Al Ameen J Med Sci 2014; 7(2):99-103 ● US National Library of Medicine enlisted journal ● ISSN 0974-1143

ORIGINAL ARTICLE CODEN: AAJMBG

Is motor slowing a universal phenomenon of Aging? Study correlates the effect of aging on psychomotor speed

Kamini Ramdas Ilamkar*

Department of Physiology, Government Medical College, Medical Square, Hanuman nagar, Nagpur-440003 Maharashtra, India

Abstract: *Objectives:* The purpose of study was to determine the direct effects of aging on dopamine receptor & prefrontal cortex & their indirect effect on psychomotor speed. *Background:* Reaction time is mainly a centrally determined function. Its slowing with advancing age is based on various age- induced changes that occur in central nervous system. Damage or dysfunction of basal ganglia may be the basis for psychomotor slowness of speed and reduced D_2 dopamine receptor density, a potential biomarkers of aging. After age 20 years, D_1 receptors disappear at 3.2% per decade while D_2 receptors disappear at about 2.2% per decade. Overall, therefore, the D_1/D_2 ratio falls with age. *Methods:* Reaction time is measured by using a response analyzer in healthy controls with age ranging from 20-82 years. *Result:* It was observed that there is slowness of psychomotor speed with increasing age. *Conclusion:* Speed of perception, speed of initiating the response and speed of movement are all involved in psychomotor performance. Neural system related to all these functions would be most importantly implicated in the slowing seen as a consequence of aging. It might lead to the delayed or slower reaction time to auditory and visual stimuli in elderly subjects. Damage or dysfunction of basal ganglia may be the basis for psychomotor slowness of speed and reduced D_2 dopamine receptor density, a potential biomarkers of aging. Thus motor slowing is a universal feature of human aging commonly expressing human senescence.

Keywords: Aging; Dopamine receptor; Psychomotor speed; Prefrontal cortex (PFC)

Introduction

Most significant intellectual developments of the twentieth century are the increasing recognition that all aspects of human behaviour and experience are actually functions of a material structure, the Nervous system. Ageing in humans refers to a multidimensional process of physical, psychological and social change. Steady decline in many cognitive processes is seen across the lifespan, accelerating from the twenties or thirties [1].

There are changes in the brain: though neuron loss is minor, after 20 years of age there is a 10% reduction, each decade in the total length of the brain's myelinated axons [2]. Few cells in motor cortex changed their firing rate before the response. Stimulus and response time interval comprises of sense organ time, brain time, nerve time and muscle time is the reaction time which included speed of perception, speed of initiating the response and speed of movement, are all involved in psychomotor performance [3].

More recently, converging evidence from patient studies, animal research, pharmacological intervention and molecular genetics indicates that dopamine is critically implicated in higher-order cognitive functioning which decline across adulthood and aging [4]. Interdependencies exist between fronto-striatal functional connectivity, dopamine and working memory performance and that this system is functioning sub-optimally in normal aging [5].

Aims: The study was designed to evaluate the effect of normal aging on the psychomotor speed with in healthy controls by focusing on tasks related to prefrontal cortex functioning and was compared within groups to reach any statistical difference.

Objectives: The purpose of study was to determine the direct effects of aging on dopamine receptor & prefrontal cortex & their indirect effect on psychomotor speed.

Material and Methods

The study was conducted in the Department of Physiology, in association with the Medicine and Psychiatry Department of Government Medical College and Hospital Nagpur, with due permission of the ethical committee. Reaction time was measured in healthy controls after taking the informed consents from the random population in the age group 20-82 years. Study population consisted of 141 healthy volunteers were 46, 52 and 43 participants in early adulthood 20 to 39 years, middle adulthood 40 to 59 years and late adulthood 61 to 82 years group respectively.

Order of the procedure included: (A) Measurement of simple reaction time by an instrument: response analyzer, subjects included in the study were right handed and they responded with their right hands with normal hearing and vision. (B) Folstein Mini Mental State Examination (MMSE) for cognitive disability and Hamilton Rating Scale for Depression (Ham-D) [6-7] were used to exclude the significant cognitive impairment diseases such as Alzheimer's disease, Parkinson's disease, Dementia and Clinical depression as a cause of psychomotor slowing. (C) A survey questionnaire (questions including health, demographics and dominance) [8].

Minimum educational qualification as higher secondary school passed was included in this study. Subjects underwent a clinical examination by a physician, psychiatrist, and neurologist to evaluate any neurological or psychiatric disease, alcohol or substance abuse and visual or auditory impairment sufficient to compromise in evaluation and testing. Psychotropic medications or drugs known to affect the brain dopamine system were excluded from study. Screening procedures such as ECG, thyroid function studies, were also done to exclude cardiovascular diseases, hyperthyroidism and hypothyroidism disorder.

Statistical Analysis: The performances were expressed in mean ± standard deviation of the reaction time by using the Student's unpaired 't' test.

Results

The reaction time was measured by using the following parameters: For the auditory reaction time [ART], a low frequency sound [LF] and a high frequency sound [HF] were included and for the visual reaction time, a red colour light [RL] and a green colour light [GL] were included [Table-1 & 2, Fig-1].

Table-1: Showing neuropsychological data

Neuropsychological data	Mean ± SD
MMSE scores	28.0±1.2
Hamilton Depression Scale	4.1±2.2

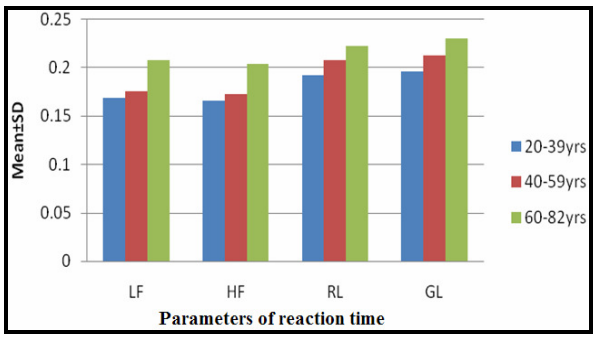
MMSE scores for these subjects ranged from 26 to 30 (28.0±1.2) indicating no significant cognitive impairment. Ham-D scores ranged from 0 to 8 (4.1±2.2), indicating no significant depressive symptoms.

Table-2: Showing mean with standard deviation of ART and VRT of healthy controls in various age groups

Parameter		Early adulthood (20 to 39 yrs) group [Mean ± SD]	Middle adulthood (40 to 59 yrs) Group [Mean ± SD]	Late adulthood (60 to 82 yrs) Group [Mean ± SD]
ART	low frequency sound	0.168±0.009	0.175±0.007	0.207±0.009
	High frequency sound	0.165±0.010	0.172±0.007	0.203±0.011
VRT	Red colour light	0.192±0.010	0.207±0.009	0.222±0.007
	Green colour light	0.196±0.010	0.212±0.012	0.230±0.009

There is increase in both [Mean ± SD] of ART and VRT with increasing age and it is statistically significant in the late adulthood [60 to 82 yrs] group.

Fig-1: Showing comparison and analysis between healthy controls in various age groups for reaction time.



Discussion

In the present study it was observed that reaction time increases as the age advances resulting slowness of psychomotor speed might be due spinal synaptic delay increases with age suggesting that decrease in ventral root conduction velocity and 10% decrease in ventral root fibers [9]. Older were slower in reaction time than younger. The variation in reaction time, suggesting that major changes in reaction time occurs in central nervous system rather than in the periphery [10]. With advancing age, various changes occur in nerves e.g. increased fibrosis, segmental demyelination and degeneration. These changes might lead to decrease in motor nerve conduction velocity mainly beyond 50 years of age which is more apparent in upper than lower extremities [11].

Older individuals might be much more sensitive to small changes in movement complexity than younger and suggested that this sensitivity is movement complexity to progressive over the adult life span and this increases reaction time with advance age [12]. As the age advances, there is age-related decline in psychomotor speed leading to delayed response by elderly individuals [13]. This may be due to direct effect of age on perceptual speed in addition to the indirect effect through sensory motor speed [14]. Slowing in reaction time with age suggested that there is decline in speed of information processing as the person ages [15] and progressively from 20 s up to the age of 60 years and above, might be due to age related decline in frontal cortex function which is involved in attentional task [16-17]. The elderly subjects tend to be more cautions and their speed of reaction is slower as well, hence they take longer time to respond. Reaction time is mainly a centrally determined function and its slowing with advancing age is based on various age-induced changes that occur in central nervous system. Speed of perception, speed of initiating the response and speed of movement are all involved in psychomotor performance. Neural system related to all these functions would be most importantly implicated in the slowing seen as a consequence of aging. It might lead to the delayed or slower reaction time to auditory and visual stimuli in elderly subjects. They also suggested that damage or dysfunction of basal ganglia may be the basis for psychomotor slowness of speed and reduced D_2 dopamine receptor density, a potential biomarkers of aging [18].

Decreased in catecholamines turnover in CNS (Central nervous system) of senescent rats and metabolism of nor-epinephrine and dopamine in some brain region with increased age and potential markers of aging included slower reaction times, reduced D_2 dopamine receptor density suggesting that reaction time is related to multiple aspects of Nigrostriatal dopamine function [19-20]. In childhood, the densities of D_1 and D_2 dopamine receptors in the brain striatum rise and fall together. After age 20 years, D_1 receptors disappear at 3.2% per decade while D_2 receptors disappear at about 2.2% per decade. Overall, therefore, the D_1/D_2 ratio falls with age [21].

Dopamine exerts different modulatory effects on medial PFC activity during tasks requiring sustained attention that are dependent upon the type of D2-like receptor that is activated. The PFC has the ability to actively maintain patterns of activity, and thus it can function not only as an attentional template, but also represent rules and goals that guide behaviour. The activity of the PFC is influenced by neuromodulatory systems that originate in the midbrain and hindbrain. Among them, midbrain dopaminergic (DA) input into the

PFC plays an important role in maintaining and updating PFC representations [22]. Meredith N Braskie (2008) further supports a relationship between striatal dopamine processing and frontal lobe cognitive function [23]. The molecular-imaging studies indicated that individual differences in DA functions are linked to cognitive performance irrespective of age, and serve as powerful mediators of age-related decline in executive functioning, episodic memory, and perceptual speed [24].

Age-related deficits have been demonstrated in working memory performance and in the dopamine systems suggesting that dopamine synthesis helps modulate default network activity in younger adults and those alterations to the dopamine system may contribute to age-related changes in working memory function [25]. Van Dyck C.H. et al (2007) also found diminished nigrostriatal dopaminergic function was associated with slowing of reaction speed [26]. Dopamine signalling may be particularly important under high executive demands; the dopaminergic system playing a key role in coordinating activity patterns in distinct regions of the neocortex [27]. Thus Dopamine may play an important role in attentional and executive processes, as it modulates cortico-limbic inputs, including afferents from the prefrontal cortex.

Acknowledgment

The author wish to thank to departments of medicine and psychiatry, Government Medical College & hospital, Nagpur and all the participants for their participation and co-operation to make these study successful.

References

1. Salthouse Timothy A. When does age-related cognitive decline begin?. *Neurobiology of Aging* 2009; 30(4): 507-514.
2. Marner L, Nyengaard JR, Tang Y, Pakkenberg B. Marked loss of myelinated nerve fibers in the human brain with age. *The Journal of comparative neurology* 2003; 462(2):144-52.
3. Luchies CW, Schiffman J, Richards LG, Thompson MR, Bazuin D, Deyoung AJ. Effects of age, step direction and reaction condition on the ability to step quickly. *J Gerontolo A Bio Sci Med Sci* 2002; 57(4): M246-9.
4. Backman L. The correlative triad among aging, dopamine, and cognition: current status and future prospects. *Neurosci Biobehav Rev.* 2006;30(6):791-807.
5. Klostermann EC. Dopamine and frontostriatal networks in cognitive aging. *Neurobiol Aging.* 2012; 33(3):623. e15-623.e24.
6. Folstein MF, Folstein SE, McHugh PR. Mini-mental state: a practical method for grading the cognitive state of patients for the clinician. *J. Psychiatr. Res.* 1975; 12(3):189-198.
7. Hamilton M. A rating scale for depression. *J. Neurol. Neurosurg. Psychiatr.* 1960; 23(2):56-62.
8. Strawbridge WJ, Wallhagen MI, Cohen RD. Successful aging and well-being: self-rated compared with Rowe and Kahn. *Gerontologist* 2002; 42(6):727-33.
9. Wayner MJ, Emmers R. Spinal synaptic delay in young and aged rats. *Am J Physiol* 1958; 194(2):403-405.
10. Weiss AD. The locus of reaction time change with set, motivation and age. *J Gerontology* 1965; 20:60-64.
11. Ganeriwal SK, Reddy BV, Surdi AD, Kowale AN, Zawar PB. Influence of age on motor nerve conduction. *Indian J Physiol Pharmacol* 1983; 337-341.
12. Light KE, Spirduso WW. Effects of adult aging on the movement complexity factor of response programming. *J Gerontol* 1990; 45(3):107-9.
13. Houx PJ, Jolles J. Age related decline of psychomotor speed, effect of age, brain damage, health, sex and education. *Perceptual and Motor Skills* 1993; 76:195-211.
14. Earles JL, Salthouse TA. Interrelations of age, health and speed. *J Gerontology Psychological Sciences* 1995; 50B (1):33-41.
15. Politano V, Draper T, McCormick MR. Age related differences in reaction time of healthy older amateur golfers. LARNet - *The Cyber Journal of Applied Leisure and Recreation Research* 2002;1-4.
16. Wilkinson RT, Allison S. Age and simple reaction time, decades for 5325 subjects. *J Gerontol* 1989; 44(2):29-35.
17. Barense MD, Fox MT, Baxter MG. Aged rats are impaired on an attentional set-shifting task, sensitive to medial frontal cortex damage in young rats. *Learning and Memory* 2002; 9:191-201.
18. Hicks LH, Birren JE. Aging, brain damage and psychomotor slowing. *Psychological Bulletin* 1970; 74:377-396.
19. Ponzio F, Brunello N, Algeri S. Catecholamine synthesis in brain of ageing rat. *J Neurochemistry* 1978; 22:1617-1620.
20. MacRae PG, Spirduso WW, Wilcox RE. Reaction time and nigrostriatal dopamine function: The effects of age and practice. *Brain Research* 1988; 451: 139-146.
21. Seeman P. Human brain dopamine receptors in children and aging adults. *Synapse* 1987; 1(5): 399-404.
22. Glickstein SB. Mice Lacking Dopamine D2 and D3 Receptors Exhibit Differential Activation of Prefrontal Cortical Neurons during Tasks Requiring Attention. *Cerebral Cortex* 2005; 15:1016-1024.

23. Braskie MN. Relationship of striatal dopamine synthesis capacity to age and cognition. *J Neurosci.* 2008; 28(52):14320-8.
24. Backman L. Linking cognitive aging to alterations in dopamine neurotransmitter functioning: recent data and future avenues. *Neurosci Biobehav Rev.* 2010; 34(5):670-7.
25. Braskie MN. Correlations of striatal dopamine synthesis with default network deactivations during working memory in younger adults. *Hum Brain Mapp.* 2011; 32(6): 947-961.
26. Van Dyck CH. Striatal dopamine transporters correlate with simple reaction time in elderly subjects. *Neurobiol Aging* 2008; 29(8): 1237-1246.
27. Stuart WS, MacDonald. Aging-Related Increases in Behavioral Variability. Relations to Losses of Dopamine D1 Receptors. *The Journal of Neuroscience,* 2012; 32(24):8186-8191.

*All correspondences to: Dr. Kamini Ramdas Ilamkar, Assistant Professor, Government Medical College, Nagpur-440003. Maharashtra, India. Email: kamini_ilamkar@yahoo.co.in