

A cross-sectional study on the impact of caloric restriction on cognitive performance of young and aged males: Role of physical activity

Sreejit Ghosh^{1,2} , Subhashis Sahu^{1*} , Goutam Paul¹

ABSTRACT

Background: Stress is an acknowledged part of normal aging. Lifestyle choices can influence age-related declines in cognition. It has been discovered that caloric restriction protects aging processes and downregulates normal aging by lowering stress. This study assessed the effects of caloric restriction (CR) and physical activity (PA) on age-related cognitive changes. **Methods:** From the low-income and middle-income healthy population, 236 males with ages ranging from 20 to 35 years and 300 males with ages ranging from 55 to 70 years were selected. Information was obtained about socioeconomic status, health status, physical activity level (PAL), cognitive function as assessed by the Mini-Cog and 6 CIT, and calorie intake through the completion of questionnaires. **Results:** When comparing the elderly group to the younger members of the same calorie group, the AP diet (at-par i.e. diet consumed in an amount equal to the energy needed for 24 hours) reduced cognitive function. The CR diet enhanced the patients' cognitive abilities more in the younger group than in the older one. Regardless of age or caloric intake, subjects' cognition was positively affected by high PAL relative to low PAL. **Conclusion:** (a) Both young people on a CR diet and elderly people on an AP diet have enhanced cognitive capacities. (b) Regardless of calorie consumption, high PAL, as opposed to low PAL, improves cognition in both young and old populations.

Keywords: Aging, Diet, Caloric restriction, Cognitive function, Depression, Physical activity.

Indian Journal of Physiology and Allied Sciences (2025);

DOI: 10.55184/ijpas.v77i01.460

ISSN: 0367-8350 (Print)

INTRODUCTION

It is well recognized that one of the main health hazards of aging societies is impaired cognitive function¹. The problems related to aging-induced cognitive decline have been addressed earlier by several researchers². The updated information confirms that our cognitive capacity deteriorates with age.³ Memory is the cognitive function that deteriorates the most.^{4,5} It is still unclear if learning ability and cognitive decline may be restored, given the impact of aging on cognitive impairment. Many variables contribute to age-related cognitive deterioration. Some of these include things like personality⁶, lifestyle⁷, health⁸, cognitive style⁹, and educational level¹⁰. Cognitive impairment is also known to be caused by worry, anxiety, or sadness^{11,12}. Research has demonstrated that Caloric restriction (CR) can enhance resistance to age-related neurotoxicity and neurodegeneration in animals¹³. CR involves subjecting the organism to a low level of stress (hormetic), which may, in turn, trigger stress responses that offer protection against various aging processes¹⁴. The successful maintenance of cognitive abilities and cognitive skills with age depends on several conditions: the absence of diseases leading to loss of autonomy, the practice and maintenance of cognitive and physical activities¹⁵, adequate sleep^{16,17}, abstinence from smoking and excessive alcohol consumption^{18,19}, a socially active lifestyle²⁰, and a healthy diet²¹. Because diet is modifiable, it is of interest to what extent dietary factors cause adverse cognitive outcomes in old age. The possible beneficial (protective or delaying) role of several members of the vitamin B-complex family^{22,23} and antioxidants^{24,25}

¹Department of Physiology, University of Kalyani, Kalyani, Nadia, Pin-741235, West Bengal, India.

²Department of Physiology, Jhargram Raj College, Jhargram, Pin-721507, West Bengal, India.

***Corresponding author:** Subhashis Sahu, Department of Physiology, University of Kalyani, Kalyani, Nadia, Pin-741235, West Bengal, India, Email: skcsahu@yahoo.co.in

How to cite this article: Ghosh S, Sahu S, Paul G. A cross-sectional study on the impact of caloric restriction on cognitive performance of young and aged males: Role of physical activity. *Indian J Physiol Allied Sci* 2025;77(1):18-25.

Conflict of interest: None

Submitted: 08/11/2024 **Accepted:** 05/02/2025 **Published:** 30/03/2025

on aging-related cognitive decline have been discussed in literature along with the risk and benefit of chronic alcohol consumption²⁶ and intake of CR diet²⁷ respectively. In experimental animals, caloric restriction slows the normal aging process²⁸ and lowers the production of beta-amyloid plaques^{29,30}. Numerous studies published in the last 30 years suggest that some lifestyle decisions, such as eating a low-calorie or CR diet and consuming specific micro- and macronutrients, like unsaturated fatty acids (UFA), may benefit the aging brain³¹⁻³³. Long-term studies in humans have shown that following dietary CR can promote healthy aging and longer life.^{34,35} It is evident from animal studies that increased physical activity could lead to a perceived enhancement in cognitive function in behavioral tasks like the Morris water maze³⁶. The current study's goal is to assess how caloric restriction affects age-related cognitive

deterioration in individuals. Further, the study investigates if physical activity potentiates the effect, if any, of CR on ageing-induced natural decline of cognition in young (20–35 years) and aged (55–70 years) male subjects.

METHODS AND MATERIALS

Ethical Clearance of the Study

The work has been carried out with appropriate ethical sanction from the Institutional Ethics Committee for Human Research of the Department of Physiology, University of Kalyani [Ref No. KU/IEC (H)/1/10/2023-24].

Subjects

The Indian state of West Bengal served as the study's location. After screening a total of 672 people, 536 men (236 young and 300 aged) were chosen as the sample population (Figure 1). The young people's age range was 20 to 35 years, and the senior people's age range was 55 to 70 years. According to their self-report, none of the study participants had a history of addiction. There were no signs of morbidity or disability in the study individuals' health status. The Health Assessment Questionnaire (HAQ) was used to evaluate the state of health³⁷. The study participants were selected from among those who go for morning walks and also from free health check-up camps, construction sites, marketplaces, and educational institutions, among other places. According to Kuppaswamy's Socio-economic scale³⁸, the entire study population, as evaluated, belonged to the Middle-Education Low Income group.

General Procedure

Subject interviews were always scheduled in advance. Either the interview day and time were fixed or comparable preparations were made, or the participants' assent was acquired in advance (like with morning walkers), with institutions, neighborhood clubs that offered free health checkups to the locals, or the village chief. Without a set program, participants were chosen at random for interviews at a marketplace during business hours. In each instance, the individuals were told what they had to do and how to execute it. Depending on the respondents' priorities, the rejection rate for participating in the interview procedure ranged from 3 to 28%. Except for the mini-cog test, which required active participation from the subjects, all other data on the subjects' calorie intake, health, socioeconomic position, physical activity level (PAL), and cognitive function as assessed by the 6-item Cognitive Impairment Test were collected, by having them complete questionnaires that were administered by interviewers with training. The subjects provided only passive answers to the questions (Figure 2). The procedure was designed by skilled interviewers to take 15 to 20 minutes to gather information from a single person. Based on the data from their voter ID, male subjects from the state of West Bengal were split into two age groups for the current study: young (20–35 years old) and old (55–70 years

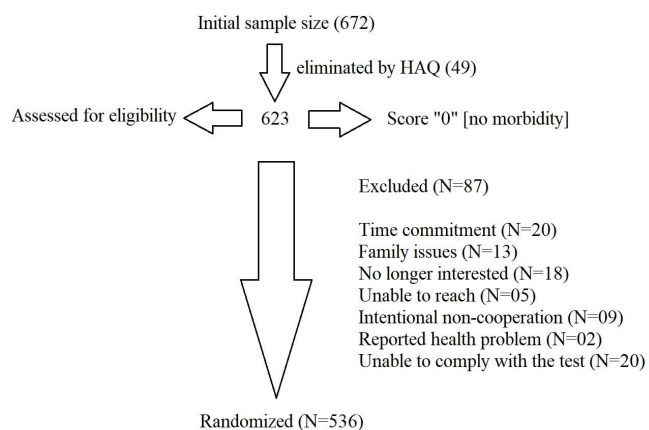


Figure 1: Protocol for subject selection

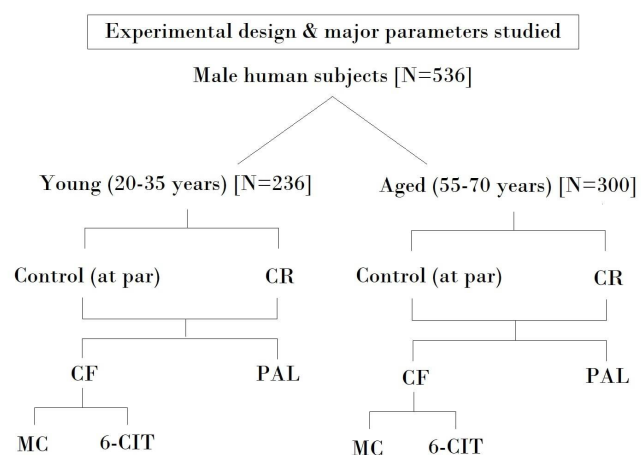


Figure 2: Experimental design and parameters studied. CR = Calorie Restricted, CF = Cognitive Function, PAL = Physical Activity Level, MC = Mini-Cog, 6CIT = 6-Item Cognitive Impairment Test

old). After the 24-hour Dietary Recall method described in this section was used for assessment, individuals of both ages were split into two calorie groups. A questionnaire method was used to assess each subject's activities over 24 hours, and the physical activity level (PAL) was computed. Using the previously mentioned established methods (Mini-Cog and 6CIT), individuals were classed as either demented or not-demented. The current investigation was conducted in three stages to assess the experimental individuals' cognitive condition. Data collection from fieldwork, which included interviewing study participants, was the first phase. In the second phase, subjects were grouped based on their level of cognitive function, age, PAL, and calorie intake (Figure 2). Using the proper statistical techniques, all of the primary data were arranged and examined in the third step.

Evaluation of Energy Consumption

The estimate of food and nutrient intake, or Diet Survey, was used to determine each subject's calorie consumption. The three-24-hour recall method, as outlined by Gibson *et al.*³⁹ and verified by Maet *al.*⁴⁰, was used to conduct the diet survey.

AP indicates an at-par diet (as per calorie requirement); The average calorie requirement of young and aged groups are 2618 ± 19.98 and 2337 ± 15.92 kcal, respectively.

CR indicates a calorie-restricted diet (<20% of calorie requirement); The average calorie requirement of young and aged groups are 2677 ± 22.20 and 2480 ± 19.09 kcal, respectively.

Assessment of Cognitive Function

Mini-Cog: The Borson *et al.* technique⁴¹ was followed to conduct the qualitative evaluation of cognitive function, whether it was demented or not. To put it briefly, the interviewer had given each respondent a 3-word sentence that was unconnected to each other. For various subjects, different word phrases were used. The individual was instructed to draw a clock face and indicate the time by sketching the clock's two hands as soon as the three unrelated word phrases were given to them. The interviewer provided the precise time to be put. The individual was instructed to recall the three unrelated word phrases as soon as he finished the clock drawing test (CDT). Based on the information indicating the subject's level of accomplishment in completing the activities, they were classified as either demented or not.

Six-Item Cognitive Impairment Test (6 CIT): Mini-Cog was first used to qualitatively assess each study participant's cognitive function. To measure this performance, the 6-item Cognitive Impairment Test (6 CIT) was used. On the 6CIT, there were several questions. The respondents were instructed to listen to and memorize a five- to six-line "Name and Address" phrase before the questions were asked and to repeat the phrase after responding to the questions. Following that, the subjects underwent 6CIT in accordance with Blessed *et al.*'s methodology⁴² which was validated by Brooke and Bullock⁴³ to determine if they were demented or not.

Evaluation of Physical Activity Level (PAL)

The subjects' level of physical activity was assessed using the standard questionnaire (PAL). Every subject's daily activities were recorded for 24 hours, and the Bharathi *et al.* method⁴⁴ was used to calculate the average energy consumption of each person in finishing the various activities throughout 24

hours. Factorial estimation of total energy expenditure and physical activity level was the method used.⁴⁵

Statistical Analysis

The demographic characteristics, BMI, and calorie intake were displayed as mean \pm SEM. The significance of the difference between the study groups' obtained BMI and calorie consumption was assessed using the student's t-test⁴⁶. The study employed one-way analyses of variance (ANOVA) and the post-hoc Tukey's HSD test⁴⁷ to investigate the significance of variations in population change (%) across study groups concerning the cognitive performance of young and senior individuals who consumed various amounts of calories. The significance of the difference between the percent population with respect to cognitive status in different age and calorie groups with varying physical activity levels was ascertained using the student's t-test between the relevant groups. P values were deemed significant if they were less than 0.05; Version 20.0 of SPSS software (IBM) was used for statistical calculations.

RESULTS

Demographic Features and Calorie Consumption

In Table 1, The percentage of each group's population in each category in relation to the group's overall population was denoted in parenthesis. The BMI of both the young and aged participants on the CR diet had a mean that was considerably ($p < 0.01$) lower than the BMI of the subjects in the same age groups but on the AP calorie diet (21.55 and 20.98%, respectively). The calorie intake of young and aged subjects in the AP group consumed considerably ($p < 0.01$) more mean calories (15.70 and 18.47%, respectively) than the comparable CR diet-receiving group.

Cognitive Performance by Caloric Intake

It is observed that of the 137 young participants on an AP calorie diet, 21.17% were demented and 78.83% were not. Among 99 participants in the same age group on the CR diet, 88.89% were not demented while 11.11% were demented (Table 2).

Table 2 also shows that young participants who consumed

Table 1: Demographic features and calorie consumption of young and aged male subjects

Parameters Observed	Male Subjects			
	Young (N=236)		Aged (N=300)	
Calorie Group	AP	CR	AP	CR
Number of subjects (%)	137 (58.05)	99 (41.95)	134 (44.67)	166 (55.33)
Age (Years.)	32.2 ± 0.36	28.85 ± 0.28	58.66 ± 0.29	58.85 ± 0.43
BMI (kg/m ²)	22.56 ± 0.18	18.56 ± 0.34^a	22.03 ± 0.24	18.21 ± 0.063^b
Calorie Intake (kcal)	2344 ± 29.86	1976 ± 17.94^a	2220 ± 22.76^a	$1810 \pm 16.24^{b,c}$

Data are presented as mean \pm SEM of individual observations; significant ($p < 0.05$) differences are indicated by ^a(for Young AP), ^b(for Aged AP), ^c(for Young CR).

Table 2: Effect of calorie intake on cognitive performance in young and aged male subjects

Age group (N)	Calorie group (N)	Cognitive status	
		ND*	D**
Young (236)	AP (137)	4.16 ± 0.15 (108)	10.05 ± 0.3 (29)
	CR (99)	3.16 ± 0.15 (88) ^a	10.53 ± 0.37 (11) ^a
Aged (300)	AP (134)	4.32 ± 0.14 (88) ^a	9.7 ± 0.14 (46) ^a
	CR (166)	3.85 ± 0.21 (96) ^{b,c}	9.84 ± 0.12 (70) ^{b,c}

Data are presented as mean ± SEM of individual observations; significant ($p < 0.05$) differences are indicated by ^a(for Young AP), ^b(for Aged AP), ^c(for Young CR). *Cognitive score 0-7 indicates ND = NOT-DEMENTED, **Cognitive score > 7 indicates D = DEMENTED.

CR diet had a significant increase in the not-demented population (12.76%, $p < 0.05$) and a significant decrease in the demented population (47.52%, $p < 0.005$) when compared to young people consuming an AP calorie diet. In contrast, the subjects in the aged group as revealed from the results of Table 2 shows that out of 134 participants on the AP calorie diet, 65.67% were not demented and 34.33% were demented; while on the CR diet, out of 166 aged subjects 57.83% were not demented and 42.17% were demented. In contrast to the young population, the old population's consumption of the CR diet resulted in a substantial increase (22.84%, $p < 0.01$) in the number of demented subjects and at the same time significant decrease (11.94%, $p < 0.05$) in the number of non-demented participants (Table 2).

Furthermore, Table 2 revealed that the demented population in aged subjects on an AP calorie diet was significantly higher (62.16%, $p < 0.01$) than that in young subjects on an AP calorie diet, while the not-demented population was significantly lower (16.69%, $p < 0.01$) compared to the results observed in young subjects consuming an AP calorie diet. Table 2 further reveals that the percent population of not-demented subjects aged with CR diet was significantly less (34.94%;

$p < 0.001$) than that observed in young subjects with CR diet. On the other hand, the demented population aged with the CR diet was significantly higher (279.57%; $p < 0.001$) than the young people consuming the CR diet. Table 2 shows that of the 137 young participants fed an AP calorie diet, 21.17% had dementia and 78.83% did not. Again, out of 99 subjects on the CR diet and in the same age group, 88.89% were not demented and 11.11% were demented. The findings (Table 2) also show that, when compared to the young population consuming an AP calorie diet, the consumption of the CR diet significantly increased (by 12.76%, $p < 0.05$) the not-demented population while concurrently significantly reducing (by 47.52%, $p < 0.005$) the demented population. In contrast, the aged group revealed that of 134 participants on an AP calorie diet, 65.67% were not demented and 34.33% were; of 166 aged subjects on a CR diet, 57.83% were not demented and 42.17% were demented. Unlike the young population, the old population's consumption of the CR diet resulted in a substantial increase (22.84%, $p < 0.01$) in the demented subjects and a significant decrease (11.94%, $p < 0.05$) in the non-demented participants (Table 2). Furthermore, Table 2 revealed that the demented population in aged subjects on an AP calorie diet was significantly higher (62.16%, $p < 0.01$) than that in young subjects on an AP calorie diet, while the not-demented population was significantly lower (16.69%, $p < 0.01$) compared to the results observed in young subjects consuming an AP calorie diet. Table 2 further shows that compared to young participants on the CR diet, the percentage of not-demented subjects in the older population was substantially lower (34.94%; $p < 0.001$) in the former group. Conversely, compared to young subjects on the CR diet, the demented population in elderly adults on the CR diet was much greater (279.57%; $p < 0.001$).

Impact of Physical Activity Level

Table 3 demonstrates that 94.39% of the 137 young subjects on an AP calorie diet and high PAL were not demented,

Table 3: Effect of physical activity level (PAL) on calorie-induced changes in cognitive performance of young and aged participants

Age group	Calorie group	PAL group (N)	Cognitive status	
			ND*	D**
Young (N=236)	AP (N= 137)	HIGH (107)	3.87 ± 0.16 (101)	12.0 ± 2.0 (6)
		LOW (30)	5.26 ± 0.33 (19) ^a	9.88 ± 0.25 (11) ^a
	CR (N= 99)	HIGH (60)	3.28 ± 0.16 (60) ^a	--
		LOW (39)	4.617 ± 0.27 (30) ^{c,e}	10.13 ± 0.32 (9) ^{c,e}
Aged (N=300)	AP (N= 134)	HIGH (85)	4.305 ± 0.15 (69)	10.42 ± 0.89 (16)
		LOW (49)	4.461 ± 0.49 (18) ^b	9.625 ± 0.13 (31) ^b
	CR (N= 166)	HIGH (70)	3.47 ± 0.2 (58)	9.857 ± 0.55 (12)
		LOW (96)	5.27 ± 0.45 (33) ^d	9.915 ± 0.13 (63) ^d

Data are presented as mean ± SEM of individual observations; significant ($p < 0.05$) differences are indicated by ^a(for Young AP with high PAL), ^b(for Aged AP with high PAL), ^c(for Young CR with high PAL), ^d(for Aged CR with high PAL), and ^e(for Young AP with low PAL). *Cognitive score 0-7 indicates ND = NOT-DEMENTED, **Cognitive score > 7 indicates D = DEMENTED. HIGH indicates when the PAL value is ≥ 1.70, and LOW indicates when the PAL value is 1.40 to 1.69

while only 5.61% of them were demented. Conversely, of 30 participants with low PAL who were in the same age and calorie group, 36.66% were found to be demented while 63.3% were not. Table 3 further demonstrated that compared to the young AP calorie group with high PAL, the not-demented population in the AP low-PAL group had a considerable

lower percentage (32.94%, $p < 0.001$). Results of Table 3 also showed that, compared to the young AP group with high PAL, the demented population in those on an AP calorie diet and with low PAL was substantially greater (553.47%, $p < 0.001$). Analysis of the results of Table 3 further highlighted that, of the young individuals on a CR diet with high PAL, 100% were not demented; in contrast, 76.92% of the young CR participants with low PAL were not demented and 23.07% were demented. The not-demented population was dramatically reduced (23.08%, $p < 0.001$) by low PAL in the young population with the CR diet with a simultaneous accountable increase in the demented population under comparable conditions. In contrast to the young, 82.14% of the elderly subjects who followed an AP calorie diet and had high PAL scores were not demented, while 18.82% were. Within the same calorie group, 36.73% of participants did not exhibit dementia and 63.26% did (Table 3). There was a large increase (236.13%, $p < 0.001$) in the demented population and a significant decrease (55.28%, $p < 0.001$) in the not-demented population among elderly adults on an AP diet and with low PAL. In contrast to the AP calorie diet, the CR diet was consumed by 82.86% of the not-demented and 17.14% of the demented-aged subjects with high PAL. However, in the same age and calorie group, the proportion of the demented and not-demented population with low PAL was found to be 34.37 and 65.62%, respectively (Table 3). The table (Table 3) also reveals that Compared to aged subjects with a CR diet and high PAL, the percentage of the population without dementia was considerably lower in those with a CR diet and low PAL (58.52%, $p < 0.001$). Conversely, the aged subjects having a CR diet with low PAL significantly increased (282.85%, $p < 0.001$) the demented population in comparison to the aged population with a CR diet with high PAL.

DISCUSSION

The current study evaluates the impacts of caloric restriction on age-related cognitive deterioration in young (20–35 years) and aged (55–70 years) male subjects. Additionally, the study investigates if physical activity potentiates the effect, if any, of CR on aging-induced natural decline of cognition. Tables 2 and 3 show that the effects of the CR diet on cognitive performance appear to be opposing in young and aged participants. The hypothesis that the CR diet reduces oxidative damage by reducing energy flux and metabolism^{48,49} may be the reason for the percent increase in the population that is not demented and the corresponding decrease in the population that is demented among young subjects on the CR diet compared to young subjects on the

AP diet (Table 2). The CR diet appears to lower the rate of metabolism in the population of young adults.⁵⁰ Because a change in food or nutrient intake may result in the creation of a new or different steady state⁵¹, multiple types of metabolic adaptation with CR diet are not even unlikely⁵². Heilbronn and Ravussin's research⁵³ makes it clear that the CR diet promotes the development of metabolic adaptability. The literature that is now available thus supports the theory that the rise in cognitive function induced by the CR diet in young subjects is probably caused by a decreased metabolic rate as a result of some kind of metabolic adaptation. Further, lower metabolic rates reduce the generation of superoxide radicals, which lessens the harm that these radicals do to brain neurons and may account for the possibility of improved cognitive function⁵⁴.

In contrast to the younger group, the elderly population on a CR diet exhibits diminishing cognitive ability (Table 2). This decline may be caused by malnourishment of the brain tissue. It is not justifiable to completely rule out the chance that an aging population's inability to maintain the functional integrity of brain neurons by supplying the necessary amount of energy through a CR diet may result from the GI system's declining functional capability.⁵⁵ This opinion is further reinforced by the current study's findings, which demonstrate that older participants on a CR diet performed less cognitively than their AP-calorie-consuming counterparts (Table 2). The current study suggests that the reasons behind the consumption of the CR diet by the elderly population may differ from those of the younger population, who follow a regular low-calorie diet. Furthermore, the health status questionnaire fails to identify any clinical justification for the CR diet among older participants. Differential insulin sensitivity and glucose utilization may be the cause of the elderly subjects' poor and the young subjects' better cognitive performance when following a similar low-calorie diet (Table 2). Evidence exists that macronutrients (energy providers) have an impact on cognitive function⁵⁶ supporting the fact that brain tissue uses glucose for energy.⁵⁷ It has previously been demonstrated that physical activity and both physical and mental well-being are positively correlated⁵⁸⁻⁶⁰. The current investigation shows that among young and aged groups, as well as among those following AP or CR diets, high PAL increases the population that is not demented in comparison to low PAL (Table 3). Regarding age and calorie consumption, the low PAL not demented population of the comparable group is smaller than the high PAL, not demented population among young people following a CR diet. A favorable effect of high PAL on cognitive function is indicated by the rise in the not-demented population of young participants with an AP diet and high PAL compared to the low PAL population of the same age and calorie group (Table 3). Regarding age and calorie consumption, the low PAL not demented population of the comparable group is smaller than the high PAL, not demented population among young participants following a

CR diet. A favorable effect of high PAL on cognitive function is indicated by the rise in the not-demented population of young subjects with an AP diet and high PAL compared to the low PAL population of the same age and calorie group (Table 3). However, a study on mentally stable young people who had high PAL with CR and AP diets (Table 3) indicates that CR diets have a stronger favorable impact on cognitive function than AP diets. The results can be explained by the fact that, regardless of age, high PAL increases glucose utilization by peripheral tissues, including brain tissue, allowing the brain to perform better⁶¹ in comparison to low PAL, where there is a greater chance that brain tissue will starve to death from lack of fuel.

Reduced risks of cognitive decline are linked to physical activity. Likewise, there is a noteworthy tendency of protection against cognitive deterioration associated with increased physical exercise, as demonstrated by Laurin D *et al.*¹ In response to high levels of physical activity, the metabolism of proteins and carbohydrates is more tightly regulated than that of fat⁶². Thus, the current data may imply that elevated PAL provides improved control over the metabolism of carbohydrates, which in turn affects brain cell function and influences cognition.

Engaging in physical activity is linked to a decreased risk of cognitive deficit. The majority of human populations exposed to naturally occurring episodes of CR are deficient in micronutrients and protein⁵³. In the context of the current study, the prevalence of poor cognition among older adults on CR diet (Table 3) may be the result of malnourished brain tissue as well as its degraded function and/or fuel deprivation^{63,64}. According to reports, high PAL⁶¹ within a certain calorie group slows down the natural phenomena of cognitive decline with aging. On the other hand, high PAL in a young population with a specified calorie intake can also positively influence brain development and functioning⁶⁵. In summary, the current cross-sectional study involving both young and old populations consuming either an AP or CR diet leads to the following conclusions: (a) CR diet improves cognitive function in the young population (20–35 years old) when compared to AP diet in the same age group; (b) AP diet is preferred over CR diet in the elderly population (55–70 years old) due to better cognitive function; (c) Regardless of calorie consumption, high PAL, in contrast to low PAL, preserves cognitive abilities in both young and old populations. This may be accomplished by interfering with cellular physiological and biochemical components. Further research is required to clarify how other confounding factors, such as food quality, socioeconomic status, occupation, education, sex, and stress besides many others, affect cognitive abilities in humans at various ages, rather than just calories or PAL.

ACKNOWLEDGMENT

The authors would like to thank all the participating subjects. The authors acknowledge support from the Departmental Research Committee, University of Kalyani.

REFERENCES

- Laurin D, Verreault R, Lindsay J, MacPherson K, Rockwood K. Physical activity and risk of cognitive impairment and dementia in elderly persons. *Arch Neurol*. 2001;58(3):498-504. DOI: 10.1001/archneur.58.3.498.
- Das A, Ghosh SK, Paul G, Poddar MK. Role of Physical Activity on Calorie-Induced Changes in Cognition and Depression of Young and Aged Humans. *IJND*. 2017;54(3):316-35. <https://DOI.org/10.21048/ijnd.2017.54.3.15460>
- Jorm AF, Korten AE. Assessment of cognitive decline in the elderly by informant interview. *Brit Jour Psych*. 1988;152(2):209-13. DOI: 10.1192/bjpp.152.2.209.
- Hedden T, Gabrieli JDE. Insights into the ageing mind: a view from cognitive neuroscience. *Nat Rev Neurosci*. 2004;5(2):87-96. DOI: 10.1038/nrn1323.
- Park DC, Reuter-Lorenz P. The adaptive brain: aging and neurocognitive scaffolding. *Annu Rev Psychol*. 2009;60(1):173-96. DOI: 10.1146/annurev.psych.59.103006.093656.
- Crowe M, Andel R, Pedersen NL, Fratiglioni L, Gatz M. Personality and risk of cognitive impairment 25 years later. *Psychol Aging*. 2006;21(3):573-80. DOI: 10.1037/0882-7974.21.3.573.
- Fratiglioni L, Paillard-Borg S, Winblad B. An active and socially integrated lifestyle in late life might protect against dementia. *Lancet Neurol*. 2004;3(6):343-53. DOI: 10.1016/S1474-4422(04)00767-7.
- Stewart R, Hirani V. Dental health and cognitive impairment in an English national survey population. *J Am Geriatr Soc*. 2007;55(9):1410-14. DOI: 10.1111/j.1532-5415.2007.01298.x.
- Meyer TD, Gudgeon E, Thomas AJ, Collerton D. Cognitive style and depressive symptoms in elderly people—Extending the empirical evidence for the cognitive vulnerability-stress hypothesis. *Behav Res Ther*. 2010;48(10):1053-57. DOI: 10.1016/j.brat.2010.06.003.
- Paradise M, Cooper C, Livingston G. Systematic review of the effect of education on survival in Alzheimer's disease. *Int Psychogeriatr*. 2009;21(1):25-32. DOI: 10.1017/S1041610208008053.
- Byrne GJ, Pachana NA. Anxiety and depression in the elderly: do we know any more? *Curr Opin Psychiatry*. 2010;23(6):504-09. DOI: 10.1097/YCO.0b013e32833f305f.
- Potter GG, Steffens DC. Contribution of depression to cognitive impairment and dementia in older adults. *Neurologist*. 2007;13(3):105-17. DOI: 10.1097/01.nrl.0000252947.15389.a9.
- Carter CS, Leeuwenburgh C, Daniels M, Foster TC. Influence of Caloric restriction on Measures of Age-Related Cognitive Decline: Role of Increased Physical Activity. *J Gerontol A Biol Sci Med Sci*. 2009;64(8):850-59. DOI: 10.1093/gerona/glp060.
- Witte AV, Fobker M, Gellnar R, Knecht S, Floel A. Caloric restriction improves memory in elderly humans. *Proc Natl Acad Sci USA*. 2009;106(4):1255-60. DOI: 10.1073/pnas.0808587106.
- Aartsen MJ, Smiths CHM, Van Tilburg T, Knopscheer KCPM, Deeg DJH. Activity in older adults: Cause or consequence of cognitive functioning— A longitudinal study on everyday activities and cognitive performance in older adults. *J Gerontol B Psychol Sci Soc Sci*. 2002;57(2):153-62. DOI: 10.1093/geronb/57.2.p153.
- Sateia MJ. Neuropsychological impairment and quality of life in obstructive sleep apnea. *Clin Chest Med*. 2003;24(2):249-59. DOI: 10.1016/s0272-5231(03)00014-5.
- Beebe DW, Gozal D. Obstructive sleep apnea and the prefrontal cortex: towards a comprehensive model linking nocturnal upper airway obstruction to daytime cognitive and behavioral deficits. *Jour Sleep Res*. 2002;11(1):1-16. DOI: 10.1046/j.1365-

- 2869.2002.00289.x.
18. Rosendorff C, Beeri MS, Silverman JM. Cardiovascular risk factors for Alzheimer's disease. *Am J Geriatr Cardiol.* 2007;16(3):143-49. DOI: 10.1111/j.1076-7460.2007.06696.x.
 19. Elwood PC, Gallacher JE, Hopkinson CA, et al. Smoking, drinking, and other life style factors and cognitive function in men in the Caerphilly cohort. *J Epidemiol Commun Health.* 1999;53(1):9-14. DOI: 10.1136/jech.53.1.9.
 20. Nakazawa K, Quirk MC, Chitwood RA, et al. Requirement for hippocampal CA3 NMDA receptors in associative memory recall. *Science.* 2002;297(5579):211-18. DOI: 10.1126/science.1071795.
 21. Greenwood CE, Winocur G. High-fat diets, insulin resistance and declining cognitive function. *Neurobiol Aging.* 2005;26(1):42-45. DOI: 10.1016/j.neurobiolaging.2005.08.017.
 22. Morris MS. The role of B vitamins in preventing and treating cognitive impairment and decline. *Adv Nutr.* 2012;3(6):801-12. DOI: 10.3945/an.112.002535.
 23. Enderami A, Zarghami M, Darvishi-Khezri H. The effects and potential mechanisms of folic acid on cognitive function: a comprehensive review. *Neurol Sci.* 2018;39(10):1667-75. DOI: 10.1007/s10072-018-3473-4.
 24. Terracina S, Petrella C, Francati S, et al. Antioxidant Intervention to Improve Cognition in the Aging Brain: The Example of Hydroxytyrosol and Resveratrol. *Int J Mol Sci.* 2022;23(24):15674. DOI: 10.3390/ijms232415674.
 25. Abrego-Guandique DM, Bonet ML, Caroleo MC, et al. The Effect of Beta-Carotene on Cognitive Function: A Systematic Review. *Brain Sci.* 2023;13(10):1468. DOI: 10.3390/brainsci13101468.
 26. Hendriks H, van de Rest O, Snippe A, Kieboom J, Hogenelst K. Alcohol Consumption, Drinking Patterns, and Cognitive Performance in Young Adults: A Cross-Sectional and Longitudinal Analysis. *Nutrients.* 2020;12(1):200. DOI: 10.3390/nu12010200.
 27. Dias IR, Santos CS, Magalhaes CODE, et al. Does calorie restriction improve cognition? *IBRO Rep.* 2020;9:37-45. DOI: 10.1016/j.ibror.2020.05.001.
 28. Daviglius ML, Plassman BL, Pirzada A, et al. Risk factors and preventive interventions for Alzheimer disease: state of the science. *Arch Neurol.* 2011;68(9):1185-90. DOI: 10.1001/archneurol.2011.100.
 29. Morgan TE, Xie Z, Goldsmith S, et al. The mosaic of brain glial hyperactivity during normal ageing and its attenuation by food restriction. *Neurosci.* 1999;89(3):687-99. DOI: 10.1016/s0306-4522(98)00334-0.
 30. Patel NV, Gordon MN, Connor KE, et al. Caloric restriction attenuates A β -deposition in Alzheimer transgenic models. *Neurobiol Aging.* 2005;26(7):995-1000. DOI: 10.1016/j.neurobiolaging.2004.09.014.
 31. Schroeder JE, Richardson JC, Virley DJ. Dietary manipulation and caloric restriction in the development of mouse models relevant to neurological diseases. *Biochem Biophys Acta.* 2010;1802(10):840-46. DOI: 10.1016/j.bbadis.2010.04.007.
 32. Luchsinger JA, Tang MX, Shea S, Mayeux R. Caloric intake and the risk of Alzheimer disease. *Arch Neurol.* 2002;59(8):1258-63. DOI: 10.1001/archneur.59.8.1258.
 33. Stranahan AM, Mattson MP. Impact of Energy Intake and Expenditure on Neuronal Plasticity. *Neuromol Med.* 2008;10(4):209-18. DOI: 10.1007/s12017-008-8043-0.
 34. Parrott MD, Greenwood CE. Dietary influences on cognitive function with aging: From high-fat diets to healthful eating. *Ann NY Acad Sci.* 2007;1114:389-97. DOI: 10.1196/annals.1396.028.
 35. Gomez-Pinilla F. The influences of diet and exercise on mental health through hormesis. *Ageing Res Rev.* 2008;7(1):49-62. DOI: 10.1016/j.arr.2007.04.003.
 36. Willcox BJ, Willcox DC, Todoriki H, et al. Caloric restriction, the traditional Okinawan diet, and healthy aging: The diet of the world's longest-lived people and its potential impact on morbidity and life span. *Ann NY Acad Sci.* 2007;1114:434-55. DOI: 10.1196/annals.1396.037.
 37. Ramey DR, Raynauld JP, Fries JF. The Health Assessment Questionnaire 1992: Status and Review. *Arthritis Care Res.* 1992;5(3):119-29. DOI: 10.1002/art.1790050303.
 38. Kumar N, Gupta N, Kishore J. Kuppuswamy's Socioeconomic Scale: Updating Income Ranges for the Year 2012. *Indian J Public Health.* 2012;56(1):103-04. DOI:10.4103/0019-557X.96988
 39. Gibson RS, Ferguson EL. An interactive 24-hr recall for assessing the adequacy of iron and zinc intakes in developing countries. Washington DC: International Life Sciences Institute (ILSI) Press; (Ed: Harvest Plus: Technical Monograph Series 8; c/o IFPRI). 2008;pp:1-161. ISBN 978-0-9818176-1-3.
 40. Ma Y, Olendzki BC, Pagoto SL, et al. Number of 24 hour diet recalls needed to estimate energy intake. *Ann Epidemiol.* 2009;19(8):553-59. DOI: 10.1016/j.annepidem.2009.04.010.
 41. Borson S, Scanlan J, Brush M, Vitaliano P, Dokmak A. The mini-cog: a cognitive "vital signs" measure for dementia screening in multi-lingual elderly. *Int J Geriatr Psychiatry.* 2000;15(11):1021-27. DOI: 10.1002/1099-1166(200011)15:11<1021::aid-gps234>3.0.co;2-6.
 42. Blessed G, Tomlinson BE, Roth M. The association between quantitative measures of dementia and of senile change in the cerebral grey matter of elderly subjects. *Brit J Psychiatry.* 1968;114(512):797-811. DOI: 10.1192/bjpp.114.512.797.
 43. Brooke P, Bullock R. Validation of a 6 item cognitive impairment test with a view to primary care usage. *Int J Geriatr Psychiatry.* 1999;14(11):936-40. PMID:10556864.
 44. Bharathi AV, Sandhya N, Vaz M. The development and characteristics of a physical activity questionnaire for epidemiologic studies in urban middle class Indians. *Ind Jour Med Res.* 2000;111:95-102. PMID:10937385.
 45. Food and Agriculture Organization of the United Nations, World Health Organization, eds. Human energy requirements: report of a Joint FAO/WHO/UNU expert consultation: Rome, 17-24 October 2001. Edition/Series: FAO Food and Nutrition Technical Report Series 1; Rome; 2004. ISBN 92-5-105212-3.
 46. Feng Y, Huang Y, Ma X. The application of Student's t -test in internal quality control of clinical laboratory. *Front Lab Med.* 2017;1(3):125-28. https://DOI.org/10.1016/j.flm.2017.09.002.
 47. Silva F.A.S., Azevedo C.A.V. Comparison of means of agricultural experimentation data through different tests using the software Assistat. *Afr J Agric Res.* 2016;11(37):3527-31. DOI: 10.5897/AJAR2016.11523
 48. Sacher GA. Evolution of Longevity and Survival Characteristics in Mammals. In: Schneider, E.L. (eds) "The Genetics of Aging". 1st ed. Springer New York, NY; 1978;pp:151-168. ISBN 978-1-4684-2445-4 (online)/ ISBN 978-1-4684-2447-8 (print). https://DOI.org/10.1007/978-1-4684-2445-4_7.
 49. Sacher GA, Duffy PH. Genetic relation of life span to metabolic for inbred mouse strains and their hybrids. *Fed Proc.* 1979;38(2):184-88. PMID:761651.
 50. Redman LM, Heilbronn LK, Martin CK, et al. Metabolic and behavioral compensations in response to caloric restriction: implications for the maintenance of weight loss. *PLoS One.* 2009;4(2):e4377. DOI: 10.1371/journal.pone.0004377.

51. Energy and protein requirements. Report of a joint FAO/WHO/UNU Expert Consultation. *World Health Organ Tech Rep Ser.* 1985;724:1-206. PMID:3937340.
52. Widdowson E. Biology of Human Starvation. *Nature.* 1952;170:177. <https://doi.org/10.1038/170177a0>.
53. Heilbronn LK, Ravussin E. Caloric restriction and aging: review of the literature and implications for studies in humans. *Am J Clin Nutr.* 2003;78(3):361-69. DOI: 10.1093/ajcn/78.3.361.
54. Sohal RS, Weindruch R. Oxidative Stress, Caloric Restriction and Aging. *Science.* 1996;273(5271):59-63. DOI: 10.1126/science.273.5271.59.
55. Ahmed T, Haboubi N. Assessment and management of nutrition in older people and its importance to health. *Clin Interv Aging.* 2010;5:207-16. DOI: 10.2147/cia.s9664.
56. Solfrizzi V, Panza F, Capurso A. The role of diet in cognitive decline. *J Neural Transm.* 2003;110(1):95-110. DOI: 10.1007/s00702-002-0766-8.
57. Miekle A, Riby LM, Stollery B. The impact of glucose ingestion and gluco-regulatory control on cognitive performance: a comparison of younger and middle aged adults. *Hum Psychopharmacol.* 2004;19(8):523-35. DOI: 10.1002/hup.643.
58. Booth FW, Gordon SE, Carlson CJ, Hamilton MT. Waging war on modern chronic diseases: primary prevention through exercise biology. *J Appl Physiol.* 2000;88(2):774-87. DOI: 10.1152/jappl.2000.88.2.774.
59. Myers J, Prakash M, Froelicher V, Do D, Partington S, Atwood JE. Exercise capacity and mortality among men referred for exercise testing. *N Engl J Med.* 2002;346(11):793-801. DOI: 10.1056/NEJMoa011858.
60. Larson EB, Wang L, Bowen JD, et al. Exercise is associated with reduced risk for incident dementia among persons 65 years of age and older. *Ann Int Med.* 2006;144(2):73-81. DOI: 10.7326/0003-4819-144-2-200601170-00004.
61. Van Loon LJ, Greenhaff PL, Constantin-Teodosiu D, Saris WH, Wagenmakers AJ. The effects of increasing exercise intensity on muscle fuel utilization in humans. *Jour Physiol.* 2001;536(1):295-304. DOI: 10.1111/j.1469-7793.2001.00295.x.
62. Melzer K. Carbohydrate and fat utilization during rest and physical activity, e-SPEN, *Eur e-Jour Clin Nutr Metab.* 2011;6(2):e45-e52. <https://doi.org/10.1016/j.eclnm.2011.01.005>.
63. Ortega RM, Requejo AM, Andres P, et al. Dietary intake and cognitive function in a group of elderly people. *Am J Clin Nutr.* 1997;66(4):803-09. DOI: 10.1093/ajcn/66.4.803.
64. Payne ME. Nutrition and late-life depression: etiological considerations. *Aging health.* 2010;6(1):133-43. DOI: 10.2217/ah.09.90.
65. Meeusen R. Exercise, nutrition and the brain. *Sports Med.* 2014;44(Suppl 1):47-56. DOI: 10.1007/s40279-014-0150-5.

PEER-REVIEWED CERTIFICATION

During the review of this manuscript, a double-blind peer-review policy has been followed. The author(s) of this manuscript received review comments from a minimum of two peer-reviewers. Author(s) submitted revised manuscript as per the comments of the assigned reviewers. On the basis of revision(s) done by the author(s) and compliance to the Reviewers' comments on the manuscript, Editor(s) has approved the revised manuscript for final publication.