Feasibility of enhancing well-child visits with family nutrition and physical activity risk assessment on body mass index

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Summary

Objective

Integration of behavioural risk assessment into well-child visits is recommended by clinical guidelines, but its feasibility and impact is unknown.

Methods

A quasi-experimental study evaluated the feasibility and effectiveness of risk assessment on body mass index (BMI) at 1-year follow-up. Children with assessments (intervention) were compared with those who did not complete assessments (non-respondent) and those who received standard care (non-exposed).

Results

Analyses included 10,647 children aged 2–9 years (2,724 intervention, 3,324 non-respondent and 4,599 non-exposed). Forty-five per cent of parents completed the assessments. Intervention and non-respondent groups differed in change in BMI z-score at 1 year by /C0.05 (confidence interval [CI]: /C0.08, /C0.02; /P = 0.0013); no difference was observed with non-exposed children. The intervention group had a smaller increase in BMI z-score (0.07 ± 0.63) than non-respondent group (0.13 ± 0.63). For children with normal weight at baseline, intervention versus non-respondent groups differed in BMI z-score change by /C0.06 (CI: /C0.10, /C0.02; /P = 0.0025). However, children with overweight at baseline in the intervention versus the non-exposed group differed in BMI z-score change (0.07 [CI: 0.02, 0.14]; /P = 0.016). When analysed by age, results were similar for 2- to 5-year-olds, but no differences were found for 6- to 9-year-olds.

Conclusion

Automating risk assessment in paediatric care is feasible and effective in promoting healthy weight among preschool but not older children.

Keywords: BMI, paediatrics, prevention, primary care.

Introduction

Childhood obesity remains a pervasive problem, and in response, obesity prevention guidance has been established for paediatric primary care (1). Endorsed universal protocols for identification, prevention and treatment of paediatric obesity include body mass index (BMI) screening, risk assessment, preventive counselling and education for parents and children, regardless of weight status (2,3). Screening should include growth (e.g. age-specific and sex-specific BMI percentile) and risk assessment of factors including parental weight status, family income, nutrition, physical and sedentary activity, and sleep (3).

Paediatric primary care providers can play a critical role by improving parental understanding of healthy growth patterns and obesity risk factors (4). Mothers prefer physicians as the source of feeding, growth and health information, and paediatric providers agree it is their role to discuss these topics (5–7). Primary prevention strategies

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to minimize risk may be more effective and efficient than treating obesity after it occurs. Because most children attend well-child visits (WCVs) and paediatric providers are trusted healthcare professionals (4), the paediatric clinical setting provides a promising, broad-scale opportunity to conduct primary obesity prevention. To capitalize on this opportunity, it is important to develop and test tools that can be integrated into the providers’ practice and workflow (8).

The family nutrition and physical activity (FNPA) risk assessment tool could serve as a standardized tool to meet these clinical guidelines as it assesses parenting practices, child behaviours and home environmental characteristics that predispose children to becoming obese (9). In school-aged children, the tool demonstrates utility in longitudinal analyses to predict a child’s risk for obesity (10,11), and the summary risk score has been related to adiposity measures, severity of obesity, cardiovascular disease risk and glucose intolerance (12–14). Furthermore, the FNPA has been shown to enable paediatric providers to quickly assess risk and provide behaviourally anchored counselling during WCVs (15).

The present study examined the feasibility of collecting FNPA assessments under real-world conditions and the effectiveness of integrating the assessment during WCV on child BMI over a 1-year period. Given that exposure to FNPA has been associated with parent intent to reduce risk behaviours associated with childhood obesity (16), the hypothesis was that children whose parents completed the FNPA at baseline would have lower BMI at follow-up when compared with children whose parents did not complete FNPA, regardless of whether this was due to failure or lack of opportunity to respond. Results are reported by child baseline weight category to evaluate the effectiveness of the intervention on primary and secondary prevention. Additionally, we report associations between FNPA risk assessment score and obesity risk to validate FNPA in a new population, preschool-aged children (9,11). Likewise, results for preschool-aged and school-aged children are reported separately given that FNPA was originally designed for school-aged children.

Methods

Experimental design

A quasi-experimental study design was used to evaluate the effectiveness of the multilevel intervention that consisted of (a) health system programming that fired the FNPA risk assessment at scheduled WCVs, captured parent-reported data and integrated those data into the child’s electronic health record (EHR) as clinical decision support; (b) parent completion of the risk assessment; and (c) provider use of the child’s EHR to observe the risk assessment data and deliver preventive counselling. The feasibility of collecting FNPA data at WCV was evaluated as the frequency of parents being offered and completing the assessment in association with a scheduled WCV. Change in BMI z-score at 1-year follow-up was the primary outcome.

Participants

Intervention group participants included children who had a baseline WCV at an intervention clinic between 1 November 2013 and 31 October 2014 with weight and height data (BMI screening) and a completed FNPA assessment and a second (1-year follow-up) WCV with height and weight data within 10–18 months of baseline. The non-respondent group participants were children from the same clinics as intervention participants and FNPA assessments were offered but not completed at baseline. Non-exposed group participants included children who completed a baseline WCV at a non-intervention clinic with weight and height data between 1 November 2013 and 31 October 2014 and had follow-up WCVs within the described timeline earlier. Participants were aged 2–9 years at baseline. Child sex, date of birth, height, weight, FNPA completion status, demographic information and clinical diagnoses were extracted from the EHR. Children diagnosed with type 1 diabetes or cancer were excluded.

The participants were derived from paediatric clinics at Geisinger, a large, integrated health system in Pennsylvania. The health system made the FNPA risk assessment available as standard of care during WCVs in conveniently selected intervention clinics, and all providers in those clinics had been trained to use the FNPA. Intervention and non-respondent participants were derived from 14 clinics, and all providers were employed by Geisinger. Non-exposed participants were derived from six clinics where FNPA was not available nor were providers trained on the intervention. The intervention group was compared with both control groups separately.

Risk assessment intervention

The intervention was designed to promote assessment of behavioural and environmental risk factors for obesity and to help parents and providers engage in discussion about healthy lifestyles. The intervention was initiated with an automated email generated 10 days prior to a scheduled WCV via the patient portal (at home). The email included a link to the FNPA risk assessment. As shown in
Parents completed the FNPA risk assessment in the patient portal or waiting room (via iPad, kiosk) and received immediate feedback that displayed their response, the recommended practice and a behaviourally anchored strategy to consider adopting. At home, parents could print results; in the clinic, this was incorporated into the printed summary report provided after the visit. The risk assessment took less than 2 min to complete, a process acceptable to both parents and providers (15). Front desk support staff and rooming nurses at intervention clinics were trained to encourage parent completion of the risk assessment before the patient was roomed for examination.

The health system’s programming enabled automated FNPA risk assessment data collection; integration of these data with BMI screening data into the EHR in real-time; and display as clinical decision support. This support included responses for each FNPA item, the summary score, talking points for promoting health and reducing risk, and printable educational materials. Providers were trained to use motivational interviewing and goal setting to counsel parents on the FNPA topics and provide tailored feedback with educational materials. Educational materials (English and Spanish) were mapped to FNPA topics and developed by the Academy of Nutrition and Dietetics Foundation Kids Eat Right Program (17).

**Anthropometric measures**

All clinics were trained on anthropometric methods for height and weight measurement and used standardized, calibrated scales (Healthometer 599KL) and stadiometers (SECA 264) to screen BMI with EHR documentation. As a standard procedure at WCVs, height was measured to the nearest 0.1 cm and weight to the nearest 0.1 kg. Sex-specific BMI-for-age percentiles were calculated in EHR to identify the children by weight status: underweight (≤5th percentile), normal weight (>5th and <85th), overweight (≥85th and <95th), obese (≥95th and <99th) and severely obese (≥99th).

**Statistical analysis**

All analyses were performed using a SAS (version 9.4) statistical software (SAS Institute, Inc., Cary, NC). Differences in the baseline demographic characteristics between the intervention, non-respondent and non-exposed groups were assessed. Categorical variables (e.g. race) were compared using chi-squared test, and continuous variables (e.g. age and BMI z-score) were tested for normality. If variables were normally distributed,
then t-tests were used. Mann–Whitney U-tests were used to test for nonparametric differences in distribution.

Outcomes of interest were frequency of FNPA risk assessment completion by opportunity, odds of obesity by FNPA risk score tertile and changes in BMI z-score from baseline to 1-year follow-up WCV. An analysis of variance was used to determine the association between weight status category and mean baseline FNPA risk score. Logistic regression was used to calculate the odds of obesity by baseline FNPA risk score tertile and changes in BMI z-score from baseline to 1-year follow-up WCV. An analysis of variance was used to determine the association between weight status category and mean baseline FNPA risk score. Logistic regression was used to calculate the odds of obesity by baseline FNPA risk score tertile to evaluate validity of the tool. Differences in BMI z-score at baseline and 1 year were compared between groups using two-sample t-tests. Generalized linear regression models were used to test for differences in change in BMI z-score between groups. Post hoc analyses used multilevel models to examine and account for clustering within clinic site and care provider. For these models, the intraclass correlation coefficient was calculated to evaluate the amount of variation explained by site and provider clusters. This analysis included the introduction of clinic-level characteristics (clinic-level tertiles for the number of WCVs per year, per cent of WCV with completed FNPA, number of providers and number of WCV per provider) and provider characteristic (tertile for number of WCVs per year) to test for site-level and provider-level effects and effect modification with the intervention. Data are reported as means ± standard deviations, and results were considered significant at \( P < 0.05 \).

### Results

#### Participant characteristics

As shown in Table 2a, 2,724 intervention, 3,324 non-respondent and 4,599 non-exposed children were evaluated. Children were primarily non-Hispanic White and male, 27.2% received medical assistance (a proxy for low-income status) and 12.9% of children were obese, which is lower than national estimates (1). Demographics were similar for the two age groups of 2–5 years (Table 2b) and 6–9 years (Table 2c) at baseline. As shown, a total of 6,496 children were aged 2–5 (1,617 intervention, 2,133 non-respondent and 2,746 non-exposed), and 4,151 were aged 6–9 (1,107 intervention, 1,191 non-respondent and 1,853 non-exposed). At baseline, on average, the intervention children significantly differed from the non-respondent and the non-exposed children in age, BMI z-score and weight category (all \( P \)s ≤ 0.05). Intervention and non-exposed children also significantly differed in medical assistance and race/ethnicity (all \( P \)s ≤ 0.05). Among children aged 2–5, intervention children significantly differed from non-respondent and non-exposed children in age, BMI z-score and weight category (all \( P \)s ≤ 0.05). Additionally, children aged 2–5 in the intervention and non-exposed groups significantly differed by sex, medical assistance and race/ethnicity. In contrast, among children aged 6–9, the intervention and

| Intervention (N = 2,724) | Non-respondent (N = 3,324) | Non-exposed (N = 4,599) | \( P \)-value\(^{†}\) | \( P \)-value\(^{‡}\) |
|-------------------------|-----------------------------|-------------------------|-------------------------|-------------------------|
| **Baseline age (years)** | 5.4 ± 2.1 | 5.0 ± 2.4 | 5.3 ± 2.1 | \(<0.0001\) \(0.021\) |
| **BMI z-score**         | 0.46 ± 1.12 | 0.36 ± 1.17 | 0.29 ± 1.14 | \(<0.0001\) \(0.0009\) |
| **Category**            | \(N\) | % | \(N\) | % | \(N\) | % | \(P\)-value\(^{†}\) | \(P\)-value\(^{‡}\) |
| **Sex**                 | 1,351 | 49.6 | 1,622 | 48.8 | 2,208 | 48.0 | 0.536 | 0.189 |
| Male                    | 1,373 | 50.4 | 1,702 | 51.2 | 2,391 | 52.0 | \(0.014\) \(<0.0001\) |
| **Weight category**     | 90 | 3.3 | 156 | 4.7 | 202 | 4.4 | \(<0.0001\) | \(<0.0001\) |
| Underweight             | 1,810 | 66.5 | 2,242 | 67.5 | 3,271 | 71.1 | \(<0.0001\) | \(<0.0001\) |
| Normal                  | 435 | 16.0 | 475 | 14.3 | 590 | 12.8 | \(<0.0001\) | \(<0.0001\) |
| **Medical assistance**  | 389 | 14.3 | 451 | 13.6 | 536 | 11.7 | \(<0.0001\) | \(<0.0001\) |
| No                      | 1,802 | 66.2 | 2,187 | 65.8 | 3,767 | 81.9 | \(0.770\) \(<0.0001\) |
| Yes                     | 922 | 33.9 | 1,137 | 34.2 | 832 | 18.1 | \(0.328\) \(<0.0001\) |
| **Race/ethnicity**      | 2,232 | 81.9 | 2,771 | 83.4 | 4,203 | 91.4 | \(<0.0001\) | \(<0.0001\) |
| Non-Hispanic White      | 279 | 10.2 | 319 | 9.6 | 120 | 2.6 | \(<0.0001\) | \(<0.0001\) |
| Hispanic                | 213 | 7.8 | 234 | 7.0 | 276 | 6.0 | \(<0.0001\) | \(<0.0001\) |

BMI, body mass index; FNPA, family nutrition and physical activity; SD, standard deviation.

\(^{†}\)Comparing intervention group versus non-respondent group.

\(^{‡}\)Comparing intervention group versus non-exposed group.

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non-respondent groups significantly differed in age ($P \leq 0.01$), and the intervention and non-exposed group differed in medical assistance and race/ethnicity (all $P$'s $\leq 0.0001$).

Table 2b  Characteristics of 2- to 5-year-old children at baseline in the FNPA risk assessment intervention group and comparison non-respondent and non-exposed groups

| Category                  | Intervention ($N = 1,617$) | Non-respondent ($N = 2,133$) | Non-exposed ($N = 2,746$) | $P$-value$^1$ | $P$-value$^2$ |
|---------------------------|-----------------------------|------------------------------|---------------------------|--------------|--------------|
| Baseline age (years)      | 3.9                         | 3.7                          | 3.7                       | $<0.0001$    | $<0.0001$    |
| BMI z-score               | 0.44                        | 0.17                         | 0.17                      | $<0.0001$    | $<0.0001$    |
| Sex                       | Female 826 51.1 1,041       | 48.8                         | 1,315                     | 47.9         | 0.167        |
|                           | Male 791 48.9 1,092         | 51.2                         | 1,431                     | 52.1         | 0.042        |
| Weight category           | Underweight 56 3.5 121     | 5.7                          | 157                       | 5.7          | 0.0013       |
|                           | Normal 1,096 67.8 1,485    | 69.6                         | 2,007                     | 73.1         |              |
|                           | Overweight 252 15.6 291    | 13.6                         | 326                       | 11.9         |              |
|                           | Obese 213 13.2 236         | 11.1                         | 256                       | 9.3          |              |
| Medical assistance        | No 1,054 65.2 1,381        | 64.7                         | 2,208                     | 80.4         | 0.781        |
|                           | Yes 563 34.8 752           | 35.3                         | 538                       | 19.6         |              |
| Race/ethnicity            | Non-Hispanic White 1,305 80.7 | 83.2                      | 2,504                     | 91.2         | 0.056        |
|                           | Hispanic 179 11.1 222      | 10.4                         | 71                        | 2.6          |              |
|                           | Other 133 8.2 136          | 6.4                          | 171                       | 6.2          |              |

Table 2c  Characteristics of 6- to 9-year-old children at baseline in the FNPA risk assessment intervention group and comparison non-respondent and non-exposed groups

| Category                  | Intervention ($N = 1,107$) | Non-respondent ($N = 1,191$) | Non-exposed ($N = 1,853$) | $P$-value$^1$ | $P$-value$^2$ |
|---------------------------|-----------------------------|------------------------------|---------------------------|--------------|--------------|
| Baseline age (years)      | 7.7                         | 7.7                          | 7.7                       | 0.0006       | 0.363        |
| BMI z-score               | 0.49                        | 0.48                         | 1.07                      | 0.187        | 0.713        |
| Sex                       | Female 529 47.4 581         | 48.2                         | 893                       | 48.1         | 0.515        |
|                           | Male 582 52.6 610           | 51.8                         | 960                       | 51.9         | 0.686        |
| Weight category           | Underweight 34 3.1 35      | 2.9                          | 45                        | 2.4          | 0.553        |
|                           | Normal 714 64.5 757        | 63.6                         | 1,264                     | 68.2         | 0.159        |
|                           | Overweight 183 16.5 184    | 15.5                         | 264                       | 14.3         |              |
|                           | Obese 176 15.9 215         | 18.1                         | 280                       | 15.1         |              |
| Medical assistance        | No 748 67.6 806            | 67.7                         | 1,559                     | 84.1         | 0.958        |
|                           | Yes 359 32.4 385           | 32.3                         | 294                       | 15.9         |              |
| Race/ethnicity            | Non-Hispanic White 927 83.7 | 83.6                         | 1,699                     | 91.7         | 0.529        |
|                           | Hispanic 100 9.0 97        | 8.1                          | 49                        | 2.6          |              |
|                           | Other 80 7.2 98            | 8.2                          | 105                       | 5.7          |              |

BMI, body mass index; FNPA, family nutrition and physical activity; SD, standard deviation.

Feasibility

At baseline, parent response rate to FNPA risk assessment was 45% overall, 43% among parents of children...
aged 2–5 and 48% among parents of children aged 6–9. Data were collected in the clinic waiting room for 85% of the patients, whereas 15% completed the risk assessment in the patient portal.

Validation of risk score

The association between FNPA risk assessment summary scores and weight category at baseline was evaluated to validate the risk score. As shown in Table 3, the summary scores for intervention children significantly differed by weight category for children aged 2–9 and in both age groups (all $P$'s $\leq 0.01$). The summary scores were grouped into tertiles by age and evaluated for odds of overweight or obesity at baseline (9,11). In the present study, on average, the lowest tertile of summary scores was associated with greater odds ratio of being overweight (1.45, confidence interval [CI]: 1.22, 1.72), obese (1.70, CI: 1.36, 2.12) and severely obese (1.56, CI: 1.07, 2.29). Likewise, for children aged 2–5, the lowest tertile scores were associated with greater odds of being overweight (1.47, CI: 1.17, 1.85), obese (1.48, CI: 1.10, 1.99) and severely obese (1.34, CI: 0.84, 2.16). Similarly, for 6- to 9-year-olds, lower tertile scores were associated with greater odds ratio of being overweight (1.43, CI: 1.09, 1.87), obese (2.03, CI: 1.46, 2.83) and severely obese (2.09, CI: 1.09, 4.00).

Baseline to 1-year follow-up

For children aged 2–9 (reported in parentheses) and aged 2–5 (reported in brackets), the observed effects followed the same pattern. The change in BMI $z$-score from baseline to 1 year differed significantly by $-0.05$ (CI: $-0.08$, $-0.02$; $P = 0.0013$) $-0.09$, CI: $-0.13$, $-0.04$; $P = 0.0002$] between the intervention and non-respondent children, but no difference was observed between the intervention and non-exposed children as shown in Table 4a (Table 4b). Children in the intervention group had a smaller increase in mean BMI $z$-score ($0.07 \pm 0.63$) $[0.10 \pm 0.71]$ than non-respondent group ($0.13 \pm 0.63$) $[0.19 \pm 0.71]$. In a subsample of children with normal weight at baseline, the intervention versus non-respondent group differed significantly in BMI $z$-score change by $-0.06$ (CI: $-0.10$, $-0.02$; $P = 0.0025$) $-0.09$, CI: $-0.14$, $-0.03$; $P = 0.0013$). Children in the intervention group had a smaller increase in mean BMI $z$-score ($0.09 \pm 0.62$) $[0.13 \pm 0.68]$ than non-respondent group ($0.15 \pm 0.61$) $[0.21 \pm 0.67]$. However, children with overweight at baseline in the intervention versus the non-exposed group differed significantly in BMI $z$-score change by $0.07$ (CI: $0.02$, $0.14$; $P = 0.016$) $0.12$, CI: $0.03$, $0.22$; $P = 0.0096$). Children in the intervention group had an increase in mean BMI $z$-score ($0.02 \pm 0.50$) $[0.02 \pm 0.58]$, whereas those in the non-exposed group decreased BMI $z$-score ($-0.06 \pm 0.48$) $-0.11 \pm 0.56$.

For children aged 6–9, there were no significant differences in BMI $z$-score change at 1-year follow-up between intervention and non-respondent or non-exposed comparison groups (Table 4c). The BMI $z$-score outcomes are reported from unadjusted models. In sensitivity analysis, adjusted models were evaluated (adjusting for baseline BMI $z$-score, medical assistance, race and ethnicity) and found to be consistent with the reported unadjusted results (data not shown).

Post hoc clustering analyses

In multilevel modelling (data not shown), the percent of variation explained by clinic site and provider was $<1\%$ in the overall model, suggesting that clinic and provider characteristics accounted for a minimal amount of change in BMI $z$-score (similar results were observed when stratifying within intervention, non-respondent and non-exposed participants). To confirm this assumption, multilevel models that account for clinic and provider clustering, clinic characteristics and a provider characteristic resulted in parameter estimates of a similar magnitude and significance level to those derived from simple analyses.

| Table 3 | Association between baseline total FNPA risk assessment score with baseline weight status in the intervention group age 2–9 years ($N = 2,724$) and by age group 2–5 years ($N = 1,617$) and 6–9 years ($N = 1,107$) |
|---------|-------------------------------------------------|
| Baseline FNPA, mean (SD) | Underweight | Normal | Overweight | Obese | $P$-value |
| Age 2–9 | $N = 90$ | $N = 1,810$ | $N = 435$ | $N = 389$ | $<0.0001$ |
| 64.2 (6.3) | 65.6 (5.9) | 65.0 (5.8) | 63.7 (6.0) |
| Age 2–5 | $N = 56$ | $N = 1,096$ | $N = 252$ | $N = 213$ | $0.0033$ |
| 64.3 (4.9) | 65.5 (5.8) | 64.6 (5.7) | 64.2 (5.6) |
| Age 6–9 | $N = 34$ | $N = 714$ | $N = 183$ | $N = 176$ | $<0.0001$ |
| 64.1 (8.1) | 65.9 (6.1) | 65.5 (6.1) | 63.0 (6.4) |

FNPA, family nutrition and physical activity; SD, standard deviation.
Table 4a BMI z-scores at baseline, 1-year follow-up and changes from baseline to 1-year follow-up compared between the intervention and non-respondent or non-exposed groups for subjects aged 2–9 years and by baseline weight category

| Weight category | Timing  | Intervention N | Mean (SD) | Non-respondent N | Mean (SD) | Difference Mean [95% CI] | P-value |
|-----------------|---------|----------------|-----------|------------------|-----------|--------------------------|---------|
| Age 2–9         | Baseline| 2,724          | 0.46 (1.12)| 4,599            | 0.29 (1.14)| 0.17 [0.12, 0.22]       | <0.0001 |
|                 | 1 year  | 0.54 (1.13)    | 0.38 (1.14)|                 | 0.16 [0.10, 0.21]     |              | <0.0001 |
|                 | Change  | 0.07 (0.63)    | 0.09 (0.62)|                 | 0.00 [0.04, 0.02]     |              | 0.392  |
| Underweight     | Baseline| 90             | –2.23 (0.70)| 202              | –1.42 (0.92) | 0.12 [–0.26, 0.11]     | 0.298   |
|                 | 1 year  | –1.54 (0.99)   | 0.80 (0.90) |                 | 0.11 [–0.39, 0.17]    |              | 0.446  |
|                 | Change  | 0.69 (1.20)    |            |                  |            |                          |         |
| Normal          | Baseline| 1,810          | 0.02 (0.64) | 3,271            | –0.05 (0.67) | 0.07 [0.03, 0.11]      | 0.0002  |
|                 | 1 year  | 0.11 (0.81)    | 0.04 (0.83) |                 | 0.07 [0.03, 0.12]    |              | 0.0029 |
|                 | Change  | 0.02 (0.62)    | 0.09 (0.62)|                 | 0.00 [0.04, 0.04]    |              | 0.990  |
| Overweight      | Baseline| 435            | 1.31 (0.18) | 590              | 1.32 (0.17) | –0.02 [–0.04, 0.01]    | 0.158   |
|                 | 1 year  | 1.33 (0.53)    | 1.27 (0.52) |                 | 0.06 [–0.01, 0.12]   |              | 0.076  |
|                 | Change  | 0.02 (0.50)    |            |                  | 0.06 [0.02, 0.14]    |              | 0.016  |
| Obese           | Baseline| 389            | 2.21 (0.52) | 536              | 2.22 (0.49) | –0.01 [–0.08, 0.06]    | 0.752   |
|                 | 1 year  | 2.11 (0.64)    | 2.16 (0.59) |                 | –0.04 [–0.12, 0.04]  |              | 0.305  |
|                 | Change  | –0.10 (0.54)   | –0.07 (0.45)|                 | –0.03 [–0.10, 0.03]  |              | 0.354  |

BMI, body mass index; SD, standard deviation.

**Discussion**

This is the first study to demonstrate the feasibility and clinical effectiveness of implementing an automated risk assessment during WCV for paediatric preventive care. Lower FNPA summary scores were associated with greater odds for overweight, obesity and severe obesity among school-aged children, consistent with prior research, as well as among preschool-aged children, a population previously uninvestigated (9,11). On average, children whose parent completed the FNPA risk assessment, and thus had exposure to the intervention, experienced smaller increases in BMI z-score compared with non-respondent children. Results were similar for children with a normal weight at baseline and those aged 2–5 but not for children aged 6–9. However, children (aged 2–9 and 2–5) with overweight at baseline had a larger increase in BMI z-score compared with the non-exposed group. These results suggest that exposure to the risk assessment may promote healthier growth patterns for children aged 2–9 and 2–5, specifically for children with normal weight at baseline. It is not clear why children with overweight exposed to the intervention had unhealthier growth patterns and why significant effects were not observed among children aged 6–9. Results could be due to the intervention...
emphasizing behavioural and environmental factors within the family and at home, a setting where preschool-aged children spend more time as compared with school-aged children (18).

Poor nutrition, inadequate physical activity, excessive screen time and sedentary activity, and poor sleep habits are risk factors for childhood obesity; however, clinicians need a reliable mechanism to assess risk, assurance that the score is evidence based, and skills for translating the risk score into preventive counselling. This study demonstrates feasibility and sustainability of this approach. In the present health system, FNPA risk assessment completion rates, 5 years after introduction, exceed 50% confirming the sustainability of automated mechanism to prompt data collection, but additional efforts are needed to improve completion rates. In comparison with paper-version or interview-administered tools that require human resources, automation is feasible, sustainable and aided with multiple collection opportunities (e.g. parent portal and clinic staff encouragement) (19). Implementing an automated mechanism to collect and integrate risk assessment data for use in routine WCV into providers’ workflow is feasible, but clinician utilization of the risk score and counselling practices should be continuously evaluated.

The FNPA risk assessment is a valid tool to identify risk factors associated with overweight and obesity among school-aged and preschool-aged children. The results

**Table 4b** BMI z-scores at baseline, 1-year follow-up and changes from baseline to 1-year follow-up compared between the intervention and non-respondent or non-exposed groups for subjects aged 2–5 years and by baseline weight category

| Weight category | Timing | Intervention N | Mean (SD) | Non-respondent N | Mean (SD) | Difference Mean [95% CI] | P-value |
|-----------------|--------|----------------|----------|------------------|----------|--------------------------|---------|
| **Age 2–5**     |        |                |          |                  |          |                          |         |
| Baseline        |        | 1,617          | 0.44 (1.14) | 2,746            | 0.17 (1.17) | 0.27 [0.20, 0.35]        | <0.0001 |
| 1 year          |        | 0.54 (1.16)    | 0.31 (1.14) | 0.24 [0.17, 0.31] | <0.0001 |
| Change          |        | 0.10 (0.71)    | 0.14 (0.70) | 0.04 [0.08, 0.00] | 0.081   |
| **Underweight** |        |                |          |                  |          |                          |         |
| Baseline        |        | 56             | −2.26 (0.75) | 157              | −2.23 (0.51) | −0.03 [−0.24, 0.19]      | 0.810   |
| 1 year          |        | −1.47 (1.15)   | −1.29 (0.96) | −0.17 [−0.51, 0.17] | 0.271   |
| Change          |        | 0.79 (1.25)    | 0.94 (0.92) | 0.15 [−0.51, 0.22] | 0.350   |
| **Normal**      |        |                |          |                  |          |                          |         |
| Baseline        |        | 1,096          | 0.02 (0.65) | 2,007            | −0.10 (0.68) | 0.12 [0.08, 0.17]        | <0.0001 |
| 1 year          |        | 0.15 (0.82)    | 0.04 (0.84) | 0.10 [0.04, 0.16] | 0.0010  |
| Change          |        | 0.13 (0.68)    | 0.15 (0.67) | −0.02 [−0.07, 0.03] | 0.421   |
| **Overweight**  |        |                |          |                  |          |                          |         |
| Baseline        |        | 252            | 1.30 (0.18) | 326              | 1.31 (0.17) | 0.01 [−0.03, 0.02]       | 0.713   |
| 1 year          |        | 1.32 (0.60)    | 1.20 (0.61) | 0.12 [0.02, 0.22] | 0.020   |
| Change          |        | 0.02 (0.58)    | −0.11 (0.56) | 0.12 [0.03, 0.22] | 0.0096  |
| **Obese**       |        |                |          |                  |          |                          |         |
| Baseline        |        | 213            | 2.31 (0.62) | 256              | 2.32 (0.61) | −0.01 [−0.12, 0.10]      | 0.877   |
| 1 year          |        | 2.19 (0.78)    | 2.21 (0.77) | −0.02 [−0.16, 0.12] | 0.738   |
| Change          |        | −0.12 (0.68)   | −0.10 (0.61) | −0.02 [−0.13, 0.10] | 0.799   |

BMI, body mass index; SD, standard deviation.

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with children aged 6–9 align with previous research demonstrating that the FNPA summary score is associated with odds of overweight, obesity and severe obesity (9,11,14). Prevention prior to age 5 years is essential as obesity tends to persist once established (20,21); however, no risk assessments were identified for children as young as age 2 years when the study was designed. Recently, the Healthy Kids obesity risk assessment has shown face, content, convergent and predictive validity in preschool-aged children with lower household income (22). Both FNPA and Healthy Kids risk assessments offer potential value to clinicians to involve parents in self-assessing risk using validated tools. This study did not evaluate parent experience or behaviour change following FNPA risk assessment; however, promising results have been observed in earlier studies (15,16).

Enhancing BMI screening with FNPA risk assessment at routine WCVs was beneficial for children aged 2–9 and 2–5 overall, specifically those with normal weight at baseline, compared with children whose parents did not complete FNPA at baseline. Change in BMI z-score between baseline and 1-year follow-up favoured the intervention indicating healthier growth patterns. The overall difference in favour of the intervention group was in the same direction and equivalent to the effect of school-based preventive interventions using more intense...
approaches and rigorous study designs (23). This study addresses the lack of preventive interventions in primary care with promising results (23). Albeit, these findings may be confounded by survey response bias. Because of lack of randomization, we cannot rule out that parents of children who completed the FNPA assessment were more motivated to address obesity risk factors than those who failed to respond.

Overall, no significant differences were observed when comparing the intervention and non-exposed groups. However, change in BMI z-score among children with overweight favoured the non-exposed group rather than the intervention group. Preventive counselling related to healthy eating and physical activity could have occurred in non-intervention clinics to benefit the non-exposed group but was unlikely (24–26). Alternatively, provider-level factors such as years of practice or duration of the relationship with the family may offer insight for future research.

Inconsistent findings between the intervention and comparison groups may be attributed to differences in socio-economic status or race and ethnicity as prior research indicates strong influences from these social determinants on BMI (27). Regardless, comparisons between the intervention versus non-respondent groups are more ecologically valid as these individuals were offered the same opportunity and received care from the same providers in the same clinics. Importantly, these comparisons underscore the value of the automated risk assessment and clinical decision support tools over provider training alone (28,29). Without access to the risk assessment data, summary score, talking points and educational materials, provider training offered no observed protective benefit on BMI.

The null findings observed among children aged 6–9 reinforce the guidance from the US Preventive Services Task Force (3). In this older group, BMI screening plus risk assessment should inform preventive counselling and the provision of or referral to proven, evidence-based interventions. One occurrence of a WCV with BMI screening, risk assessment and preventive counselling may not be enough to prevent or reduce obesity. Future studies should utilize longitudinal designs to evaluate whether regular, sequential WCVs with BMI screening and risk assessment are associated with prevention and remission of obesity, consistent with natural observations (30).

Strengths of the study include the large sample size and the quasi-experimental design that compared outcomes under ecologically sound, naturalized conditions. The gains in external validity in this case outweigh the loss of internal validity associated with the lack of a truly experimental design. However, these findings should be considered in context of observed limitations. Implementation feasibility was evaluated by one factor, but clinic staff, patient experience, provider acceptance and years of practice should be evaluated. The relationship between FNPA risk score and baseline weight did not control for parent BMI, a covariate that influenced this relationship in prior studies (9,11). Significant differences between the groups on nearly all characteristics and inconsistent results between the intervention and comparison groups could be explained by lack of random assignment at the clinic or individual level and uncollected variables.

Conclusion

The present study is the first to demonstrate the feasibility of an automated and validated risk assessment in routine paediatric well-child care for preschool-aged and school-aged children. Integrating routine BMI screening with parental assessment of family practices, child behaviours and home environmental risk factors as clinical decision support in the child’s EHR resulted in favourable weight outcomes, particularly among preschool-aged children, when compared with children who received care in the same clinics but who did not have a completed risk assessment. This approach is feasible, useful and sustainable and can be applied to improve childhood obesity prevention efforts in clinical settings.

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Conflict of Interest Statement

The authors declared no conflicts of interest.

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