may react more sensitively to problem drinking in terms of cognitive and behavioural development, transmission of STDs and unplanned pregnancies. An important consideration for future research is the effect of MUP on these broader measures of social welfare.

We concede that more evidence is needed of the medium- to long-term impacts of minimum unit pricing on child wellbeing and other social harm that alcohol abuse may cause. We are unable to identify those longer-term impacts, primarily due to a range of other alcohol-related interventions introduced shortly following the Liquor Supply Plan, both in Alice Springs and across the entire Territory.

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Appendix A: Alcohol policy in the Northern Territory

Figure A 1 Timeline of key alcohol-related policies in the Northern Territory & Australia

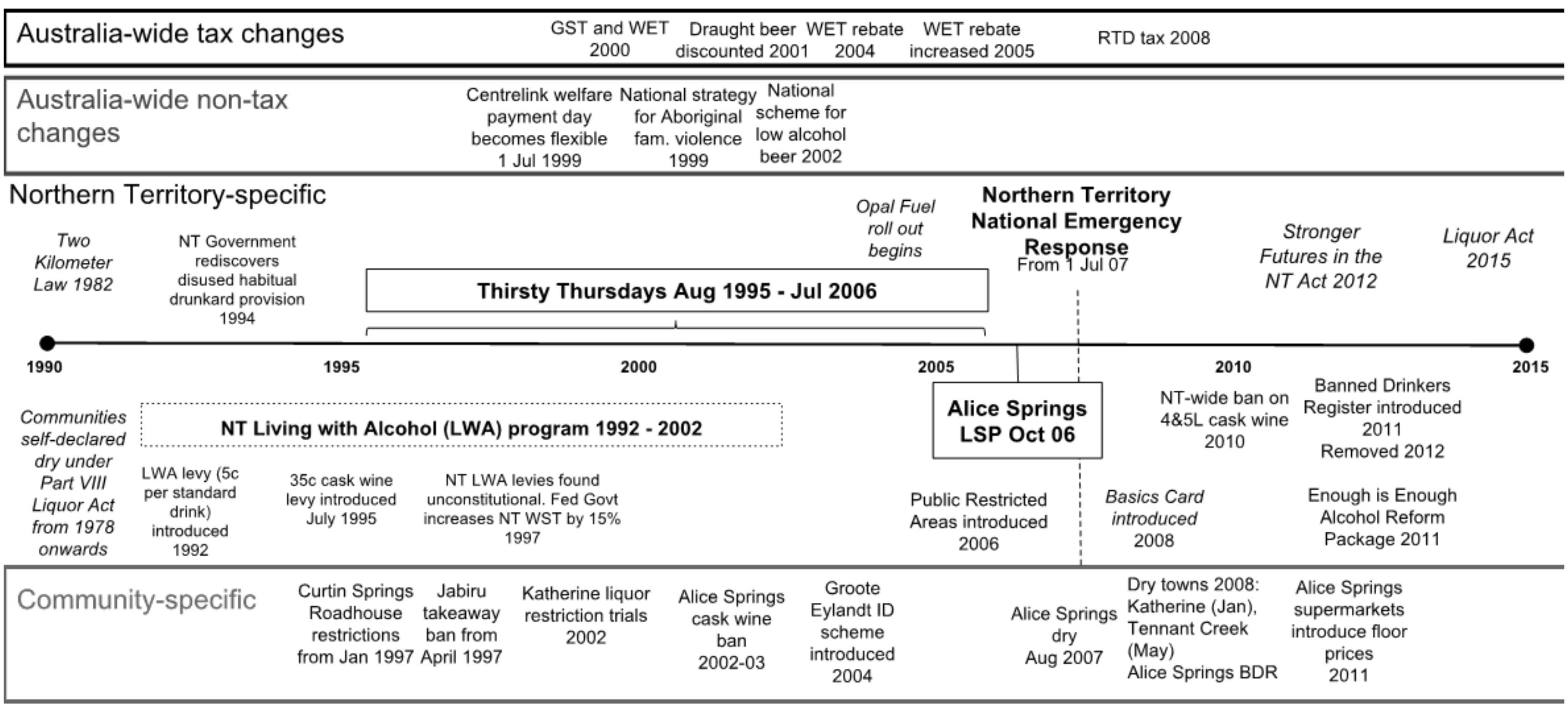

Table A 1 Full estimation results - Pure alcohol sales in litres, total volume and by category

	(1) Total	(2) Cask / Fortified Wine	(3) Full strength beer	(4) Low-strength beer	(5) Wine	(6) Other / cider	(7) Spirits
Treatment area	-5076.45***	403.41**	-2024.72***	-580.13***	-1172.66***	-690.12***	-1664.65***
	(127.85)	(187.92)	(82.97)	(9.10)	(21.43)	(16.94)	(20.04)
Post period	60.88	47.91**	14.26	14.53*	7.56	0.39	-25.87
	(51.70)	(22.97)	(22.95)	(7.49)	(8.05)	(16.73)	(16.38)
Post × Treatment	-309.42**	-491.70***	174.22**	-16.05	39.06*	-25.54	-16.63
	(131.73)	(188.36)	(83.28)	(12.18)	(21.66)	(21.95)	(23.07)
Base: Year 2005							
Year 2006	37.03	-22.86	4.68	4.17	-8.31	41.75**	61.46***
	(43.80)	(14.86)	(16.86)	(7.64)	(6.96)	(19.04)	(18.26)
Year 2007	92.16	-11.40	13.19	-5.14	2.75	48.54	98.39***
	(79.64)	(21.45)	(31.85)	(12.53)	(12.78)	(31.75)	(30.98)
Base: Quarter 1							
Quarter 2	346.40***	16.52	130.75***	48.34***	32.00***	121.94***	117.90***
	(65.47)	(26.23)	(21.08)	(9.92)	(9.31)	(24.70)	(22.54)
Quarter 3	485.24***	33.66	166.42***	56.22***	56.21***	150.80***	170.90***
	(68.32)	(25.09)	(23.78)	(10.56)	(9.89)	(25.43)	(25.35)
Quarter 4	360.37***	-20.19**	133.76***	40.51***	36.57***	129.02***	166.87***
	(55.35)	(8.65)	(21.33)	(8.32)	(7.93)	(22.90)	(24.35)
Year × Quarter							
2006 × q2	-30.23	21.70	-24.43	-7.91	8.75	-33.01*	-30.48*
	(56.30)	(25.83)	(18.02)	(8.39)	(7.99)	(19.81)	(18.32)
2006 × q3	-73.54	11.20	-22.84	-8.72	18.65**	-49.70**	-75.75***
	(53.62)	(31.04)	(16.09)	(7.45)	(9.43)	(22.44)	(23.92)

2006 × q4	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	(.)	(.)	(.)	(.)	(.)	(.)	(.)
2007 × q1	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	(.)	(.)	(.)	(.)	(.)	(.)	(.)
2007 × q2	-29.24	-0.04	-38.89**	0.67	18.84**	-15.70	-10.79
	(54.96)	(26.23)	(15.88)	(6.83)	(9.22)	(15.02)	(17.18)
2007 × q3	21.53	-11.23	-23.92	44.17***	21.33**	41.03***	-10.41
	(46.02)	(23.23)	(15.29)	(10.48)	(9.66)	(15.06)	(15.28)
Constant	4976.02***	49.59***	1742.49***	546.14***	1082.46***	587.23***	1515.83***
	(66.45)	(17.22)	(22.54)	(12.15)	(9.35)	(28.12)	(26.35)
Observations	3661	3661	3661	3661	3661	3661	3661

Note: Model presents full estimates from difference-in-difference model for benchmark model (Equation 1). Retailer sales data from Quarter 2 2005 until Quarter 3 of 2007.

Table A 2 Robustness checks pure alcohol sales per quarter, by category

Outcome in litres	(1) No controls	(2) Retailer fixed effects	(3) Year * Quarter fixed effects	(4) Treatment group specific year trends
Total alcohol	-331.11**	-308.60**	-309.42**	-198.62
	(129.11)	(131.53)	(131.73)	(138.10)
Cask / Fortified wine	-471.97***	-491.71***	-491.70***	-429.74**
	(171.22)	(188.10)	(188.36)	(191.52)
Full strength beer	156.23**	174.52**	174.22**	174.28*
	(78.45)	(83.17)	(83.28)	(91.68)
Low-mid strength	-21.62*	-15.96	-16.05	-9.05
	(12.75)	(12.15)	(12.18)	(10.19)
Regular wine	32.50	39.12*	39.06*	46.78**
	(21.31)	(21.64)	(21.66)	(20.98)
Cider / other	-35.99	-25.23	-25.54	14.76
	(23.98)	(21.89)	(21.95)	(26.25)
Spirits	-27.10	-16.26	-16.63	17.77
	(24.77)	(23.04)	(23.07)	(30.52)
Observations	3661	3661	3661	3661

Note: Estimation Model as in Equation (1), where we are adding subsequently control variables. Column (1) presents estimates include treatment group, post policy dummies plus interaction terms. Column (2) adds retailer fixed effects; column (3) adds year by quarter fixed effects; column (4) adds treatment group specific time trends. The outcome is quarterly sales of pure alcohol by retailer. Clustered standard errors in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A 3 Estimated price elasticity of demand

	(3) Alice Springs	(4) Darwin / Palmerston
Log Price Lag 1	-0.473***	-0.652***
	(0.121)	(0.103)
Linear time trend	-0.001	0.006***
	(0.001)	(0.001)
Quarter 2	0.010	-0.004
	(0.031)	(0.020)
Quarter 3	-0.004	0.014
	(0.031)	(0.020)
Quarter 4	0.009	0.001
	(0.031)	(0.019)
Constant	3.341***	3.045***
	(0.387)	(0.431)
Observations	41	41
Wholesale prices 2006		
Quarter 2	66.08	77.98
Quarter 3	67.51	78.6
Quarter 4	83.61	76.84
Wholesale Quantity 2006		
Quarter 2	3.51	3.51
Quarter 3	3.35	3.37
Quarter 4	3.11	3.43

Note: The model is estimated with a linear regression model where the outcome variable is log of pure alcohol quantity sold by wholesaler (in litres of pure alcohol) and the main dependent variable is a lagged measure of log of price per litre of pure alcohol. Clustered standard errors in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. The analyses use publicly available wholesale data (quantity and price) obtained from Symons et al. (2012) for both treatment and control regions over the period 2003 and 2010.

Table A 4 Summary statistics of estimation sample pre-policy period

	Panel A: Non-Aboriginal Mothers									
	Darwin / Palmerston City + Region					Alice Springs City + Region				
	N	Mean	SD	Min	Max	N	Mean	SD	Min	Max
Female baby	8,467	0.49	0.50	0	1	1,972	0.47	0.50	0	1
Age mother	8,466	29.09	5.68	14	47	1,972	29.65	5.78	14	46
Number of pregnancies*	8,111	2.56	1.77	0	10	1,952	2.46	1.47	0	10
Aboriginal mother	8,467	0.00	0.00	0	0	1,972	0.00	0.00	0	0
Alice Springs	8,467	0.00	0.00	0	0	1,972	1.00	0.00	1	1
Outcomes										
Birthweight in grams	8,465	3348.52	579.42	308	5720	1,972	3415.27	576.32	290	5300
Prob low birth weight	8,467	0.06	0.25	0	1	1,972	0.05	0.22	0	1
Prob high birth weight	8,465	0.10	0.30	0	1	1,972	0.13	0.34	0	1
Gestational age	8,467	38.88	1.99	20	42	1,972	39.09	1.92	20	42
Prob of Premature	8,467	0.08	0.27	0	1	1,972	0.07	0.25	0	1
Mechanisms										
Smoking 1st ant. visit	8,084	0.19	0.39	0	1	1,831	0.21	0.41	0	1
Smoking Week 36	7,249	0.15	0.36	0	1	1,767	0.18	0.38	0	1
Alcohol 1st ant. visit	7,795	0.07	0.26	0	1	1,781	0.12	0.32	0	1
Alcohol Week 36	7,071	0.03	0.18	0	1	1,698	0.06	0.23	0	1
Gestational diabetes	8,467	0.04	0.20	0	1	1,972	0.03	0.18	0	1
Pre-eclampsia	8,467	0.05	0.21	0	1	1,972	0.03	0.17	0	1
	Panel B; Aboriginal Mothers									
	N	Mean	SD	Min	Max	N	Mean	SD	Min	Max
Female child	1,227	0.50	0.50	0	1	718	0.48	0.50	0	1
Age mother	1,227	25.21	5.89	13	51	718	24.83	5.91	13	43
Number of pregnancies*	1,215	3.35	2.12	0	10	717	3.05	1.98	0	10
Aboriginal mother	1,227	1.00	0.00	1	1	718	1.00	0.00	1	1
Alice Springs	1,227	0.00	0.00	0	0	718	1.00	0.00	1	1

Outcomes										
Birthweight in grams	1,227	3189.90	665.90	290	5655	718	3282.33	650.81	420	5280
Prob low birth weight	1,227	0.12	0.33	0	1	718	0.10	0.30	0	1
Prob high birth weight	1,227	0.08	0.27	0	1	718	0.11	0.32	0	1
Gestational age	1,227	38.51	2.60	20	43	718	38.73	2.09	24	42
Prob of premature	1,227	0.14	0.34	0	1	718	0.10	0.30	0	1
Mechanisms										
Smoking 1st ant. visit	1,092	0.54	0.50	0	1	635	0.48	0.50	0	1
Smoking Week 36	943	0.48	0.50	0	1	609	0.41	0.49	0	1
Alcohol 1st ant. visit	1,049	0.13	0.33	0	1	611	0.21	0.41	0	1
Alcohol Week 36	908	0.08	0.28	0	1	584	0.16	0.36	0	1
Gestational diabetes	1,227	0.05	0.22	0	1	718	0.07	0.25	0	1
Pre-eclampsia	1,227	0.04	0.19	0	1	718	0.03	0.18	0	1

Note: *Top-coded at 10, Pre-treatment period refers to pregnancies that were conceived before Q2 2006.

Table A 5 Robustness checks birth outcomes

	(1) No controls	(2) Demographic controls	(3) Retailer Fixed effects	(4) Year-month fixed effects	(5) YM-trend × Retailer
Panel A; Birth weight in grams					
Aboriginal					
LSP × Post	-171.825*	-158.315*	-176.571**	-158.190**	-109.483
	(89.345)	(84.518)	(73.992)	(70.786)	(89.207)
N	3341	3341	3341	3341	3341
Non-Aboriginal					
LSP × Post	27.031	22.657	17.646	18.047	81.340
	(49.357)	(49.659)	(47.734)	(44.934)	(70.756)
N	16861	16861	16861	16861	16861
Panel B: Low birth weight (< 2500 g)					
Aboriginal					
LSP × Post	0.081**	0.075**	0.082**	0.077**	0.090**
	(0.036)	(0.035)	(0.033)	(0.032)	(0.041)
N	3342	3342	3342	3342	3342
Non- Aboriginal					
LSP × Post	-0.006	-0.005	-0.005	-0.004	-0.021
	(0.019)	(0.019)	(0.019)	(0.016)	(0.022)
N	16862	16862	16862	16862	16862
Panel C: Probability of premature birth (gestation<37 weeks)					
Aboriginal					
LSP × Post	0.065**	0.057*	0.068**	0.048	0.065
	(0.030)	(0.029)	(0.030)	(0.032)	(0.046)
N	3342	3342	3342	3342	3342
Non-Aboriginal					
LSP × Post	-0.001	-0.001	-0.002	-0.000	-0.026
	(0.028)	(0.028)	(0.029)	(0.028)	(0.042)
N	16862	16862	16862	16862	16862

Note: Reported is the estimated coefficient (Interaction between Alice Springs region LSP and Post period indicator) from difference-in-difference model, where the outcomes is birth weight in grams (Panel A), a binary measure of whether the child was born with low birth weight (<2500 g) (Panel B), and a binary measure of whether the baby was born premature (gestation < 37 weeks) (Panel C). Each column is a separate regression model that adds a new set of control variables. Each model controls for treatment group (Alice Springs region) and Post period indicator (post Q1 2006 conception). Clustered standard errors in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A 6 Robustness check - widen and shorten treatment window and policy period for Aboriginal mothers

	Widen Post period in 2006			Narrow policy period overall		
	Include Jan-March	Include February-March	Include March	Include up to March 2007	Include up to February 2007	Include up to January 2007
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Birth weight						
LSP × Post	-202.903**	-209.234**	-223.549**	-202.686***	-234.559***	-282.604***
	(88.470)	(81.736)	(83.560)	(66.669)	(63.421)	(66.381)
N	3341	3341	3341	3341	3341	3341
Panel B: Low Birth weight						
LSP × Post	0.069**	0.071**	0.085**	0.081**	0.098***	0.116***
	(0.030)	(0.031)	(0.033)	(0.032)	(0.032)	(0.034)
N	3342	3342	3342	3342	3342	3342
Panel C: Premature						
LSP × Post	0.071**	0.056	0.058	0.067*	0.089**	0.109**
	(0.032)	(0.034)	(0.038)	(0.036)	(0.039)	(0.043)
N	3342	3342	3342	3342	3342	3342

Note: Robustness checks in columns (1)-(3) widen the treatment definition. Benchmark treatment includes babies conceived on or after Q2 in 2006 up until end of Q1 2007, while robustness checks include babies who would be affected in their last term of pregnancies. Columns (4)-(6) shortens the policy period. The benchmark policy period included babies conceived up until the end of Quarter 1 2007. Benchmark estimates are reported in Standard errors in parentheses, * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A 7 Placebo test - Birth outcomes

	Benchmark Estimate Police period: Q2/05-Q1/07		Placebo Policy period: Q2/03-Q1/05	
	(1) Aboriginal mothers	(2) Non-Aboriginal mothers	(3) Aboriginal mothers	(4) Non-Aboriginal mothers
Panel A: Birthweight in grams				
Treatment effect	-158.190**	18.047	0.739	11.764
	(70.786)	(44.934)	(94.301)	(30.410)
N	3341	16861	3341	16861
Panel B: Probability of low birth weight (<2500 grams)				
Treatment effect	0.077**	-0.004	0.034	-0.019
	(0.032)	(0.016)	(0.044)	(0.014)
N	3342	16862	3342	16862
Premature	0.048	-0.000	0.070	-0.001
	(0.032)	(0.028)	(0.044)	(0.015)
N	3342	16862	3342	16862

Note: Reported is the estimated coefficient for the treatment effect of main interest (Interaction between Alice Springs region LSP and Post period indicator) from difference-in-difference model, where the outcomes is birth weight in grams (Panel A), a binary measure of whether the child was born with low birth weight (<2500 g) (Panel B), Each model controls for treatment group (Alice Springs region) and Post period indicator (post Q1 2006 conception). And the full set of control variables. Benchmark estimates refer to the main estimates reported in Table 2. Placebo estimates report the treatment effect for the same model but shifted the treatment window to the placebo period (Q2/2003 until Q1 2005). This is the only available period where no other policies affected treatment and control group. Standard errors, clustered by location, are reported in parentheses * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.