Randomised pilot trial of cash incentives for reducing paediatric asthmatic tobacco smoke exposures from maternal caregivers and members of their social network

Mandeep S Jassal ,[1] Cassia Lewis-Land,[1] Richard E Thompson,[2] Arlene Butz[1]

ABSTRACT

Background The primary aim was to evaluate the efficacy of financial incentives for reducing paediatric tobacco smoke exposures (TSEs) through motivating cigarette usage reduction among low-income maternal caregivers and members of their social network.

Design Randomised control pilot trial over a 6-month study follow-up time period. The study was undertaken from May 2017 to -May 2018. Once monthly follow-up visits occurred over the 6-month study period.

Setting Baltimore City, Maryland, USA.

Participants We grouped 135 participants into 45 triads (asthmatic child (2–12 years of age), maternal caregiver and social network member). Triads were assigned in a 1:1 allocation ratio. The maternal caregiver and social network members were active smokers and contributed to paediatric TSE.

Interventions Triads were randomised to receive either usual care (TSE education and quitline referrals) or usual care plus financial incentives. Cash incentives up to $1000 were earned by caregivers and designated social network participants. Incentives for either caregivers or social network participants were provided contingent on their individual reduction of tobacco usage measured by biomarkers of tobacco usage. Study visits occurred once a month during the 6-month trial.

Main outcome measures The main outcome measure was mean change in monthly paediatric cotinine levels over 6 months of follow-up interval and was analysed on an intention-to-treat basis.

Results The mean change in monthly child cotinine values was not significantly different in the intervention cohort over the 6-month follow-up period, compared with the control group (p=0.098, CI −0.16 to 1.89). Trends in child cotinine could not be ascribed to caregivers or social network members. Despite decreasing mean monthly cotinine values, neither the intervention cohort's caregivers (difference in slope (control–intervention)=3.30 ng/mL/month, CI −7.72 to 1.13, p=0.144) or paired social network members (difference in slope (control–intervention)=−1.59 ng/mL/month, CI −3.57 to 6.74, p=0.546) had significantly different cotinine levels than counterparts in the control group.

Conclusions Financial incentives directed at adult contributors to paediatric TSE did not decrease child cotinine levels.

Trial registration number NCT03099811.

What is already known on this topic?

► Financial incentives provide a motivating reinforcement for smoking cessation. It is unclear if health-promoting financial incentives could be of additive benefit for paediatric tobacco smoke exposure (TSE) reduction in low-income populations where both asthma and cigarette usage are highly prevalent.

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What this study adds?

► Cash incentives were unable to reduce paediatric TSE when targeted at smoking caregivers and their social network. The trial shows the limitations of financial incentives for promoting changes in adult smoking behaviours when the study is framed in the context of paediatric TSE reduction.

INTRODUCTION

Tobacco smoke exposure (TSE) is a modifiable environmental contributor often linked with poorly controlled paediatric asthma.[1][2] It is estimated that up to 70% of low-income children with asthma are exposed to TSE, and one in six children are exposed to in-home smoke.[3][4] For decades, researchers have employed a multitude of individual-directed (eg, cognitive/behavioural approaches and nicotine pharmacotherapies) and societal-directed strategies (eg, TSE indoor smoking bans and/or legislative instruments).[5][6] The diversity in strategies and target populations is logical, given the complexities in nicotine addiction and multitude of TSE sources.

Trials from the past decade have predominantly focused on the reduction of paediatric indoor TSE using single or a combination of approaches, including self-help materials, counselling, phone support, nicotine replacement therapies (NRTs), biochemical feedback, air cleaner, air pollution

feedback (eg, particulate matter or air nicotine) and motivational interviewing.[7–12] Reviews of these well-designed interventions demonstrated that they are efficacious for TSE reduction only to a limited degree; however, none could eliminate paediatric exposures.[13 14] A recent review of behavioural-based and/or medication-based interventions aimed at primarily increasing parental cessation showed a 4% absolute difference between parental quit rates in the intervention and control groups, though over three-quarters of parents in both intervention and control groups continued to smoke, leaving the overwhelming majority of children potentially exposed to their parents' smoke.[15] The major conclusion that stems from these studies is significant indoor exposures remain in paediatric populations in lower-income settings.

One major source of persistent tobacco exposures is based in the realities of childcare in low-income environments where care often occurs by more than one non-primary caregiver (eg, grandmother, aunt and/or biological father).[16 17] Up to one-third to one-half of paediatric asthmatic TSEs are occurring under the care of these alternative caregivers.[18] The key question that we derived from these paediatric TSE reduction studies is what novel strategy can we undertake to better protect children from adult-based cigarette smoking exposures?

One strategy that has proven to be effective in academic settings for adult-centric studies, yet not formally incorporated in most public health programmes, is the use of financial incentives to promote smoking cessation.[19] However, this approach has yet to be tested in a paediatric-centric study where the primary endpoint is the reduction of TSE from multiple adult contributors. Financial incentives have classically been used to reduce adult smoking based on adult-specific outcomes (eg, reduction in nicotine biomarkers). Incentives are particularly appealing for a trial among paediatric asthmatics since the disease is more prevalent in lower socioeconomic populations and incentives are likely to be more efficacious among lower-income groups.[20–24] During the time that the incentives are being administered, researchers have noted enhanced smoking cessation behaviours, most notably in those who initially did not express a strong desire to quit or reduce smoking.[19 25 26] The most successful of these incentive strategies have been ones that employed financial reimbursement schema based on behavioural economic models that are crafted for the target population.[27 28] In other words, it is not simply the size of the incentive that influences smoking behaviour change, but it is the understanding and application of the complex psychological insights of the target population that provide a more effective incentive schema (eg, framing, size, timing and outcomes).[29 30]

Using insight from our previously published mixed methodology study among caregivers of asthmatic children with known TSE,[31] we constructed a paediatric TSE reduction intervention targeting smoking cessation among maternal caregivers of children with asthma and members of their social network. Maternal caregivers who smoke were the preferred target, given their presumed primary role in daily parental activities and therefore higher likelihood of smoke exposures.[32 33] Moreover, we inferred that maternal caregivers would be more amenable to smoking reduction for the betterment of their children. In addition to the female-centric approach, we focused our tobacco intervention on members of the caregivers' social network, regardless of gender, since they were reportedly frequent contributors to paediatric TSE due to increased amount of time spent around the child.

The primary aim of this work was to examine the feasibility and efficacy of a cash incentive schema to reduce TSE among urban caregivers of paediatric asthmatics using a randomised controlled trial (RCT) over a 6-month study period. The incentives were directed at maternal caregivers and a selected member of their social network who were active smokers of conventional cigarettes and known contributors to indoor paediatric TSE. We hypothesised that financial incentives provided to adult contributors to TSE, when framed as an intervention promoting personal and child respiratory health, will motivate them to reduce smoking activities and result in lower paediatric TSE.

METHODOLOGY

Study design

We conducted a 6-month RCT comparing usual care (TSE education and state quitline referrals) with an incentive programme targeting smoking reduction among adult caregivers of children with asthma and an adult member of their social network. The primary outcome was change in mean change in monthly paediatric salivary cotinine levels over the 6-month follow-up study period.

Study population

We used a recruitment schema that enrolled cohorts in triads (see figure 1). Recruitment of triads began through the identification of children with persistent asthma (as per National Heart, Lung, and Blood Institute criteria) in paediatric pulmonary specialty clinics and inpatient units at Johns Hopkins Children's Centre. Additional recruitment approaches included community-wide dissemination of study flyers. We selected the age range (2–12 years old) based on the difficulties of reliably diagnosing asthma below the age of 2 years and the lower likelihood that children above the age of 12 years in our local setting spend significant time indoors with their adult caregivers. We designated salivary cotinine of ≥1.0 ng/mL as exposure to tobacco smoke based on previous TSE studies.[34 35] Exclusion criteria among children included the presence of major pulmonary comorbidities (eg, bronchopulmonary dysplasia, interstitial lung disease and recurrent aspiration) that could potentially confound linkage of TSE and paediatric asthma-related outcomes. Participants were recruited from May 2017 to May 2018.

Along with the child, we recruited their primary maternal caregiver who self-identified as being an active smoker and expressed a desire to reduce cigarette smoking. Confirmation of active smoking status used capillary cotinine and exhaled carbon monoxide (CO) levels, both with cut-off values that exceeded established active smoking status standards.[36] We excluded caregivers if they used electronic nicotine devices (ENDs) whose usage would erroneously affect the incentive schema, as described further. No adult participants were excluded based on reports of sole or dual use of cigarettes and ENDS. We anticipated the low likelihood of END use, given recent prevalence studies indicating a lower likelihood of usage in low-income, African–American populations mirroring our study population.[37]

The final component of each triad was recruitment and enrolment of a member of the maternal caregiver's social network. The individual was a known active smoker who the maternal caregiver identified as being a contributor to the child's TSE. There were no constraints placed on the time that the social network member spent with the child. The individual could be of any gender. Quantitative criteria confirming active smoking status, as well as exclusion variables were similar among caregivers and social network participants.
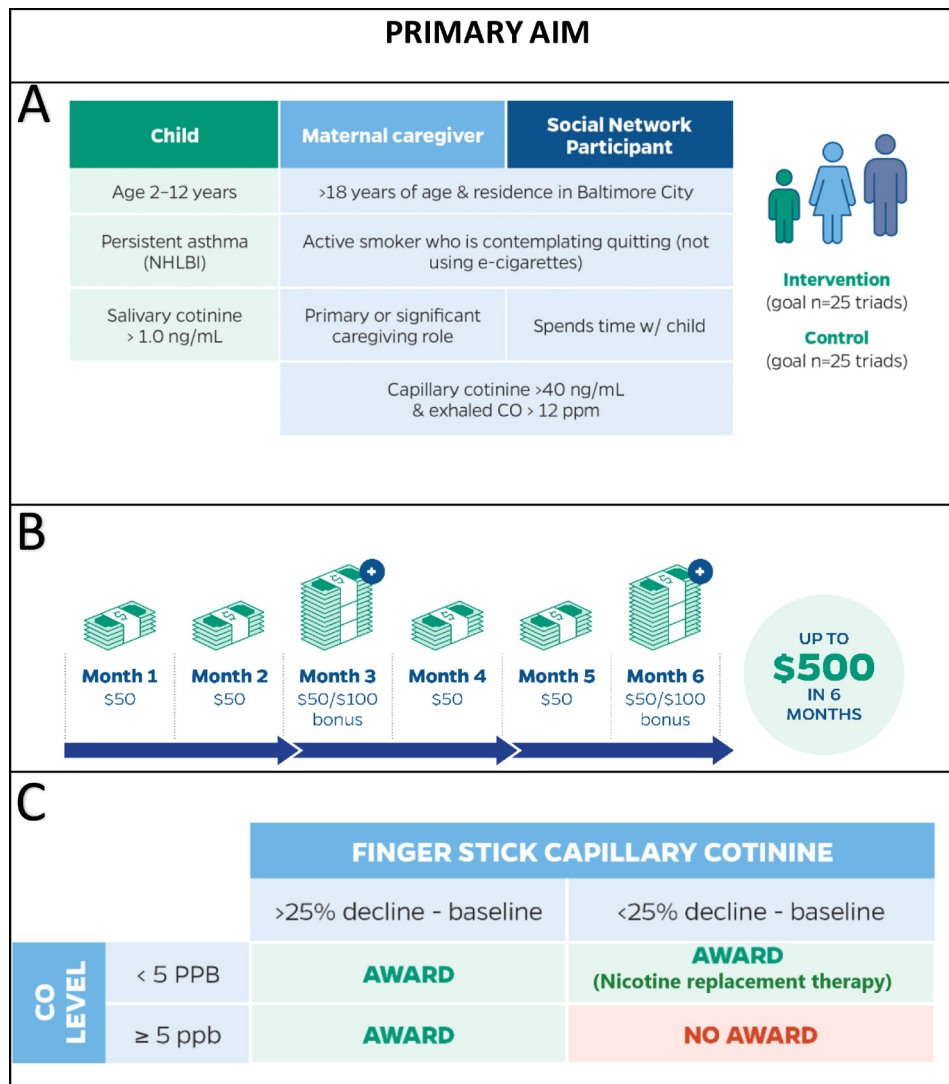
Figure 1 Methodology overview. (A) Overview of study triad inclusion criteria, (B) financial incentive reward mechanisms contingent on nicotine diagnostic testing and (C): financial incentive award schema. CO, carbon monoxide; NHLBI, National Heart, Lung, and Blood Institute.

Randomisation

We allocated triads in permuted blocks of two to either the intervention or control conditions. The randomisation ratio was 1:1. The randomisation outcome was not disclosed until entry of eligibility criteria in the trial database. Blinding was not possible, given the nature of the intervention.

Study protocol

All study participants received standard TSE education by senior research assistants based on Centers for Disease Control and Prevention fact sheets.[38] Participants received monthly encouragement to use the state tobacco quitline, which had the capacity to offer individual phone-based counselling and up to 4 weeks of NRT.

Caregivers and their social network members assigned to the incentive cohorts were eligible to receive financial incentives contingent on the reduction of monthly exhaled CO or capillary cotinine values, compared with baseline levels (see figure 1).

Point-of-care diagnostics (eg, exhaled CO or capillary cotinine) were used to immediately recognise fulfilment of incentivisation criteria. The reduced time from testing to incentivisation delivery was intentionally designed using behavioural economic principles to promote instant gratification and immediate feedback.[39]

Measurement of variables
Exhaled CO and cotinine

Measurement of exhaled CO used Vitalograph BreathCO (Vitalograph, Kansas, USA) and capillary cotinine results used PTS Detect cotinine system (PTS Diagnostics, Indiana, USA). The cotinine and exhaled CO levels were used for biochemical verification of smoking reductions of 7 and 1 day, respectively.[40] The PTS test required a 40 μL sample obtained from lancing a finger and provided values ranging from 25 to 200 ng/mL; values beyond those ranges were designated by the testing device using '<' or '>' symbol. Values of <25 and >200 were defaulted to 24 and 201, respectively, for statistical analyses.

Classification of meaningful declines in exhaled CO and cotinine levels

We designated exhaled CO levels of ≥4 ppm as an active smoker.[36] We had intended to use exhaled CO levels as a marker of smoking activities in those using NRTs. However, since no

participant availed of NRTs provided by the state quitline, we solely relied on cotinine levels to determine incentivisation. We designated a >25% decline in cotinine as meaningful, as opposed to cessation, since our primary focus was on smoking reduction and not cessation. Similar incentive-based strategies targeting smoking outcomes have often relied on abstinence-contingency measures, as delineated by exhaled CO or urine/saliva cotinine.[26 41 42] We preferred smoking reduction since it is a more attainable goal compared with complete cessation and, once achieved, it may encourage further efforts to achieve cessation.[43 44] A 25% decline seemed achievable and clinically impactful based on several past smoking intervention studies.[44–46]

Smoking characteristics

Caregiver and social network participant tobacco smoking characteristics were ascertained at baseline and at each follow-up visits using TSE measures (eg, self-reported indoor smoking bans, frequency/amount of cigarette smoking), monthly cigarette expenditures, past quit attempt measures (eg, methods used, number of attempts 1 year prior to enrolment and reasons for continued smoking) and demographics. We collected at both baseline and each monthly follow-up the usage of state quitline services (behavioural counseling±NRT usage), as well as the intensity of the caregiver's addiction to nicotine using the Fagerstrom Test for Nicotine Dependence (FTND).[47]

Asthma control

We measured asthma control in participants aged ≤3 years using the Test for Respiratory and Asthma Control in Kids (TRACK)[48] and aged ≥4 years using the Asthma Control Test (ACT).[49] TRACK is a validated questionnaire to monitor respiratory symptom control in preschool-aged populations, while the ACT has been extensively studied in school-aged children. Anxiety and depression for the caregiver was measured using Patient Health Questionnaire-4 (PHQ-4).[50]

Financial incentive protocol

Adult participants in the intervention cohort were eligible up to $500 over the 6-month study period. Participants received $50 per month for low cotinine levels (>25% decrease compared with baseline values), with an additional $100 given at 3 and 6 months for low cotinine levels during the previous 2 months (see figure 1). Triads enrolled in the control condition received $20 each month as reimbursement for study participation for a total of $120 over the study period. All participants were reimbursed in cash, as opposed to cash equivalents (eg, gift cards or vouchers), to motivate trial retention and provide greater freedom in usage of study rewards. Participants were awarded the incentive immediately after obtainment of the capillary cotinine level results within our point-of-care test (as described previously)—approximately 5 min.

Outcomes

The primary outcome was the monthly mean change in children's monthly salivary cotinine levels over the 6-month study period. Triads were excluded from analyses if we were unable to collect at least 3 monthly salivary cotinine from the group's child member over the 6-month study. Secondary outcomes included smoking characteristics of adult participants, social network–caregiver interactions, paediatric asthma control and caregiver mental health outcomes (anxiety and depression).

Statistical analysis

We compared the baseline characteristics of cohorts in the RCT trial using summary statistics (eg, mean or medians). Regression models were fit using generalised estimating equations (GEEs)[51] to model the longitudinal relationship between the trial's monthly follow-up visits and cotinine levels among children, caregivers and social network participants. Standard GEE models allowed for missing data to be modelled by making use of all available data without the need to use imputations to replace missing values.[52] Data were analysed on an intention-to-treat basis.

Non-transformed, raw paediatric cotinine values were positively skewed, and log transformation of the monthly data did not increase the symmetric distribution. Non-transformed and log-transformed data did not pass the Shapiro-Wilk normality test (see online supplemental figure 1). Transformation of the data does not alter the results for the primary outcome and the GEE modelling displayed henceforth using non-transformed data.

Descriptive statistics using measures of central tendency described baseline paediatric asthma control and caregiver mental health (anxiety and depression). Inferential statistics of these two clinical outcomes were not reported since valid statistical comparisons are not possible, given the lack of significant decrease in paediatric cotinine levels in intervention and control cohorts. Given that our study's primary outcome was a decrease in paediatric cotinine for secondary effects on improvement in both asthma control and caregiver mental health, we could not satisfy this premise, given the lack of fulfilment of the first objective, and therefore no further inferential analyses were performed. Due to budget restraints, we also did not collect the multitude of socioeconomic, environmental and clinical data from adult participants that would have allowed for more meaningful understanding of caregiver mental health outcomes—all of which are needed elements since we could not ascribe the results to an intervention.

Power calculations were based on the hypothesis of a 50% reduction in salivary cotinine levels at the 6-month study interval between study groups—derived from previous nicotine measurements in CM and smoking ban studies by our group.[4] To detect a mean monthly cotinine difference of 75% (salivary cotinine SD 2.2 ng/mL) difference between groups, with 80% power using a cut-off for statistical significance of 0.05, we needed to have a sample size of at least 45 caregiver–child triads. We attempted to increase the enrolled triad goal by 10% to account for loss to follow-up but we were unable to consistently maintain the additional required five triads in the study. Missing data were limited to approximately 10% over the total 6-month study. Missing data were not imputed in the analyses due to the relatively low missing data, as well as the possibility that there is no random loss to the missing data. There were no observed differences in patterns of missing data among randomised children, caregivers and social network members between the two cohorts. Given our lower-income study participants who may have multiple clinical and non-clinical factors for undertaking smoking behaviours and missing appointments, we believe that potential bias could be introduced if imputation processes were undertaken. If individual missing study visits were more likely to be engaging in more smoking behaviours or allowing children to be in high TSE environments, imputation methods could have have resulted in downward biases Individuals not available for follow-up could have been a different subset of participants (eg, higher psychosocial stressors, poorer medical supervision of paediatric asthmatics or lesser interest in personal well-being),

whose inclusion could have yielded lesser monthly change in individual and paediatric cotinine levels over the 6-month study follow-up period. Statistical significance was defined as two-sided, with significance level set at $p < 0.05$. Statistical analyses were conducted using STATA V.15.1.

RESULTS

Participant characteristics

Among the 418 individuals assessed for eligibility, 64% were excluded for predominantly the lack of fulfilment of inclusion criteria (online supplemental figure 2). Approximately 23% of maternal caregivers screened had expressed interest in the study but were excluded due to the inability to locate a member of their social network who is known to smoke in proximity to their child and willing to participate in the study. Less than 5% of participants were lost to follow-up but no reasons could be ascertained due to the inability to contact the participants. Only participants randomised to the intervention were lost to follow-up. We could not ascertain the reasons for participant attrition after multiple unsuccessful attempts at contacting them through home visits and phone messaging. No difference in baseline characteristics were noted in those lost to follow-up, compared with those who remained in the study. No attrition was noted among the control group despite being paid less than the intervention cohort, a monetary feature that the cohort was made aware in the institutional review board-approved informed consent forms.

The intervention cohort (total n=63) consisted of 21 participants equally distributed among linked triads of children, caregivers and social network members (table 1). The control cohort (total n=72) consisted of 24 participants within each triad population. The gender distribution of enrolled children was approximately equal, and >70% resided in households with an annual income that was below the federal poverty level.[53] Greater than 90% of the maternal caregivers were the biological mothers. There was an equal gender distribution among social network participants and represented a broad spread of assigned social roles. The social network member was in the majority of occasions a first-degree female relative of the maternal caregiver (eg, grandmother and aunt), while approximately 20% were the biological father. The median PHQ-4 scores among caregivers was suggestive of a diagnosis of mild anxiety; no indications of depression were present on baseline screening.

Smoking characteristics were equally distributed across adult participants with most reporting continued cigarette usage at each follow-up time point to address stress or a combination of stress and addiction. Median FTND scores indicated a moderate to high level of nicotine dependence in both social network and caregiver participants. Greater than 70% of participants reported living with at least one smoker and >65% did not enforce a home smoking ban. Younger children (<4 years) were classified as having mild suboptimal asthma control based on the TRACK questionnaire; however, children aged ≥4 years assessed using the ACT showed on average adequate asthma control. High levels of TSE was noted in both cohorts (median levels of >5 ng/mL), though median cotinine levels were 2 ng/mL higher in children randomised to the intervention. Median cotinine and exhaled CO levels confirmed the presence of active, daily smoking status in all caregivers and social network participants.

Cotinine outcome measures

No significant difference was demonstrated in the intervention cohort's mean monthly child cotinine levels over the 6-month follow-up study period, compared with the control cohorts (difference in slope (control–intervention)=−0.86 ng/mL/month, p=0.098, CI −0.160 to 1.887) (figure 2). The upward trend in cotinine levels within both paediatric cohorts could not be attributed to smoking patterns in maternal caregivers or social network members, both of whom had declining cotinine values over time. Maternal caregivers in the intervention group had no significant differences in their decline of cotinine values when compared with the caregivers in the control group (difference in slope (control–intervention)=3.30 ng/mL/month, p=0.144, CI −7.717 to 1.127). Social network participants allocated to the intervention also had no significant differences in cotinine values compared with their counterparts in the control group (difference in slope (control–intervention)=−1.59 ng/mL/month, p=0.546, CI −3.569 to 6.745). No adult participant reported use of the state tobacco quit line and subsequent prescription of NRTs to potentially confound cotinine data.

Financial incentive outcomes

The median monthly incentive earned by the intervention cohort's caregivers was $100, as compared with $50 for the social network member (online supplemental table 1). In total, caregivers allocated to the intervention earned $3150 of incentives, in contrast to $1300 earned by social network members. Solely one individual (caregiver) earned the maximum of $500 over the 6-month interval.

Social network measures

Approximately half (52%) of all social network participants were accessible for monthly study outcome follow-up in which the caregiver was available (online supplemental figure 3). Caregivers acknowledged that the lack of accessibility to the social network participant was also accompanied by their own lack of communication with the designated social network participant. Despite the lack of engagement by the social network participant with the caregiver, the majority of social network participants (>95%) re-engaged with the study team after being lost to follow-up by the final study visit.

DISCUSSION

This study did not support our primary hypothesis that paediatric cotinine levels will decrease using our financial incentive schema directed at caregivers and a member of their social network. Regardless of the randomisation status, maternal caregivers had declining cotinine levels over the 6-month trials—as evidenced by effect size. Despite the decrease in cotinine levels from caregivers who are a likely source of high amounts of TSE, paediatric cotinine values continued to rise in both cohorts. Our approach to using cash incentives to motivate reduction in smoking patterns among social network participants also did not prove successful. Moreover, maternal caregivers did not appear to have a cohesive relationship with their designated social network members over the trial period, which in turn made it difficult for constant enforcement of the study methodology among network participants.

The study's findings are contrary to a recent Cochrane systematic review showing high-certainty evidence that incentives improve smoking cessation rates during short-term and long-term follow-up.[19] The trials included in the review used mixed populations of whom none mirrored our study population, especially given our unique paediatric component. The objective of these trials, with the exception of studies focused on pregnant women, was the achievement of smoking cessation for

Table 1 Baseline characteristics of study participants (N=135) participants were recruited in triads and randomised by incentive administration

	Child (n=45)		Caregiver (n=45)		Social network (n=45)	
	I (n=21)	C (n=24)	I (n=21)	C (n=24)	I (n=21)	C (n=24)
Age (years)						
2–4	9 (43%)	5 (21%)				
5–11	11 (52%)	17 (71%)				
12–17	1 (5%)	2 (8%)				
18–30			7 (33%)	8 (33%)	9 (43%)	4 (16%)
31–50			13 (62%)	15 (63%)	8 (38%)	10 (42%)
>50			1 (5%)	1 (4%)	4 (19%)	10 (42%)
Gender						
Male	9 (43%)	14 (58%)			11 (52%)	11 (46%)
Female	12 (57%)	10 (42%)	21 (100%)	24 (100%)	10 (48%)	13 (54%)
Income						
<20K			15 (73%)	20 (83%)	15 (73%)	22 (92%)
20–40k			3 (14%)	4 (17%)	4 (19%)	2 (8%)
>40K			2 (9%)		2 (8%)	
Refused			1 (4%)			
Asthma control (median value)						
TRACK	70	65				
ACT	18	21				
Relationship to child						
Biological mother			20 (92%)	22 (92%)	1(4%)	1 (4%)
Biological father					6 (30%)	5 (21%)
Maternal grandmother			1 (4%)	2 (8%)	4 (19%)	7 (29%)
Maternal friend					3 (14%)	2 (8%)
Other family			1 (4%)		7 (33%)	9 (38%)
PHQ-4 (median value)			2	1.5		
Depression			5	4		
Anxiety						
FTND (median value)			7	8	8	8
Monthly cigarette expenditures (US$)						
<20			1 (4%)	2 (8%)	1 (4%)	2 (8%)
21–75			10 (48%)	12 (50%)	10 (48%)	5 (21%)
>76			10 (48%)	10 (42%)	10 (48%)	17 (71%)
Number of quit attempts in the last year						
0			8 (38%)	8 (33%)	7 (33%)	13 (55%)
1–2			7 (33%)	12 (50%)	11 (53%)	7 (29%)
>2			6 (29%)	4 (17%)	3 (14%)	4 (16%)
Methods used to quit in the last year						
Abrupt cessation only			8 (62%)	9 (56%)	6 (43%)	9 (82%)
Behavioural counselling only			1 (8%)	2 (13%)		
NRT only			2 (15%)	1 (6%)	7 (50%)	
Combination of the above			2 (15%)	4 (25%)	1 (7%)	2 (18%)
Reasons for smoking						
A				1 (4%)	1 (4%)	1 (4%)
SR			10 (45%)	12 (50%)	6 (28%)	8 (34%)
Other			3 (14%)	1 (4%)	1 (4%)	3 (12%)
A±SR±weight control			9 (41%)	10 (42%)	14 (64%)	12 (50%)
Additional smokers living in home						
0			4 (19%)	7 (29%)	4 (19%)	4 (17%)
1			15 (71%)	17 (71%)	14 (67%)	20 (83%)
2			2 (10%)		3 (14%)	
Indoor home smoking ban						
Yes			7 (33%)	6 (25%)	4 (19%)	6 (25%)
No			14 (67%)	18 (75%)	17 (81%)	18 (75%)
Cotinine (ng/mL) (median value)	7.3	5.28	192	187	187.1	201
Exhaled CO (ppm) (median value)			11	9	9	14.5

A, addiction/craving; ACT, Asthma Control Test; C, randomisation to the control cohort; CO, carbon monoxide; FTND, Fagerstrom Test for Nicotine Dependence; I, randomisation to the incentive intervention cohort; NRT, nicotine replacement therapy; PHQ-4, Patient Health Questionnaire-4; SR, stress relief; TRACK, Test for Respiratory and Asthma Control in Kids.
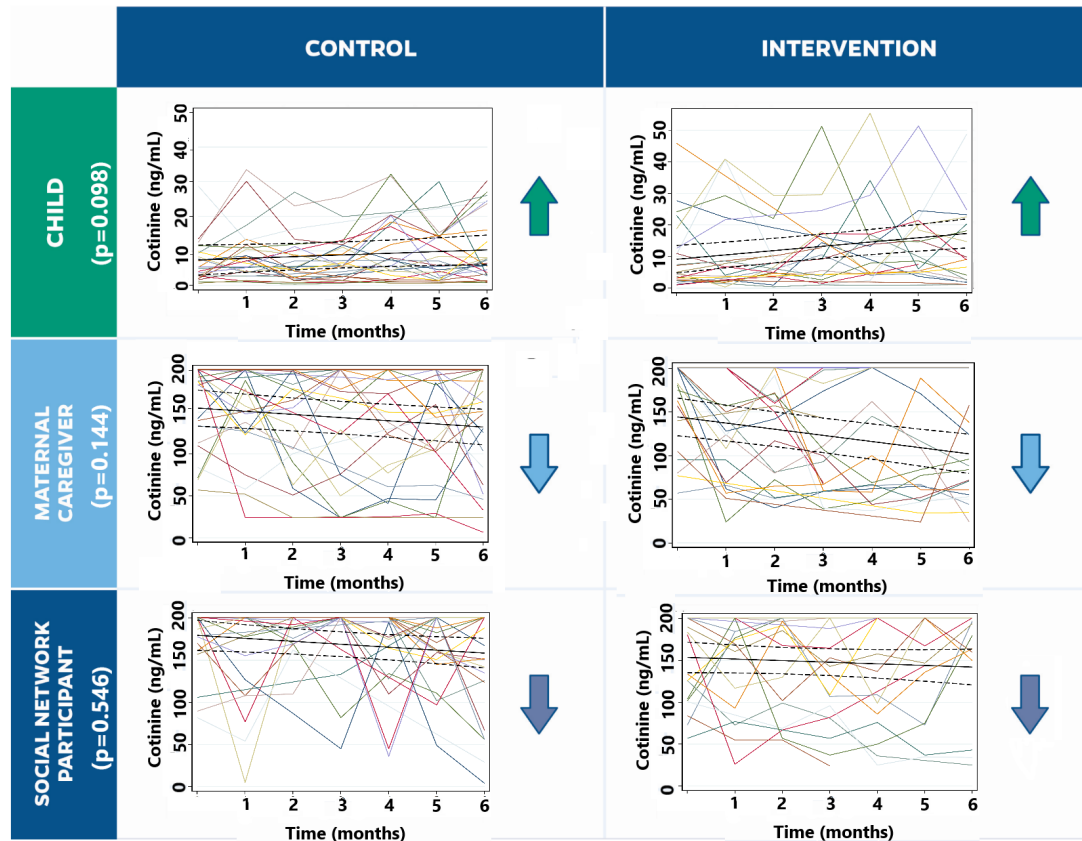
Figure 2 Trend of cotinine levels among triads (child, maternal caregiver and social network member) randomised to the incentive-based intervention, compared with the control cohort. Children, maternal caregivers and social network members in the intervention cohort did not have a significant difference in the mean monthly cotinine levels, compared with the control population. The effect size, p value and CI for each of the populations were the following: children (difference in slope (control–intervention)=−0.86 ng/mL/month; p=0.098, CI −0.160 to 1.887), maternal caregiver (difference in slope (control–intervention)=3.30 ng/mL/month; p=0.144, CI −7.717 to 1.127) and social network (difference in slope (control–intervention)=−1.59 ng/mL/month; p=0.546, CI: −3.569 to 6.745). Colourful lines represent individual trajectories of cotinine over time. Solid black line represents the regression line based on generalised estimating equation models, with CIs designated with dashed lines Arrows represent the cumulative trend in cotinine levels.

the primary benefit of the smoker themselves; pregnant women have an obvious motivation that transcends their own health. However, our incentive approach is unique in that we framed the intervention on the immediate betterment of children's respiratory well-being. We did not exclude conversations on the benefits to adult participants' health, but we provided a far greater focus on the secondhand smoke education and feedback of paediatric cotinine levels to our adult participants. Another key aspect that differentiates our work from other incentive studies is that our population was predominantly low-income versus middle-income populations predominantly enrolled in prior studies.

Prior paediatric financial incentive studies are limited with only one study that provided financial incentives for the expressed purpose of reducing secondhand smoke exposures in children.[54] This work was distinctly different from ours in that it provided low-value incentives ($5 or $10 gift cards) and were given to preteens for them to be motivated to remove themselves from high TSE environments. Unlike our approach that used a behavioural economic-derived incentive schema, the prior study used individualised behavioural coaching and delayed cotinine feedback (≥14 days) for TSE reduction.

The reason for the lack of efficacy of our incentive strategy may have a psychosocial and/or biological basis. Lower-income populations in Baltimore City may have a greater degree of nicotine

addiction, resembling that of other similar low-income African–American populations, for which the degree of addiction may be too high for an incentive-based approach to overcome.[55 56] Moreover, lower socioeconomic populations are under intense and evolving stressors (eg, employment, food security, housing, family disruptions or stressors), which can undermine cessation efforts.[57 58] Cigarettes may be seen as one of the few available modalities to address the stressors through its nicotine-based anxiolytic, antidepressant or through habit usage during stressful events.[58 59] The lack of incorporation of accessible mental health therapy, despite screening positive for anxiety and/or depression using PHQ-4 scoring, could have contributed to the lack of efficacy of our approach.

Furthermore, the fragile connections between the maternal caregiver and their designed social network member, as well the inability to adequately contact participants lost to follow-up due to unstable mobile phone numbers, showed the social limitations of our project and support systems of these caregivers. Linking the social and psychological limitations, we may have exacerbated the anxiety levels of maternal caregivers who felt obligated or responsible for the participation and improved cotinine values of their designated social network member. Moreover, the generalisability of our work could have been limited to maternal caregivers capable of possessing a large enough social network to locate at least one member willing to participate in

tobacco reduction. Given the prevalence of social isolation in low-income populations, our results may not be translatable to caregivers who lack such a large or diverse social network to include as co-participants.[60 61]

One ecological element that was not sufficiently emphasised in this intervention was the greater promotion and monitoring of home smoking bans. We provided handouts and briefly discussed the importance of home smoking bans at each study visit. This material was provided by senior research assistants, but this component may have had a different uptake or interpretation if we chose for it to be delivered by clinically affiliated staff (eg, community health workers and physician investigators). Our educational approach to a smoking ban was not too dissimilar than other studies using equatable minimalist approaches, including self-reported home smoking bans using solely promotional material delivered by mail and phone call.[62] However, the benefits of more intensive efforts are evident in recent work showing reduced paediatric cotinine and uptake in maternal cessation rates when implementing a smoking ban that includes the joint advocacy for incrementally more challenging smoking behaviour change (smoke free-zones within the home to a complete smoking ban) and social reinforcement (eg, praise).[63 64] A purposeful decision was made during our study design development to not overemphasise the promotion of the indoor smoking ban based on our earlier mixed-method work among our target study population.[31] Our earlier study showed that most of our study population preferred to address tobacco reduction not in terms of a home smoking ban but more directly towards the active smoker contributing to paediatric TSE. The participants alluded to the lack of social capital to influence the behaviours of other smokers within the home setting and therefore wanted more practical strategies directed at individual-based smoking reduction. Despite these conclusions, our paediatric-centric work could have benefited from regularly investing more resources into behavioural-based approaches promoting indoor smoking bans through improvement of caregiver self-efficacy to protect childhood TSE.

It is reasonable to consider that incentives might be more useful within a more holistic intervention that includes core components seen in other notable paediatric and adult TSE studies, including social (eg, smoke-free public housing and media education campaigns), behavioural (eg, skills training for physical avoidance of child exposures), physician level (eg, paediatrician advice) and biological (eg, personalise cessation approaches and diversify tobacco medication options that includes non-nicotine pharmacotherapies (Varenicline, Bupropion)).[65–69] Linking incentives to other non-cotinine measures or combining with cotinine biomarkers could have resulted in greater efficacy. For example, incentives may have been proven more useful if contingent on the continual usage of quitline services. Incentives could have had a more durable impact if linked with equity-positive outcomes and more ambitious social efforts (eg, improving material, human and social capital)–both of which, if lacking, can hinder smoking abatement or mitigation in lower socioeconomic populations.[70–72] The utility of incentives could have been enhanced if more practically tied to clinical outcomes, including the introduction of paediatric asthma or other environmentally influenced clinical outcomes. However, such disease-specific results often have multiple social, environmental and clinical covariates that result in difficulty in achievement of clinical outcomes based on limited intervention components.

Another limitation of our strategy was the inability to accommodate for thirdhand smoke exposures. Thirdhand smoke refers to the tobacco-related gases and particles that become embedded in various materials found in homes, cars and other indoor places.[73 74] Therefore, children will continue to show detectable cotinine levels due to the lingering effects of smoking even after the direct exposure has ceased. Thirdhand smoke in housing settings is particularly relevant for our study's lower-income population residing in Baltimore City since all participants resided in multiunit housing (apartment or duplex/triplex row homes). For these residents of multiunit housing, the source of high paediatric TSE can extend beyond their residence due to shared air spaces, ventilation systems, windows, elevator shafts, hallways, holes in walls, pipes and electric outlets.[75] If further work is to target paediatric cotinine in similar urban settings, then broader interdiscplinary efforts will be required (eg, promotion and enforcement of multiunit housing smoking bans).[76 77]

Other potential limitations associated with this study was that it may not have been powered to sufficiently test key moderator effects from covariates associated with paediatric TSE (eg, child age, proportion of a larger network of friends and family who smoke, and caregiver demographics (income, education and ethnicity)). These moderator effects may have explained the lack of decreasing cotinine values in children, but we were unable to discern their contribution in our control and intervention cohorts who had similar sociodemographic variables. Selection bias was also likely present due to the differing motivations of lower-income caregivers and select social network participants. Although all have verbally stated they wished to reduce or cease smoking behaviours, some individuals may have not have been as forthright in stating their motivation in joining the study (eg, enrolling primarily to acquire the cash incentives). If receipt of cash was the primary motivation, which is understandable in populations residing at or below poverty levels, then we can reasonably doubt if the participants were at a stage to prepare or act on reducing cigarette usage. We also relied on a reduction and not abstinence as the basis for incentive delivery, which is contrary to several incentive studies that target tobacco cessation.[26 41 42] This focus on reduction may make it more difficult to extrapolate our results to these cessation-oriented studies. However, we felt strongly that reduction is more practical since it is an outcome that is more likely to engage smokers to quit and ultimately increase and maintain the likelihood of cessation.[78 79] Abstinence was also a difficult variable for us to measure based on the device used for assessment cotinine values; our point-of-care serum-based device was preferable given its immediate results (5 min), but it registered quantitative cotinine values starting at >25 ng/mL, a value below which cannot distinguish between smoking abstinence, infrequent usage or low-daily smoker.[80 81] Furthermore, the role of feedback of paediatric cotinine levels on caregiver smoking behaviours were not acknowledged, a notably beneficial component in past paediatric TSE studies.[82 83] The results of children's salivary cotinine levels were not be acquired in a timely manner and could not be optimally used as a feedback tool for smoking modification. Our study only examined TSE from a single non-caregiver source, limiting our understanding of the exact source(s) of paediatric TSE. Perhaps our conclusions would support our hypothesis if we had diagnostics or applied advanced social network analyses to measure all TSE contributors among each child participant. Lastly, we had initially considered the utility of air nicotine measurements that could have been impactful for feedback-based results. However, we deferred its usage based both on our previous work[31] among our target population, who preferred only individual-based tobacco measures (eg, cotinine values), and limitations in acquiring timely results from air nicotine analyses.

In summary, our pilot work shows the limitations of cash incentives for motivating reductions in smoking behaviours for adult contributors to paediatric TSE, especially when the incentive is framed as predominantly a paediatric TSE reduction intervention. We observed that caregivers in both intervention and control cohorts had no significant change in cotinine levels, despite up to $500 being offered to those randomised to the incentive strategy. Paediatric cotinine levels trended upward as the study progressed, indicating the complex and multiple routes of exposures that likely require comprehensive, community-based interventions.

ORCID iD
Mandeep S Jassal http://orcid.org/0000-0001-9452-3328

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