Universal Cash Transfers Reduce Childhood Obesity Rates

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Abstract

We evaluate the impact of universal income on childhood obesity. While the goals of implementing universal income are many, its influence on childhood obesity is of particular interest given the growing obesity epidemic and its future threat to global public health. We use evidence from Alaska's universal income program, the Permanent Fund Dividend (PFD), which has provided annual, unconditional, and universal income to Alaskan residents for over thirty-five years. We use both survey and administrative data to evaluate how the availability of unconditional resources at an early developmental stage, in terms of PFD payments to the child, affects a child's body mass index (BMI). Using date-of-birth eligibility cut-offs as an identification strategy, we find that an additional one thousand dollars in PFD payments decreases the probability of an Alaskan child being obese by as much as 4.5 percentage points. Back-of-the-envelope calculations for Alaska suggest these reduction may avert 500 cases of obesity and achieve medical cost savings of $2-10 million per year. These findings highlight just one of the potential social benefits of universal income and the potential it has as a tool for addressing the obesity epidemic.

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1 Introduction

There has been increased interest in universal basic income (UBI) and its role in our economy's future and the welfare of its citizens. Many politicians and thought leaders have argued for the implementation of a universal and unconditional cash transfer program on a national scale (Alba, 2016), citing its potential to improve economic security (Thigpen, 2016) or as a substitute for existing welfare programs (Murray, 2008). Universal transfers are provided to all residents on a long-term basis, regardless of income, while unconditional payments are those distributed with no "strings attached" (Marinescu, 2017). Several countries, such as Finland and India, have recently implemented UBI experiments (Khosla, 2017; Authors and Research at Kela, 2016). The United States has also shown interest in UBI, as demonstrated by an ongoing random control trial aimed at understanding the effectiveness of universal income on people's well-being across several dimensions, such as employment, social networks, and health (Weller, 2017). On the political front, the Democratic Party nominee for the 2016 US presidential election, Hillary Clinton, considered integrating a UBI proposal into her campaign platform (Matthews, 2017). The program was intended to be named "Alaska for America" given the inspiration it drew from the Alaska Permanent Fund Dividend (PFD), which is an annual universal and unconditional income payment provided to all Alaska residents. While skeptical of costs, conservative thinkers have also written favorably about UBI's potential as a replacement for complex means-tested and strings-attached programs (Tanner, 2015).

Despite the growing interest in UBI, there is little accumulated knowledge regarding its effects on the well-being of recipients. In this paper, we evaluate the impact of universal and unconditional cash transfers on childhood obesity. Although universal income is expected to influence well-being in several different ways, we focus on childhood obesity given the growing obesity epidemic and its future threat to global public health (Lancet, 2011).

We use the Alaska PFD to evaluate how universal and unconditional income affect childhood obesity. The PFD is the world's only continuous universal income program, providing

ongoing annual unconditional payments to all residents (subject to minor eligibility rules) since 1982. The PFD amount varies year-to-year according to the investment earnings of the Alaska Permanent Fund, the state's sovereign wealth fund, and establishes an income floor below which the cash income of residents cannot fall. This cash payment represents a non-negligible portion of Alaskans' earnings, and is particularly important in rural areas where economies lack economic bases and are still a mixture of subsistence and a small formal economy (Goldsmith, 2010). The program is very popular and the public expects it to run in perpetuity. The PFD is distributed to all residents of the state—both adults and children, as well as green-card holders and refugees—regardless of income or wealth, making it universal. Further, residents are not required to meet any conditions before receiving the PFD, nor are they restricted in how they spend the PFD, making it unconditional. Thus, the Alaska PFD is the closest example to a UBI program worldwide and provides a unique opportunity for understanding how unconditional and universal payments influence well-being.[1]

While the goals of UBI are many, its influence on childhood obesity is of particular interest given that the obesity rate in the U.S. has more than doubled (from 5.0% to 12.4%) among children aged 2-5 years since 1980 (Ogden et al., 2014). Childhood obesity has been associated with type II diabetes mellitus, hyperlipidemia, atherosclerosis, hypertension, depression, nonalcoholic fatty liver disease, and obstructive sleep apnea (Daniels, 2006; Dietz, 1998; Krebs et al., 2003; American Academy of Pediatrics, 2003). Additionally, there is evidence that obese children are considerably more likely to be obese as adults, which may negatively affect labor market outcomes (Lindeboom et al., 2010; Cawley, 2015), and early childhood medical interventions have significant impacts on adolescent education outcomes (Bharadwaj et al., 2013; Chyn et al., 2019). Childhood obesity is also responsible for significant health care costs (Biener et al., 2017): the incremental lifetime direct medical cost from the perspective of a 10-year-old obese child relative to a 10-year-old normal-weight child

[1]Other recent empirical studies of the PFD focus on the impact of the PFD on consumption and spending (Hsieh, 2003; Kueng, 2018), crime (Watson et al., 2019), and employment (Jones and Marinescu, 2018; Bibler et al., 2019; Feinberg and Kuhn, 2018)

ranges from $12,660 to $19,630 (Finkelstein et al., 2014).

Income, and how it interacts with obesity, is an important part of almost all policy interventions. In theory, additional income could lead to an increase or decrease in childhood obesity. Higher income, for example, can prevent weight gain by allowing parents to substitute healthier, more costly, food for cheaper energy-dense food or by increasing demand for a child's health care. On the other hand, additional income could promote weight gain by allowing children to consume more calories and spend more time in sedentary pursuits (Cawley, 2015). Several studies have found that conditional cash transfers improve health and nutritional outcomes for children in early life (Rasella et al., 2013; Reis, 2010; Schmeiser, 2012; Fernald et al., 2008); however, it is likely that these benefits are achieved in part due to the conditions imposed by the program. Existing studies of unconditional income payments and obesity have found mixed results: Swedish lottery payments have been shown to reduce obesity across income groups (Cesarini et al., 2016), but dividends from a tribal casino have been found to increase obesity among the poorer households with non-offsetting reduction in obesity for wealthier households (Akee et al., 2013). These payments, however, do not stem from a universal program and focus on obesity outcomes for young adults, as opposed to children. Chung et al. (2016) show the Alaska PFD has large, positive effects on birth weights, particularly for mothers with less education.

The Alaska PFD presents an ideal setting to understand the impact of unconditional, anticipated, and universal cash on children's obesity at a very early age. Indeed, the PFD may be more conducive to influence childhood obesity compared to other cash transfer programs. The PFD payments we study are nominally assigned to the child; thus, a labeling effect may induce parents to spend this cash disproportionately on the child relative to other sources of income (Kooreman, 2000). The income effect of the PFD also causes mothers to reduce their labor supply (Bibler et al., 2019), enabling more maternal time with children. Schaller and Zerpa (2019) show maternal job loss can improve children health through such a pathway, while Jo (2018) finds a similar effect acting on childhood obesity. Finally, the

PFD is universal and is thus distributed across the entire income distribution, including segments of the population for which obesity may be more responsive to income payments (Lakdawalla and Philipson, 2009; Jo, 2014).

We determine the causal effect of universal income receipt on childhood obesity by exploiting quasi-experimental variation in the cumulative amount of PFD received by a child. We find that a one-thousand dollar PFD payment significantly decreases the probability of being obese as a child by as much as 4.5 percentage points, which equates to a 22.4% reduction in the number of obese 3-year-old Alaskans. Consistent with theory (Lakdawalla and Philipson, 2009), the effect of the PFD on obesity is nonlinear in household income: middle-income households are particularly responsive to the PFD payments while there is no detectable response from high- and low-income households. Back-of-the-envelope calculations suggest that a one-thousand dollar investment per child avert 500 annual obesity cases in Alaska, resulting in obesity-related medical expenditure savings of $2-10 million before those children turn 18. Importantly, our results suggest that UBI could have far-ranging benefits to society and has potential for combating the growing obesity epidemic.

2 Background, Data, and Research Design

2.1 The Alaska Permanent Fund Dividend

Since 1982, the Alaska has distributed annual dividend payments to residents based on the earnings of the state's sovereign wealth fund, the Alaska Permanent Fund. The fund was established in 1976 and capitalized with royalties generated from oil production. Today, its value stands at over 63 billion dollars with investment in a diverse set of assets. The annual dividends paid from fund earnings represents a non-negligible portion of Alaskans' income. For example, between 2000 and 2016, the average PFD size per-person was $1,600, representing 6.28% of overall household income. PFD payments are determined by a formula that is based on a rolling average of the Fund's income over five years in order to produce

more stable dividend amounts from year to year. It is important to note that the Fund is well-diversified across different regions and asset classes. State oil revenue, which originally capitalized the fund, currently represent only 2-3% of annual fund additions; since 1985, investment returns are the main growth mechanism. The Fund's returns, and thus the size of the PFD payments, are therefore not necessarily reflective of Alaska's economic conditions.

2.2 Identification Strategy

The influence of income on childhood obesity is challenging to identify empirically: if household income is determined by unobserved factors that also influence a child's weight, then the estimated relationship between income and obesity will be spurious (Kuehnle, 2014). We are able to address this issue by exploiting two forms of quasi-experimental variation in income. First, an Alaskan resident adult may sponsor a newborn child to receive a PFD if the child is born before December 31st of the qualifying year. Because of this rule, a child born on December 31st will receive one more PFD than a child born one day later on January 1st (Fig. 1). So long as a child's date-of-birth has no independent effect on obesity, the additional PFD amount received from being born before the eligibility cut-off can be considered exogenous. Second, the PFD has seen considerable variation in size over the last two decades: the PFD has averaged approximately $1,600 per person, with a high of $3,200 in 2008 and low of $900 in 2012 (all in nominal terms). As a result, the cumulative amount of PFDs received by a particular age will differ across children. The exogenous nature of these two sources of income variation supports a causal interpretation of our estimated effect of the PFD on childhood obesity.

2.3 Data

Our analysis relies on linked survey and administrative data called the Alaska Longitudinal Child Abuse and Neglect Linkage Project (or ALCANLink), an ongoing project which combines two surveys conducted by the Alaska Department of Health and Social Services—

the Pregnancy Risk Assessment Monitoring System (PRAMS) survey and the Childhood Understanding Behaviors Survey (CUBS)—with administrative data from vital records and the Alaska Permanent Fund Dividend Division.[2] The PRAMS survey samples one-sixth of all mothers delivering live births in Alaska and collects information on pre- and post-natal behaviors and outcomes of mothers and their newborn children. The survey is administered by mail two to six months after birth (with follow-up by phone) and has historically had a ∼65% response rate. The survey oversamples mothers of low birth weight children and Alaska Native people. CUBS is an Alaska-specific program developed as a three-year follow-up survey to the PRAMS survey to understand the behavior and outcomes of toddlers. It is administered two months after their child's third birthday to all PRAMS survey respondents who remain in-state. Linked vital statistics includes information on birth weight, birth date, presence of birth defect, use of c-section, place of birth, race, ethnicity, mother's age at delivery, smoking status, martial status, mother height and weight, gestational diabetes, drinking indicator, and Kessner index. Figure 1 illustrates how birth timing and the date a mother returns the CUBS follow-up survey determines the number of PFDs that children accumulate before a mother records a child's obesity at age three.

Our data is a subset of the ALCANLink project, covering children who were the subject of a CUBS follow-up survey. These data cover children born between January 2009 and December 2011, have mothers who were sampled by (and responded to) the PRAMS survey two to six months later, and include mothers who received and responded to CUBS between 2012-2015. The time period of coverage for the current data contained in ALCANLink was chosen principally for administrative reasons. Data from the Permanent Fund Division allow us to observe the application status of each child in each year between birth and their CUBS survey, which provides the information to calculate the accumulated dollars of dividend received by each child. Table 1 shows the number of children in each obesity category.

Our primary outcome of interest is the obesity status of children at the age of three,

[2]We obtained these confidential data via data use agreement with Alaska DHSS.

the age at which the follow-up survey (CUBS) is administered. We adopt the conventional measure of child obesity, which is based on body mass index (BMI) referenced to Centers for Disease Control and Prevention (CDC) growth charts.[3] The CDC defines four weight categories for children's BMI: underweight, normal weight, overweight, and obese, with category cutoffs based on the 5th, 85th, and 95th percentiles, respectively, of the U.S. population in the 1970s. Mothers report their children's height and weight in CUBS, and BMI and obesity status are calculated before inclusion in ALCANLink. While our analysis is focused on obesity outcomes ($\geq$95th percentile), we also examine the prevalence of overweight children ($\geq$85th percentile). Table 1 describes the distribution of three-year-old children across these BMI classes. Table 1 includes all children who's obesity status was calculable (n=1225), while our main analysis locks the sample with a number of controls where some data are missing (n=885).

We categorize control variables into three groups: child characteristics, mother characteristics, and early nutrition characteristics. We test the balance for each control variable across the number of PFDs a child receives at the time that CUBS is administered (Tables A.1, A.2, and A.3). For the child characteristics, balance is rejected for: child age, with younger children (indicating a prompt response to the CUBS follow-up survey) receiving fewer dividends; and birth weight, with children receiving one PFD having a lighter birth weight than children receiving two or three PFDs. Mother characteristics are balanced for all control variables. Critically, we observe no statistically significant difference in race, age, or income across mother's whose children receive one, two, or three PFDs. For early nutrition characteristics, children receiving a single PFD tend to have later introductions to solid foods and liquids other than breast milk.

[3]BMI is calculated as $weight/height^2$.

2.4 Empirical Model

We estimate the probability of being obese at age three ($t = 3$) using the following model:

$$P(Ob_{i,t=3} = 1) = \Lambda\big(\beta TotalPFD_i + \gamma X_i\big), \tag{1}$$

where Ob_i is a binary variable equal to one if a child is obese (i.e., BMI is $\geq$95th percentile cutoff) and zero otherwise; $TotalPFD_i$ is the total amount of PFDs (in 1,000 dollars) the child received on or before a mother completes CUBS; X_i is a vector of control variables: demographics and child, mother, and early nutrition characteristics (Tables A.1, A.2, and A.3); $\Lambda(\cdot)$ denotes the logit function; and the parameter β is the coefficient of interest. To explore whether the effect of the PFD on childhood obesity varies across income groups, we also interact the total amount of the PFD received by a child with a categorical variable indicating household income (less than \$25,000; between \$25,000 and \$75,000; and above \$75,000).[4] Note that we do not use birth-month fixed effects given that a significant portion of our variation stems from the number of PFDs received, which is driven by birth month. The marginal effect of the PFD is the reduction in the probability of being obese at age three from a marginal increase in the PFD:

$$R = P(Ob_{i,t=3} = 1)[1 - P(Ob_{i,t=3} = 1)]\beta. \tag{2}$$

3 Results

The accumulation and investment of universal and unconditional income generates significant and meaningful reductions in childhood obesity in our sample. We find that an additional \$1,000 in accumulated PFD reduces the relative probability of being obese as a three-year-old to 0.691, which is equivalent to reducing the average probability of being obese by 5.2 percentage points, all else equal (Table 2, column 1). The estimates in Table 2 are

[4]These income groups roughly define terciles for our sample.

sample average treatment effects and do not account for the over-sampling of low-birth-weight children and Alaska Native mothers on the part of the survey design. Using survey sample weights slightly reduces the marginal effect of the PFD to 4.5 percentage points. Our results hold even when using different constructions of obesity status by comparing obese children to only normal-weight children (column 2); obese *and* overweight children to both normal and underweight children (column 3); or overweight and obese children to only normal-weight children (column 4). Furthermore, the marginal effect of the PFD increases by $\sim$1 percentage point when moving from columns (1-2) to columns (3-4), which implies that the PFD also lowers incidences of overweight status. We test our primary model specification across a number of alternative dependent and independent variable specifications and present these results in Table 3. The effect of the PFD on obesity and overweight status is negative and statistically significant at the 95% level for all models that condition on child characteristics and nutrition or mother characteristics.

We also find evidence of a nonlinear relationship between household income and the effect of the PFD on childhood obesity (Table 4). Specifically, we find that the obesity-reduction effect is driven by middle-income families (those households earning between \$25,000-\$75,000 per year). We find no evidence of an effect of the PFD for low- and high-income families. The same analysis across a more refined disaggregation across the eight income categories in the survey instrument reveals a relatively smooth U-shaped relationship between the PFD effect and household income (Table 5 and Figure 2). As before, the PFD's effect is statistically significant and negative for the middle-income group (\$25,000-\$75,000), but generally not for high- and low-income families.

3.1 Robustness Checks

The causal interpretation of our results relies on the quasi-experimental nature in how PFD payments are accumulated over a child's first three years. Nonetheless, there are four potential threats to our identification strategy, which we address in this section. First, a parent

may forget or opt out of applying for a child's PFD for reasons that may be related to the child's weight. Second, parents could also be strategic in timing their child's conception in order to be eligible for an additional PFD. If these decisions are associated with a child's home and nutritional environments, then the estimated effect of the PFD will be biased. Third, there is evidence that birth season is associated with a mother's characteristics (Buckles and Hungerman, 2013); for example, winter births are disproportionately realized by teenage and unmarried women. Thus, children may be systematically heavier or lighter depending on the month in which they are born. Fourth, there are several federal tax benefits—e.g., Earned Income Tax Credit (EITC), child tax credit, dependent deduction, etc.—that a child born before December 31st will qualify for when filing taxes the following spring (2-3 months after birth). In contrast, a child born after January 1st will not qualify for such benefits until the following year. As a final robustness check, we compare our estimates to a reference distribution of placebo effects, where the amount of PFD accumulated by a child at the age of three is artificially reassigned across all subjects in the sample.

3.1.1 Application Status

A parent may forget or opt out of applying for a child's PFD for reasons that may be related to the child's weight. If these decisions are correlated with home and nutrition environments to which the child is exposed, then the estimated PFD effect will be biased. We conduct two tests to address this concern. First, we condition on whether parents applied for every dividend a child was eligible for. The model results, Table 6, with the added control are quite similar to those of our primary specification. Second, we employ a two-stage least squares (2SLS) approach to address the potential endogeneity in application status.[5] We test three different instruments for $TotalPFD$: the number of years a child was eligible to receive a PFD (either 2 or 3), the total dollar value of PFD a child was eligible to receive over the period, and an over-identified model using both the number and average dollar value of PFD

[5]Note that since 2SLS is a linear estimator, the second stage results will differ from the those of the logit model estimated in our primary specification.

a child receives. The results of the 2SLS estimations are presented in Table 7. There is a strong relationship between the instruments in the first stage as we reject the hypothesis of weak instruments at any conventional level. The Wu-Hausman test suggests that the value of cumulative PFD received is exogenous to application status. Further, the Sargan test for endogeneity enabled by our over-identified models in columns 3 and 6 indicates that we can't reject the null hypothesis that our instruments are valid. The coefficients in Table 7 are qualitatively similar to our estimates of the marginal effects estimated in our logit regressions. Altogether, these robustness checks provide support that our results are not invalidated by any PFD application selection bias.

3.1.2 Endogenous Birth Timing and Birth Season

Birth season may also be related to obesity outcomes in ways that are unrelated to receiving the PFD. If our control variables do not capture these effects, our estimates of the PFD's effect on obesity will be biased. While it may be tempting to address this issue by using birth-month fixed effects, such effects are subsumed by the variation we exploit in the birth-date cutoff (Jan. 1) in our identification strategy. Approximately 70% of the variation we observe in the cumulative PFD a child receives is explained by birth month, with the balance split between year of birth, application status, and mother's survey response timing. Thus, rather than relying on birth-month fixed effects, we address this concern by controlling for strategic birth timing and trimming our sample to include only the fourth and first quarters of the year (October through March).

It is useful to distinguish between two possible sources of birth season endogeneity. The first is strategic birth timing, where parents manipulate conception timing in order for children to be born in a particular season, potentially to receive an additional PFD. Endogeneity arises if these parents have unobservable characteristics that are correlated with obesity (e.g., "savvy" parents may also be more health conscious). We are able to construct a variable from the survey that controls for such strategic timing. The PRAMS survey asks directly

if a mother was: a) trying to get pregnant at the time she became pregnant, and b) if the mother was happy with the pregnancy timing (or whether she would have preferred to be pregnant soon or later). Our constructed variable for strategic timing "Intended Timing Preg." is the intersection of a "yes" response to each of these two questions—i.e., the mother was trying to become pregnant *and* she was happy with the timing. While this control is positively associated with obesity, its inclusion has no meaningful effect on the PFD effect of interest (Table 8), suggesting that any strategic birth timing does not result in increased PFD accumulation.

The second source of birth-season endogeneity arises if birth season itself has an effect on obesity. To address this concern, we narrow our sample to only the 1st and 4th quarters of the year, which include months (October-March) that have similar characteristics (temperature, daylight hours, etc.). The results of estimating the model on this narrower sample are presented in Table 9. Dropping approximately half the sample decreases the precision of the PFD estimate, but increases their magnitude. In Table 10 we present results from a particularly aggressive specification, using the control variables for application status and strategic birth timing along with the trimmed sample (Q1 and Q4 only). This specification leads to the largest estimate of the PFD effect in our study, nearly a 8.5 percentage-point decrease in obesity, suggesting that our primary estimate is mostly driven by children born during the winter months.

3.1.3 Tax Benefits

Birth timing around the birth-date eligibility cutoff (Jan. 1) also defines child tax benefits, as well as PFD receipt. This potentially conflates our estimate with tax benefits that families receive sometime during the late winter/spring as part of their annual tax return. Such tax benefits are determined by whether a child is born during the tax year, income, marital status, and the number of other dependents/children in the family. The marginal tax benefit reflected in CUBS will also depend on CUBS survey timing relative to when families file

income tax returns. Figure 3 shows a timeline of the tax return dates and PFD payout dates by a child's birth month. The interactions of the various timings define four groups. Group one, children born in January or February, is eligible for between two and three tax returns and is eligible for two PFDs. Group two, children born in March through June, is eligible for three tax returns and between two and three PFDs. Group three, born in July, is eligible for three to four tax returns and between two and three PFDs. The final group, born August through December, could receive between three to four tax returns and is eligible for three PFDs. We attempt to flexibly model tax effects by adding the following interaction term (along with its primals) to the model in Eq. 1: $TaxPFDGroup \times Income \times Married \times Dependants$, where $TaxPFDGroup$ denotes the group assignment based on birth month (as described above). Since income tax benefits also depend on household income ($Income$), marital status ($Married$), and the number of other children and dependants ($Dependents$), we interact birth group with these variables to create a flexible way to disentangle the effects of income tax benefits from the effect of the PFD.[6] We note that by controlling for the number of eligible PFDs in $TaxPFDGroup$, we are removing an important source of variation from our estimate of the PFD's effect on obesity. Specifically, in this model we are exploiting only the annual variation in PFD amount and the actual application status of the children. The results presented in Table 11 show that the while the magnitude of the PFD's effect on obesity increases (with a decrease in precision), the inference remains relatively unchanged.

3.1.4 Placebo Tests

As a final robustness check, we compare our estimates to a reference distribution of placebo effects, where the amount of PFD accumulated by a child at the age of three is artificially reassigned across all subjects in the sample. Obtaining similar or larger estimates when the

[6] *Income* is a categorical measure of self reported income, categorized into 5 bins. *Married* is a binary variable equal to one if the mother is married when responding to PRAMS. *Dependants* is the number of people a mother reports as being dependent on *Income*, such as themselves, their partners, children, other family members, etc. While dependent children under 18 are especially important for tax treatments, such as the EITC and the child tax credit, the number of total dependents is also relevant for the purpose of exemptions.

accumulated PFD is artificially reassigned across subjects would suggest that we have found our effect by chance (Abadie et al., 2010). Such permutation tests are based on the fact that, under the null hypothesis of no effect, arbitrarily reassigning accumulated PFD across subjects should have no influence on the incidence of obesity. As demonstrated in Figure 4, our estimate (the point) lies below the 0.5 percentile of the distribution of placebo effects, providing additional evidence of the significance of our effect.

3.2 Estimated Savings in Medical Expenditures

To get a sense of the economic significance of our results, we simulate how the estimated effect of the PFD on childhood obesity might persist through adolescence and reduce associated medical expenditures for an average Alaskan child and the average cohort of Alaskan-born children. The simulation, described in complete detail in Appendix A, accounts for the fact that as children mature, they transition into and out-of obesity, which attenuates the age-three treatment effect. While obesity in adolescence has been shown to persist into adulthood, we focus only on the benefits accrued before age 18 since these are incurred soonest after our treatment effect. Additionally, while many studies have shown that there are substantial market and non-market costs associated with obesity apart from direct medical expenses (Dee et al., 2014; Goettler et al., 2017), we focus narrowly on the medical costs since indirect costs are typically measured for adults.

We estimate the difference in medical expenditures of the average cohort of Alaskan-born obese three-year-olds, relative to their cost had they not been obese. We measure this difference over a 15-year horizon, from four through seventeen years of age. To determine the effect of the PFD on obesity-related medical-cost savings, we hypothetically reduce the amount of PFD received by a three-year-old by one-thousand dollars and predict how many additional obesity cases are created at age three, how these persist into adolescence, and how a child's cumulative medical expenses are impacted by the age of seventeen.

The expected medical-cost savings for a child at age $t > 3$ from a marginal increase in

the PFD are:

$$c \times \Big[P(Ob_t = 1|Ob_{t=3} = 1) - P(Ob_t = 1|Ob_{t=3} = 0)\Big] \times \hat{R} \tag{3}$$

where c is the annual medical cost of being obese (relative to not being obese), $\hat{R}$ is the reduction in the probability of being obese at age three given a marginal increase in the PFD, and the expression in the brackets represents the relative influence of a child's obesity status at the age of three ($Ob_{t=3}$) on the likelihood of being obese at age t.[7] We estimate a child's medical-cost savings between the ages of four and seventeen from the PFD for a cohort of size N by summing the savings in Eq. 3 across all N individuals and ages.

Estimates of the relative medical cost of being obese c are taken from the literature (Biener et al., 2017) and $\hat{R}$ is the predicted marginal effect of the PFD on a child's obesity status (Eq. 2). The probability of a child being obese at age three, $P(Ob_{t=3} = 1)$, is determined for Alaska from the CUBS sample in combination with the PRAMS survey-design weights. These probabilities for an individual three-year-old are then multiplied by the average size (11,000) of the 2009-2011 Alaska birth cohorts (Martin et al., 2011, 2012, 2013).

To project a child's obesity status over a 15-year horizon, we employ a standard modeling approach from the literature and estimate transition probabilities to and from obesity-status categories. We estimate transition probabilities as a function of a child's initial obesity status and their mother's BMI (Whitaker et al., 1997):

$$P(Ob_{i,t} = 1|Ob_{i,t=3}) = \Lambda\big(\gamma_1 Ob_{i,t=3} + \gamma_2 BMI_i^{mtr}\big)t, \tag{4}$$

where $Ob_{i,t=3}$ denotes a child's obesity status at age three; BMI_i^{mtr} denotes the BMI of the child's biological mother, averaged over the child's observed lifetime; and γ is a vector of parameters to be estimated. Since CUBS is not a longitudinal study, we do not observe the

[7] See Appendix A for a derivation of this result.

long-run obesity outcomes of the children in our sample. Instead, we estimate Eq. 4 using the National Longitudinal Survey of Children and Young Adults. Confidence intervals for our estimated medical-cost savings are obtained via a bootstrapping procedure (See Appendix A for additional details regarding the cost-savings projections).

Table 12 presents the results from our medical-cost-savings simulaiton exercise. Obesity rates for Alaskan-born three-year-olds between 2012 and 2014 were 0.203, which translates into 2,230 Alaskan obesity cases. Extrapolating the estimated marginal effect of the PFD (-4.5 percentage points) to the Alaska three-year-old population, we find that 500 cases of obesity were averted from an additional $1,000 in PFD payments, which is equivalent to a 22.4% reduction in the number of obese three-year-olds. Drawing from the literature (Cawley, 2010), obese children incur $1,400 more in medical expenses per year relative to children who are not obese, on average. Assuming that all averted three-year-old obesity cases remained non-obese through the age of seventeen, a $1,000 universal income payment would result in medical-cost savings of $10.3 million for the average Alaska-born cohort, respectively, which equates to $920 per person. If we assume instead that obesity is not fully persistent (i.e., the effect of the initial cash transfer on obesity decays with age), we find medical-cost savings that are approximately one-quarter of the previously estimated size—$2.3 million for the average Alaska-cohort, respectively, which translates into 21 cents in savings for every dollar spent.

4 Conclusion

The use of universal and unconditional cash transfers to address the fast-pace of economic change has gained momentum, but we have yet to understand its consequences on health and well-being. We contribute to furthering this understanding by focusing on an issue that is expected to be a significant threat to global public health (Lancet, 2011). Indeed, obesity has become an epidemic throughout the world, with the United States leading the way with

an obesity rate of approximately 36.5% among U.S. adults between 2011-2014. According to Cawley (2010), the estimated annual cost of treating obesity in the U.S. for the adult non-institutionalized population is $168.4 billion, or 16.5% of national spending on medical care.

We find that a one-thousand dollar unconditional and universal income payment decreases the probability of being obese as a child by 4.5 percentage points, which equates to a 22.4% reduction in the number of obese 3-year-old Alaskans. The averted obesity cases result in average medical-cost savings between 20 and 92 cents per PFD dollar by the age of 17, depending on how the effect of the cash transfer is assumed to persist over time. These estimates represent a lower bound since they do not account for lifetime medical-cost savings, they do not include additional medical-cost savings that might be realized from receiving PFD payments beyond the age of three, and they ignore the indirect effects of obesity, which tend to be larger than the direct ones that we estimate (Dee et al., 2014).

The magnitude of our estimates are larger than some previously reported in the literature on benefit transfers and obesity (e.g., Jo, 2018; Schmeiser, 2012; Akee et al., 2013; Cesarini et al., 2016). However, these differences can be largely explained by three distinct features of the PFD and the timing of cash transfer. First, while classic economic theory assumes the fungibility of money, evidence from the behavioral economics literature suggests that mental accounting might drive PFD recipients to experience a labelling effect, whereby money is mentally assigned to particular forms of consumption based on how it is acquired. Because the PFD payments we study are assigned to the children (rather than the parents or the household), parents may spend this cash disproportionately on children relative to other sources of income. Indeed, previous research has demonstrated that "child benefit" payments issued in the Netherlands are disproportionately spent on child expenditures (Kooreman, 2000). Second, the PFD is distinct from wage subsidy payments, such as the EITC, which tend to draw mothers into the labor force, and in turn, decrease a mother's home production, particularly activities like preparing healthy meals or physical activities

with children (Jo, 2018). In contrast, the PFD has been demonstrated to have the reverse effect, allowing mothers to substitute unearned for earned income and decrease their labor supply (Bibler et al., 2019). Third, our study population is younger and has a higher baseline obesity rate than those of past studies. Bharadwaj et al. (2013) and Chyn et al. (2019) find early health interventions can have significant impacts on educational outcomes later in life. In the context of obesity, younger children (particularly under the age of three) are much more responsive to changes in nutrition than older children (Schroeder et al., 1995; Martorell, 2017). Finally, the PFD is distributed to households across the entire income distribution, and thus, our sample is comprised of families with relatively larger household incomes. The samples of previous studies are comprised of relatively poor families with average incomes ranging from \$19,000-\$24,000 per year (Schmeiser, 2012; Akee et al., 2013; Cesarini et al., 2016). Consistent with our findings, these studies find a small (if any) effect of unearned income or wage subsidy payments on obesity for this income group. In contrast, the average household income in our study is \$45,000, with approximately 40% of our sample lying in the household income category (\$25,000-\$75,000) that is responsible for driving our estimated effect. Thus, the universal nature of the PFD is a rare opportunity to explore how all segments of the population respond to cash transfers, not just those portions of the population that have been targeted in previous programs.

Identifying the mechanisms through which additional income reduces obesity is of great interest for policy (Currie, 2009; Kuehnle, 2014). Unfortunately, our data do not allow us to conduct a thorough evaluation of how additional PFD resources are used by families.

While our study has important implications for universal income programs, we are somewhat limited by the nature of survey data, such as non-response bias and measurement error. For example, we find that not reporting height and/or weight (used to calculate our BMI measure) tends to be correlated with observable characteristics such as race and income. Future research would benefit from administrative data with more systematic collection.

Our investigation documents the causal relationship between universal cash transfers and

childhood obesity. The medical cost savings we estimate are considerably larger than those found in most school interventions (Cradock et al., 2017), but are much smaller than the ones obtained from sugar sweetened beverage excise tax, and nutrition standards (Gortmaker et al., 2015). It is important to note, however, that the reductions we observe are a byproduct of the unconditional cash transfer and not one of its stated goals; thus, the benefits we identify are only a small portion of the intended effects of universal income and should not taken as a complete cost/benefit accounting of such a policy. Nevertheless, our results make it clear that universal income has the possibility of improving children's health, which can have long-lasting monetary and non-monetary benefits. It is also encouraging that these health improvements are a result of a non-targeted obesity intervention. It is therefore possible that universal and unconditional cash transfers have far-ranging benefits to society that go beyond those intended by a UBI program.

References

Abadie, A., A. Diamond, and J. Hainmueller (2010). Synthetic Control Methods for Comparative Case Studies: Estimating the Effect of Californias Tobacco Control Program. *Journal of the American Statistical Association 105*(490), 493–505.

Akee, R., E. Simeonova, W. Copeland, A. Angold, and E. J. Costello (2013, 1). Young Adult Obesity and Household Income: Effects of Unconditional Cash Transfers. *American Economic Journal: Applied Economics 5*(2), 1–28.

Alba, D. (2016, 10). We Must Remake Society in the Coming Age of AI: Obama. *Wired.com*.

American Academy of Pediatrics (2003). Prevention of Pediatric Overweight and Obesity. *Pediatrics 112*(2), 424–430.

Authors and Research at Kela (2016). From idea to experiment: Report on universal basic income experiment in Finland.

Bharadwaj, P., K. V. Løken, and C. Neilson (2013). Early life health interventions and academic achievement. *American Economic Review 103*(5), 1862–1891.

Bibler, A., M. Guettabi, and M. N. Reimer (2019). Short-Term Labor Responses to Unconditional Cash Transfers.

Biener, A., J. Cawley, and C. Meyerhoefer (2017). The Medical Care Costs of Youth Obesity: An Instrumental Variables Approach. Technical report.

Buckles, K. S. and D. M. Hungerman (2013). Season Of Birth And Later Outcomes: Old Questions, New Answers. *The Review of Economics and Statistics 95*(3), 711–724.

Cawley, J. (2010). The economics of childhood obesity. *Health Affairs 29*(3), 364–371.

Cawley, J. (2015, 9). An economy of scales: A selective review of obesity's economic causes, consequences, and solutions. *Journal of Health Economics 43*, 244–268.

Cesarini, D., E. Lindqvist, R. Östling, and B. Wallace (2016, 5). Wealth, Health, and Child Development: Evidence from Administrative Data on Swedish Lottery Players. *The Quarterly Journal of Economics 131*(2), 687–738.

Chung, W., H. Ha, and B. Kim (2016). Money transfer and birth weight: Evidence from the Alaska permanent fund dividend. *Economic Inquiry 54*(1), 576–590.

Chyn, E., S. Gold, and J. S. Hastings (2019). The Returns to Early-life Interventions for Very Low Birth Weight Children. Technical report.

Cradock, A. L., J. L. Barrett, E. L. Kenney, C. M. Giles, Z. J. Ward, M. W. Long, S. C. Resch, A. A. Pipito, E. R. Wei, and S. L. Gortmaker (2017). Using cost-effectiveness analysis to prioritize policy and programmatic approaches to physical activity promotion and obesity prevention in childhood. *Preventive Medicine*.

Currie, J. (2009, 3). Healthy, Wealthy, and Wise: Socioeconomic Status, Poor Health in Childhood, and Human Capital Development. *Journal of Economic Literature 47*(1), 87–122.

Daniels, S. R. (2006). Consequences of Childhood Overweight and Obesity. *The Future of Children 16*(1), 46–67.

Dee, A., K. Kearns, C. O'Neill, L. Sharp, A. Staines, V. O'Dwyer, S. Fitzgerald, and I. J. Perry (2014). The direct and indirect costs of both overweight and obesity: A systematic review. *BMC Research Notes*.

Dietz, W. H. (1998, 3). Health consequences of obesity in youth: childhood predictors of adult disease. *Pediatrics 101*, 518–525.

Feinberg, R. and D. Kuhn (2018). Guaranteed Non-Labor Income and Labor Supply: The Effect of the Alaska Permanent Fund Dividend. *BE Journal of Economic Analysis and Policy Vol. 18*(No. 3).

Fernald, L. C., P. J. Gertler, and L. M. Neufeld (2008). Role of cash in conditional cash transfer programmes for child health, growth, and development: an analysis of Mexico's Oportunidades. *The Lancet*.

Finkelstein, E. A., W. C. K. Graham, and R. Malhotra (2014). Lifetime Direct Medical Costs of Childhood Obesity. *PEDIATRICS*.

Goettler, A., A. Grosse, and D. Sonntag (2017). Productivity loss due to overweight and obesity: A systematic review of indirect costs. *BMJ Open*.

Goldsmith, S. (2010). The Alaska Permanent Fund Dividend: A Case Study in Implementation of a Basic Income Guarantee. In *13th Basic Income Earth Network Congress*, Sao Paulo, Brazil.

Gortmaker, S. L., Y. C. Wang, M. W. Long, C. M. Giles, Z. J. Ward, J. L. Barrett, E. L. Kenney, K. R. Sonneville, A. S. Afzal, S. C. Resch, and A. L. Cradock (2015). Three interventions that reduce childhood obesity are projected to save more than they cost to implement. *Health Affairs*.

Hsieh, C.-T. (2003). Do Consumers React to Anticipated Income Changes? Evidence from the Alaska Permanent Fund. *The American Economic Review 93*(1), 397–405.

Jo, Y. (2014, 12). What money can buy: Family income and childhood obesity. *Economics & Human Biology 15*, 1–12.

Jo, Y. (2018, 7). Does the earned income tax credit increase children's weight? The impact of policy-driven income on childhood obesity. *Health Economics 27*(7), 1089–1102.

Jones, D. and I. E. Marinescu (2018, 2). The Labor Market Impacts of Universal and Permanent Cash Transfers: Evidence from the Alaska Permanent Fund. *Working Paper 24312. National Bureau of Economic Research*.

Khosla, S. (2017). India's Universal Basic Income: Bedeviled by the Details. *Carnegie India*.

Kooreman, P. (2000). The labeling effect of a child benefit system. *American Economic Review 90*(3), 571–583.

Krebs, N. F., M. S. Jacobson, and American Academy of Pediatrics Committee on Nutrition (2003, 8). Prevention of pediatric overweight and obesity. *Pediatrics 112*(2), 424–430.

Kuehnle, D. (2014). The causal effect of family income on child health in the UK. *Journal of Health Economics*.

Kueng, L. (2018, 11). Excess Sensitivity of High-Income Consumers. *The Quarterly Journal of Economics 133*(4), 1693–1751.

Lakdawalla, D. and T. Philipson (2009, 12). The growth of obesity and technological change. *Economics & Human Biology 7*(3), 283–293.

Lancet (2011, 8). Urgently needed: a framework convention for obesity control. *378*(9793), 741.

Lindeboom, M., P. Lundborg, and B. van der Klaauw (2010, 12). Assessing the impact of obesity on labor market outcomes. *Economics & Human Biology 8*(3), 309–319.

Marinescu, I. (2017). No Strings Attached: The Behavioral Effects of U.S. Unconditional Cash Transfer Programs. Technical report, Roosevelt Institute.

Martin, J. A., M. P. H. . Brady, E. Hamilton, S. J. Ventura, M. A. . Michelle, J. K. Osterman, S. Kirmeyer, T. J. Mathews, and E. C. Wilson (2011). Births: Final data for 2009. National Vital Statistics Reports, Volume 60, Number 1. Technical report, National Center for Health Statistics, Hyattsville, MD.

Martin, J. A., M. P. H. . Brady, E. Hamilton, S. J. Ventura, M. A. . Michelle, J. K. Osterman, and T. J. Mathews (2013). Births: Final data for 2011. National Vital Statistics Reports Volume 62, Number 1. Technical report, National Center for Health Statistics, Hyattsville, MD.

Martin, J. A., M. P. H. . Brady, E. Hamilton, S. J. Ventura, M. A. . Michelle, J. K. Osterman, E. C. Wilson, and T. J. Mathews (2012). Births: Final Data for 2010. National Vital Statistics Reports; vol 61 no 1. Technical report, National Center For Health Statistics, Hyattsville, MD.

Martorell, R. (2017, 3). Improved nutrition in the first 1000 days and adult human capital and health. *American Journal of Human Biology 29*(2), e22952.

Matthews, D. (2017, 9). Hillary Clinton almost ran for president on a universal basic income. *Vox.com*.

Murray, C. (2008). Guaranteed income as a replacement for the welfare state. *The Foundation of Law, Justice and Society*.

Ogden, C. L., M. D. Carroll, B. K. Kit, and K. M. Flegal (2014). Prevalence of childhood and adult obesity in the United States, 2011-2012. *JAMA - Journal of the American Medical Association*.

Rasella, D., R. Aquino, C. A. Santos, R. Paes-Sousa, and M. L. Barreto (2013). Effect of a conditional cash transfer programme on childhood mortality: A nationwide analysis of Brazilian municipalities. *The Lancet*.

Reis, M. (2010). Cash transfer programs and child health in Brazil. *Economics Letters*.

Schaller, J. and M. Zerpa (2019, 1). Short-Run Effects of Parental Job Loss on Child Health. *American Journal of Health Economics 5*(1), 8–41.

Schmeiser, M. D. (2012). The Impact Of Long-Term Participation In The Supplemental Nutrition Assistance Program On Child Obesity. *Health Economics 21*(4), 386–404.

Schroeder, D. G., R. Martorell, J. A. Rivera, M. T. Ruel, and J.-P. Habicht (1995, 4). Age Differences in the Impact of Nutritional Supplementation on Growth. *The Journal of Nutrition 125*(suppl_4), 1051S–1059S.

Tanner, M. (2015). The Pros and Cons of a Guaranteed National Income. Technical report, Cato Institute, Washington, DC.

Thigpen, D. E. (2016). Universal Income: What Is It, and Is It Right for the U.S.? Technical report, Roosevelt Institute.

Watson, B., M. Guettabi, and M. Reimer (2019, 4). Universal Cash and Crime. *The Review of Economics and Statistics*, 1–45.

Weller, C. (2017, 9). One of the biggest VCs in Silicon Valley is launching an experiment that will give 3,000 people free money until 2022. *Business Insider*.

Whitaker, R. C., J. A. Wright, M. S. Pepe, K. D. Seidel, and W. H. Dietz (1997, 9). Predicting Obesity in Young Adulthood from Childhood and Parental Obesity. *New England Journal of Medicine 337*(13), 869–873.

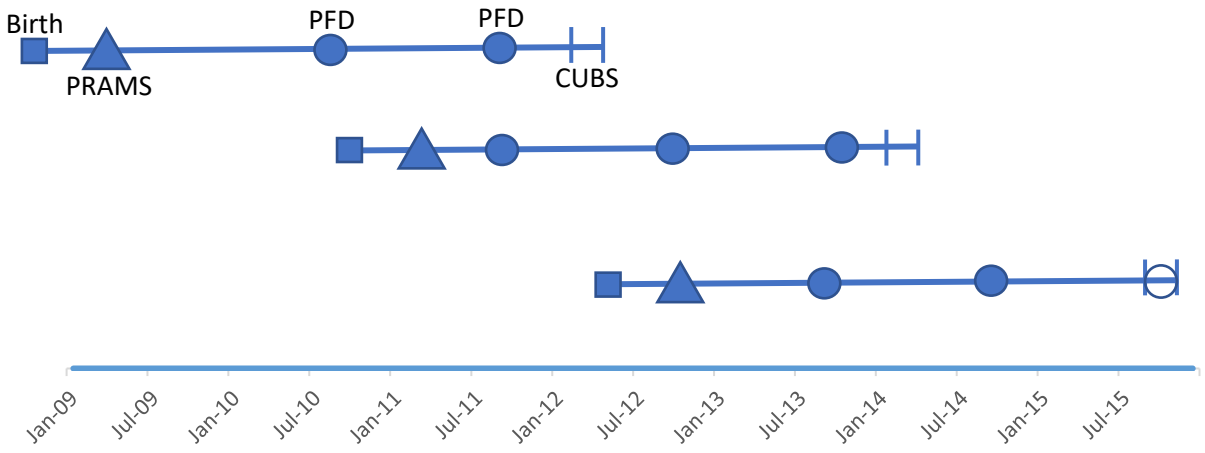
Tables and Figures

Table 1: Sampled three-year-old children, by BMI weight classification

Percentile	Underweight <5th	Healthy 5th-85th	Overweight 85-95th	Obese >95th
N Children (Unweighted)	75	679	190	281
% of Children (Unweighted)	6%	55%	16%	23%
% of Children (Weighted)	6%	54%	15%	24%

Table includes all children who's obesity status was calculable (n=1225) while our main analysis locks the sample with a number of controls where some data are missing (n=885). Fractions are calculated with (weighted) and without (unweighted) survey sample design weights.

Figure 1: Timeline showing the relationship between date-of-birth, survey design, and accumulated PFD income.



PRAMS is a survey is administered 4-6 months after birth, CUBS is a survey is administered 2-7 months after 3rd birthday. Top case: child born Jan-Feb will only receive 2 PFDs before mother returns CUBS survey. Middle case: children born Aug-Dec will receive 3 PFDs before mother returns CUBS survey. Bottom case: children born Mar-July may receive 2 or 3 PFDs, depending on the CUBS survey date.

Table 2: Un-weighted Estimated Effect of Total PFD ($1,000s) on the Probability of Being Obese and Overweight as a Three-year-old Child

Risk of being:	≥95th	≥95th	≥85th	≥85th
Compared to:	<85th	5th-85th	<85th	5th-85th
	(1)	(2)	(3)	(4)
Logit coefficient	−0.370***	−0.380**	−0.360***	−0.349***
	(0.140)	(0.152)	(0.117)	(0.120)
Odds ratio	0.691***	0.684**	0.697***	0.705***
	(0.096)	(0.104)	(0.082)	(0.085)
Marginal effect	-0.052	-0.058	-0.065	-0.064
Observations	885	698	885	830

*p<0.1; **p<0.05; ***p<0.01; Robust standard errors in parentheses. Columns (1) and (2) measure the effect of the PFD on the risk of being classified obese (BMI ≥ 95th percentile for sex/age) relative to all other weights (BMI < 85th percentile, column 1) or only normal weight children (5th-85th percentile, column 2). Columns (3) and (4) are estimates for both obese and overweight children relative to all other weight classes (column 3) or only normal-weight children (column 4).

Table 3: Logit Coefficient Estimates for Different Control Variable Specifications

	Obese					*Obese & Overweight*				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Total PFDs ('000$)	−0.055	−0.172	−0.304**	−0.268**	−0.370***	−0.149	−0.224**	−0.303***	−0.286***	−0.360***
	(0.109)	(0.120)	(0.132)	(0.129)	(0.140)	(0.094)	(0.103)	(0.112)	(0.108)	(0.117)
Logit ME at mean	-0.009	-0.027	-0.044	-0.039	-0.052	-0.035	-0.046	-0.057	-0.055	-0.065
Child characteristics		X	X	X	X		X	X	X	X
Mother characteristics			X		X			X		X
Pre/post natal nutrition				X	X				X	X
Observations	885	885	885	885	885	885	885	885	885	885

*p<0.1; **p<0.05; ***p<0.01; Robust standard errors in parentheses. Columns (1-5) measure the effect of the PFD on the risk of being classified obese (BMI ≥ 95th percentile for sex/age) relative to all other weights (BMI < 85th percentile). Columns (6-10) are estimates for both obese and overweight children relative to all other weight classes. Child characteristics are listed in Table A.1, mother characteristics are listed in Table A.2, pre/post natal nutrition variables are are listed in Table A.3.

Table 4: Odds Ratios of Estimated Effect of Total PFD ($1,000s) on the Probability of Being Obese and Overweight as a Three-year-old Child for low, middle, and high income terciles

Risk of being:	≥95th	≥95th	≥85th	≥85th
Compared to:	<85th	5th-85th	<85th	5th-85th
	(1)	(2)	(3)	(4)
PFD	0.888	0.935	0.914	0.958
	(0.186)	(0.254)	(0.191)	(0.212)
PFD x 25-75K	0.5	0.461**	0.546**	0.518**
	(0.148)	(0.161)	(0.148)	(0.146)
PFD x >75k	1.056	1.063	0.916	0.89
	(0.367)	(0.422)	(0.283)	(0.284)
Observations	885	698	885	830

*p<0.1; **p<0.05; ***p<0.01. Robust standard errors in parentheses. Estimates presented are the results of PFD × Income tercile interactions.

Table 5: Estimated Effect of Total PFD ($1,000s) on the Probability of Being Obese and Overweight as a Three-year-old Child, by Income Group

	Obese					*Obese & Overweight*				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
PFD x 10,000	0.100	−0.151	−0.357	−0.226	−0.396	0.319	0.170	0.039	0.225	0.076
	(0.252)	(0.290)	(0.301)	(0.292)	(0.297)	(0.249)	(0.295)	(0.304)	(0.309)	(0.317)
PFD x 10,000-14,999	0.296	0.219	0.412	0.117	0.388	−0.258	−0.458	−0.358	−0.709	−0.536
	(0.440)	(0.486)	(0.485)	(0.484)	(0.499)	(0.462)	(0.502)	(0.523)	(0.516)	(0.554)
PFD x 15,000-19,999	0.661	0.955	1.156*	0.941	1.090	−0.275	−0.149	−0.068	−0.381	−0.328
	(0.542)	(0.604)	(0.644)	(0.688)	(0.688)	(0.495)	(0.552)	(0.594)	(0.573)	(0.595)
PFD x 20,000-24,999	0.231	0.438	0.384	0.493	0.352	−0.118	0.114	0.038	0.027	−0.066
	(0.552)	(0.579)	(0.633)	(0.614)	(0.643)	(0.492)	(0.539)	(0.581)	(0.561)	(0.596)
PFD x 25,000-34,999	−0.671	−0.485	−0.256	−0.367	−0.179	−0.713*	−0.588	−0.364	−0.598	−0.319
	(0.419)	(0.472)	(0.487)	(0.483)	(0.491)	(0.389)	(0.437)	(0.453)	(0.457)	(0.473)
PFD x 35,000-49,999	−0.355	−0.286	−0.226	−0.385	−0.306	−0.688*	−0.642	−0.557	−0.873**	−0.740*
	(0.411)	(0.459)	(0.485)	(0.459)	(0.484)	(0.370)	(0.410)	(0.430)	(0.426)	(0.446)
PFD x 50,000-74,999	−0.682*	−0.556	−0.611	−0.588	−0.677	−0.917***	−0.889**	−0.944**	−0.997**	−1.065***
	(0.378)	(0.418)	(0.446)	(0.435)	(0.455)	(0.332)	(0.378)	(0.393)	(0.397)	(0.409)
PFD x 75,000 +	−0.055	0.175	0.343	0.187	0.324	−0.452	−0.279	−0.167	−0.389	−0.257
	(0.356)	(0.400)	(0.411)	(0.405)	(0.405)	(0.317)	(0.362)	(0.374)	(0.378)	(0.389)
Child characteristics		X	X	X	X		X	X	X	X
Mother characteristics			X		X			X		X
Pre/post natal nutrition				X	X				X	X
Observations	885	885	885	885	885	885	885	885	885	885

*p<0.1; **p<0.05; ***p<0.01; Robust standard errors in parentheses. Columns (1-5) measure the effect of the PFD on the risk of being classified obese (BMI ≥ 95th percentile for sex/age) relative to all other weights (BMI < 85th percentile). Columns (6-10) are estimates for both obese and overweight children relative to all other weight classes. Child characteristics are listed in Table A.1, mother characteristics are listed in Table A.2, pre/post natal nutrition variables are are listed in Table A.3.

Table 6: Logit Coefficient Estimates, Conditional on Appliers

	Obese					*Obese & Overweight*				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Total PFDs ('000$)	−0.054	−0.195	−0.337**	−0.279**	−0.391***	−0.154	−0.235**	−0.327***	−0.286**	−0.370***
	(0.114)	(0.128)	(0.141)	(0.136)	(0.147)	(0.099)	(0.111)	(0.121)	(0.115)	(0.125)
Always Applied	−0.010	0.167	0.252	0.084	0.170	0.036	0.082	0.186	−0.0001	0.075
	(0.302)	(0.333)	(0.390)	(0.329)	(0.386)	(0.262)	(0.289)	(0.331)	(0.296)	(0.339)
Logit ME at mean	-0.009	-0.03	-0.049	-0.041	-0.055	-0.036	-0.048	-0.062	-0.055	-0.067
Child characteristics		X	X	X	X		X	X	X	X
Mother characteristics			X		X			X		X
Pre/post natal nutrition				X	X				X	X
Observations	885	885	885	885	885	885	885	885	885	885

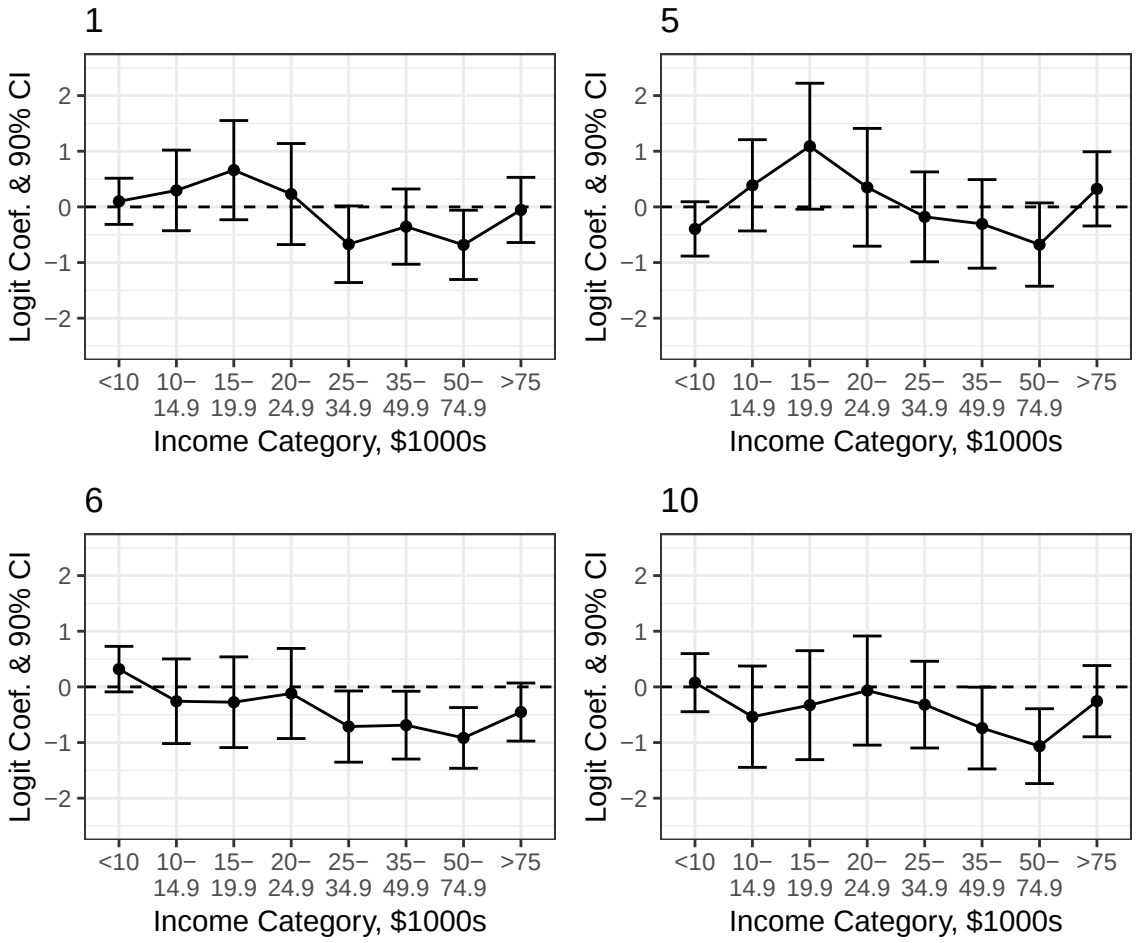
*p<0.1; **p<0.05; ***p<0.01; Robust standard errors in parentheses. Columns (1-5) measure the effect of the PFD on the risk of being classified obese (BMI ≥ 95th percentile for sex/age) relative to all other weights (BMI < 85th percentile). Columns (6-10) are estimates for both obese and overweight children relative to all other weight classes. Child characteristics are listed in Table A.1, mother characteristics are listed in Table A.2, pre/post natal nutrition variables are are listed in Table A.3.

Table 7: Two-stage Least Squares Estimates of PFD Effect

	Obese			*Obese & Overweight*		
	(1)	(2)	(3)	(4)	(5)	(6)
Total PFDs	−0.043*	−0.044**	−0.041**	−0.076***	−0.062***	−0.062***
	(0.023)	(0.020)	(0.020)	(0.026)	(0.024)	(0.024)
IV: Eligible PFDs	Number	Tot. $'s	# + avg$	Number	Tot. $'s	# + avg$
Child characteristics	X	X	X	X	X	X
Mother characteristics	X	X	X	X	X	X
Pre/post natal nutrition	X	X	X	X	X	X
Weak instruments	1286.14***	2656.6***	1350.96***	1286.14***	2656.6***	1350.96***
Wu-Hausman	0.01	0.01	0.15	0.6	0	0.01
Sargan	NA	NA	0.1	NA	NA	1.64
Observations	885	885	885	885	885	885
Adjusted R^2	0.144	0.144	0.144	0.156	0.156	0.156

*p<0.1; **p<0.05; ***p<0.01; Robust standard errors in parentheses. *p<0.1; **p<0.05; ***p<0.01; Robust standard errors in parentheses. Columns (1-3) measure the effect of the PFD on the risk of being classified obese (BMI ≥ 95th percentile for sex/age) relative to all other weights (BMI < 85th percentile). Columns (4-6) are estimates for both obese and overweight children relative to all other weight classes. Child characteristics are listed in Table A.1, mother characteristics are listed in Table A.2, pre/post natal nutrition variables are are listed in Table A.3.

Figure 2: Logit Coefficients for Effect of Total PFD ($1,000s), by Income Category



Panels 1, 5, 6, and 10 corresponds to specification 1, 5, 6, and 10 in Table 5. The outcome for panels 1 and 5 is obese versus all other weight status. The outcome for panels 6 and 10 is obese and overweight versus other weight status. Panels 1 and 6 have no control variables. Panels 5 and 10 have all controls.

Figure 3: Timeline of PFD and Income Tax Returns

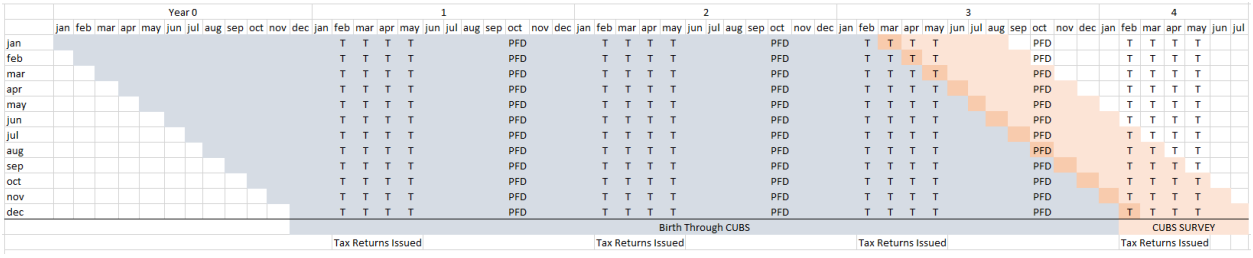


Table 8: Controlling for Strategic Birth Timing

	Obese					*Obese & Overweight*				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Total PFDs ('000$)	−0.051	−0.170	−0.316**	−0.266**	−0.384***	−0.148	−0.224**	−0.313***	−0.289***	−0.370***
	(0.109)	(0.120)	(0.133)	(0.129)	(0.140)	(0.094)	(0.103)	(0.114)	(0.108)	(0.119)
Intended Timing Preg.	−0.305*	−0.242	0.889*	−0.107	0.993*	−0.114	−0.029	0.847*	0.138	0.918*
	(0.176)	(0.193)	(0.525)	(0.203)	(0.544)	(0.147)	(0.161)	(0.463)	(0.171)	(0.481)
Logit ME at mean	-0.009	-0.026	-0.046	-0.039	-0.054	-0.034	-0.046	-0.059	-0.056	-0.067
Child characteristics		X	X	X	X		X	X	X	X
Mother characteristics			X		X			X		X
Pre/post natal nutrition				X	X				X	X
Observations	885	885	885	885	885	885	885	885	885	885

*p<0.1; **p<0.05; ***p<0.01; Robust standard errors in parentheses. Columns (1-5) measure the effect of the PFD on the risk of being classified obese (BMI ≥ 95th percentile for sex/age) relative to all other weights (BMI < 85th percentile). Columns (6-10) are estimates for both obese and overweight children relative to all other weight classes. Child characteristics are listed in Table A.1, mother characteristics are listed in Table A.2, pre/post natal nutrition variables are are listed in Table A.3.

Table 9: Birth Timing: Only First and Fourth Quarter Births

	Obese					*Obese & Overweight*				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Total PFDs ('000$)	−0.172	−0.363*	−0.566**	−0.475**	−0.651**	−0.266*	−0.419***	−0.490***	−0.464***	−0.549***
	(0.159)	(0.196)	(0.237)	(0.219)	(0.255)	(0.136)	(0.162)	(0.185)	(0.178)	(0.201)
Logit ME at mean	-0.029	-0.05	-0.07	-0.062	-0.077	-0.061	-0.082	-0.082	-0.084	-0.085
Child characteristics		X	X	X	X		X	X	X	X
Mother characteristics			X		X			X		X
Pre/post natal nutrition				X	X				X	X
Observations	421	421	421	421	421	421	421	421	421	421

*p<0.1; **p<0.05; ***p<0.01; Robust standard errors in parentheses. Columns (1-5) measure the effect of the PFD on the risk of being classified obese (BMI ≥ 95th percentile for sex/age) relative to all other weights (BMI < 85th percentile). Columns (6-10) are estimates for both obese and overweight children relative to all other weight classes. Child characteristics are listed in Table A.1, mother characteristics are listed in Table A.2, pre/post natal nutrition variables are are listed in Table A.3.

Table 10: Complete Robustness Specification

	Obese					*Obese & Overweight*				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Total PFDs ('000$)	−0.175	−0.418**	−0.623**	−0.552**	−0.730***	−0.274*	−0.446***	−0.509***	−0.489***	−0.558***
	(0.165)	(0.202)	(0.247)	(0.228)	(0.270)	(0.143)	(0.172)	(0.193)	(0.186)	(0.207)
Always Applied	0.114	0.571	0.680	0.674	0.726	0.139	0.277	0.433	0.231	0.323
	(0.459)	(0.632)	(0.724)	(0.650)	(0.700)	(0.405)	(0.507)	(0.644)	(0.561)	(0.659)
Intended Timing Preg.	−0.239	−0.146	1.037	−0.035	1.428	−0.192	−0.060	1.085	0.097	1.406
	(0.268)	(0.319)	(0.897)	(0.345)	(1.048)	(0.226)	(0.258)	(0.833)	(0.283)	(0.956)
Logit ME at mean	-0.029	-0.057	-0.076	-0.071	-0.084	-0.062	-0.087	-0.084	-0.088	-0.085
Child characteristics		X	X	X	X		X	X	X	X
Mother characteristics			X		X			X		X
Pre/post natal nutrition				X	X				X	X
Observations	421	421	421	421	421	421	421	421	421	421

*p<0.1; **p<0.05; ***p<0.01; Robust standard errors in parentheses. Columns (1-5) measure the effect of the PFD on the risk of being classified obese (BMI ≥ 95th percentile for sex/age) relative to all other weights (BMI < 85th percentile). Columns (6-10) are estimates for both obese and overweight children relative to all other weight classes. Child characteristics are listed in Table A.1, mother characteristics are listed in Table A.2, pre/post natal nutrition variables are are listed in Table A.3.

Table 11: Robustness Check, Controlling for Flexible Child Tax Incentives

	Obese					*Obese & Overweight*				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Total PFDs ('000$)	−0.287	−0.418**	−0.579***	−0.644***	−0.723***	−0.191	−0.274	−0.375*	−0.422**	−0.491**
	(0.225)	(0.211)	(0.221)	(0.222)	(0.226)	(0.191)	(0.197)	(0.207)	(0.200)	(0.214)
Logit ME at mean	-0.037	-0.049	-0.065	-0.072	-0.079	-0.034	-0.043	-0.056	-0.064	-0.071
Flexible Tax Incentives	X	X	X	X	X	X	X	X	X	X
Child characteristics		X	X	X	X		X	X	X	X
Mother characteristics			X		X			X		X
Pre/post natal nutrition				X	X				X	X
Observations	885	885	885	885	885	885	885	885	885	885

*p<0.1; **p<0.05; ***p<0.01; Robust standard errors in parentheses. Columns (1-5) measure the effect of the PFD on the risk of being classified obese (BMI ≥ 95th percentile for sex/age) relative to all other weights (BMI < 85th percentile). Columns (6-10) are estimates for both obese and overweight children relative to all other weight classes. Child characteristics are listed in Table A.1, mother characteristics are listed in Table A.2, pre/post natal nutrition variables are are listed in Table A.3.

Table 12: Childhood Medical-Cost Savings from a $1,000 Universal Cash Transfer

	Obesity at 3yos	Effect of $1k at 3yo	Obesity Cost[a]	Medical Cost Savings, 4-17 Fully Persist	Medical Cost Savings, 4-17 Linear Age Decay
Individual	0.203	-0.045	$1,365	$920	$210
95% CI	[0.177,0.228]	[-0.091,-0.005]	[794,2011]	[80,2050]	[10,500]
AK Cohort	2,230	-500		$10.3m	$2.3m
95 CI	[1951,2511]	[-1003,-51]		[1.1,22.7]	[0.1,5.5]

95% confidence intervals are in brackets.

[a] Biener et al. (2017)

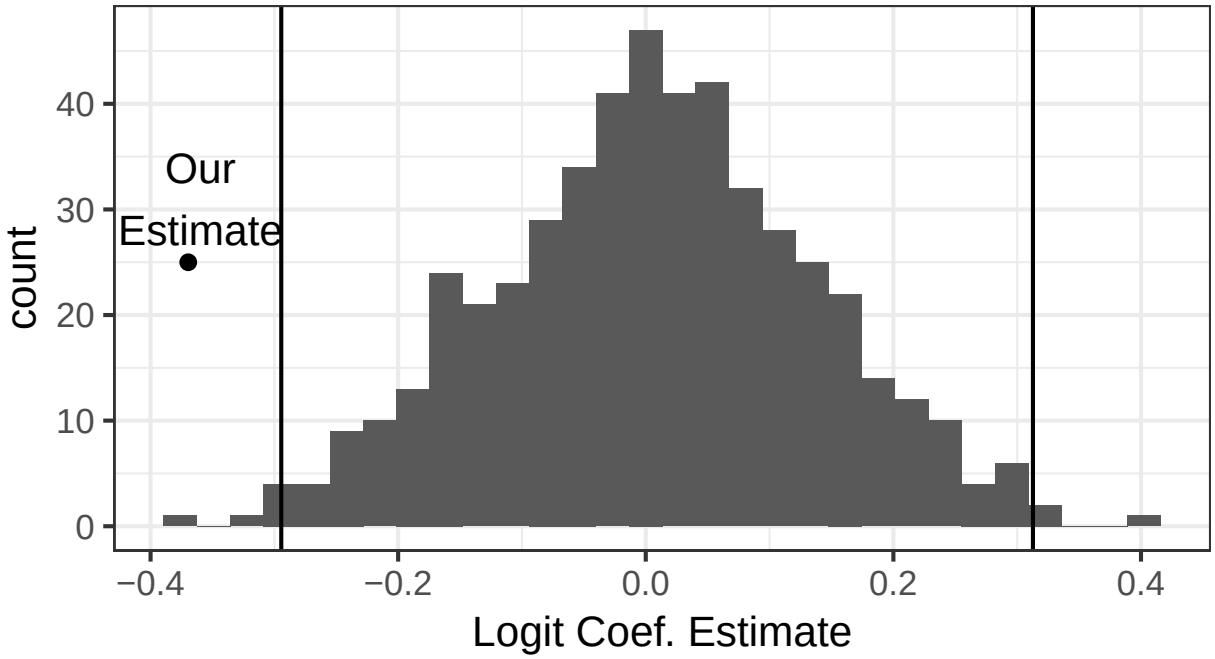


Figure 4: Our estimated unweighted logit coefficient (Point) from Table 2 (col. 1), distribution of 500 placebo effects, and 0.5 and 99.5 percentiles of placebo effects (vertical lines). Placebo effects estimated by randomly re-assigning each child's accumulated PFDs by age 3.

Appendix A Cost-Savings Simulation

The goal of our simulation is to estimate the obesity-caused difference in medical expenditures for the average cohort of Alaskan-born 3-year-olds, relative to their cost had they been non-obese. We measure this difference over a 15-year horizon, from 4 through 17 years old. We then shock this estimate by hypothetically removing some of the PFD money from the Alaskan cohort that 3-year-olds actually received to see how many additional obesity cases are created at age three, how these persist into adolescence, and how that may impact medical expenses.

To project health outcomes, we utilize a standard modeling approach from the literature and calculate transition probabilities to and from health-status categories. Our estimated transition probabilities are based on a child's initial BMI status an the BMI status of their mothers. As CUBS is not a longitudinal study, we do not observe the actual long run outcomes of the children surveyed. Instead, we fit a model on data from the National Longitudinal Survey of Youth (NLSY) and the corresponding National Longitudinal Survey of Children and Young Adults. Using the parameter values from a model fitted to a representative U.S. child through age 17, the parameters are used to calculate the transition probabilities of the average Alaskan cohort in the data. This assumes that the transition probabilities are similar for the average U.S. child/young adult born to a mother who was born 1957-1965, to the cohort of children represented in the Alaskan data (after accounting for child and mother's BMI status). This assumption is necessary in order to utilize longitudinal data with respondents up to age 17, but we recognize it as a potential limitation.

A.1 Derivation of Expected Medical Cost Savings

Let c_1 and c_0 denote the average annual medical cost associated with being obese and non-obese, respectively. Then the expected annual medical cost, $Cost_t$, for a child at age $t > 3$ conditional on the cumulative amount of PFD income received by the age of three is equal

to:

$$E(Cost_t|PFD) = c_1 P(Ob_t = 1|PFD) + c_0\big[1 - P(Ob_t = 1|PFD)\big]$$
$$= (c_1 - c_0)P(Ob_t = 1|PFD) + c_0, \tag{A.1}$$

where $Ob_t \in \{0, 1\}$ denotes a child's obesity status and $P(Ob_t = 1|PFD)$ denotes the conditional probability of being obese at age $t > 3$. The conditional probability of being obese at age $t > 3$ depends on whether a child was obese at the age of three and how receiving PFD influences the likelihood that a child is obese at the age of three. We can incorporate this relationship be rewriting Eq. (A.1) as:

$$E(Cost_t|PFD) = (c_1 - c_0) \sum_{h=0}^{1} P(Ob_t = 1|Ob_{t=3} = h)P(Ob_{t=3} = h|PFD) + c_0,$$

where $P(Ob_{t=3} = h|PFD)$ represents the influence of PFD on the likelihood of being obese (or non-obese) at the age of three and $P(Ob_t = 1|Ob_{t=3} = h)$ denotes the transition probability representing the likelihood of being obese at age t conditional on having obesity status $h =$ obese or non-obese at age three.

Now suppose that we are interested in the annual medical-cost savings, S_t, associated with an additional \$1,000 in cumulative PFD income received by a child by the age of three. Let $PFD = 1$ if a child receives an additional \$1,000 in PFD and $PFD = 0$ if a child does not receive any additional PFD income. Then the expected annual savings for a child at age $t > 3$ is equal to:

$$E(S_t) = E(Cost_t|PFD = 1) - E(Cost_t|PFD = 0)$$
$$= c \sum_{h=0}^{1} P(Ob_t = 1|Ob_{t=3} = h)\Big[P(Ob_{t=3} = h|PFD = 1) - P(Ob_{t=3} = h|PFD = 0)\Big], \tag{A.2}$$

where $c = (c_1 - c_0)$ is the annual medical cost of being obese relative to not being obese. The expression in the brackets is equal to the marginal effect of the PFD on the probability of being obese or non-obese. Let R_h denote the marginal effect of the PFD on the probability that a child's obese status is equal to $h \in \{0, 1\}$. Then recognizing that $R_0 = -R_1$, we can rewrite the expression in Eq. (A.2) as:

$$E(S_t) = c\Big[P(Ob_t = 1|Ob_{t=3} = 1) - P(Ob_t = 1|Ob_{t=3} = 0)\Big]R_1, \tag{A.3}$$

which gives us the expression in Eq. (3) of the main paper. To obtain an estimate of the expected medical-cost savings from an additional \$1,000 in PFD for a cohort of size N over the ages four to seventeen, we sum Equation (A.3) over all ages and individuals:

$$E(S) = \sum_{t=4}^{17} \sum_{i=1}^{N} E(S_{i,t}). \tag{A.4}$$

A.2 Re-weighting to Population and Bootstrapping Confidence Intervals

Population-weighted estimates and confidence intervals are obtained through a bootstrap procedure. We take 1,000 sample draws-with-replacement from our data, with draw probability corresponding to the survey sample design weights. This sampling procedure re-weights to survey sample to reflect the population. We estimate Eq. 1 on each of these sample draws, recording the estimated average marginal effect and the predicted obesity rate. For each draw, we predict the obesity rate in the counterfactual, having removed \$1,000 of PFD income. Applying the difference of these rates to the Alaska cohort (11,000) yields the number of averted cases. To estimate cost savings, we sample from of the annual marginal cost of obesity, taken from the literature Biener et al. (2017) (which we assume is normally distributed). These costs are multiplied over a 15-year horizon to capture the potential savings if obesity reductions fully persist through childhood and adolescence. The mean and 95%

confidence intervals are calculated from the distribution of estimated parameters, predicted cases and cost savings. Because obesity shocks are not in reality fully persistent (e.g. children tend toward the obesity outcomes of their parents), in the next subsection we describe our procedure to allow the obesity-reduction effect to decay with child age.

A.3 Estimating Age-decay model

To estimate the transitory nature of an obesity shock at age-3, we estimate an obesity transition model over a 15-year horizon (Eq. 4) on a nationally representative survey dataset, the National Longitudinal Survey of Children and Young Adults (NLS-CYA). These survey data are re-weighted to the population by the same bootstrapping procedure outlined in the previous section. The procedure is modified because NLS-CYA samples mothers and each of their children, so we account for the clustered nature of the data by sampling from the household (rather than child) level.

Eq. 4 interacts child age with child's BMI at age 3 and mother's BMI, allowing for the transition probability to change with the child's age based on their initial continuations and mother's weight status. The resulting parameter estimates are presented in Table A.4. These parameters are used to estimate the likelihood a reduction in obesity at age 3 will be maintained at each age through age 17. A more flexible interaction of age (discrete age bins) was also estimated, but yielded similar inference.

Supplementary Tables and Figures

Table A.1: Child Characteristics: Descriptive Statistics by #PFDs and associated sample balance tests

	1	2	3	Combined	P-value
	$N = 31$	$N = 492$	$N = 361$	$N = 884$	
Obese	26% (8)	22% (108)	22% (78)	22% (194)	0.86[1]
Gender: 2	61% (19)	51% (250)	47% (171)	50% (440)	0.26[1]
Age (months): 38	55% (17)	47% (229)	41% (147)	44% (393)	<0.001[1]
39	32% (10)	36% (179)	28% (102)	33% (291)	
40	10% (3)	14% (70)	19% (70)	16% (143)	
41	3% (1)	2% (10)	11% (39)	6% (50)	
42	0% (0)	1% (3)	1% (2)	1% (5)	
43	0% (0)	0% (1)	0% (1)	0% (2)	
Birth weight (grams)	2188 2494 3316 (2743 ± 900)	2494 3345 3706 (3158 ± 808)	2466 3260 3657 (3095 ± 816)	2466 3300 3670 (3118 ± 817)	0.016[2]
Birth Defect? (No)	97% (30)	97% (475)	97% (351)	97% (856)	0.85[1]
C-SECTION Used	26% (8)	28% (139)	28% (102)	28% (249)	0.96[1]
Time In Hosp. After Birth: < 1 DAY	10% (3)	7% (36)	6% (20)	7% (59)	0.83[1]
1-2 DAYS	32% (10)	47% (230)	49% (176)	47% (416)	
3-5 DAYS	26% (8)	21% (105)	22% (81)	22% (194)	
6-14 DAYS	10% (3)	6% (29)	6% (23)	6% (55)	
MORE THAN 14 DAYS	19% (6)	11% (55)	11% (40)	11% (101)	
NOT BORN IN HOSPITAL	3% (1)	7% (35)	6% (20)	6% (56)	
STILL IN HOSPITAL	0% (0)	0% (2)	0% (1)	0% (3)	
Gestational Age (Weeks): <28 weeks	3% (1)	2% (9)	1% (3)	1% (13)	0.89[1]
28-33	6% (2)	7% (33)	6% (23)	7% (58)	
34-36	16% (5)	14% (68)	16% (57)	15% (130)	
37-42	74% (23)	75% (369)	75% (270)	75% (662)	
>42 weeks	0% (0)	3% (13)	2% (8)	2% (21)	
Place of Birth : HOSPITAL	97% (30)	91% (450)	93% (337)	92% (817)	0.52[1]
BIRTHING CENTER	3% (1)	7% (32)	6% (21)	6% (54)	
RESIDENCE	0% (0)	2% (10)	1% (3)	1% (13)	
Region : Anch	32% (10)	42% (206)	39% (141)	40% (357)	0.73[1]
Gulf Coast	16% (5)	10% (49)	9% (32)	10% (86)	
Interior	19% (6)	16% (80)	14% (51)	15% (137)	
Northern	0% (0)	3% (15)	4% (13)	3% (28)	
Southeast	6% (2)	8% (39)	11% (39)	9% (80)	
Southwest	6% (2)	8% (40)	8% (29)	8% (71)	
Mat-Su	19% (6)	13% (63)	16% (56)	14% (125)	
				Joint F-test for significance: 0.6891 on 22 and 861 DF, p-value: 0.8534	
Variables not included in child controls, but shown for description:					
Number PFD Entitled : 3	13% (4)	9% (42)	100% (361)	46% (407)	<0.001[1]
Height (inches)?	37.0 37.5 38.9 (38.0 ± 2.3)	36.5 37.5 39.0 (37.6 ± 2.3)	36.0 37.8 39.0 (37.5 ± 2.3)	36.5 37.5 39.0 (37.6 ± 2.3)	0.92[2]
Weight	32.0 35.0 38.0 (35.1 ± 4.5)	30.8 34.0 37.0 (34.2 ± 5.2)	30.0 33.0 37.0 (33.8 ± 5.6)	30.0 33.6 37.0 (34.0 ± 5.4)	0.14[2]
Month of Birth (numeric)	4.0 5.0 7.0 (5.4 ± 2.7)	3.0 4.0 6.0 (4.5 ± 2.5)	9.0 10.0 11.0 (9.9 ± 1.6)	4.0 7.0 10.0 (6.7 ± 3.4)	<0.001[2]
Month of Birth dummy : 1	10% (3)	12% (60)	0% (0)	7% (63)	<0.001[1]
2	3% (1)	11% (54)	0% (0)	6% (55)	
3	10% (3)	13% (65)	0% (0)	8% (68)	
4	16% (5)	17% (86)	0% (0)	10% (91)	
5	13% (4)	13% (65)	0% (0)	8% (69)	
6	19% (6)	13% (65)	2% (9)	9% (80)	
7	13% (4)	12% (60)	6% (20)	10% (84)	
8	6% (2)	1% (7)	15% (54)	7% (63)	
9	3% (1)	1% (7)	19% (68)	9% (76)	
10	0% (0)	1% (4)	16% (59)	7% (63)	
11	3% (1)	1% (7)	21% (76)	10% (84)	
12	3% (1)	2% (12)	21% (75)	10% (88)	

$_a\, b\, _c$ represent the lower quartile a, the median b, and the upper quartile c for continuous variables. $x \pm s$ represents $\bar{X} \pm 1$ SD.Numbers after percents are frequencies.Tests used:[1]Pearson test; [2]Kruskal-Wallis test

Table A.2: Mother Characteristics: Descriptive Statistics by #PFDs and associated sample balance tests

	1	2	3	Combined	P-value
	$N = 31$	$N = 492$	$N = 361$	$N = 884$	
Race : NH/OPI	3% (1)	3% (14)	3% (10)	3% (25)	0.19[1]
White	65% (20)	64% (317)	63% (226)	64% (563)	
Other, (inc. Black, Asian)	10% (3)	4% (22)	5% (19)	5% (44)	
AI/AN	19% (6)	26% (129)	29% (106)	27% (241)	
unknown	3% (1)	2% (10)	0% (0)	1% (11)	
Non-Hispanic : Yes	87% (27)	96% (470)	95% (344)	95% (841)	0.1[1]
Mother age at delivery: <20	10% (3)	6% (31)	5% (19)	6% (53)	0.17[1]
20-24	13% (4)	17% (83)	22% (81)	19% (168)	
25-29	42% (13)	34% (166)	31% (113)	33% (292)	
30-34	19% (6)	31% (151)	25% (89)	28% (246)	
35-39	13% (4)	11% (54)	13% (47)	12% (105)	
40+	3% (1)	1% (7)	3% (12)	2% (20)	
Household Income: ≤ $10,000	19% (6)	12% (58)	11% (41)	12% (105)	0.56[1]
$10,000 - $14,999	10% (3)	4% (21)	7% (25)	6% (49)	
$15,000 - $19,999	10% (3)	3% (17)	5% (18)	4% (38)	
$20,000 - $24,999	6% (2)	7% (32)	6% (23)	6% (57)	
$25,000 - $34,999	3% (1)	9% (46)	10% (35)	9% (82)	
$35,000 - $49,999	13% (4)	14% (69)	14% (51)	14% (124)	
$50,000 - $74,999	19% (6)	19% (92)	16% (56)	17% (154)	
≥$75,000	19% (6)	32% (157)	31% (112)	31% (275)	
Household Size	2.0 3.0 3.0 (2.6 ±1.1)	2.0 3.0 4.0 (2.9 ±1.3)	2.0 3.0 4.0 (3.0 ±1.4)	2.0 3.0 4.0 (3.0 ±1.4)	0.44[2]
Marital status at birth: Unmarried	32% (10)	31% (153)	34% (124)	32% (287)	0.6[1]
Mom BMI before preg.	23.0 26.2 30.1 (27.0 ± 5.8)	21.8 24.9 29.1 (26.1 ± 5.8)	22.1 25.1 29.3 (26.5 ± 6.1)	21.9 25.0 29.2 (26.3 ± 5.9)	0.47[2]
Trying to Get Pregnant: Yes	45% (14)	57% (279)	58% (211)	57% (504)	0.35[1]
Gestational diabetes: Yes	6% (2)	10% (47)	12% (42)	10% (91)	0.47[1]
Drank last 3 month preg:: Yes	61% (19)	62% (306)	64% (232)	63% (557)	0.81[1]
Smoked last 3 month preg: Yes	13% (4)	13% (65)	13% (46)	13% (115)	0.98[1]
Wanted to get Preg: Sooner	13% (4)	22% (106)	20% (73)	21% (183)	0.55[1]
Later	42% (13)	27% (133)	27% (96)	27% (242)	
Then	39% (12)	45% (219)	44% (160)	44% (391)	
Did not want	6% (2)	7% (34)	9% (32)	8% (68)	
WIC Assistance – dur preg : Yes	48% (15)	37% (182)	42% (151)	39% (348)	0.21[1]
				F-statistic: 1.091 on 26 and 829 DF, p-value: 0.3448	

$_a\, b\, _c$ represent the lower quartile a, the median b, and the upper quartile c for continuous variables. $x \pm s$ represents $\bar{X} \pm 1$ SD.Numbers after percents are frequencies.Tests used:[1]Pearson test; [2]Kruskal-Wallis test

Table A.3: Early Nutrition: Descriptive Statistics by #PFDs and associated sample balance tests

	1	2	3	Combined	P-value
	$N = 31$	$N = 492$	$N = 361$	$N = 884$	
Kessner Index : Adequate PNC	55% (17)	61% (299)	61% (222)	61% (538)	0.38[1]
Intermediate PNC	19% (6)	25% (122)	27% (97)	25% (225)	
Inadequate PNC	6% (2)	5% (26)	4% (14)	5% (42)	
Unknown PNC	19% (6)	9% (45)	8% (28)	9% (79)	
Breastfeeding : Still at PRAMS	65% (20)	72% (354)	67% (241)	70% (615)	0.22[1]
First week for food (if ate yet)	0.0 0.0 15.5 (6.1 ± 7.9)	0.0 0.0 0.0 (3.4 ± 6.7)	0.0 0.0 0.0 (2.9 ± 6.2)	0.0 0.0 0.0 (3.3 ± 6.6)	0.034[2]
Did eat food	61% (19)	78% (382)	80% (290)	78% (691)	0.044[1]
First week drank liquid (if drank yet)	0.0 0.0 5.0 (3.1 ±4.8)	0.0 0.0 4.0 (2.9 ±5.2)	0.0 0.0 3.0 (2.3 ±4.1)	0.0 0.0 4.0 (2.7 ±4.8)	0.6[2]
Did drink liquid : Yes	19% (6)	36% (178)	35% (126)	35% (310)	0.16[1]
Mom drank last 3 months preg.: Yes	61% (19)	62% (306)	64% (232)	63% (557)	0.81[1]
Mom smoked last 3 months preg: Yes	13% (4)	13% (65)	13% (46)	13% (115)	0.98[1]
Mom Did smoke: No	94% (29)	88% (434)	86% (312)	88% (775)	0.44[1]
				F-statistic: 1.573 on 11 and 872 DF, p-value: 0.1014	

$_a\, b\, _c$ represent the lower quartile a, the median b, and the upper quartile c for continuous variables. $x \pm s$ represents $\bar{X} \pm 1$ SD.Numbers after percents are frequencies.Tests used:[1]Pearson test; [2]Kruskal-Wallis test

Table A.4: Persistence Regression Model

	Logit Coef.	Std dev	5th	95th
(Intercept)	-16.52	0.09	-16.61	-16.35
Age	-0.01	0.01	-0.03	0.01
$Ob_{t=3} = 1$	4.13	0.41	3.33	4.95
$Ob_{t=3} = 1$ x Age	-0.23	0.05	-0.33	-0.15
$BMI^{mtr} = Normal$	13.29	0.28	12.68	13.80
$BMI^{mtr} = Normal$ x Age	0.04	0.03	-0.01	0.10
$BMI^{mtr} = Overweight$	13.66	0.35	12.98	14.32
$BMI^{mtr} = Overweight$ x Age	0.05	0.03	-0.01	0.10
$BMI^{mtr} = Obese$	14.59	0.34	13.91	15.22
$BMI^{mtr} = Obese$ x Age	0.02	0.03	-0.04	0.08

Standard deviation and confidence intervals are bootstrapped, with samples clustered at the household level.