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Retrospective Cohort Study

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Stress-Associated Cognitive Impairments amongst Faculty of a Southern Nigeria University: Investigating the Effects of Perceived Stress on Cognition Scores

Promise Tamunoipiriala Jaja[1,2*] *and Datonye Victor Dapper*[1]

[1]*Department of Human Physiology, College of Health Sciences, University of Port Harcourt, Nigeria*

[2]*Department of Neurological Surgery, University College Hospital, Ibadan, Nigeria*

Abstract

Stress-induced cognitive-decline is emphasized by stress-hormone receptors presence in brain cognition-control areas in animal models; human studies are few. We established the burdens of stress and mild cognitive impairments and their causal associations amongst faculty of University of Port Harcourt. Using a two-phased retrospective cohort study, 115 subjects (28 were controls; others were recruited following multi-staged sampling of University of Port Harcourt lecturers) were examined using the ten-item PSS and MOCA-B scales for perceived-stress levels and mild cognitive impairments (MCI) respectively. Unlike the controls ($Chi^2 = 0.70$, $p > 0.05$), the lecturers showed significant ($Chi^2 = 8.23$; $p < 0.05$ and $F = 14.50$; df 2.84; $p = 0.01$) association between stress and MCI using Chi-squared and age-controlled multiple linear regression. The mean stress and cognition scores were dissimilar ($p > 0.05$) for the study groups, with 16.82 ± 4.98 and 26.32 ± 1.67 respectively for the main study. Of 62.0% of the respondents with above-average perceived-stress, 37% had MCI; unlike 9.1% amongst those with below-average perceived-stress and 26.4% for the whole sample. The MCI development risk ratio was 4:1. The predominantly above-average perceived-stress levels amongst lecturers were positively associated with the presence of mild cognitive impairments, even when age-controlled. A four-fold additional risk of developing MCI exists for the stressed lecturers.

Keywords

Stress, Cognitive impairment, Mild cognitive impairment, Human stress, Neuroendocrinology

Introduction

Background

Stress is of multi-factorial origin physiological, physical or psychological [1-3] and triggers a central cascade according to Hans Selye's general adaptation syndrome, which was criticised for using physical stressors [2]. John Mason set to correct this by deducing psychological stressors that potentially override physical stressors and highlighted cardinal features of a putative stressor as being *novel*, *unpredictable*, show *feeling of lack of control over the situation* and/or *a threat to a socially evaluative situation* [2].

Stress describes the infinite spectrum of demands [2,3] and the comprehensive response to demands [4] on man. ***Absolute stressors*** always elicit the stress response, unlike ***relative stressors*** that do so at times; a phenomenon exploited where some stressors are perceived as non-stressful [2].

Stress triggers the hypothalamo-pituitary-adrenal (HPA) axis, ultimately elaborating glucocorticoids and adrenaline [2,3,5] which increase blood glucose, peripheral vascular resistance, cardiac inotropy and chronotropy, amongst other [2] actions to provide energy.

Receptors for the mediators of the stress response from the adrenal cortex (glucocorticoids and mineralocorticoids especially) have been identified in the areas mediating the cognitive faculties of learning and memory (amygdala, hippocampus, pre-frontal cortex, medial pre-frontal and orbitofrontal cortical areas) putting these cognition control centres under possible control by stress and stress hormones [6]. Glucocorticoid receptors are present in virtually all cells in the body [2,3,5,7]. The stress mediators from the adrenal medul-

***Corresponding author:** Dr. Jaja Promise Tamunoipiriala, Department of Neurological Surgery, University College Hospital, Ibadan, Nigeria

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la (Adrenaline) have receptors that have also been identified in the Amygdala (limbic system area with roles in fear and emotionally relevant memory), even though the catecholamines are unable to cross the blood-brain-barrier [2]. The receptors for norepinephrine and glucocorticoids have also been identified in the glial cell types [8].

The elevation of glucocorticoids [either from exposure to stress or exogenously [9] have been linked with declarative memory deficits [10-12]. There have also been reports of improved memory when exogenously administered in the afternoon (when the normal circadian blood levels of cortisol is low in man), it is thought to aid in memory consolidation by spreading the salient parts of events to the hippocampus via activation of the baso-lateral amygdala, partly due to activation of its noradrenergic connections [5]. The effects of the glucocorticoids on memory processes have been reported to go beyond the hippocampus [13], although hippocampal signalling pathways have been shown to be a mediator of stress-induced cognitive deficits in rats [14]; putatively by glucocorticoids ultimately causing major synaptic deficits in the hippocampus [15]. Stress ultimately stimulates recall of emotional memories while it impairs the formation of non-emotional memories [16] in addition to reducing the pre-frontal cortex dependent task-related cognitive activities in wistar rats [17]. Essentially, the stress-induced cognitive deficits are mediated through the ability of stress to activate the HPA axis [18].

Rationale

Stress-induced and/or stress-hormone induced cognitive deficits have been shown in animal studies [6,9,10,19-26]. This is corroborated in animal models with the presence of organic deficits in the brain areas (especially the limbic system) controlling cardinal cognitive faculties [3,5,7,9]. These changes have been linked to the initiation of neurodegenerative changes in animals as well [3,27,28]. There is paucity of data on this subject in human studies, although some reports exist describing cognitive deficits in the stressed; the same were replicated in humans using glucocorticoids [7,10] and noradrenaline [29].

Cognitive deficits are reported from stressors occurring prenatally [23,30], early-in-life [3,19,23], including childhood maternal deprivation [19,31] and childhood maltreatment [32-34]; from anaesthesia [35], chronic immobilization [6], chronic social defeat [36], illness-related [37-40], noise [41-43], sleep deprivation [19,22] and trauma [24,26,44-47]. Human stress and its causation of mild cognitive impairment is reported as being intermediary in the development of neurodegeneration [48] and dementias [49].

Justification

High stress levels reported in Nigerian teachers (secondary schools and universities) [50,51] presents the possible development of cognitive impairments in Nigerian University's faculty. Stress's multi-aetiological origin worsens this [2] as people are exposed to multiple stressors, putatively initiating an iteration loop as stressed individuals potentially develop cognitive deficits, resulting in unsatisfactory work output, culminating in more stress (especially from reprimanding) and so goes the loop. It's imperative to assess the burden of stress and its possible cognition impairing effects to support provision of solutions.

Hypothesis and objectives

We tested the hypothesis, *if high levels of perceived-stress amongst university faculty is associated with increased serum levels of stress hormones (glucocorticoids and adrenaline), then stress-associated cognitive impairments will occur from the effects of stress hormones on the brain cognition control areas (prefrontal cortex, hippocampus, amygdala, etc)*. The assessment of the hypothesis involved evaluating perceived-stress levels and the cognition scores of subjects. The study aimed to determine the prevalence of above-average perceived-stress, to elucidate the prevalence of mild cognitive impairments (MCI), to establish the proportion of respondents with above-average perceived-stress that exhibit MCI and to determine the presence of a causal relationship between above-average perceived-stress and MCI amongst faculty in the University of Port Harcourt, Nigeria.

Materials and Methods

Study design

The study set in the University of Port Harcourt followed a **retrospective cohort** study design, using academic staff of the institution as the **study population**. The respondents were assessed for the prior existence of perceived stress; for this study, a tool testing the presence of stress in the previous month was used, this put the exposure at a retrospective location in time despite the potential cross-sectional nature of the data collection at baseline.

Study sample

A ***pilot study*** was done to serve as ***controls*** using twenty eight respondents; these were House officers (lowest cadre medical doctors) from the Braithwaite Memorial Specialist Hospital in Port Harcourt. Essentially, the pilot study respondents were a youthful group with significant level of education and anecdotally expected to have significant work stress by virtue of their placement in the clinical echelon, as lowest rung medical practitioners; they are often charged with executing all the low-level tasks in the various clinical services they work in, a responsibility that translates to longer work hours and work intensities, responsibilities they hitherto have not been exposed to (a novel and fairly unpredictable situation), although they do not have teaching responsibilities. These nascent doctors being largely youthful are expectedly of reduced predisposition to chronic illnesses (e.g., hypertension, diabetes mellitus).

Study eligibility criteria

The criteria for inclusion and exclusion of the academic staff of the University for the Study are presented below:

Inclusion Criteria include consenting:

- Academic staff of the University of Port Harcourt
- Aged between 25 and 70 years of age.

Exclusion Criteria:

- All non-academic staff of the University of Port Harcourt.
- Academic staff of the University of Port Harcourt that were younger than 25 years of age or greater than 70 years (albeit these would have been retired from service); also excluding contract, sabbatical, adjunct and emeritus staff.
- All academic staff with moribund ailment that was likely to significantly constitute a confounder as a stressor. These persons would in fact not be fit to work and can't be seen in the work environment.
- All individuals suspected of being mentally ill and persons who volunteered information, suggesting that a pre-study mental ailment existed that may confound cognitive deficits.

Sampling technique

The study sample was selected using a **multi-stage sampling technique**. Stage one was a clustered sampling of the faculties (schools as they are called) of the University of Port Harcourt from which three clusters were selected from the eleven faculties. Second stage was by clustered sampling of the departments in the selected faculties (basic medical sciences, engineering and humanities). Third stage was via simple random sampling, here the subjects were selected by simple random sampling of the academic staff of the selected departments from the second stage, using the list of staff in the departments organized alphabetically and assigned numbers, numbers corresponding to the study subjects were generated using the first digit from random numbers generated from a scientific calculator. In the event of the generation of a previously selected number, the first two digits were taken, if the number generated exceeds the available number of assigned staff numbers, the first two numbers were added. This sequence was repeated until a new number is generated. Attempts at contacting all the recruited respondents were made and those unreached were replaced by generation of an alternate number.

Sample size determination

Sample size was estimated using Epi-Info™ 7.1.5.2., using the unmatched cohort and cross-sectional studies option, the two-sided confidence level was set to 95%, power to 90%, ratio of the unexposed to the exposed group to 2, percentage outcome in the unexposed group to 30% and percentage outcome in the exposed group to 60%; the sample size yielded was a minimum of 112 respondents.

Study period

Study period was between October and December 2016 for the pilot (control) study and the main study was between April and August 2017.

Confounders and bias

Confounders were excluded as much as possible, like advancing age (controlled for statistically), pre-existing morbidities capable of altering cognitive capacity (dementias and other neurodegenerative disorders) or that may suppose that there may have been illness related physiological demands (hypertension, diabetes mellitus, malignant neoplasms especially at the metastatic stage).

Study instruments

Study Instruments used were the 10-item Perceived Stress Scale (PSS-10) to assess the perception of the level of stressful circumstances the respondents had been exposed to within the month prior to testing, thus it presents stress-level scores indicative of the extent of stress the respondent was exposed to at least a month prior to the assessment. It measures items in a likert-scale manner on a scale of 0-4, representing responses of 'never', through to 'very often'; except for items 4, 5, 7, 8 for which the scoring is reversed. All the responses are summed up to get the final available scores. Its scores range between 0-40. Its use for research purposes is granted without permission requests and it has been validated [52,53]. The Montreal Cognition Assessment Basic (MOCA-B) test was also used for the assessment of the mild cognitive impairment status of the subjects. It tests various cognitive domains like reasoning, memory (short term), calculation, abstraction, language, concentration, objects identification etc. It has a score range between 0-30; a score less than 26 is indicative of mild cognitive impairment. Adjustments made for level of education include adding a point to the total score if the subject has less than four years of education or illiterate. It is accessible without permission online at www.mocatest.org; however, permission was requested for and received from the MOCA clinic, Quebec, Canada to use the test for this study, after their review of an abridged study proposal. The study data were summarized in the study proforma.

Study approvals

Ethical approval was received from the research ethics committee of the University of Port Harcourt with reference number UPH/R&D/REC/04, prior to commencing the study. Written permission was gotten also from the MOCA clinic and institute, Quebec, Canada to use the Montreal cognitive assessment for this study.

Study variables and measures

The **study variables** were both categorical and continuous; the categorical included the binary outcomes on the nominal scale for the retrospective above average perceived stress status and the cognitive impairment status, the stress scale score grade was also categorical on the ordinal scale. The continuous variables were the stress scale score on a ratio scale as well as the MOCA score also on a ratio scale.

Study measures were the ***exposure (retrospective above-average perceived-stress)*** and the ***outcome (mild cognitive impairment)***.

Data analyses

For statistical analysis, data from the subjects were col-

lated and sorted before being entered into a spreadsheet, followed by entry into statistical programs, Epi-Info™ 7.1.5.2. (Centers for Disease Control and Prevention, Atlanta, USA) and STATA/IC 15.1™ (StatsCorp LLC, Texas, USA) for analyses. Proportions were generated for the categorical variables; means, standard deviations and standard errors were estimated for the continuous variables. Causality was assessed for using contingency tables and the cohort study analysis commands on STATA/IC 15.1™ yielding the chi-squared statistic, risk data as well as other measures of association (multiple linear regression; age-controlled). The regression models were also computed. Significant P value was set a values less than 0.05.

Study limitations

The sample size was not large because, there was a time frame restriction for data collection and many respondents recruited from the sampling considered the stress and cognition aasessments as strenous, hence declining consent.

Results

This study using 115 subjects in a retrospective cohort study showed that the 28 subjects in the pilot study (control) group were evenly distributed amongst the sexes, unlike the main study group that showed male (60.92%) preponderance as presented in Table 1 and Table 2. As presented in Table 3, the average perceived-stress scores in both groups showed no significant difference (p = 0.10); albeit the average perceived-stress scores were above-average, with 16.82 for the academic staff of the University of Port Harcourt. About 62.00% of the main study subjects had above-average perceived-stress scores (as depicted in Table 4 and Table 5), most (59.77% of 62.07%) of these had grade I and II stress scores as presented in Table 5. The average cognition scores (as shown in Table 3) also showed no statistically significant difference (p = 0.29) across the study groups. Thirty two point one four percent (that is about 1 in 3) of the subjects in the pilot study group unlike 26.44% (that is about 1 in 4) of the main study group subjects had MCI; these are shown in Table 5.

Findings from Table 6 show that there were no significant (p > 0.05) association in the control group, unlike (Chi^2 = 8.23; p = 0.01) the main study group; although, nearly 90% of the main study subjects with MCI were stressed as in Table 4. Table 7 showed that subjects with high stress levels showed a four times more risk of developing MCI than the unstressed; above average perceived stress was the sole likely cause of MCI in twenty eight percent of the subjects that were stressed (with the attributable fraction for the exposed as seen in Table 7); about seventeen percent of the whole study sample were likely to develop MCI due solely to stress (as shown from the attributable fraction for the study population in Table 7). Results following multiple linear regression (controlled for age) showed the presence of statistically significant F = 14.50; df 2.84; p = 0.01) association between perceived stress and the presence of mild cognitive impairment as presented in Table 7.

The age controlled multiple regression analysis done and reported in Table 7 to test for an association between the

Table 1: Main study group age-sex distribution.

	Sex		
Age groups (in years)	**Male**	**Female**	**Total**
20-29	6 (46.15)	7 (53.85)	**13 (14.94)**
30-39	26 (63.41)	15 (36.59)	**41 (47.13)**
40-49	14 (60.87)	9 (39.13)	**23 (26.44)**
50-59	5 (62.50)	3 (37.50)	**8 (9.20)**
60 and above	2 (100.00)	0 (00.00)	**2 (2.30)**
Total	**53 (60.92)**	**34 (39.08)**	**87 (100.00)**

Values presented as absolute and percentages in parenthesis.

Table 2: Pilot study group age-sex distribution.

	Sex		
Age groups (in years)	**Male**	**Female**	**Total**
20-24	0 (0.00)	2 (100.00)	**2 (7.14)**
25-29	7 (38.89)	11 (61.11)	**18 (64.29)**
> 30	7 (87.50)	1 (12.50)	**8 (28.57)**
Total	**14 (50.00)**	**14 (50.00)**	**28 (100.00)**

Values presented as absolute and percentages in parenthesis.

Table 4: Proportions of the main study subjects based on perceived stress scores and mild cognitive impairment statuses.

Mild cognitive impairment status	**Above average stress score status**		
	Exposed‡	**Unexposed‡**	**Total†**
Cases‡	20 (37.04) **86.96**	3 (9.09) **13.04**	**23 (0.2644)**
Non-cases‡	34 (62.96) **53.13**	30 (90.91) **46.88**	**64 (0.7356)**
Total	54 (0.6207)	33 (0.3793)	**87 (1.0000)**

†Values presented as 'frequency (proportions)'; ‡Values presented as 'frequency (column percentage) **row percentage**'.

Table 3: Summary statistics of the PSS and MOCA scores.

	Means		**Standard deviation**		**Standard error**		**Two-sample z-test for pilot versus main study**	
	Pilot (n = 28)	**Main (n = 87)**	**Pilot (n = 28)**	**Main (n = 87)**	**Pilot (n = 28)**	**Main (n = 87)**	**P value**	**Significance**
PSS score	16.821	16.816	5.930	4.983	1.130	0.534	0.997	No
MOCA Score	26.82	26.32	2.326	1.667	0.440	0.179	0.292	No

PSS: Perceived stress scale; MOCA: Montreal cognitive assessment.

Table 5: Distribution of the proportions of the above average stress score status, grades of the PSS scores and based on MCI status amongst the pilot and **main** study groups.

		Frequency (percentages)	**Standard error**
	Yes	17 (60.71)	0.094
		54 (62.07)	**0.052**
	No	11 (39.29)	0.094
		33 (37.93)	**0.052**
	Total	28 (100.00)	
Above average stress score status		**87 (100.00)**	
	0	11 (39.29)	0.094
		33 (37.93)	**0.052**
	I	7 (25.00)	0.083
		27 (31.03)	**0.05**
	II	8 (28.57)	0.087
		25 (28.74)	**0.049**
	III	2 (7.14)	0.05
		2 (2.30)	**0.016**
	Total	28 (100.00)	
Grade of PSS score		**87 (100.00)**	
	Yes	9 (32.14)	0.09
		23 (26.44)	**0.048**
	No	19 (67.86)	0.09
		64 (73.56)	**0.048**
	Total	28 (100.00)	
MCI status		**87 (100.00)**	

Values in bold characters represent those from the main study group; those not in bold characters represent values from the pilot study groups. PSS: Perceived stress scale, MCI: Mild cognitive impairment

Table 6: Contingency table, cohort study and risk data analysis of the main study group, based on MCI and above-average perceived stress statuses.

		Above-average perceived stress status			
		Exposed	**Unexposed**	**Total**	
Mild cognitive impairment status	**Cases**	20	3	**23**	
	Non-cases	34	30	**64**	
	Total	**54**	**33**	**87**	
	Risk	0.3704	0.9091	0.2644	
		Point estimate		**95% Confidence interval**	
Risk difference		0.2795		0.1176	0.4414
Risk ratio		4.0704		1.3114	12.6572
Attributable fraction in exposed		0.7546		2.3743	0.9210
Attributable fraction in population		0.6561			
		Chi^2 = 8.23 Pr > Chi^2 = 0.0041			

Perceived Stress status (average and below or above average) and MOCA score variables controlling for age (a principal confounder for cognitive decline), testing the hypothesis whether there is a statistically significant association between *hipss* and *mocascore* controlling for *Age*. Here the coefficient of interest is β_1= *Coefficient of stress score*; here, the *hipss* = 0 represents subjects without above average stress scores and *hipss* = 1 represents those with above average stress scores. The regression equation for this analysis is as follows:

$$mocascore_i = \beta_0 + \beta_1 * hipss_i + \beta_2 * Age_i + e_i$$

The filled out regression equation from the analysis is:

Table 7: Multiple linear regression of the *mocascore* and *hipss* controlling for *Age* in the main study group.

Source	**SS**	**df**	**MS**	**Number of observations**		87
				F (2, 84)		14.50
Model	61.3182	2	30.6591	**Prob > F**		0.0001
Residual	177.6703	84	2.1151	**R-squared**		0.2566
Total	**238.9885**	**86**	**2.7789**	**Adjusted R-squared**		0.2389
				Root MSE		1.4543
mocascore	**Coefficient**	**Standard error**	**t**	**P > t**	**95% Confidence interval**	
hipss	-1.7128	0.3247	-5.28	0.001	-2.3584	-1.0671
Age	-0.0059	0.1883	-0.31	0.754	-0.0434	0.0315
Constant	27.6085	0.7289	37.88	0.001	26.1591	29.058

mocascore: Montreal cognitive assessment score; Hipss: Above-average perceived stress score status.

$mocascore_i = 27.61-1.71*hipss_i-0.006*Age_i$

Discussion

Perceived stress scale scores discussion

The average perceived-stress score of the academic staff of University of Port Harcourt was 16.82, it showed no statistically significant difference ($p > 0.05$) when compared to that of the pilot study group; it was however higher than the 14.70 reported by Cohen [52] for Negroes in their probability sample of Americans using the Perceived Stress Scale, thus the academic staff had averagely above average stress scores; this finding was corroborated by the works of Omoniyi and Ogunsami [51] and Ekundayo & Kolawole [50] who reported high levels of stress amongst University lecturers and teachers in secondary schools in South-Western Nigeria. The teaching staff in Nigeria are thought to be exposed to multiple stressors, some peculiar to their professions and others related to the general challenges of the Nigerian environment. They are exposed to enormous teaching chores, compounded with the straining teacher-to-student ratio, persistent complaints of abyssmal remuneration packages, amidst the other non-specific challenges from the environment.

The distribution of the above 60% - in disparity with the works of Khan and colleagues who reported about 36% of their sample as being stressed [54] - of the subjects with above-average stress scores was such that, they were almost evenly distributed amongst the grades I and II stress score groups but for a negligible 3.7% of the academic staff of the University with grade III stress scores. Khan and colleagues [54] reported an almost 2:1 relationship between the high and very high stress groups as the distribution for the respondents in their student population with above-average stress levels. The large number of the subjects reporting above average stress levels is not unexpected as presented in the paragraph immediately above; such that either one or a combination of stressors will be present in each individual. The fact that the study using students reported a lower stress prevalence is also expected as the burden from life's stressors were most like transferred to the parents or guardians of the students.

Cognitive assessment scores discussion

The mean *mocascores* for the pilot and main study groups showed no statistically significant difference ($p > 0.05$). A third of the subjects in the pilot study group had mild cognitive impairments; unlike about a quarter of the academic staff of the University of Port Harcourt with mild cognitive impairments.

The gender dynamics for developing MCI was statistically significant ($chi^2 = 8.23$; $p < 0.05$) in favour of higher risks in the males, with a 3:1 Male:Female ratio. These findings were in discord with the works of Katz and colleagues [55] who reported that the American septuagenarian women studied were more likely to develop amnesic MCI when stressed; the findings of Kluen and colleagues [29] were also in disagreement with our findings as the female respondents (from Hamburg, Germany) in their study exhibited more generalization memory deficits when stressed than their male counterparts; here they received adrenrenoceptor stimulation, via the use of yohimbine. Wright and colleagues had reported no gender disparity between stress's (noisy conditions) induced cognitive impairments in their study using healthy individuals in England [43]. Our study had male preponderance, this may have conferred males more chances for displaying MCI, and however the discussed literature presented Caucasian works.

Associations between above average stress scores and cognitive impairment

The pilot study group showed no significant ($p > 0.05$) association between the above-average stress levels and the presence of MCI, this was the reverse ($Chi^2 = 8.23$; $p < 0.05$) amongst the lecturers in University of Port Harcourt. Most (86.96%) of the main study group subjects with MCI reported above-average stress scores; many (60%) of whom had grade II stress scores, with about a third (35%) with grade I and five percent (5%) having grade III stress scores. The subjects with above average stress levels had four times the risk of developing MCI than their unstressed colleagues. Twenty eight percent (28%) of the subjects with above average stress scores were likely to develop MCI from factors related to stress only; it however was true that 17.4% of the academic staff were likely to develop MCI of causes solely due to stress. Katz and colleagues [55] had in 2016 reported that the PSS score was

able to predict onset of amnesic MCI, expressing that this became more apparent after a three year follow-up period in their elderly subjects; a finding supporting the associations between PSS derived stress scores and MCI in this study. The perceived-stress scores represent a retrospective assessment of the levels of stress with the past month, hence the subjects reporting above average stress levels, correlate putatively with elevated serum levels of the stress hormones. The theoretical ensuing cognitive decline from these stress hormone surges is seen as mild cognitive impairments in our subjects; as reported by Mo and colleagues as well as Zborowski using animal models [11,12]. The associations between high stress levels and cognitive deficits from synaptic disruptions and volume reduction in the hippocampus [13,15] as well prefrontal cortex changes [6] have been shown in animal models.

Some (34%) of the participants with above-average perceived-stress did not have MCI; representing 63% of the participants with above average stress and 53% of participants without MCI. Putatively, these persons may have better coping strategies that prevent the stressors from evoking the stress response, the stress hormones elaborated failed to significantly alter cognitive functions or they may engage in activities (like optimal exercise, satisfactorily optimized environment conditions) that ameliorate or rescind stress's effects on cognition; these were in concert with reports from Shilpa and colleagues [6] and Wearick-Silva [31].

Regression analysis (using multiple linear regression) controlling for age, established significant association between the dichotomous predictor perceived stress variable (presence or absence of above average stress levels) and the continuous dependent variable - cognitive assessment score (mocascore), controlling for age (a confounder for cognition impairment).

Conclusion

Academic staff of the University of Port Harcourt reported above-average perceived stress score levels averagely; although the average cognitive assessment scores were indicative of absence of mild cognitive impairment. Above average stress scores were positively associated with the presence of mild cognitive impairment; even when controlled for age. There is a four times additional risk of developing mild cognitive impairments in academic staff with above average stress scores. Almost 90% of the subjects with mild cognitive impairment also had above average stress levels.

The cognitive assessment score for an individual can be estimated using:

$$mocascore_i = 27.61 - 1.71 * hipss_i - 0.006 * Age_i$$

Further studies to accumulate evidence for stress's cognitive impairing properties with adjustments for confounders; possibly using the randomized clinical trial design as much as ethically permissible is required. Stratification of the risk capacity of various stressors may help policy makers in deciding the more consequential stressors to combat on a population basis.

Declaration of Interests Statement

No conflict of interests to disclose. The authors received no external funding to execute this study.

References

1. Guez J, Rotem SA, Keha E, et al. (2016) The effect of trier social stress test (TSST) on item and associative recognition of words and pictures in healthy participants. Front Psychol 7: 507.
2. Lupien SJ, Maheu F, Tu M, et al. (2007) The effects of stress and stress hormones on human cognition: Implications for the field of brain and cognition. Brain Cogn 65: 209-237.
3. Lupien SJ, McEwen B, Gunnar MR, et al. (2009) Effects of stress throughout the lifespan on the brain, behaviour and cognition. Nat Rev Neurosci 10: 434-445.
4. Staal M (2004) Stress, cognition, and human performance: A literature review and conceptual framework. National Aeronautics and Space Administration, California.
5. Erickson K, Drevets W, Schulkin J (2003) Glucocorticoid regulation of diverse cognitive functions in normal and pathological emotional states. Neuroscience & Biobehavioral Reviews 27: 233-246.
6. Shilpa B, Bhagya V, Harish G, et al. (2017) Environmental enrichment ameliorates chronic immobilisation stress-induced spatial learning deficits and restores the expression of BDNF, VEGF, GFAP and glucocorticoid receptors. Prog Neuropsychopharmacol Biol Psychiatry 76: 88-100.
7. Judd L, Schettler P, Brown E, et al. (2014) Adverse consequences of glucocorticoid medication: Psychological, cognitive and behavioural effects. Am J Psychiatry 171: 1045-1051.
8. Pearson LJ, Osborne D, McNay E (2016) Role of glia in stress-induced enhancement and impairment of memory. Front Integr Neurosci 9: 63.
9. Dachir S, Kadar T, Robinzon B, et al. (1993) Cognitive deficits induced in young rats by long-term corticosterone administration. Behav Neural Biol 60: 103-109.
10. Coluccia D, Wolf O, Kollias S, et al. (2008) Glucocorticoid therapy-induced memory deficits: Acute versus chronic effects. J Neurosci 28: 3474-3478.
11. Mo C, Pang T, Ransome M, et al. (2014) High stress hormone levels accelerate the onset of memory deficits in male Huntington's disease mice. Neurobiol Dis 69: 248-262.
12. Zborowski V, Sari M, Heck S, et al. (2016) p-Chloro-diphenyl diselenide reverses memory impairment-related to stress caused by corticosterone and modulates hippocampal [^{3}H]glutamate uptake in mice. Physiology & Behavior 164: 25-33.
13. Atsak P, Guenzel F, Kantar GD, et al. (2016) Glucocorticoids mediate stress-induced impairment of retrieval of stimulus-response memory. Psychoneuroendocrinology 67: 207-215.
14. Sardari M, Rezayof A, Khodagholi F (2015) Hippocampal signaling pathways are involved in stress-induced impairment of memory formation in rats. Brain Research 1625: 54-63.
15. Kvarta M, Bradbrook K, Dantrassy H, et al. (2015) Corticosterone mediates the synaptic and behavioral effects of chronic stress at rat hippocampal temporoammonic synapses. J Neurophysiol 114: 1713-1724.
16. Arnau BG, Maria GG, Srivastava RK, et al. (2016) Peripheral and

central CB1 cannabinoid receptors control stress-induced impairment of memory consolidation. Proc Natl Acad Sci U S A 113: 9904-9909.

17. Devilbiss D, Spencer R, Berridge C (2016) Stress degrades prefrontal cortex neuronal coding of goal-directed behavior. Cerebral Cortex 27: 2970-2983.

18. Hood A, Pulvers K, Spady T, et al. (2015) Anxiety mediates the effect of acute stress on working memory performance when cortisol levels are high: A moderated mediation analysis. Anxiety Stress Coping 28: 545-562.

19. Alzoubi K, Malkawi B, Khabour O, et al. (2017) *Arbutus andrachne* L. reverses sleep deprivation-induced memory impairments in rats. Molecular Neurobiology 55: 1150-1156.

20. Bhagya V, Christofer T, Shankaranarayana RBS (2016) Neuroprotective effect of *Celastrus paniculatus* on chronic stress-induced cognitive impairment. Indian Journal of Pharmacology 48: 687-693.

21. Kato H, Tsuji M, Miyagawa K, et al. (2016) Ethanol withdrawal-induced impaired recognition is reversed by chronic exposure to stress and the acute administration of corticosterone in mice. Biol Pharm Bull 39: 1631-1637.

22. Lu C, Shi Z, Dong L, et al. (2017) Exploring the effect of ginsenoside Rh1 in a sleep deprivation-induced mouse memory impairment model. Phytother Res 31: 763-770.

23. Pascuan C, Di Rosso M, Pivoz-Avedikian J, et al. (2017) Alteration of neurotrophin and cytokine expression in lymphocytes as novel peripheral markers of spatial memory deficits induced by prenatal stress. Physiol Behav 173: 144-155.

24. Patarroyo W, García-Perez M, Lamprea M, et al. (2017) Vibrissal paralysis produces increased corticosterone levels and impairment of spatial memory retrieval. Behavioural Brain Research 320: 58-66.

25. Ranjbar H, Radahmadi M, Alaei H, et al. (2016) The effect of basolateral amygdala nucleus lesion on memory under acute, mid and chronic stress in male rats. Turk J Med Sci 46: 1915-1925.

26. Stricker N, Lippa S, Green D, et al. (2017) Elevated rates of memory impairment in military service-members and veterans with posttraumatic stress disorder. J Clin Exp Neuropsychol 39: 768-785.

27. Wolozin B, Behl C (2000) Mechanisms of neurodegenerative disorders: Part 1: Protein aggregates. Arch Neurol 57: 793-796.

28. Wolozin B, Behl C (2000) Mechanisms of neurodegenerative disorders: Part 2: Control of cell death. Arch Neurol 57: 801-804.

29. Kluen L, Agorastos A, Wiedemann K, et al. (2017) Noradrenergic stimulation impairs memory generalization in women. J Cogn Neurosci 29: 1279-1291.

30. Lee Y, Kim Y, Goto Y (2016) Cognitive and affective alterations by prenatal and postnatal stress interaction. Physiol Behav 165: 146-153.

31. Wearick-Silva L, Marshall P, Viola T, et al. (2017) Running during adolescence rescues a maternal separation-induced memory impairment in female mice: Potential role of differential exon-specific BDNF expression. Dev Psychobiol 59: 268-274.

32. Burri A, Maercker A, Krammer S, et al. (2013) Childhood trauma and PTSD symptoms increase the risk of cognitive impairment in a sample of former indentured child laborers in old age. PLoS One 8: e57826.

33. Cowell R, Cicchetti D, Rogosch F, et al. (2015) Childhood maltreatment and its effect on neurocognitive functioning: Timing and chronicity matter. Dev Psychopathol 27: 521-533.

34. Cromheeke S, Herpoel L, Mueller S (2014) Childhood abuse is related to working memory impairment for positive emotion in female university students. Child Maltreat 19: 38-48.

35. Liu G, Zhu T, Zhang A, et al. (2016) Heightened stress response and cognitive impairment after repeated neonatal sevoflurane exposures might be linked to excessive GABAAR-mediated depolarization. J Anesth 30: 834-841.

36. Duque A, Vinader CC, Monleón S (2017) Indomethacin counteracts the effects of chronic social defeat stress on emotional but not recognition memory in mice. PLoS One 12: e0173182.

37. Fayette D, Gahérová Ľ, Móciková H, et al. (2017) Chemotherapy-related cognitive impairment in patients with hodgkin lymphoma - pathophysiology and risk factors. Klin Onkol 30: 93-99.

38. Rubin L, Meyer VJ, Conant R, et al. (2016) Prefrontal cortical volume loss is associated with stress-related deficits in verbal learning and memory in HIV-infected women. Neurobiol Dis 92: 166-174.

39. Rubin L, Wu M, Sundermann E, et al. (2016) Elevated stress is associated with prefrontal cortex dysfunction during a verbal memory task in women with HIV. J Neurovirol 22: 840-851.

40. Tanimukai H (2015) Cancer-related cognitive impairment: Current knowledge and future challenges. Seishin Shinkeigaku Zasshi 117: 585-600.

41. Azman K, Zakaria R, Abdul AC, et al. (2016) Tualang honey attenuates noise stress-induced memory deficits in aged rats. Oxid Med Cell Longev 2016: 1549158.

42. Hu L, Han B, Zhao X, et al. (2016) Chronic early postnatal scream sound stress induces learning deficits and NMDA receptor changes in the hippocampus of adult mice. Neuroreport 27: 397-403.

43. Wright B, Peters E, Ettinger U, et al. (2016) Moderators of noise-induced cognitive change in healthy adults. Noise Health 18: 117-132.

44. Cui J, Guo Y, Yang W, et al. (2018) Effects of exercise on learning and memory, oxidative stress and nNOS expression in marginal division of striatum of the ovariectomized rats. J Sports Med Phys Fitness 58: 356-365.

45. Patki G, Allam F, Atrooz F, et al. (2013) Grape powder intake prevents ovariectomy-induced anxiety-like behavior, memory impairment and high blood pressure in female Wistar rats. PLoS One 8: e74522.

46. Sanz-García A, Knafo S, Pereda-Pérez I, et al. (2016) Administration of the TrkB receptor agonist 7,8-dihydroxyflavone prevents traumatic stress-induced spatial memory deficits and changes in synaptic plasticity. Hippocampus 26: 1179-1188.

47. Wang Y, Cao X, Ma H, et al. (2016) Prior stressor exposure delays the recovery of surgery-induced cognitive impairment and prolongs neuroinflammation in aged rats. Brain Res 1648: 380-386.

48. Wong W, Hassed C, Chambers R, et al. (2016) The effects of mindfulness on persons with mild cognitive impairment: Protocol for a mixed-methods longitudinal study. Front Aging Neurosci 8: 156.

49. Eshkoor S, Hamid T, Mun C, et al. (2015) Mild cognitive impairment and its management in older people. Clin Interv Aging 10: 687-693.

50. Ekundayo H, Kolawole A (2013) Stress among secondary school teachers in Ekiti State, Nigeria. Journal of Education and Social Research 3: 311-315.

51. Omoniyi M, Ogunsanmi J (2012) Stress among academic staff in South West Nigeria. The African Symposium 12: 126-132.

52. Cohen S, Janicki DD (2012) Who's stressed? Distributions of psychological stress in the United States in probability samples from 1983, 2006 and 2009. Journal of Applied Social Psychology 42: 1320-1334.

53. Cohen S, Kamarck T, Mermelstein R (1983) A global measure of perceived stress. J Health Soc Behav 24: 385-396.

54. Khan Z, Lanin AB, Ahmad N (2015) The level of stress in male and female school students. Journal of Education and Practice 6: 166.

55. Katz MJ, Derby CA, Wang C, et al. (2016) Influence of perceived stress on incident amnestic mild cognitive impairment: Results from the Einstein aging study. Alzheimer Dis Assoc Disord 30: 93-98.

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