Shift in Diurnal Feeding Patterns in Nursing Home Residents With Alzheimer’s Disease

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Background. Individuals with Alzheimer’s disease (AD) are highly susceptible to weight loss and malnutrition, which, to date, have not been associated with decreased food consumption. The current study examined food intake patterns and how they change in relation to body mass index (BMI), behavioral function, and cognitive status in institutionalized seniors with AD.

Methods. Twenty-one consecutive days of investigator-weighed food intake collections were conducted on 25 subjects with likely AD residing at a home for the aged. All subjects maintained the ability to self-feed.

Results. Eighty-eight percent of participants did not meet targeted energy needs, including an estimated 37% prevalence of protein inadequacy. Subjects with increased behavioral difficulties, based on the London Psychogeriatric Rating Scale, had reduced meal-related intakes that were highly associated with decreased energy consumption at dinner. With behavioral changes, particularly increased mental disorganization and confusion, there was a shift in circadian eating patterns such that the greatest proportion of daily energy was consumed at breakfast. Individuals with low BMIs tended to be those with more behavioral difficulties, such that BMI was also associated with the shift in overall eating patterns.

Conclusions. Changes in behavioral function in seniors with AD result in a circadian shift in intake patterns with the preponderance of calories consumed at breakfast in those with increased behavioral difficulties. This shift in eating patterns associates both with poor overall intake and poor BMI.

Eliminating weight loss and malnutrition is a priority because of their association with an increased risk of morbidity (1), mortality (2,3), and greater rates of disease progression in seniors with Alzheimer’s disease (AD; 3). Although weight loss in AD is well documented (4–10) and considered a clinical feature consistent with this diagnosis (11), its etiology remains elusive. Reports of lower energy intakes in institutionalized AD patients compared to non-AD controls (12) and an association between weight change and decreased independence in self-feeding (8) support the hypothesis that weight loss may be secondary to decreased intakes. However, observations of weight loss in a group of AD outpatients with a concomitant increase in food intake (13), and reports of dietary intakes that were similar (4,14,15) and higher (6) in AD patients compared to controls, led others to suggest that weight loss is due to factors beyond reduced food intake.

Perhaps contributing to this ambiguity is the fact that measures of disease progression were not previously considered; rather, the AD population was compared, as a whole, to cognitively intact controls. Yet this clustering of the AD population is inconsistent with a neurodegenerative disease known for progressive behavioral deterioration. Rather, it is likely that intake patterns deteriorate with disease progression and that altered eating patterns are masked if disease status is not considered.

This study examines intake patterns in a group of senior residents in cognitive impairment (CI) units with likely AD, and how these patterns change in relation to measures of behavioral function, cognitive ability, and body weight status. Accurate methods to assess food intake were employed (16) and sufficient numbers of days of intake were examined (17) to capture habitual intake of the individual for the first time in this population.

Methods

Subjects

All residents of the CI units of the Jewish Home for the Aged at the Baycrest Centre for Geriatric Care (Toronto, Canada) were considered. By examining medical histories, only residents with likely AD were included. Although AD diagnoses using National Institute of Neurological and Communicative Disorders-Alzheimer’s Disease and Related Disorders Association criteria (11) were unavailable, residents were excluded if there was a diagnosis of CI secondary to other causes, such as vascular dementia or other neurodegenerative disorders. Subjects also met the following criteria: (i) maintains the ability to self-feed or requires only minimal assistance (e.g., tray set-up) and (ii) absence of other diseases requiring nutritional intervention (e.g., types 1 or 2 diabetes mellitus). Twenty-five individuals (3 men and 22 women; mean age 85.9 ± 7.6), comprising an ~60% participation rate of eligible subjects, were included. Following protocol approval by the Baycrest ethics committee, informed consent was obtained from the family or legal guardian.
Cognitive and Behavioral Function Assessments

Cognitive status was determined using the Mini-Mental State Examination (MMSE) (18) and behavioral function was assessed by the London Psychogeriatric Rating Scale (LPRS; 19). A higher score on the LPRS indicates greater disability and consists of four subscales: Mental Disorganization/Confusion (MENT), Physical Disability (PD), Socially Irritating Behavior (SIB), and Disengagement (DIS). In anticipation that the MMSE may “bottom-out,” the LPRS was included because it was designed to specifically assess usual behavior of geriatric patients (19).

Food Intake Collection

Twenty-one consecutive days of investigator-weighed food intake and delivery were monitored on each subject. The nutrient profile of the meals was determined using Dietary Food Management software, which contains all in-house recipes and calculates the nutrient composition on the basis of individual ingredients using the Canadian Nutrient Database (20). Afternoon snack intake, usually muffins, juices, and nutrient supplements (e.g., Ensure), was estimated visually and standard weights assumed.

Statistics

Meal-related nutrient intakes and delivery, as well as within-individual variability, expressed as standard deviation (SD) and coefficient of variation (CV), were compared using a repeated measures analysis of variance, followed by Tukey’s Honestly Significant Difference test, using SAS for Windows (v6.12). Data were examined for normality prior to analyses using the Shapiro-Wilk statistic. Estimation of the prevalence of inadequate protein intakes was performed by probability analysis (21). Regression analyses were used to determine the associations between cognitive status, behavioral function, BMI, mean energy intake of the individual, and mean percentage of contribution of each meal to the total energy intake ([meal kcal/total kcal]*100).

RESULTS

Subject Descriptions

Details of the subjects are contained in Table 1. In terms of body weight as a risk factor for increased mortality (22), 48% of the subjects (2 males, 10 females) had high-risk BMIs (<22 kg/m²). Although all subjects were able to eat independently, 23 of 25 subjects were severely cognitively impaired, based on the MMSE (score: ≤17 [23]). The remaining subjects were mildly impaired (score 18–23).

Dietary Intake and Its Variability

Mean 24-hour and meal-related energy and macronutrient intakes are given in Table 2. All three meals contributed equally to mean 24-hour energy consumed. Because energy delivered was the lowest at breakfast (Table 3), on average, subjects consumed the greatest percentage of energy delivered ([consumed/delivered]*100%) at breakfast (p < .05). Energy needs (24), calculated as 1.3*basal metabolic rate, were not met by energy consumed for 88% of the subjects. Although the contributions of protein, fat, and carbohydrate to total energy consumed (14.8 ± 1.9%, 32.4 ± 4.1%, and 54.6 ± 5.7%, respectively) were in line with current recommendations (24), the estimated prevalence of inadequate protein intakes (<0.8 g protein/kg body weight) was 37% of the subjects. The protein density of the diet (14.8% of calories) would be appropriate if adequate levels of energy were consumed; thus the low energy intake was the likely contributor to the high level of protein inadequacy observed.

Mean protein intake was lowest at breakfast and highest at lunch, while mean fat consumed at breakfast was lower than at lunch and dinner, which were not different from each other. By contrast, mean carbohydrate intake was highest at breakfast, likely reflecting the carbohydrate-dense foods offered. Snacks also provided mostly carbohydrate foods (Table 2).

The within-individual variabilities in intake (SD and CV) were lower at breakfast compared to lunch and dinner, which did not differ from one another, for energy or the macronutrients (Table 2). This consistency of breakfast intake occurred in tandem with lower variability for energy delivered (Table 3).

Snacks

Table 2 shows the mean energy consumed at all snack times for 25 individuals (i.e., all snack times included, even those of 0 kcal), while Table 4 provides snack data that includes only snacks that were consumed. Snacks tended to be “treats,” which, if received, were almost always consumed

Table 1. Average Age, BMI, MMSE, LPRS, and LPRS Subscales of Study Population

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th></th>
<th>Men</th>
<th></th>
<th>Women</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>85.9 ± 7.6 (65–98)</td>
<td>25</td>
<td>86.7 ± 9.0 (74–93)</td>
<td>3</td>
<td>85.8 ± 7.2 (65–98)</td>
<td>22</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>21.7 ± 3.3 (14.7–26.6)</td>
<td>22</td>
<td>20.6 ± 1.5 (18.9–22.5)</td>
<td>22</td>
<td>21.8 ± 3.4 (14.7–26.6)</td>
<td>22</td>
</tr>
<tr>
<td>MMSE (30)</td>
<td>7.4 ± 6.5 (0–21)</td>
<td>22</td>
<td>1.7 ± 2.1 (0–4)</td>
<td>22</td>
<td>8.3 ± 6.5 (0–21)</td>
<td>22</td>
</tr>
<tr>
<td>LPRS (72)</td>
<td>33.2 ± 10.7 (13–55)</td>
<td>22</td>
<td>34.3 ± 9.7 (26–45)</td>
<td>22</td>
<td>33.1 ± 11.1 (13–55)</td>
<td>22</td>
</tr>
<tr>
<td>PD (18)</td>
<td>6.9 ± 3.5 (0–13)</td>
<td>22</td>
<td>10.0 ± 3.0 (7–13)</td>
<td>22</td>
<td>6.4 ± 3.3 (0–13)</td>
<td>22</td>
</tr>
<tr>
<td>DIS (12)</td>
<td>8.4 ± 2.1 (4–12)</td>
<td>22</td>
<td>9.0 ± 3.5 (5–11)</td>
<td>22</td>
<td>8.4 ± 2.0 (4–12)</td>
<td>22</td>
</tr>
<tr>
<td>SIB (16)</td>
<td>5.3 ± 3.5 (0–12)</td>
<td>22</td>
<td>2.7 ± 0.6 (2–3)</td>
<td>22</td>
<td>5.7 ± 3.6 (0–12)</td>
<td>22</td>
</tr>
<tr>
<td>MENT (26)</td>
<td>12.6 ± 5.2 (3–23)</td>
<td>22</td>
<td>12.7 ± 4.7 (9–18)</td>
<td>22</td>
<td>12.6 ± 5.3 (3–23)</td>
<td>22</td>
</tr>
</tbody>
</table>

Notes: Values are expressed as mean ± SD (range). BMI = body mass index.
†Maximum possible score. Higher scores on the London Psychogeriatric Rating Scale (LPRS) and its subscales, Physical Disability (PD), Disengagement (DIS), Socially Irritating Behavior (SIB), and Mental Disorganization/Confusion (MENT), indicate greater functional deterioration; the reverse is true for the Mini-Mental State Examination (MMSE): lower scores indicate greater cognitive deterioration.
Table 2. Group Mean and Variability of Energy and Macronutrient Consumption Based on 21-day Estimates for 25 Individuals

| Meal            | Mean energy consumed (kcal) | Mean ± SD
d | Range         | Mean Within-Individual SD ± SD | Mean Within-Individual CV ± SD |
<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>Breakfast</td>
<td>387 ± 104</td>
<td>202–664</td>
<td>92.3 ± 32.7\a</td>
<td>24.6 ± 8.6\a</td>
<td></td>
</tr>
<tr>
<td>Lunch</td>
<td>384 ± 107</td>
<td>248–594</td>
<td>153.7 ± 60.6\a</td>
<td>41.8 ± 16.9\a</td>
<td></td>
</tr>
<tr>
<td>Dinner</td>
<td>374 ± 113</td>
<td>150–564</td>
<td>155.3 ± 34.7\a</td>
<td>44.5 ± 14.3\a</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>1247 ± 217</td>
<td>844–1789</td>
<td>259.0 ± 69.1\a</td>
<td>21.3 ± 7.6</td>
<td></td>
</tr>
<tr>
<td>Mean protein consumed (g)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breakfast</td>
<td>13.1 ± 4.3\b</td>
<td>5.0–22.6</td>
<td>3.8 ± 1.2\a</td>
<td>31.1 ± 10.8\a</td>
<td></td>
</tr>
<tr>
<td>Lunch</td>
<td>16.2 ± 6.0\b</td>
<td>7.7–29.4</td>
<td>9.0 ± 4.6\a</td>
<td>58.6 ± 28.8\b</td>
<td></td>
</tr>
<tr>
<td>Dinner</td>
<td>15.0 ± 5.7\b</td>
<td>5.2–27.1</td>
<td>8.1 ± 2.6\a</td>
<td>58.2 ± 17.0\b</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>2.2 ± 1.5</td>
<td>0.3–5.8</td>
<td>2.2 ± 0.8</td>
<td>148.3 ± 103.9</td>
<td></td>
</tr>
<tr>
<td>Mean fat consumed (g)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breakfast</td>
<td>11.4 ± 4.4\b</td>
<td>4.7–22.5</td>
<td>3.7 ± 1.2\a</td>
<td>35.8 ± 14.8\b</td>
<td></td>
</tr>
<tr>
<td>Lunch</td>
<td>15.4 ± 5.2\b</td>
<td>7.4–27.0</td>
<td>8.7 ± 3.7\a</td>
<td>58.9 ± 23.1\b</td>
<td></td>
</tr>
<tr>
<td>Dinner</td>
<td>15.4 ± 6.0\b</td>
<td>4.1–27.1</td>
<td>9.0 ± 3.1\a</td>
<td>63.2 ± 18.9\b</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>2.8 ± 1.9</td>
<td>0.4–7.9</td>
<td>2.7 ± 0.8</td>
<td>150.1 ± 108.7</td>
<td></td>
</tr>
<tr>
<td>Mean carbohydrate consumed (g)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breakfast</td>
<td>60.4 ± 17.0\b</td>
<td>31.2–101.9</td>
<td>15.0 ± 5.6\a</td>
<td>25.9 ± 10.2\b</td>
<td></td>
</tr>
<tr>
<td>Lunch</td>
<td>47.1 ± 12.0\b</td>
<td>29.0–70.8</td>
<td>18.6 ± 5.5\a</td>
<td>41.0 ± 11.9\b</td>
<td></td>
</tr>
<tr>
<td>Dinner</td>
<td>45.7 ± 13.9\b</td>
<td>21.5–75.2</td>
<td>18.9 ± 4.3\a</td>
<td>43.8 ± 12.7\b</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>16.5 ± 9.7</td>
<td>2.0–32.1</td>
<td>14.8 ± 3.9</td>
<td>141.3 ± 108.5</td>
<td></td>
</tr>
</tbody>
</table>

SD represents the variability between individuals only.
\(\bar{X}_{\text{SD}} \pm SD_{\text{SD}}\).
Mean snack intake calculated from the mean consumption of each subject (snack events of 0 kcal were included).
Means with different letters are significantly different \((p < .05)\). Tests of significance done across meals only (did not include snack or total intakes) for energy, protein, fat, and carbohydrate separately.

entirely. This “all-or-none” phenomenon contributed to the high within-subject variability in snack intake.

Relationship Between BMI, Behavioral Function, and Cognitive Status

When the relationships between current BMI and MMSE, LPRS, and its subscales were investigated, a trend for a negative association between BMI and the MENT subscale was found \((p = .061, r^2 = .14)\). All other associations were not significant. Thus, even in this small group of reasonably homogeneous individuals, those with increased mental disorganization/confusion tended to show the poorest body weight status.

The MMSE was correlated with the LPRS \((p = .002, r^2 = .28)\) and the MENT subscale \((p = <.001, r^2 = .33)\). Thus, in-line with the MMSE, the LPRS, particularly the MENT subscale, is likely sensitive to disease progression \((25)\).

Table 3. Mean Energy Delivered Based on 21-day Means for 25 Individuals

<table>
<thead>
<tr>
<th>Meal</th>
<th>Mean Energy Delivered (kcal ± SD)</th>
<th>Range (kcal)</th>
<th>Mean Within-Individual SD ± SD</th>
<th>Mean Within-Individual CV ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breakfast</td>
<td>645 ± 193a</td>
<td>415–1031</td>
<td>89.6 ± 42.0a</td>
<td>14.2 ± 5.8a</td>
</tr>
<tr>
<td>Lunch</td>
<td>724 ± 121b</td>
<td>527–1088</td>
<td>183.0 ± 67.8b</td>
<td>25.0 ± 7.3b</td>
</tr>
<tr>
<td>Dinner</td>
<td>729 ± 112b</td>
<td>575–1103</td>
<td>189.7 ± 46.0b</td>
<td>26.0 ± 4.7b</td>
</tr>
<tr>
<td>Meal Total\a</td>
<td>2097 ± 364</td>
<td>1571–3196</td>
<td>262.5 ± 67.2</td>
<td>12.6 ± 2.9</td>
</tr>
</tbody>
</table>

SD represents the variability between individuals only.
\(\bar{X}_{\text{SD}} \pm SD_{\text{SD}}\).
Meal Total does not include snacks delivered (discussed in Results).
Means with different letters are significantly different \((p < .05)\). Tests of significance done across meals only.

Relationship Between Energy Consumed and Behavioral Function

Measures of behavioral dysfunction associated with total intake and demonstrated their strongest relationship with dinner consumption \((Figure 1)\), such that individuals with greater behavioral difficulties, indicated by higher total LPRS, PD, and SIB scores, consumed less energy at dinner. When expressed as a percentage of total intake \((\text{meal kcal/total kcal}) \times 100)\), a marked shift in the time of day when intake and demonstrated their strongest relationship with total kcal was found \((p < .001, r^2 = .33)\). Thus, in-line with the MMSE, the LPRS, particularly the MENT subscale, is likely sensitive to disease progression \((25)\).

Relationship Between BMI and Energy Intake

Individuals with poor BMIs had breakfast intakes that were indistinguishable from those with higher BMIs, but

Table 4. Mean Energy and Macronutrient Values of Consumed Snacks \((n = 279)\)

<table>
<thead>
<tr>
<th>Snacks (n = 279)</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (kcal)</td>
<td>188 ± 73</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>4.1 ± 2.4</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>5.2 ± 2.9</td>
</tr>
<tr>
<td>Carbohydrate (g)</td>
<td>31.0 ± 12.0</td>
</tr>
</tbody>
</table>

\(^a\) Snack events of 0 kcal were not included. The mean of all snacks was calculated, rather than the mean of each individual’s mean snack consumption.
showed poor consumption profiles at lunch and dinner (Figure 3). This resulted in a positive association between BMI and total meal intake (snack not included). By contrast, 24-hour intakes were not related to BMI, in part due to the greater snack consumption of those with low BMIs.

With decreasing BMI, there is clearly a shift in the contribution of each meal to total intake (Figure 4). Those with low BMIs consumed a greater percentage of the day’s total energy at breakfast and less at lunch and dinner. Compared to those with higher BMIs, subjects with lower BMIs also tended to consume a higher percentage of energy as snacks.

**DISCUSSION**

This study is the first to demonstrate a change in the diurnal eating patterns of patients with AD that associates with measures of behavioral function. Noteworthy is the fact that decreased meal-related intakes in those with increased behavioral dysfunction are associated with lower intakes at dinner, but not breakfast. Thus, with increased behavioral difficulties and disease progression individuals no longer engage in patterns of eating consistent with those who are less impaired. These findings question the validity of attempting to maintain meal-time patterns traditionally provided to cognitively intact individuals for those with increased behavioral difficulties.

In healthy young adults (18–41), there is a tendency for meal sizes to increase over the day with peak intakes at noontime and early evening (26). Thus breakfast contributes proportionately less to daily intake in comparison to lunch and dinner. With AD progression, reversal of this pattern occurs such that those with more behavioral difficulties achieve the lowest intakes at dinner, with breakfast contributing the greatest percentage to total energy intake (Figures 1 and 2). These observations are consistent with known disruptions in circadian rhythms in AD (27,28) and suggest that increased confusion or agitation in the afternoon or early evening, “sundowning” (29), likely contributes to the change in eating patterns. That is, scoring poorly on the MENT subscale, which associates with these disruptions in eating patterns, is a possible proxy indicator for patients exhibiting the sundowning syndrome.

Although the MMSE was not associated with a shift in eating patterns, this is likely due to “bottoming-out” of the scores. Indeed, 48% of the subjects scored 5 or lower. Nevertheless, it is likely that both disease progression and behavioral difficulties are predictive of the changes in eating patterns observed because the MMSE was highly correlated with the LPRS and MENT subscale.

Although these data demonstrate that behavioral function is important, it is unlikely the sole contributor to the eating patterns observed. Hunger and satiety signals are likely disturbed secondary to neuronal degeneration impacting on neurochemical (30,31) and anatomical (32) pathways involved in food intake regulation, which may involve cell loss in the paraventricular nuclei (33). Thus, diminished hunger signals may not be sufficient to drive individuals to increase their breakfast intake to compensate for reduced dinner intake. Indeed, if this were the case, greater breakfast intakes would have been observed in those with increased behavioral difficulties. Additionally, this study did not account for the myriad of other factors reported to contribute
Figure 3. Relationship between body mass index (BMI) and 21-day mean energy consumed (n = 25). A. Breakfast: p = .764, r² = .004. B. Lunch: p = .017, r² = .22. C. Dinner: p = .009, r² = .26. D. Meal Total (B + L + D); p = .026, r² = .20. E. Snack: p = .015, r² = .24. F. 24-hour Total (B + L + D + S); p = .109, r² = .11.

Figure 4. Relationship between the percent contribution of each meal to total energy consumption and body mass index (BMI; n = 25). A. Breakfast: p = .043, r² = .17. B. Lunch: p = .033, r² = .18. C. Snack: p = .009, r² = .26. D. Dinner: p = .019, r² = .22.
implement due to staffing profiles, our data suggest that these assessments are best made during the evening meal and that assistance needs may change throughout the day.

In summary, this study provides the most extensive measures of food intake in a population of institutionalized seniors with AD to date. Collectively, these data suggest that poor food intake, which associates with increased behavioral difficulties, possibly related to sundowning, likely contributes to poor BMI. Consistent with a disturbance in circadian rhythms, peak food consumption levels shift from evening to morning in those with decreased behavioral function. Importantly, this suggests that with behavioral changes and AD progression the provision of high energy, nutrient-dense afternoon and evening meals is unlikely to meet the nutritional needs of this population and that consideration must also be given to changes in meal-time assistance needs throughout the day.

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References


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