A Pilot Study of Obesogenic Eating Behaviors in Children With Migraine

Stephen Ray, MRCPCH, MPhil, MBChB, Shashi Bhushan Singh, MPhil, MBChB, Jason C. G. Halford, PhD, Joanne A. Harrold, PhD, and Ram Kumar, MRCP, MA

Abstract

We studied associations between migraine severity and obesogenic eating behaviors in children with a prospective cross-sectional, clinic-based study. Migraine severity was quantified using the PedMIDAS tool and attack frequency. Eating behaviors were assessed using the Dutch Eating Behaviour Questionnaire and the Child Eating Behaviour Questionnaire. Food intake was assessed using a Food Intake Questionnaire. Statistical tests of association between eating behavior, food intake, and adiposity with migraine severity were performed. Sixty children (mean age = 10.9 years, standard deviation = 3.1; 26 males) were recruited. There was a positive correlation between the Child Eating Behaviour Questionnaire desire to drink subscale and PedMIDAS scores (r = 0.41, P = .01). Attack frequency was associated with higher intake of high fat or sugar content food and drink (r = 0.27, P = .04). No association between migraine severity and adiposity was found. Suggestion that migraine severity in children is associated with certain obesogenic eating behaviors requires further large study investigation.

Keywords

pediatric, migraine, eating behavior, obesogenic, nutritional

A large amount of recent research has highlighted an association between migraine and adiposity, in adults and children. Although the association between established obesity and migraine chronification has been studied, there has been no extensive work addressing whether the relationship is bidirectional; whether migraineurs exhibit habitual obesogenic eating behaviors, even before the establishment of obesity.

Excess energy intake, resulting from ingestion of calorie-dense food and drink, is receiving increasing attention as the dominating causal factor in the establishment of obesity in children. There are overlapping neurobiological mechanisms in migraine, eating behaviors, and adiposity. We hypothesize that migraine severity could act via eating behaviors and subsequent caloric intake, leading to increasing adiposity. We aimed to identify whether there are associations between migraine severity and obesogenic eating behaviors in children.

Methods

A single-center, cross-sectional, clinic based pilot study was conducted from August 2009 to August 2010. All participating families provided informed written consent following approval from the local research ethics committee. Participants were screened for recruitment to the study from new referrals to the general pediatric and pediatric neurology outpatient services at our institution from local family physicians for evaluation of headache. The inclusion criteria were as follows: children aged 5 to 16 years inclusive and a diagnosis of migraine supported by ICHD-II criteria. Exclusion criteria were as follows: children receiving treatment from a pediatrician or pediatric subspecialist for a chronic medical condition, current or previous treatment with prophylactic migraine medications, and pre-existing medical condition that was likely to be a cause of secondary headache. The target sample size was 60 migraine cases.

There is no standardized tool for measuring migraine headache intensity. We choose the following ways of measuring migraine severity: (1) the PedMIDAS migraine impact tool and (2) the number of days with acute migraine attacks per month (attack frequency). Eating behaviors were assessed using the Child Eating Behaviour Questionnaire.

1 Department of Neurology, Alder Hey Children’s NHS Foundation Trust, Liverpool, United Kingdom
2 Institute of Infection and Global Health, University of Liverpool, Liverpool, United Kingdom
3 Manchester Royal Infirmary Hospital, Manchester, United Kingdom
4 Department of Experimental Psychology, University of Liverpool, Liverpool, United Kingdom

Corresponding Author:
Stephen Ray, MRCPCH, MPhil, MBChB, Department of Neurology, Alder Hey Children’s NHS Foundation Trust, Eaton Road, Liverpool, L12 2AP, United Kingdom.
Email: dr.stj.ray@gmail.com
Questionnaire for children under 12 years of age (parent report). The Child Eating Behaviour Questionnaire measures tendencies to excessive consumption (food responsiveness, emotional overeating, enjoyment of food, desire to drink), and under-consumption (satiety responsiveness, slowness of eating, emotional undereating, and food fussiness). The Dutch Eating Behaviour Questionnaire was used in children older than 12 years (self-report); Dutch Eating Behaviour Questionnaire—Child form was used in children 7 to 12 years old (self-report).6

Habitual dietary intake was recorded with a Food Intake Questionnaire,9 a food and drink item inventory based on preceding 24-hour period recall of ingestion (self-report). The Food Intake Questionnaire has negative and positive marker food and drink items based on nutritional value and obesogenic tendency. Negative marker items are subdivided into fatty foods (eg, pizza) and sugary foods/drinks. The sum of negative marker items ingested is calculated, with higher scores representing greater intake of high-fat and sugary food and drinks.

There are a number of ways of measuring adiposity; we used body mass index z scores as an indirect measure and weight categories assigned according to International Obesity Task Force centiles.10 The child’s body mass index was calculated from measured height and weight at recruitment. Nonparametric tests of association between the measures of eating behavior, food intake and adiposity with the measures of migraine severity were performed since the data did not show normal distribution. Statistical significance was set at uncorrected P value .05.

Results

Sixty children were recruited (Table 1) with no missing data. With regard to coexistent conditions, one child had attention-deficit hyperactivity disorder (ADHD) and another child had a conduct disorder. Neither had received treatment with medications. Two children had asthma treated with fluticasone inhaler at low dose. Nine children were overweight and 5 were obese according to International Obesity Task Force centiles,10 with no underweight children. We did not demonstrate a significant association between migraine severity and adiposity (Table 2).

Among the eating behavior measures, the desire to drink subscale of the Child Eating Behaviour Questionnaire was positively associated with migraine severity as measured with the PedMIDAS (Spearman rank coefficient, $r = 0.41, P = .01$). The Child Eating Behaviour Questionnaire desire to drink scores in this study ($mean = 2.71, standard deviation = 1.11$) mirrored scores seen in the population in which the questionnaire was validated ($mean = 2.71, standard deviation = 1.1$).6 Migraine severity as measured by monthly attack frequency was associated with increasing Food Intake Questionnaire negative marker food and drink item intake score ($r = 0.27, P = .04$). The Food Intake Questionnaire negative marker food scores in this study ($mean = 0.27, standard deviation = 0.15$) were higher than those seen in the population where the questionnaire was validated ($mean = 0.015, standard deviation = 0.0010$).9 Other eating behavior subdomains of the Child Eating Behaviour Questionnaire, the Dutch Eating Behaviour Questionnaire, and Dutch Eating Behaviour Questionnaire—Child form were not significantly associated with either measure of migraine severity (Table 2).

Discussion

In relation to our study objective, we did detect positive associations of pediatric migraine severity and obesogenic eating behaviors as represented by measures of their desire to drink, and their habitual intake of high-fat or sugar dietary items. We did not detect an association between migraine severity and obesity. A strength of our study is that the children had not been exposed to migraine prophylaxis medications, many of which have effects on appetite and weight.

Food and drink ingestion are behaviors controlled by homeostatic and hedonic mechanisms. The role of hedonic feeding drives, that is, feeding driven by the “liking” of pleasurable taste and “wanting” of such reward, and subsequent increased energy intake in the etiology of obesity in children has been increasingly appreciated.6 The brain’s reward matrix involves structures such as the insular cortex, hypothalamus, and basal ganglia and mediators such as monoamine (dopamine and serotonin) and peptidergic (eg, orexin) neurotransmitters and hormones. Dysfunction of this reward matrix and its mediators is implicated in studies of feeding behavior, obesity, and migraine, implying there may be shared causal mechanisms.2

The desire to drink subscale of the Child Eating Behaviour Questionnaire consists of 3 items: “My child is always asking for a drink”; “If given the chance, my child would drink continuously throughout the day”; and “If given the chance, my child would always be having a drink.” This wording is consistent with interpreting “desire to drink” as a hedonic drive. Hedonic drinking behavior and the ready availability of energy-dense, sugary drinks may contribute to the rising prevalence of obesity in children. Wardle et al4 demonstrated a

<table>
<thead>
<tr>
<th>Variable</th>
<th>Value</th>
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<tbody>
<tr>
<td>Mean Age (y) [SD, range]</td>
<td>10.9 [3.1, 6.3 to 16.1]</td>
</tr>
<tr>
<td>Age: &lt;12 y; ≥12 y, n</td>
<td>37; 23</td>
</tr>
<tr>
<td>Sex: male; female, n</td>
<td>26; 34</td>
</tr>
<tr>
<td>Migraine: with aura; without aura, n</td>
<td>41; 19</td>
</tr>
<tr>
<td>Vomiting during migraine attack, n</td>
<td>32</td>
</tr>
<tr>
<td>Loss of appetite during migraine attack, n</td>
<td>39</td>
</tr>
<tr>
<td>Migraine attack frequency per month: &lt;4; 4-10; &gt;10, n</td>
<td>20; 20; 20</td>
</tr>
<tr>
<td>Mean monthly headache frequency [SD, range]</td>
<td>9 [6, 1 to 30]</td>
</tr>
<tr>
<td>Mean PedMIDAS score [SD, range]</td>
<td>28.8 [32.0, 0 to 161]</td>
</tr>
<tr>
<td>PedMIDAS disability grade categories (score thresholds): little (0-10); mild (11-30); moderate (31-50); severe (&gt;50), n</td>
<td>22; 16; 11; 11</td>
</tr>
<tr>
<td>Mean body mass index z score [SD, range]*</td>
<td>0.62 [1.14, -1.69 to 3.57]</td>
</tr>
<tr>
<td>Weight categories: underweight; normal weight; overweight; obese, n</td>
<td>0; 46; 9; 5</td>
</tr>
</tbody>
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Abbreviation: SD, standard deviation.

*Weight categories and body mass index z scores are based on the International Obesity Task Force age-group centiles.9
significant positive association between the (Child Eating Behaviour Questionnaire) desire to drink subscale score and body mass index z scores in young nonobese children. An increasing desire to drink subscale score may thus indicate an obesogenic tendency in children with increasing migraine severity, although we could not demonstrate a relationship with body mass index z score in our small study. Alternatively, the desire to drink subscale score may reflect homeostatic thirst as a result of a state of underhydration, which may predispose to increased migraine severity. Increasing water consumption has been used as a preventative intervention to reduce migraine severity, although we could not demonstrate a relationship with body mass index z scores in young nonobese children. An alternative explanation is that the desire to drink subscale score may thus indicate an associated with baseline dietary fat intake, which is in line with the findings of our study.\textsuperscript{12} Reductions in dietary fat and carbohydrate intake in these women were associated with decreased headache frequency, which may indicate dietary fat or overall caloric intake as a potentiator of migraine severity. Plausible molecular mechanisms include diet-induced release of key mediators of acute migraine attacks, such as calcitonin gene–related peptide and prostaglandins.\textsuperscript{2}

A limitation of our study is that there are newer validated food intake questionnaires, rather than the Food Intake Questionnaire, that estimate habitual dietary patterns over a week and may have offered a more accurate estimate of dietary patterns in our subjects. Another limitation is the sample size of the study, which was small, because of recruitment of only medication-naïve new-diagnosis migraine patients. A statistical limitation in our methodology is that we performed uncorrected comparisons because of the exploratory nature of the study.

In conclusion, our small study highlights novel associations between migraine and eating behaviors. However, these require confirmation with a larger population-based questionnaire study using robust methods for measuring habitual macronutrient caloric intake in food and drinks, including carbonated and high-fructose drinks. Whether there is a causal relationship linking migraine and eating behavior, and its direction, remains to be clarified. Studies using validated biomarkers of migraine severity, hydration status, adiposity, hedonic feeding drive, and energy expenditure are required. Longitudinal studies to identify the temporal relationship between the onset of migraine in children, increase in adiposity, and eating behaviors along with other potential mediators and confounders are required. The role of macronutrient dietary manipulation for reducing migraine severity, for example, reducing sugary drink intake or adopting a low–glycemic index diet, should be investigated further.

**Author Contributions**

RK, SR, JCGH and JAH conceived the study design. SR and RK wrote the protocol and ethical application. SR and SS conducted recruitment. RK, SS, JCGH, JAH and SR analysed the data. RK, SS and SR wrote the manuscript and all authors contributed to editing the manuscript.
Declaration of Conflicting Interests
The authors declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: JCGH and JAH disclose funding of their Kissileff Laboratory for the Study of Human Ingestive Behavior from the United Kingdom Biotechnology and Biological Sciences Research Council, Medical Research Council–National Prevention Research Initiative, and European Union (EU) Frame Work 7. All other authors report no potential conflict. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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Ethical Approval
The study was approved by the Chester Local Research Ethics Committee, United Kingdom (09/H1017/51). Every participant’s parent or guardian provided informed written consent.

References