

RESEARCH SUBMISSION

Chinook winds and migraine attack onset in children and adolescents: A prospective longitudinal clinical cohort study

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ABSTRACT

Objective: To explore the relationship between migraine attack onset in children and adolescents and Chinooks, which are dry and warm westerly winds that generally occur in the winter and bring about abrupt weather changes to the east of the Rocky Mountains in Southern Alberta, Canada.

Methods: This was a prospective longitudinal clinical cohort study with recruitment from November 2020 to May 2024. Participants were: 8–18 years old, had migraine as per International Classification of Headache Disorders 3rd edition criteria, had 1–15 headache days/month, lived in the geographical location where Chinook winds occur, and had exposure to at least one pre-Chinook or Chinook day during the study period. Chinook days were defined using Nkemdirim's criteria and Environment Canada data were used to categorize day type as either Chinook, pre-Chinook, or non-Chinook. Weather data were merged with data from daily headache diaries, completed for periods of 8–30 days. The primary outcome was attack onset, defined as a day with a new migraine attack of moderate or severe severity, as per the 4-point scale (0 = none, 1 = mild, 2 = moderate, and 3 = severe). Both univariate and adjusted models were used to determine if there was an association between migraine attack onset and day type (i.e., pre-Chinook, Chinook, or non-Chinook) at the aggregate study sample level. The adjusted models controlled for age and sex, and both models included a random intercept. Subsequently, individual $n=1$ models were fitted to explore each individual participant's personal odds of migraine attack onset on both pre-Chinook and Chinook days versus non-Chinook days. Pre-Chinook/Chinook sensitivity values were calculated for each individual by dividing the model's regression coefficient by its standard error. Sensitivity values >1.96 suggest a significant association between pre-Chinook/Chinook days and attack onset.

Abbreviation: PedMIDAS, Pediatric Migraine Disability Assessment Scale

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Results: Sixty youth with 1253 days of complete data, of which 144 (12%) were attack onset days, participated in the study. There were 158 Chinook (13%), 124 pre-Chinook (10%), and 971 non-Chinook days (77%). There were 39 female participants (39 of 60; 65%), with a median age of 14 years (quartile [Q] 1=12, Q3=16), and a median headache frequency of 6.2 days/month (Q1=4, Q3=11). Neither the univariate nor the adjusted models found any significant association between day type and attack onset at an aggregate level (pre-Chinook adjusted odds ratio [OR], 0.98; 95% confidence interval [CI], 0.54–1.78, $p=0.947$; Chinook adjusted OR, 1.15; 95% CI, 0.69–1.91, $p=0.596$). No individual participants met the threshold for statistically significant pre-Chinook or Chinook sensitivity.

Conclusion: We did not find a relationship between pre-Chinook and Chinook conditions and migraine attack onset. This may be due to the lack of an association between Chinooks and attack onset in youth with migraine, or due to a lack of statistical power in our study. Future studies with greater statistical power should aim to assess for a potential relationship between Chinooks and attack onset, as it could have important treatment implications.

Plain Language Summary

In this study, we examined if the onset of migraine attacks occurs more frequently in youth with migraine during Chinooks, which are dry and warm westerly winds that generally occur in the winter and bring about abrupt weather changes to the east of the Rocky Mountains in Southern Alberta, Canada. Sixty (60) youth with migraine completed daily headache diaries for between 8–30 days, and we used Environment Canada data to determine weather conditions, which we categorized as pre-Chinook, Chinook, or non-Chinook days. Results did not support a link between pre-Chinook or Chinook days and attack onset in children and adolescents with migraine; however, studies with larger samples will be needed to continue examining this question.

KEYWORDS

children and adolescents, chinook, migraine, pediatric, pre-chinook, weather

INTRODUCTION

Migraine is a neurological disorder that encompasses a multitude of symptoms, namely frequent recurrent headache attacks, but can also involve nausea, vomiting, and sensitivity to sound and light, among others.¹ In children and adolescents, the prevalence of migraine is between 10%–11%^{2,3} and migraine can have a debilitating impact that affects youth's social, home, and school environments.^{4,5}

The vast majority of people with migraine, inclusive of children and adolescents,^{6,7} report that specific internal and external stimuli can trigger the onset of a migraine attack. However, discrepancies between self-reported triggers and prospective-collected daily diary data are common.^{8,9} Weather conditions and changes are among the most commonly reported triggers in both adults^{8,10} and children and adolescents,^{6,7} with the latter consistently reporting weather-related factors among the top three most common triggers. Studies on sunlight,¹¹ barometric pressure,¹² temperature,¹³ and precipitation¹⁴ suggest that these weather parameters may be

associated with an increased probability of migraine attacks; however, several prospective studies aiming to link weather patterns to attack onset have failed to identify any compelling associations.^{15,16} In addition, it is increasingly understood that some self-reported triggers may actually reflect symptoms of the migraine attack prodrome, and this bias may apply to weather-related triggers as well (e.g., the perception of sunlight as a trigger may reflect prodromal photosensitivity).¹⁷

In southern Germany, Austria, and Switzerland, warm westerly winds known as Föhn winds, which predominantly occur in the winter, are said to bring about a “foehn illness” to their residents, where migraine attacks and other headache types are among several symptoms.¹⁸ Similarly, in Southern Alberta, Canada, Chinooks are dry and warm winds typically occurring in the winter that induce increases in temperature and wind speed, and decreases in humidity and barometric pressure.¹⁹ Specific meteorological criteria, originally proposed by Nkemdirim,²⁰ are typically applied to define Chinook weather. Contrary to weather more broadly, in prospective work,

Chinooks have consistently been found to be associated with increased odds of migraine attack onset in adults, and this relationship appears to strengthen with increased age.^{21,22} To our knowledge, no studies have been conducted to determine whether Chinooks are associated with increased odds of migraine attack onset in children and adolescents. Given that migraine in children and adolescents can diverge from migraine in adults in terms of presentation, treatment, and causes, it is thus uncertain whether the associations between Chinooks and migraine attacks observed in adults may be similarly found in children and adolescents.²³ If Chinooks are associated with increased migraine attack odds in youth, this could potentially lead to using a preventive strategy whereby treatments are taken early before or during Chinooks to mitigate the higher attack risk period. Given that effective migraine treatment can lead to better long-term disease outcomes, understanding and detecting migraine triggers early could lead to improved outcomes.²⁴

In this study, we aimed to determine if there is a temporal association between Chinooks and migraine attack onset in youth. We hypothesized that Chinook weather conditions would increase the odds of experiencing migraine attacks in children and adolescents. Specifically, we hypothesized that days in which Chinook criteria were met would increase the likelihood of same-day migraine attacks compared to days where a Chinook did not occur.

METHODS

This was an observational longitudinal cohort study using prospective headache diary data, entered on a daily basis for periods of 8–30 days by child and adolescent participants with migraine aged

8–18 years old between November 2020 and May 2024. The present study was approved by the Conjoint Health Research Ethics Board at the University of Calgary in Calgary, Alberta, Canada (REB24-0534). Participants had provided written informed consent and/or assent to participate in one of three original studies requiring daily headache diary collection, and a waiver of consent was obtained from Conjoint Health Research Ethics Board for the data to be used for the present study's specific objectives.

Participants

Participants were diagnosed by a neurologist or a nurse practitioner trained in headache medicine, according to the criteria from the third edition of the International Classification of Headache Disorders.²⁵ Participants were eligible for inclusion if they reported a minimum of one attack and a maximum of 15 attacks per month, ensuring that both headache and non-headache days were observed. Participants were also required to have exposure to at least one non-Chinook day and either one Chinook or pre-Chinook day during the data collection period. Monthly headache frequency values were calculated by dividing the number of attacks each participant experienced by the total number of diary days completed by the participant and multiplying by 30. Participants with a headache frequency outside 1–15 headaches/month were excluded to ensure a sample of participants who had both headache and non-headache days observed. Exclusion criteria were significant medical comorbidity (e.g., cancer, systemic lupus erythematosus, etc.), psychotic disorder, significant global developmental delay, moderate–severe intellectual disability, or inability to read or understand English.

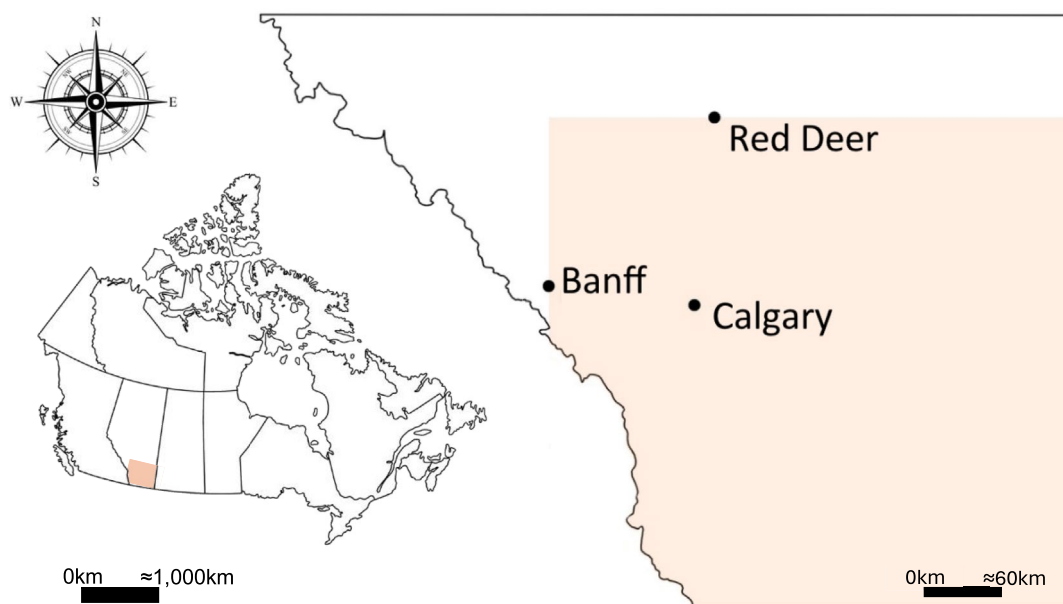


FIGURE 1 Chinook geographical boundaries established for this study.

To define participants at risk of Chinook exposure, we established a geographical location for Chinook occurrence by overlaying two published maps outlining the geographical boundaries of Chinook occurrence.^{26,27} Using these maps, we established the northern, southern, western, and eastern borders for inclusion. Potential participants living outside of these borders were excluded from the study, as determined by each participant's mailing address. Participants living on our defined borders (i.e., Red Deer and Banff) were also excluded from the study. Our chosen borders for the Chinook geographical location were as follows and are illustrated in Figure 1:

1. Northern border: city of Red Deer;
2. Southern border: Alberta-Montana border;
3. Western border: town of Banff; and
4. Eastern border: Alberta-Saskatchewan border.

Headache diaries

Participants completed daily headache diaries for 8–30 days and entered time-stamped data directly into an electronic survey housed in Research Electronic Data Capture software (REDCap, managed by the University of Calgary Clinical Research Unit). Participant-reported daily headache diary data elements included: date and time of headache onset and offset (if applicable), and on days with attacks, pain severity, whether the day was a school day, and whether the attack was associated with aura. Pain severity was measured both using the 3-point scale as per above, and using the 11-point numerical rating scale, where 0 indicates no pain, and 10 indicates the highest possible pain severity. A modified version of the 6-item Pediatric Migraine Disability Assessment Scale (PedMIDAS)²⁸ was also included in the daily diaries to ascertain migraine-related disability on a daily basis. We modified the PedMIDAS for daily use by altering the time frame assessed to the given day, as opposed to the 3-month timeframe in the original scale, as per prior work.²⁹

Outcome

Migraine attack onset was the primary outcome of this study, defined as a day with a new attack graded as moderate or severe on the 4-point severity grading scale (where “0” indicates no pain, “1” indicates mild severity, “2” indicates moderate severity, and “3” indicates high severity). For the purposes of the primary analysis, with the goal of aligning with the methods used in the seminal adult Chinook migraine study,²¹ new moderate–severe attack days were compared to days without attacks. Days with mild severity (severity=1 on the 3-point pain scale) as well as recurrent headache days (defined as headache days following a headache day) were removed from the primary analysis data set. We also performed a sensitivity analysis that included all attack onset days (exclusive of recurrent headache days), regardless of severity.

Primary exposure

The primary exposure of interest was day type in relation to Chinook occurrence, categorized as a Chinook day, a pre-Chinook day, or non-Chinook day. To ascertain the calendar day type for the study period, Environment Canada weather data, collected at the Calgary International Airport, depicting hourly meteorological conditions were extracted for analysis. Weather parameters used to define the day type included hourly temperature (°C), wind speed (km/h), wind direction (tens of degrees), relative humidity (%), and barometric pressure (mm Hg). The onset of a Chinook wind was defined using a modified version of Nkemdirim's criteria,²⁰ as per below:

1. Wind direction between south–southwest and west–northwest inclusive (20°–30° clockwise from true north; in tens of degrees);
2. Wind speeds greater than 15 km/h (noninclusive);
3. At least a 3°C rise in temperature over 1 h in the 24-h period; and
4. Any drop in relative humidity over 1 h.

Each calendar day within the study period was categorized as a pre-Chinook, Chinook, or non-Chinook day using custom code in the Python programming language (version 3.8.5). A pre-Chinook day was defined as the calendar day before a Chinook day. A Chinook day was defined as the calendar day the Chinook criteria were met simultaneously for at least 1 h of the day. A non-Chinook day was defined as any calendar day that could not be classified as a pre-Chinook or Chinook day. Once a Chinook wind had begun, any period ≤2 consecutive hours that did not meet Chinook criteria was still considered part of the Chinook wind.

Covariates

We included age and sex as covariates given their known association with attack frequency. Age was included as a covariate as migraine attack frequency has been shown to change with age.³⁰ Sex was also included as a covariate as studies show females are affected by migraine two to three times more often than males.^{31,32} Females have also been shown to have higher frequency of migraine attacks and more severe migraine attacks than males.³³

Statistical analysis

Our study used a convenience sample and no a priori sample size calculations were completed. Characteristics of participants and day types were summarized with proportions and percentages for categorical data, and with either means and standard deviations or medians and quartiles (Q1, Q3) as applicable based on the distribution of the data. The distributions of continuous outcome data were assessed using histograms, QQ plots, and Shapiro–Wilk tests. Diary days with missing data were excluded from any statistical models.

Mixed effects logistic regression models were used to examine the association between the type of day (pre-Chinook, Chinook, or non-Chinook) and the odds of migraine attack onset. First, univariate models with type of day as the independent variable were created, with inclusion of a random intercept to account for repeated measures within participants. Second, primary adjusted models were created that modeled the odds of attack onset in relation to type of day, while controlling for age and sex as covariates, and with inclusion of a random intercept, to produce the primary results. Model assumptions were verified for the primary model. The assumption of linearity between the continuous covariate age and the outcome was assessed by comparing two nested models: our primary model that assumes a linear relationship between age and the outcome, and a natural cubic spline model where age was given three degrees of freedom in the model (and thus allowed to have a nonlinear relationship to the outcome). These two models were compared using an analysis of variance likelihood ratio test to compare the goodness-of-fit of the two models. We checked for collinearity among the fixed effects variables by fitting a logistic regression model where the odds of attack onset were modeled with the dependent variables type of day, sex, and age, without the random effects included, and we computed variance inflation factors (VIF) and generalized VIF (for the day type variable, given multiple degrees of freedom). We considered VIFs and generalized VIFs >2.5 indicative of possible multicollinearity. We checked for overdispersion using a dispersion ratio test and QQ plots of model residuals. We tested whether a hierarchical model (inclusive of fixed and random effects vs. only fixed effects) was appropriate by inspecting the intraclass correlation (ICC) magnitude for the random effects, and we compared model fit between the fixed effects alone and fixed + random effects using the marginal R^2 (for fixed effects) and the conditional R^2 (for fixed + random effects).

As a sensitivity analysis, we also ran the adjusted mixed effects logistic regression model, with the same methods as above, on a data set where all attack onset days were included, regardless of severity (i.e., inclusive of mild severity days as opposed to only moderate-severe days in the primary models).

Similar to the modeling reported in the seminal Chinook study among adults with migraine,²¹ we also fitted individual logistic regression models to each participant's data to explore personalized odds of attack onset in relation to the type of day. Because these were $n=1$ models, we did not include covariates in them. These models were used to determine pre-Chinook and Chinook sensitivity for each participant, calculated by dividing the regression coefficient by its standard error to produce a Wald statistic. Sensitivity values >1.96 correspond to a statistically significant Wald statistic and would infer that the individual's odds ratio is significantly different from 1 and that the probability of attack onset in the setting of a pre-Chinook or Chinook day is different from 1.

Although we did not conduct a priori sample size calculations, when we noted a null association between our exposure (i.e., day type) and our outcome (i.e., odds of attack onset), we decided to perform *post hoc* power simulations to determine estimates for the study's current power and to ascertain the required sample size,

based on our current data, to detect a statistically significant association at a power (β) of 80% with $\alpha=0.05$. First, we simulated 1000 new data sets based on our current model and ran the model on each of these simulated data sets to estimate current power. We then created power curves to estimate the required sample size to achieve 80% power based on our observed data and effect sizes. We did this by first extending our mixed effects logistic regression model (i.e., increasing the number of participants to $n=2000$). Then we generated 500 simulated data sets (based on our collected data) at prespecified intervals (i.e., between $n=1500$ and $n=2000$ in $n=100$ increments) and refit the model at each interval to understand how power changes at increasing sample sizes. This generated a power curve whereby the estimated power at each sample size, based on the simulations, was visualized. A two-sided α level of 0.05 was considered statistically significant for all analyses. Our analyses were conducted using R³⁴ (version 4.4.2) along with the following R software packages: *lme4* (version 1.1-36), *readxl* (version 1.4.3), *dplyr* (version 1.1.4), *ggplot2* (versions 3.5.1), and *simr* (version 1.0.8).

RESULTS

Data were collected from 111 potential participants for this study. However, as shown in Figure 2, four participants were outside of our defined Chinook region, 29 participants were outside of our monthly headache frequency criteria (i.e., <1 or >15 headache days per month), 15 participants did not have daily data during a pre-Chinook and/or Chinook day, and three participants had only reported mild attacks. The final sample for the primary analysis included data from $n=60$ participants, with a total of 1253 eligible headache diary days (mean number of completed diary days per participant = 25.2, range = 8–30), of which 12% (144 of 1253) were moderate-severe attack onset days and the remainder were days with no headache (88%; 1109 of 1253). Of the attack onset days, 76% (110 of 144) were rated as moderate severity and 24% (34 of 144) were rated as severe (days with mild attacks were excluded from the present analysis). For type of day, 13% (158 of 1253) were Chinook days, 10% (124 of 1253) were pre-Chinook days, and 77% (971 of 1253) were non-Chinook days.

The majority of the participants were female (65%; 39 of 60), the median age was 14 years (Q1 = 12 year, Q3 = 16 years), and the median headache frequency was 6.21 days/month (4 days/month, 11 days/month). Thirty-seven participants lived within the Calgary city limits (37 of 60; 62%), with the remaining 23 participants living within the Chinook geographical borders but outside of Calgary's city limits (23 of 60; 38%). Further descriptive statistics for the included participants across all headache and Chinook day types (i.e., before any filtering of the data) are summarized in Tables 1 and 2. The proportion of attack onset days by day type (Chinook, pre-Chinook, or non-Chinook) is illustrated in Figure 3.

In a univariate unadjusted model, there was no significant association between attack onset and type of day (pre-Chinook day odds ratio [OR], 1.00; 95% confidence interval [CI], 0.55–1.81, $p=0.993$;

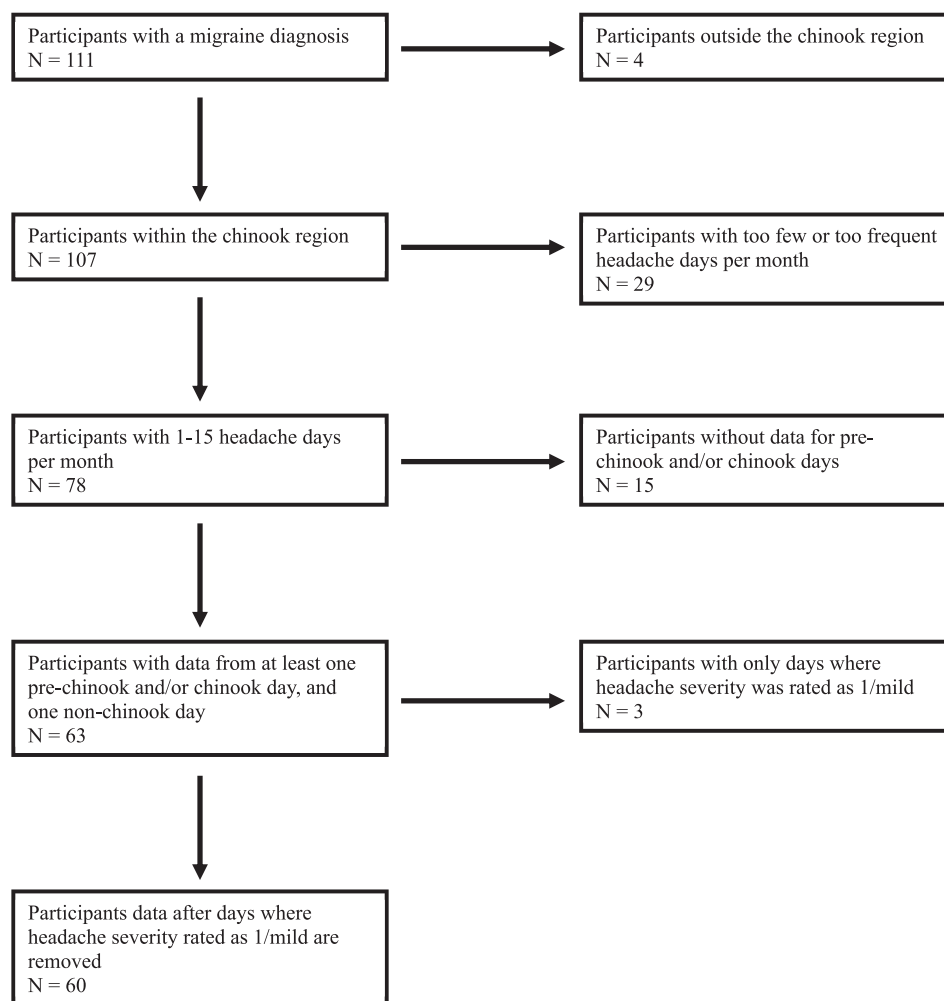


FIGURE 2 Participant recruitment flow chart.

Chinook day OR, 1.17; 95% CI, 0.70–1.95, $p=0.541$). The random effects variance (σ^2) was 3.29, with an ICC of 0.04.

In the primary adjusted model, the odds of attack onset were not significantly associated with pre-Chinook days (adjusted OR [aOR], 0.98; 95% CI, 0.54–1.78, $p=0.947$) nor with Chinook days (aOR, 1.15; 95% CI, 0.69–1.91, $p=0.596$) compared to non-Chinook days, after adjusting for age and sex. The random effects variance in the primary model was 3.29, with an ICC of 0.02. The results of the mixed effects adjusted logistic regression model are summarized in Table 3. Model assumptions were confirmed with no violations observed. For the assumption of linearity between age and the outcome, the analysis of variance likelihood ratio test comparing our primary model to the model where age was given three degrees of freedom (i.e., natural cubic splines model) was nonsignificant ($\chi^2=0.37$, $df=2$, $p=0.832$), suggesting that the splined model did not improve fit over the linear model. Variance inflation factors and generalized VIFs for the fixed effects model were all ≤ 2.5 , indicating no concerns with collinearity. The dispersion ratio of our primary model (value=0.990, $p=0.872$) and the QQ plot of the residuals did not suggest any problems with overdispersion in the model. Although the ICC value for the random effects was

relatively low (0.02), the conditional R^2 value (fixed + random effects) was slightly larger than the marginal R^2 value (fixed effects; conditional $R^2=0.046$ vs. marginal $R^2=0.023$), indicating that the random effects model provided a better fit to our data than the nonhierarchical fixed effects model. Notably, the R^2 values suggest that a low proportion of the variance in the odds of attack onset was explained by variables in our model.

In the sensitivity analyses where all attack onset days were included regardless of severity (i.e., new mild attacks included as well), 63 participants contributing 1426 headache diary days were included. Within the sensitivity analysis population, 240 days were attack onset days (240 of 1426; 17%), of which 40% were mild attack days (96 of 240), 46% were moderate attack days (110/240), and 14% were severe attack days (34 of 240). Of the days in the sensitivity analysis population, 13% (180 of 1426) were Chinook days, 10% were pre-Chinook days (145 of 1426), and 77% were non-Chinook days (1101 of 1426). Table S1 provides further details about the sensitivity analysis population. Again, as observed in the primary model, no association was found between the odds of attack onset and day type in the sensitivity analysis for pre-Chinook days (aOR, 0.99; 95% CI, 0.62–1.59, $p=0.975$) nor for Chinook days (aOR, 1.14; 95% CI,

TABLE 1 Participant baseline characteristics (n=60).

Characteristic	n (%)
Sex	
Male	21 (35%)
Female	39 (65%)
Gender	
Boy	20 (33%)
Girl	35 (58%)
Gender diverse	3 (5%)
Unknown	1 (2%)
Prefer not to answer	1 (2%)
Age (median, Q1, Q3)	14 (12, 16)
Age range, years	9–18
Headache frequency in days/month (median, Q1, Q3)	
All attack days	6.21 (4, 11)
New attack days	4.9 (3, 6.6)
Baseline aura	
No	39 (65%)
Yes	21 (35%)
Baseline PedMIDAS score (median, Q1, Q3)	19 (8, 40.5)
Baseline PedMIDAS grade	
1	23 (38%)
2	17 (28%)
3	9 (15%)
4	11 (19%)

Abbreviations: PedMIDAS, Pediatric Migraine Disability Assessment Scale; Q, quartile.

0.76–1.73, $p=0.524$), after adjusting for age and sex. The full results of the sensitivity analysis model are included in Table 3.

In the individualized $n=1$ models, there were no participants with pre-Chinook nor Chinook sensitivity Wald statistic values >1.96 , indicative that none of the $n=1$ models were statistically significant (median pre-Chinook sensitivity value = 1.17, $Q1=1.08$, $Q3=1.37$; median Chinook sensitivity value = 1.19, $Q1=1.14$, $Q3=1.33$). Figure 4 illustrates the pre-Chinook and Chinook sensitivity values derived from taking the individual model's regression coefficients and dividing them by their standard errors.

In our *post hoc* power simulations with $n=1000$ simulations, we estimated that our power to detect a statistically significant association between Chinook days and attack onset was 8.2% (95% CI, 6.6–10.1), given our very small observed z-test effect size of 0.14. For the association between pre-Chinook days and attack onset, we estimated that our power to detect a statistically significant association was 5.7% (95% CI, 4.4–7.3), given our very small observed z-test effect size of -0.02 . Next, we used simulations to generate power curves to estimate the sample required to achieve 80% power for these associations. We found that we would need a sample size of $n=1700$ to establish a statistically significant association between Chinook days and attack onset at the observed effect size of 0.14. We did not

TABLE 2 Participant and weather characteristics across all diary days (1519).

Characteristic	n (%)
Attack day types	
All attack days	361 (24%)
New attack days	237 (66%)
Consecutive attack days	92 (25%)
Not classified ^a	32 (9%)
No attack	1109 (73%)
Missing/not answered	49 (3%)
Headache severity rating	
All attack days	
Mild	134 (37%)
Moderate	170 (47%)
Severe	56 (15%)
Missing/not answered	1 (1%)
New attack days	
Mild	92 (39%)
Moderate	110 (46%)
Severe	34 (14%)
Missing/not answered	1 (1%)
Headache days with aura	
All attack days	
No	315 (87%)
Yes	42 (12%)
Missing/not answered	4 (1%)
New attack days	
No	206 (87%)
Yes	29 (12%)
Missing/not answered	2 (1%)
PedMIDAS score (0 to 6; median, Q1, Q3)	
All attack days	0 (0, 1)
Missing/not answered	3 (1%)
New attack days	0 (0, 1)
Missing/not answered	2 (1%)
Day type	
Pre-Chinook day ^b	147 (10%)
Chinook day	187 (12%)
Non-Chinook day	1177 (77%)
Date missing ^c	8 (1%)

Abbreviations: PedMIDAS, Pediatric Migraine Disability Assessment Scale; Q, quartile.

^aWe could not determine new or continuous headache days if we did not have headache data for the preceding day (e.g., first day of diary was an attack day).

^bWe have fewer pre-Chinook days compared to Chinook days because a single pre-Chinook day would precede one, or a series of consecutive, Chinook days.

^cMissed diaries or those where a date was not provided could not have their Chinook status calculated.

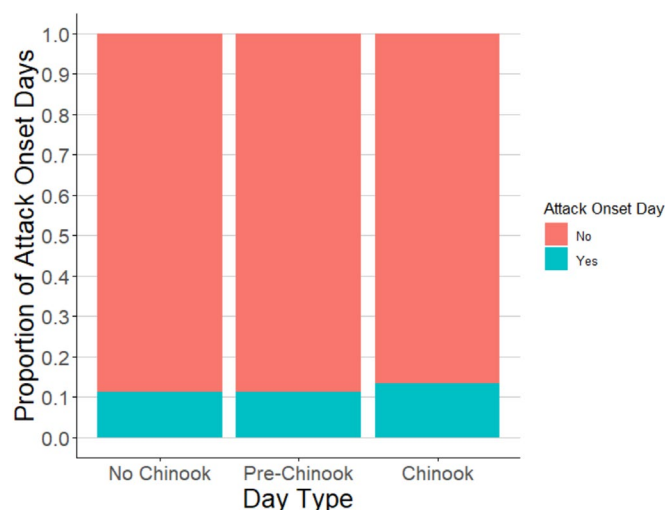


FIGURE 3 Proportion of attack onset and no attack days across Chinook weather days.

achieve 80% power for the association between pre-Chinook and non-Chinook days when extending our model to $n=2000$ participants. We then tried extending the model to $n=10,000$ participants but we were only able to achieve a power of 12.8% for these associations, with an observed effect size of -0.02 .

DISCUSSION

In this study, we did not find an association between Chinook days and pre-Chinook days and migraine attack onset in children and adolescents in aggregate models, in sensitivity analyses, nor in $n=1$ individualized models. These findings contrast our hypothesis that Chinooks increase the likelihood of migraine attack onset in children and adolescents.

Weather and migraine in adults

Most of the literature determining the association between weather conditions and migraine attack onset comprises studies performed on adults. Moreover, this literature contains contradictory findings. For example, although some studies have found that changes in temperature,¹³ sunlight,¹¹ and barometric pressure¹² are associated with migraine attack onset, other studies have not borne out significant associations between weather parameters and migraine attacks.¹⁵ The association between the onset of migraine attacks and Chinook wind conditions has only been studied in adults to date, in two seminal prospective observational studies. The first was a small study of 13 adults with migraine who experienced 369 attack onsets during the study period.²² Overall, the authors described a significant association between Chinook and pre-Chinook days and the probability of attack onset after controlling for age, sex, and season. When they stratified by age, only the older participants showed a significant association, with those <43 years not exhibiting a statistically significant effect. A second, larger prospective study with similar aims included 75 adults

TABLE 3 Results of mixed effects logistic regression models examining the association between migraine attack occurrence and Chinook days.

Variable	OR	95% CI	p value
Univariate model			
Type of day (referent = non-Chinook day)			
Pre-Chinook	1.00	0.55–1.81	0.993
Chinook	1.17	0.70–1.95	0.541
Random effects			
Variance (σ^2) = 3.29			
ICC = 0.04			
Adjusted model			
Type of day (referent = non-Chinook day)			
Pre-Chinook	0.98	0.54–1.78	0.947
Chinook	1.15	0.69–1.91	0.596
Age, years	0.97	0.89–1.05	0.431
Female sex (referent = male)	1.79	1.16–2.77	0.009 ^a
Random effects			
Variance (σ^2) = 3.29			
ICC = 0.02			
Sensitivity analysis (adjusted model with all attack severities)			
Type of day (referent = non-Chinook day)			
Pre-Chinook	0.99	0.62–1.59	0.975
Chinook	1.14	0.76–1.73	0.524
Age, years	1.00	0.94–1.07	0.926
Female sex (referent = male)	1.40	1.00–1.97	0.053
Random effects			
Variance (σ^2) = 3.29			
ICC = 0.02			

Abbreviations: CI, confidence interval; ICC, intraclass correlation; OR, odds ratio.

^aIndicates statistical significance at $p < 0.05$ level.

with migraine who had 2381 attack onsets occur during the study period.²¹ Similar to the original smaller Chinook study, the authors found that both Chinook and pre-Chinook days were associated with increased odds of attack onset, and that older adults (≥ 50 years) were much more likely to exhibit sensitivity to Chinook winds as compared to younger adults. Although we did not find that any of the participants in our pediatric study were Chinook/pre-Chinook sensitive, the adult study that estimated proportions of Chinook wind sensitive adults reported that 18% of those 16–49 years and 40% of those ≥ 50 years were sensitive to these weather conditions.²¹ These data suggest that age may modify the association between Chinook wind sensitivity and attack onset among people with migraine. Methodologically, our study appears to have been underpowered to detect a significant association in our younger participants, due to very small estimated effect sizes (i.e., effect size of 0.14 between Chinook days and odds

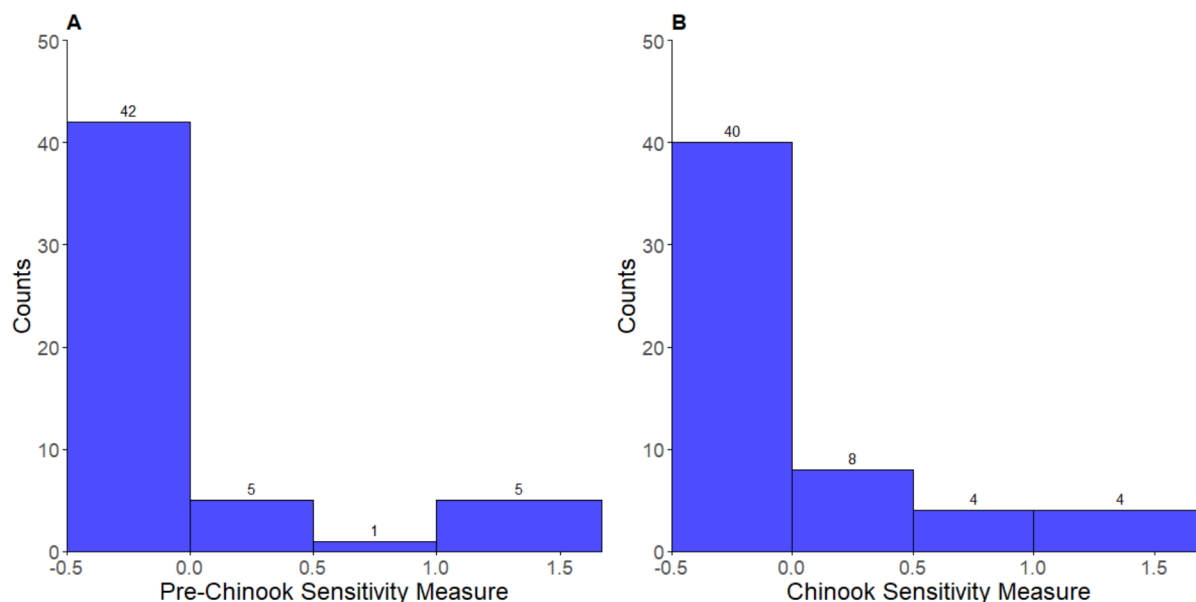


FIGURE 4 Histogram of individual (A) pre-Chinook ($n=53$) and (B) Chinook ($n=56$) sensitivity values comparing non-attack days and days with new attack occurrence. Pre-Chinook and Chinook sensitivity measures >1.96 represent a significant Wald statistic. Such values suggest an individual's odds ratio is significantly different from 1 and that their probability of experiencing a migraine attack on (A) pre-Chinook or (B) Chinook wind days is significantly different from 1.

of attack onset, and effect size of -0.02 between pre-Chinook days and attack onset). Despite having a comparable sample size to the published adult Chinook studies, the number of observed attacks (outcomes) in our study was also relatively low (144 days moderate-severe new attack days, 236 new attack days of any severity vs. 369²² and 2381 days²¹ in the adult studies). To avoid concerns with statistical power, future studies among children and adolescents with migraine will require larger sample sizes (estimated at $n=1700$ based on our power curve simulations) and/or longer observation periods to examine this question effectively. However, our data suggest that the effect sizes may be smaller than what would be deemed clinically significant, given that effect sizes <0.2 are typically considered very small. This begs the question of whether the Chinook-attack onset phenomenon is worthy of further study in children and adolescents, as it may be too small to be clinically meaningful.

Weather and migraine in children and adolescents

Although to our knowledge, no prior studies have aimed to determine the association between Chinook wind conditions (or other wind phenomena such as Foehn winds) and migraine attack onset in children and adolescents, there is a broader literature that has aimed to link other weather conditions to attacks in young people. As has been observed in the adult migraine literature, findings from these studies have been mixed. Although in one study, youth commonly retrospectively reported that sunlight could provoke the initiation of their attacks,¹⁴ in another study that used prospective data from diaries administered three times per day, there was no significant association between attack onset and sunlight.³⁵ The effect of temperature

on attack onset has also been examined and has yielded inconsistent results. In one study examining the probability of pediatric emergency department visits for headache and weather parameters, higher temperatures were associated with higher volumes of visits.³⁶ However, a prospective diary study in children and adolescents with chronic primary headaches did not find an association between temperature and attack onset.³⁵ In the two studies that have aimed to determine the relationship between relative humidity, precipitation, and attack onset in youth, both the study where youth retrospectively reported on their triggers¹⁴ and the prospective diary study³⁵ have found that youth with primary headaches may be more likely to have attacks when there is precipitation or an increase in relative humidity. Chinooks result in both a decrease in relative humidity and a rapid increase in temperature, and thus they comprise a combination of individual meteorological parameters that are often examined separately and may be predicted to differentially impact attack onset probability based on the sparse available literature. Chinooks are also associated with high wind speeds in the context of a decrease in barometric pressure, the impact of which has not been well studied among children and adolescents with migraine.

Potential mechanisms linking Chinooks to migraine attack onset

As above, it remains unclear which, if any, meteorological parameters impact the risk of attack onset in people with migraine. It is also unclear how meteorological parameters could decrease the threshold to attacks. It is likely that mechanisms would vary based on the parameter in question. For Chinooks, the most plausible

meteorological parameters that could trigger attacks are the associated drop in barometric pressure or the rapid increase in temperature. Animal work suggests that a drop in barometric pressure leads to changes in the activity of second order neurons in the superior vestibular nucleus.³⁷ This is hypothesized to occur via a barometric sensor in the inner ear, whereby the middle ear cavity may transmit signals about pressure changes in the environment to the perilymph thereby modulating vestibular nerve activity. In a neuropathic rat model, low barometric pressure has also been shown to exacerbate allodynia and hyperalgesia, with sympathectomy inhibiting the pain aggravation from low barometric pressure.³⁸ It has therefore been hypothesized that the increased superior vestibular nucleus activity in response to low barometric pressure may modulate pain through sympathetic nervous system activation. However, recent work in a mouse model of vestibular migraine has elucidated a pathway from the vestibular nuclei to the spinal trigeminal nucleus via the vestibulocerebellum³⁹; if such a pathway exists in humans, barometric pressure changes may have the ability to modulate incoming migraine pain signals from the trigeminal ganglion at the level of the spinal trigeminal nucleus through this pathway. Interestingly, vestibular function deteriorates with age in humans.⁴⁰ Perhaps this explains why our study was negative and prior Chinook studies in adults with migraine^{21,22} showed a positive association: if a deterioration in vestibular function strengthens the association between barometric pressure changes and pain in central pathways, then we may not observe this effect in children and adolescents. How a rise in temperature could lead to attack onset is also unclear. As the thermoregulatory center of the brain, the hypothalamus is also among the first brain regions to become active in the migraine prodrome and is hypothesized to play a key role in triggering attacks⁴¹⁻⁴⁴; it is therefore possible that a rapid rise in temperature could set off attacks through hypothalamic activation.

Study limitations

Our study, although novel in its aims and executed prospectively with careful linkage between objective Environment Canada data and clinical diary data, has several limitations. We did not conduct a priori sample size calculations, but our *post hoc* power calculations and power curve simulations suggest that we would need a much larger sample size ($n=1700$) and/or a longer observation period with the daily diaries to accurately determine the association of Chinooks and migraine attack onset in youth at the aggregate level. This lack of power is due to very small estimated effect sizes from our data, which does put into question whether a potential association between Chinook winds and attack onset is clinically meaningful or not in this age group. It is also possible that our study had some measurement bias in it, given that we determined participant location through their mailing addresses to ensure alignment with Chinook exposure borders but were unable to account for travel that could have decreased their exposure to Chinooks (i.e., if they were traveling outside of Chinook borders on particular study days).

Measurement of Chinook weather conditions occurred in Calgary and was extrapolated to a wider geographic area. Thirty-eight percent of participants lived outside of Calgary borders within the Chinook exposure region, but it is possible that their intensity of exposure to Chinooks was lesser, which could have biased our results toward the null. Our sample comprised children and adolescents with migraine who were being seen in tertiary care headache clinics in Calgary, Alberta, Canada. Thus, our sample was likely skewed toward participants with higher migraine-related disability and results must be interpreted in this context as they are not generalizable to all youth with migraine. We only assessed the relationship between pre-Chinook days immediately preceding the first Chinook day and Chinook days in relation to attack onset; therefore, we were unable to capture more nuanced or lagged effects that Chinook conditions may have on attack onset and severity over time. Future studies could apply time series-based models to overcome this limitation. We did not explore any other purported migraine triggers (e.g., stress, reduced sleep quality, etc.) in our analysis, and there are therefore many potential confounders that we did not include, which aligns with the fact that our model explained only a small proportion of the variance in attack onset odds. Last, we did not parse out the individual features of Chinooks that could act as migraine attack triggers, such as increased temperature, decreased barometric pressure, and decreased relative humidity. Rather, we only examined Chinook day and pre-Chinook days versus non-Chinook days.

CONCLUSION

In this study, we did not find an association between Chinook wind conditions and attack onset in children and adolescents with migraine at a population or at an individual level. Future studies should aim to revisit this question in larger samples of youth with longer observation periods, such that a larger number of attacks can be captured. Future studies could also parse out individual features of Chinook conditions that may be associated with attack onset (e.g., temperature, barometric pressure, etc). Importantly, should future studies find that a proportion of youth are sensitive to Chinook wind or other weather conditions, this could open the door to work examining the efficacy of taking acute treatment on days with high risk weather conditions to prevent attack onset.

AUTHOR CONTRIBUTIONS

Rylan Heart Villaruz: Writing – original draft; writing – review and editing; data curation; formal analysis; methodology; conceptualization. **Jonathan Kuziek:** Writing – review and editing; writing – original draft; methodology; formal analysis; supervision; data curation; conceptualization. **Kirsten Sjonnesen:** Writing – review and editing; methodology. **Lindsay Craddock:** Writing – review and editing; resources. **Werner J. Becker:** Writing – review and editing; conceptualization; methodology. **Ashley D. Harris:** Writing – review and editing; conceptualization; methodology; supervision. **Serena L. Orr:** Writing – original draft; writing – review and editing;

conceptualization; methodology; investigation; funding acquisition; supervision; project administration; formal analysis; data curation; resources.

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CONFLICT OF INTEREST STATEMENT

Werner Becker has served on medical advisory boards for Lundbeck, Teva, Pfizer, Allergan, AbbVie and Novartis, and have received speaker's honoraria from Pfizer. **Ashley D. Harris** reports research funding from the Canadian Institutes of Health Research and holds a Canada Research Chair in MR Spectroscopy in Brain Injury and Pain. **Serena L. Orr** reports royalties from Cambridge University Press. She serves on the editorial boards of *Headache*, *Neurology*, and the American Migraine Foundation. She also has research funding from the Canadian Institutes of Health Research, the Alberta Children's Hospital Research Institute, and the American Headache Society. Dr. Orr has received compensation from the Université de Sherbrooke for delivering a rounds presentation. **Rylan Heart Villaruz**, **Jonathan Kuziek**, **Kirsten Sjonnesen**, and **Lindsay Craddock** have no relevant conflicts to disclose.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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