Caffeine Intake in Eating Disorders

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\textbf{ABSTRACT}

\textbf{Objective:} The current study compares caffeine consumption in females with an eating disorder and females without an eating disorder.

\textbf{Method:} Caffeine intake in three diagnostic groups (10 females with anorexia nervosa, 27 females with bulimia nervosa, and 42 females with binge eating disorder [BED]) was compared with caffeine intake in three comparison groups \((n = 659 \text{ each}). Data were obtained from a longitudinal study of Black and White girls. Three-day food records were examined for the years before the onset of the eating disorder, the onset year, and the years after the onset of the eating disorder. Data from the same years were used for the comparison groups.

\textbf{Results:} Caffeine intake increased over time between ages 9 and 19 years across all groups and this trend was not moderated by diagnostic status. For anorexia nervosa, relative to the non-eating disorder group, the proportional intake of caffeine from soda increased significantly before onset to onset to after onset and ingestion of chocolate-containing foods decreased sharply over time.

\textbf{Conclusion:} Caffeine consumption in young girls with eating disorders differs from girls with no eating disorders only for anorexia nervosa, but not for bulimia nervosa or BED. © 2005 by Wiley Periodicals, Inc.

\textbf{Keywords:} caffeine consumption; food records; eating disorders; anorexia; bulimia

\textit{(Int J Eat Disord 2006; 39:162–165)}

Introduction

Clinical accounts report that individuals with anorexia nervosa (AN) or bulimia nervosa (BN) consume caffienated beverages excessively to boost their energy without the unwanted effect of consuming calories.\textsuperscript{1} In patient samples, caffeine use was correlated with binge frequency and purging status.\textsuperscript{2–4} The current study tested the hypothesis that caffeine intake in females with AN, BN, or binge eating disorder (BED) is elevated compared with females who do not have an eating disorder.

\textbf{Methods}

\textbf{Participants}

The sample included 2,054 females. Of these, 10 met criteria for AN, 27 for BN, and 42 for BED. Those with no eating disorder (1,977) were sorted randomly into comparison groups of 659 each (one for each eating disorder).

As described previously,\textsuperscript{5} in 1987, the National Growth and Health Study (NGHS) recruited 1,213 Black and 1,166 White girls who were 9 or 10 years old and were assessed annually for 10 years. The NGHS-Wave II was initiated in 1998 to examine risk factors for eating disorders and included 86% of the original NGHS sample.\textsuperscript{6}

\textbf{Instruments and Procedure}

Age was measured as age at last birthday and race was based on self-reported identification at study entry. Body weight and height were measured annually by research...
staff. Body mass index (BMI) was calculated by dividing weight (kg) by squared height (meters).

Caffeine intake was extracted from annual food records for Years 1-5 and Years 7, 8, and 10. By a previously validated procedure, trained and certified dieticians instructed girls to record all food and drink for 3 consecutive days and reviewed these individually with the girls. Food records were coded using Food Table Version 19 of the nutrition coordinating center (NCC) nutrient database.

Sources of caffeine were coffee/tea, soda, and chocolate-containing food (chocolate foods). Six caffeine intake variables were calculated for each girl, for each visit, and intake was averaged across the three food records: daily caffeine intake (in milligrams), daily caffeine intake per kilogram of body weight during that visit, daily caffeine intake per kilocalories, and proportion of caffeine intake from each of the three categories of caffeinated foods. Before onset data and after onset data were averaged across all visits.

Eating disorder diagnoses were based on a telephone screening interview and (if positive) a subsequent in-person diagnostic interview during NGHS-Wave II. Onset age was the age when all criteria for AN, BN, or BED were first met.

**Statistical Methods**

For computing comparable dietary measures before onset, at year of onset, and after onset, comparison girls were assigned an onset age by randomly sorting them into one of approximately equal subgroups (n = 659), corresponding to the number cases with AN, BN, or BED. All girls in the subgroup were assigned the same onset age as the corresponding eating disorder case.

A separate generalized estimating equations (GEE) model was constructed for each caffeine intake variable and each eating disorder group. These models tested differences related to group, time, and Group × Time interactions and adjusted for race and study center.

This research was reviewed and approved by an institutional review board.

**Results**

Table 1 displays mean scores (and standard deviations) of caffeine intake variables for the entire sample at each age and thus provides age-specific reference points against which to interpret the study findings regarding group differences based on time periods focused on onset age.

Comparisons of caffeine intake in girls with AN and their comparison group (Table 2) found no significant main effect for diagnostic group, $\chi^2(1) = 0.01-0.41$, $ps > .50$; and, with two exceptions, no significant Group × Time interaction, $\chi^2(2) = 0.48-3.58$, $ps > .15$. Across groups, caffeine intake increased over time, $\chi^2(2) = 16.9-72.9$, $ps < .0005$; except relative caffeine intake from coffee/tea, $\chi^2(2) = 3.88$, $p > .10$.

In the AN group, relative caffeine intake from soda increased sharply from 25% before onset to 54% during the onset year and 65% after onset, with a more gradual increase over time in the comparison group. Caffeine intake from chocolate foods decreased sharply before onset to onset and after onset in the AN patients. It decreased less markedly in the comparison group, $\chi^2(2) = 17.8$ and 11.4, respectively, $ps < .005$.

Caffeine intake indices were similar in the BN and comparison groups (data available upon request) at the before onset visits and were generally higher in the BN group at the onset visit and

**TABLE 1.**  Mean (standard deviation) of caffeine intake by age at last birthday for all eating disorder cases and the comparison groups combined

<table>
<thead>
<tr>
<th>Age</th>
<th>Average Daily Caffeine Intake (mg)</th>
<th>Average Daily Caffeine Intake (mg)/Caloric Intake</th>
<th>Average Daily Caffeine Intake (mg)/Weight (kg)</th>
<th>Proportion of Caffeine from Coffee/Tea</th>
<th>Proportion of Caffeine from Soda</th>
<th>Proportion of Caffeine from Chocolate Foods</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>16.2 (16.5)</td>
<td>0.01 (0.01)</td>
<td>0.48 (0.50)</td>
<td>0.06 (0.19)</td>
<td>0.30 (0.35)</td>
<td>0.64 (0.37)</td>
</tr>
<tr>
<td>10</td>
<td>18.2 (19.1)</td>
<td>0.01 (0.01)</td>
<td>0.47 (0.50)</td>
<td>0.06 (0.19)</td>
<td>0.35 (0.37)</td>
<td>0.59 (0.39)</td>
</tr>
<tr>
<td>11</td>
<td>22.2 (20.6)</td>
<td>0.01 (0.01)</td>
<td>0.50 (0.49)</td>
<td>0.07 (0.20)</td>
<td>0.40 (0.38)</td>
<td>0.53 (0.39)</td>
</tr>
<tr>
<td>12</td>
<td>26.9 (23.2)</td>
<td>0.02 (0.02)</td>
<td>0.54 (0.49)</td>
<td>0.07 (0.20)</td>
<td>0.51 (0.39)</td>
<td>0.43 (0.38)</td>
</tr>
<tr>
<td>13</td>
<td>29.8 (25.1)</td>
<td>0.02 (0.02)</td>
<td>0.54 (0.46)</td>
<td>0.07 (0.20)</td>
<td>0.55 (0.39)</td>
<td>0.39 (0.38)</td>
</tr>
<tr>
<td>14</td>
<td>33.4 (29.3)</td>
<td>0.02 (0.02)</td>
<td>0.57 (0.51)</td>
<td>0.09 (0.23)</td>
<td>0.59 (0.39)</td>
<td>0.33 (0.37)</td>
</tr>
<tr>
<td>15</td>
<td>40.3 (36.0)</td>
<td>0.02 (0.03)</td>
<td>0.67 (0.63)</td>
<td>0.11 (0.25)</td>
<td>0.55 (0.39)</td>
<td>0.34 (0.37)</td>
</tr>
<tr>
<td>16</td>
<td>45.1 (43.6)</td>
<td>0.03 (0.04)</td>
<td>0.73 (0.73)</td>
<td>0.12 (0.27)</td>
<td>0.57 (0.39)</td>
<td>0.31 (0.37)</td>
</tr>
<tr>
<td>17</td>
<td>50.2 (50.9)</td>
<td>0.03 (0.04)</td>
<td>0.78 (0.81)</td>
<td>0.15 (0.30)</td>
<td>0.58 (0.40)</td>
<td>0.26 (0.36)</td>
</tr>
<tr>
<td>18</td>
<td>56.7 (57.7)</td>
<td>0.03 (0.04)</td>
<td>0.90 (0.99)</td>
<td>0.18 (0.31)</td>
<td>0.56 (0.39)</td>
<td>0.26 (0.35)</td>
</tr>
<tr>
<td>19</td>
<td>62.5 (68.6)</td>
<td>0.04 (0.06)</td>
<td>0.93 (1.08)</td>
<td>0.17 (0.31)</td>
<td>0.60 (0.40)</td>
<td>0.23 (0.34)</td>
</tr>
</tbody>
</table>
The current study compared caffeine intake in females with a diagnosis of AN, BN, or BED and females with no history of an eating disorder, adjusting for the potentially confounding effect of differences in the demographic composition of the groups. Limitations include the small number of eating disorder cases, the use of 3-day diaries, the omission of diaries in Years 6 and 9, and the determination of onset age by retrospective assessment. Strengths include the use of a matched comparison group and the consideration of several measures of caffeine intake separately for before and after onset as well as for the year of onset. The prospective nature of the data, obtained from a community sample, offers a unique contribution to the literature.

Regardless of diagnostic status, all indicators of caffeine intake tended to increase over time, except relative intake from chocolate-containing foods, which tended to decrease. This is consistent with results from large population-based studies, which show that caffeine intake (and caffeine intake proportionate to body weight or total daily caloric intake) increases as children get older.10

The hypothesized greater caffeine intake in AN was not statistically significant, although an inspection of results shows that caffeine intake increased more in the AN group than in the comparison group. We did observe a significantly greater proportional increase in caffeine intake from soda with a corresponding significantly greater decrease of caffeine intake from chocolate-containing foods. We found no support for the hypothesized elevated caffeine intake in BN or BED patients.

Conclusion

After onset visits. However, these Time x Group effects were not statistically significant, $\chi^2(2) = 0.64-3.46$, $p > .15$; nor were there significant main effects for group, $\chi^2(1) = 0.11-3.18$, $p > .10$. Across groups, all caffeine indicators (except for intake from chocolate foods, which decreased) reflected increased intake over time, $\chi^2(2) = 6.37-33.8$, $p < .05$.

Comparisons of caffeine intake in BED versus the comparison group (data available upon request) found, for all variables, significant effects for time, $\chi^2(2) = 13.9-49.9$, $p < .001$; but not group, $\chi^2(2) = 0.01-3.60$, $p > .05$; or the Group x Time interactions, $\chi^2(2) = 0.37-2.09$, $p > .35$.

Note: The mean age of onset of AN was 14.9 years (SD = 3.7). The comparison group was assigned an age of onset to match the onset distribution in the AN group. The AN and comparison groups differ in their ethnic composition. Therefore, means (proportions) are shown for each ethnic group separately for descriptive purposes. In analyses comparing the two groups, race and study center were entered as covariates to adjust for the potentially confounding effect of demographic or regional differences in caffeine intake. There were no significant Race x Group interactions.
Future research might explore the role of caffeine in eating disorder symptomatology by asking specifically whether caffeine is used to decrease appetite, boost energy, or for other reasons. Moreover, as was the case in previous studies of caffeine intake in eating disorders, caffeine intake was based only on consumption of foods and beverages, leaving for future studies the question of whether medications containing caffeine might represent an important source of caffeine intake in eating disorders.

References
