

An Exploratory Research Study to observe the effect of the use of K-MEPSE kit (Kamalpuria's Mental Exercise and Physical Strength Enhancer Kit) in early-onset Dementia patients (specifically Alzheimer's patients) in a span of six months

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Abstract - Dementia is a broad term used to describe a range of conditions that affect cognitive function, including memory, reasoning, language, and the ability to perform everyday tasks. It is not a single disease but rather a syndrome caused by various brain disorders. Among the different types, Alzheimer's disease is the most common, accounting for the majority of cases. Currently, there is no known treatment to cure dementia and related disorders completely. Also, only a little is known about such disorders, specially how the brain actually works during an episode of memory loss in a dementia patient. Overviewing the studies have stated that brain exercises can actually help slow down the progression of early-onset dementia, but a specific kit has never been made which includes targeted mental exercises along with the combination of acupressure to assist in the process of slowing down the progression of early-onset dementia.

The aim of this exploratory research study was to track the mental and physiological progress of patients of dementia, specifically targeted towards Alzheimer's early-onset patients, with the periodic (weekly) use of newly developed K-MEPSE (Kamalpuria's Mental Exercise and Physiological Strength Enhancer) kit, in a specific period of six months. There were weekly sessions involving conduction of the kit exercises along with blood pressure and sugar tests. The progress was tracked monthly by the dementia screening test RUDAS (Rowland's Universal Dementia Assessment Scale). This study also reviews the existing literature and understandings about dementia and the effect of therapeutic measures on helping slow down its progression. It is also very important to have a deep research and analysis on memory loss episodes of dementia patients because it is seriously traumatizing for the patients,

which can question a person's safety and leave deep scars on their mind.

Despite the number of studies on this aspect being limited, the work carried out by researchers till date has provided great insights and valuable connections and relationships into how therapeutic interventions can be linked to dementia and its progression. Many of these studies have efficiently used large sample sizes, which is a great strength and advantage to the work. However, there still exist large gaps in the studies that future studies need to assess, hereby, this study is just a block in the bridge of fillings these gaps significantly.

Keywords – *Dementia, Mental Exercises, Alzheimer's, Treatment of Dementia, Physical Exercises*

I. INTRODUCTION

A. Dementia

Dementia is a broad term used to describe a range of conditions that affect cognitive function, including memory, reasoning, language, and the ability to perform everyday tasks. It is not a single disease but rather a syndrome caused by various brain disorders. Among the different types, Alzheimer's disease is the most common, accounting for the majority of cases. Alzheimer's is a progressive neurological condition where abnormal protein deposits—such as amyloid plaques and tau tangles—disrupt communication between brain cells and eventually lead to cell death. Over time, individuals with Alzheimer's experience worsening memory loss, confusion, difficulty with language, and changes in behaviour and personality. Although there is currently no cure, early diagnosis

and supportive care can help manage symptoms and improve quality of life.

Dementia primarily affects older adults, with its prevalence increasing significantly with age. While it is not a normal part of aging, the risk of developing dementia roughly doubles every five years after the age of 65. Globally, millions of people live with dementia, and the number is expected to rise sharply due to longer life expectancies and aging populations. Although Alzheimer's disease is the most common form, other types such as vascular dementia, Lewy body dementia, and frontotemporal dementia also contribute to the global burden. Early-onset dementia, which occurs before age 65, is less common but still affects thousands worldwide. Geographic, genetic, and lifestyle factors can influence its occurrence, with certain populations experiencing higher rates due to cardiovascular health disparities and access to medical care.

B. Symptoms of Dementia

- **Memory Loss:** One of the earliest and most common signs, especially difficulty recalling recent events, conversations, or appointments while older memories may remain intact initially.
- **Difficulty with Language:** Struggling to find the right words, repeating questions, or having trouble following or joining conversations.
- **Impaired Problem-Solving and Planning:** Challenges in making decisions, handling money, or following a sequence of steps in daily tasks such as cooking or managing medications.
- **Disorientation:** Confusion about time, place, or familiar environments, sometimes leading to getting lost even in well-known areas.
- **Poor Judgment and Decision-Making:** Making uncharacteristic choices, such as giving away large sums of money or neglecting personal hygiene.
- **Changes in Mood and Behavior:** Increased anxiety, depression, irritability, or withdrawal from social activities; sometimes aggressive or suspicious behavior.
- **Difficulty with Coordination and Motor Functions:** Problems with balance, walking, or using everyday objects like utensils or clothing.
- **Loss of Initiative:** Reduced interest in hobbies, work, or social interactions, often seeming passive or indifferent.
- **Hallucinations and Delusions:** In some types of dementia, individuals may see or hear things that are not there or hold false beliefs.
- **Difficulty Understanding Visual Images and Spatial Relationships:** Trouble judging distances, recognizing faces, or navigating stairs and curbs.

C. Causes of Dementia

Dementia arises from a diverse range of pathological processes that lead to progressive neuronal damage and loss of brain function. Alzheimer's disease is the predominant cause, characterized by the accumulation of extracellular amyloid-beta plaques and intracellular neurofibrillary tangles composed of hyperphosphorylated tau protein, which disrupt synaptic function and trigger neurodegeneration. Vascular dementia results from cerebrovascular disease, including ischemic strokes, microinfarcts, or chronic small vessel disease, which impair cerebral blood flow and oxygen delivery, causing focal or diffuse brain injury. Lewy body dementia is caused by the abnormal aggregation of alpha-synuclein protein within neurons, producing Lewy bodies that interfere with neurotransmission. Frontotemporal dementia involves selective atrophy of the frontal and temporal lobes, linked to abnormal tau or TDP-43 protein deposits. Mixed dementia, common in elderly populations, combines features of Alzheimer's and vascular pathologies. Other less common causes include prion diseases, infections like HIV or syphilis, traumatic brain injuries, and metabolic disorders. Genetic predispositions, such as mutations in the APP, PSEN1, PSEN2 genes, or the presence of APOE ε4 allele, influence susceptibility, while modifiable factors like hypertension, diabetes, smoking, and lifestyle choices also play significant roles in disease onset and progression.

D. Diagnosis of Dementia

Diagnosing dementia involves a comprehensive evaluation to determine the presence and cause of cognitive decline. Physicians typically begin with a detailed medical history and interviews with the patient and family to understand symptom progression and daily functioning. Cognitive tests, such as the Mini-Mental State Examination (MMSE) or Montreal Cognitive Assessment (MoCA), help assess memory, attention, language, and problem-solving skills. Physical and neurological exams check for other health issues that might mimic dementia symptoms. Brain imaging techniques like MRI or CT scans are often used to detect structural changes, strokes, or tumours. In some cases, blood tests rule out infections or vitamin deficiencies that can affect cognition. More advanced diagnostics, including PET scans or cerebrospinal fluid analysis, can help identify specific types of dementia. Early and accurate diagnosis is crucial for managing symptoms, planning care, and exploring treatment options.

E. Types of Dementia

Dementia encompasses several types, each with unique characteristics and underlying causes. The most common form is **Alzheimer's disease**, which is marked by gradual memory loss and cognitive decline due to the build-up of abnormal proteins in the brain. **Vascular dementia** arises from reduced blood flow to brain tissue, often following strokes or chronic cardiovascular issues, and typically leads to more sudden changes in thinking and behaviour. **Lewy body dementia** involves abnormal protein deposits called Lewy bodies and can

cause visual hallucinations, movement difficulties, and fluctuating mental states. **Frontotemporal dementia** affects the brain's frontal and temporal lobes, leading to changes in personality, behavior, and language, often at a younger age than other forms. Other less common types include **mixed dementia**, which is a combination of different pathologies, and conditions like **Parkinson's disease dementia** or **Creutzfeldt-Jakob disease**, which are associated with specific neurological disorders.

F. Comparison of Dementia Types

Type of Dementia	Key Features	Common Symptoms	Typical Onset Age	Causes/Underlying Issues
Alzheimer's Disease	Most common form; slow, progressive decline	Memory loss, confusion, language problems	65+ (can be earlier)	Amyloid plaques and tau tangles damaging brain cells
Vascular Dementia	Second most common; stepwise decline after stroke or poor circulation	Poor concentration, decision-making problems, mood swings	65+	Brain damage due to reduced blood flow
Lewy Body Dementia	Fluctuating cognition, hallucination, motor issues	Visual hallucinations, tremors, confusion, sleep issues	50-85	Lewy bodies (abnormal protein clumps) in the brain
Fronto-temporal Dementia	Affects personality and behavior; language difficulties	Apathy, inappropriate behavior, speech/language loss	45-65 (early onset common)	Degeneration of frontal and temporal brain lobes
Mixed Dementia	Combination of Alzheimer's and vascular or Lewy body dementia	Symptoms vary depending on mix of types	65+	Multiple pathologies in the brain
Parkinson's Disease Dementia	Develops in people with Parkinson's, often years after movement symptoms	Slow thinking, memory problems, hallucinations	60+	Parkinson's-related brain changes
Creutzfeldt-Jakob Disease	Rare, rapidly progressive	Sudden memory loss, motor dysfunction, personality changes	Any adult age (rare)	Prion infection causing brain cell death

G. Alzheimer's Disease

Alzheimer's disease is a progressive neurological disorder that gradually impairs memory, thinking, and behavior. It is the

most common cause of dementia and typically begins with mild memory loss, especially for recent events. As the disease advances, individuals may struggle with language, decision-making, spatial awareness, and even recognizing loved ones. The condition is driven by the accumulation of abnormal proteins in the brain—specifically, amyloid plaques between nerve cells and tau tangles within them—which interfere with communication and ultimately lead to cell death. Over time, brain tissue shrinks, especially in areas responsible for memory and reasoning. While the exact cause is still not fully understood, factors like age, genetics (such as the APOE ε4 gene), and lifestyle are known to influence risk. Although there is no cure, treatments can help manage symptoms, and research continues to explore ways to slow or prevent disease progression.

Alzheimer's disease is a neurodegenerative disorder characterized by progressive cognitive decline and memory impairment, representing the leading cause of dementia worldwide. Pathologically, it is defined by extracellular amyloid-beta plaque deposition and intracellular neurofibrillary tangles composed of hyperphosphorylated tau protein, leading to synaptic dysfunction and neuronal loss, predominantly in the hippocampus and cerebral cortex. Clinically, early symptoms include episodic memory deficits and executive dysfunction, progressing to aphasia, apraxia, and profound cognitive impairment. Genetic factors, notably the presence of the APOE ε4 allele, significantly increase susceptibility. Current therapeutic interventions focus on symptomatic management, utilizing cholinesterase inhibitors and NMDA receptor antagonists, with ongoing research targeting disease-modifying strategies.

H. Treatment of Dementia

Currently, there is no cure for dementia, but various treatments aim to slow its progression and improve quality of life. Medications such as cholinesterase inhibitors and NMDA receptor antagonists are commonly prescribed to help manage symptoms like memory loss and confusion. Alongside drug therapy, non-drug approaches play a vital role, including cognitive stimulation, physical exercise, and social engagement, which can help maintain mental function and emotional well-being. Managing underlying health conditions such as hypertension or diabetes is also important to reduce further brain damage. Support for caregivers and creating a safe, structured environment are essential components of treatment. Researchers continue to explore new therapies focused on targeting the underlying causes, with hope for more effective options in the future.

While there is no cure for dementia, several treatments can help manage symptoms and improve daily functioning. The most commonly prescribed medications include **cholinesterase inhibitors** such as donepezil, rivastigmine, and galantamine, which work by increasing levels of neurotransmitters to support memory and thinking. Another drug, **memantine**, an NMDA

receptor antagonist, helps regulate glutamate activity to protect brain cells and is often used in moderate to severe stages. Beyond medication, non-pharmacological approaches like **cognitive stimulation therapy**, regular physical exercise, and maintaining social connections have been shown to slow cognitive decline and enhance quality of life. Managing other health issues like high blood pressure, diabetes, and depression is crucial in reducing further brain injury. Additionally, therapies such as music and art therapy, occupational therapy, and creating a safe, supportive environment help reduce anxiety and behavioural symptoms. Ongoing research is exploring innovative treatments targeting amyloid plaques, tau protein, and inflammation, aiming to modify the disease course in the future.

II. REVIEW OF LITERATURE

A. Nitin Bansal and Milind Parle (2014)

Dementia encompasses a group of neurodegenerative disorders characterized by a progressive decline in cognitive abilities, including memory, language, judgment, and executive functions. It is not a singular disease but a syndrome resulting from various pathological processes, each with unique clinical presentations and aetiologies. Memory dysfunction is a core feature in dementia, and its neuropsychological basis has been well documented. Human memory is classified into sensory, short-term (working), and long-term domains, each governed by different brain systems. The hippocampus, thalamus, and associated cortical regions are instrumental in memory encoding and retrieval processes. While dementia causes irreversible memory loss, other memory disorders such as amnesia are often reversible and result from acute conditions like head trauma or substance use.

Alzheimer's disease (AD) is the most prevalent form of dementia, accounting for approximately 60% of cases. Pathologically, AD is marked by beta-amyloid plaques and neurofibrillary tangles composed of hyperphosphorylated tau protein. These abnormalities disrupt neuronal communication and lead to synaptic loss, particularly in the hippocampus and neocortex. AD also involves neurotransmitter imbalances, notably acetylcholine deficiency, which correlates with cognitive decline.

Vascular dementia (VaD), the second most common form, results from cerebrovascular events that impair blood flow to the brain. VaD can arise from large-vessel infarcts, small-vessel disease, or hypo-perfusive conditions, leading to cumulative or localized neuronal damage. This subtype often coexists with AD in mixed dementia presentations, complicating diagnosis and treatment. Other forms such as dementia with Lewy bodies (DLB) and frontotemporal dementia (FTD) further expand the spectrum of the disease. DLB is characterized by intracellular alpha-synuclein aggregates and fluctuating cognition, while FTD primarily affects younger individuals and manifests as

personality changes or language deficits depending on the affected brain region. Subcortical dementias include conditions such as Parkinson's disease dementia and Huntington's disease. These forms involve deeper brain structures like the basal ganglia and often present with motor symptoms alongside cognitive. Wilson's disease and normal pressure hydrocephalus also fall within this category and demonstrate varying degrees of cognitive impairment.

Infectious agents (e.g., HIV, prions, syphilis), metabolic disorders (e.g., hypothyroidism, vitamin B12 deficiency), chronic intoxications (e.g., alcohol, drugs, heavy metals), and tumours can also lead to secondary dementia syndromes. These forms may be reversible if identified and treated early. Pharmacological approaches to dementia remain largely symptomatic. Cholinesterase inhibitors such as donepezil and rivastigmine enhance cholinergic transmission and offer modest cognitive benefits. Memantine, an NMDA receptor antagonist, may slow progression in moderate to severe cases. Adjunct therapies, including antioxidants (e.g., vitamin E), nootropics, hormonal treatments, and anti-inflammatory agents, have shown variable efficacy in clinical trials.

Despite these efforts, the absence of a definitive cure underscores the need for preventive strategies. Epidemiological research identifies advanced age, low educational attainment, and cardiovascular risk factors as major contributors to dementia risk. Conversely, higher cognitive reserve, physical activity, and certain dietary or pharmacological interventions may offer protective effects.

B. Wallenstein et al. (2023)

The global burden of dementia, currently affecting over 55 million individuals, is intensifying as populations age, making it imperative to understand modifiable risk factors. Alzheimer's disease (AD), the most prevalent form of dementia, often follows a progression from mild cognitive impairment (MCI), which may precede dementia symptoms by years. While aging remains the most dominant risk factor for dementia, it is immutable. Consequently, research attention has shifted toward modifiable contributors such as comorbid conditions and psychosocial factors. Among the modifiable risks, depression and chronic stress have garnered considerable interest. Depression has been extensively studied and linked to elevated risks of cognitive decline and dementia. Multiple studies suggest depression may act as both a potential cause and a prodrome of dementia. The mechanisms proposed include shared pathophysiology involving neuroinflammation, hippocampal atrophy, and overlapping biomarkers such as tau protein abnormalities.

Chronic stress, although less extensively investigated, is increasingly recognized as another relevant risk factor. Previous research indicates that prolonged exposure to stress may disrupt the hypothalamic–pituitary–adrenal (HPA) axis,

increase cortisol levels, and damage brain structures critical to memory and cognition, such as the hippocampus. Moreover, stress is associated with inflammatory processes, which are believed to contribute to neurodegenerative conditions like AD. A significant gap in the literature has been the understanding of the combined or additive effects of chronic stress and depression on cognitive decline. Most prior studies have examined these variables in isolation. Furthermore, challenges in measuring chronic stress, especially non-traumatic forms, have limited the development of clear diagnostic associations. Sweden's unique diagnostic classification—Stress-Induced Exhaustion Disorder (SED)—offers a valuable tool for evaluating long-term stress impacts within a medical framework.

Wallensten et al.'s study builds upon and extends previous findings by utilizing a large administrative cohort from Region Stockholm to assess how depression and chronic stress—individually and combined—relate to the onset of MCI and dementia. Their results indicate not only that both depression and stress are independent predictors of MCI and AD but also that their coexistence significantly increases this risk. This suggests a potential additive effect, a novel contribution to the existing body of literature. Overall, this study aligns with and reinforces prior epidemiological and biological research, while contributing fresh evidence on the interaction between depression and chronic stress as compounding risks for cognitive decline. Future research is warranted to examine causality, underlying mechanisms, and the role of biomarkers and genetic predispositions in mediating these relationships.

C. Gill Livingston et. al. (2020)

Dementia, a growing global health concern, currently affects approximately 50 million individuals worldwide, with projections reaching over 150 million by 2050. This rapid increase is especially prominent in low- and middle-income countries (LMICs), where two-thirds of people with dementia reside. The 2020 Lancet Commission builds upon its 2017 report by expanding the number of modifiable risk factors from nine to twelve, based on new evidence and comprehensive meta-analyses. These factors include education, hypertension, hearing impairment, smoking, obesity, depression, physical inactivity, diabetes, and low social contact—now joined by excessive alcohol consumption, traumatic brain injury (TBI), and air pollution. The Commission emphasizes the life-course model of dementia risk, where early-life education contributes to cognitive reserve, while midlife and late-life exposures influence the development and expression of neuropathology. For example, insufficient education in early years can reduce cognitive reserve, increasing vulnerability later in life. Similarly, midlife hypertension and obesity significantly raise the likelihood of future cognitive decline. Later-life factors such as depression and social isolation may either be risk factors or early symptoms of dementia, indicating a complex, often bidirectional relationship.

Recent research suggests that addressing these modifiable risks could prevent or delay up to 40% of dementia cases globally. Notably, the potential for prevention appears even greater in LMICs, due to higher prevalence of risk factors like uncontrolled hypertension, low education access, and limited healthcare infrastructure. Interventions such as promoting childhood education, controlling blood pressure, reducing smoking and air pollution, and improving access to hearing aids have been identified as practical and impactful strategies. In terms of clinical care, the Commission argues for holistic, person-centred management that considers not only the individual with dementia but also their caregivers. Psychosocial interventions tailored to individual needs can alleviate neuropsychiatric symptoms and reduce caregiver distress. The report also critiques hospital-centric models of care, noting that people with dementia often experience poorer health outcomes and increased hospitalizations compared to their peers.

Biomarker research continues to evolve, with blood-based markers showing promise for early detection and monitoring. However, the Commission discourages routine pre-symptomatic testing due to current lack of clinical utility and potential ethical implications. It also underscores the importance of adapting interventions to diverse cultural and socioeconomic contexts, particularly in LMICs where healthcare disparities can limit effectiveness.

In summary, the 2020 Lancet Commission offers a robust synthesis of emerging evidence on dementia prevention and care. It advocates for a coordinated, life-course approach integrating public health, clinical practice, and policy reform, with an emphasis on equity and global applicability.

D. Sukhawathanakul et al. (2021)

In recent years, the exploration of psychotherapeutic interventions for individuals with dementia and cognitive impairment has gained increasing attention as non-pharmacological approaches to improving mental health and quality of life. While earlier reviews primarily focused on clinical symptoms such as depression or cognitive decline, Sukhawathanakul et al. (2021) broaden the focus by considering how these therapies influence emotional adaptation, including acceptance and adjustment to changing cognitive and life circumstances. A central premise of their review is that individuals with dementia face significant psychosocial challenges that go beyond memory loss, including the need to cope with diagnosis-related stigma, altered self-concept, and shifting interpersonal roles. Acceptance—defined as acknowledgment of a situation without resistance—and adjustment—the behavioural and emotional alignment to life changes—are considered critical to mental health outcomes in this population. Previous research has indicated that failure to adapt may lead to elevated rates of depression, anxiety, and psychological distress.

The review systematically analysed 24 studies conducted between 2003 and 2019. These studies employed a range of therapeutic modalities, including cognitive-behavioural therapy (CBT), problem-solving therapy, interpersonal therapy, mindfulness approaches, compassion-focused therapy, and reminiscence therapy. Most studies reported improvements in at least one psychological domain, particularly depressive symptoms. However, findings related to self-esteem, helplessness, quality of life, and broader well-being were less consistent. CBT emerged as the most frequently employed intervention. Several studies incorporated psychoeducation and cognitive restructuring to address maladaptive beliefs in individuals with mild cognitive impairment or early-stage dementia. Some studies showed improvements in perceived acceptance and a reduction in helplessness, though these outcomes were not universally observed. Inconsistencies were often attributed to the sensitivity of the measurement tools used. Problem-solving and meaning-based therapies also demonstrated promising outcomes. For example, the Problem Adaptation Therapy (PATH) model, which combines practical coping strategies with environmental modifications, was associated with reductions in depressive symptoms and disability in older adults. Similarly, group interventions that included emotional sharing and mutual support showed potential in improving emotional well-being and social connectedness. Another significant theme was the inclusion of caregivers in therapeutic processes. Nine of the reviewed studies engaged both patients and caregivers, reflecting the importance of dyadic support in coping with cognitive decline. These joint interventions often resulted in improved outcomes not just for patients, but also for caregiver burden and stress.

The review highlighted a number of limitations in the existing literature, including small sample sizes, short follow-up periods, and limited standardization in intervention delivery. Despite these constraints, there is growing consensus that tailored psychotherapeutic approaches can support emotional resilience and psychological adjustment among individuals with dementia, particularly when interventions are introduced in the early stages of cognitive decline.

In summary, the reviewed evidence suggests that psychotherapeutic interventions have the potential to enhance acceptance and adjustment among older adults living with dementia. While more research is needed to refine therapeutic models and evaluate long-term effectiveness, current findings support the inclusion of these interventions as part of a comprehensive care strategy that addresses both cognitive and emotional dimensions of dementia.

E. Ma et al. (2023)

Dementia, a progressive neurological condition characterized by cognitive and behavioural decline, is increasingly prevalent due to global aging trends. Traditional treatments, primarily pharmacological, offer limited efficacy and often present adverse side effects. As a result, non-pharmacological

interventions, particularly those rooted in person-centred care, are gaining traction. Among these, creative story therapy (CST) has emerged as a promising method to support cognitive, emotional, and social functioning in individuals with dementia. Creative story therapy involves the use of imaginative storytelling to engage individuals in a non-judgmental and expressive process. Unlike conventional memory-based therapies, CST does not rely on factual recall, making it accessible even for those with significant memory impairment. One widely adopted form is "Time Slips," a structured program where facilitators prompt participants to create collective stories using visual stimuli. This approach emphasizes emotional expression, social interaction, and cognitive stimulation. The theoretical foundations of CST draw from multiple psychological frameworks. Cognitive theory highlights the mental stimulation gained through narrative construction, while relationship-centred care underscores the therapeutic value of social interaction. Generativity theory suggests that storytelling provides older adults with a sense of legacy, and social-emotional selectivity theory supports the idea that meaningful engagement enhances emotional well-being in late life.

Ma et al. (2023) conducted a systematic review and meta-analysis of nine randomized controlled trials involving 597 participants. Their findings suggest that CST, when combined with routine care, significantly improves cognitive function, reduces depressive symptoms, enhances quality of life, and boosts social communication among individuals with dementia. The effect sizes for cognitive improvements and depression relief were moderate to large, indicating practical significance in clinical settings. Importantly, CST was found to outperform routine care alone in various domains, although its effect on basic needs communication was not statistically significant. The review also emphasizes that the frequency and duration of intervention matter—greater benefits were observed with programs extending beyond six weeks.

Despite these promising results, limitations remain in the current literature. Many studies reviewed had small sample sizes, short durations, and methodological shortcomings, including lack of blinding and inconsistent outcome measures. Furthermore, the impact of CST on long-term outcomes and in diverse cultural contexts remains underexplored. Nevertheless, the evidence supports the inclusion of CST as a valuable component of holistic dementia care. By promoting autonomy, creativity, and interpersonal connection, CST aligns with contemporary values of dignity and quality of life in elder care. Future research should aim to standardize intervention protocols, assess long-term effects, and explore scalability across healthcare systems.

F. Amie Robinson and Nima Moghaddam (2022)

Dementia and mild cognitive impairment (MCI) are frequently accompanied by psychological distress, particularly symptoms of depression and anxiety. This comorbidity not only

exacerbates the disease burden for patients but also contributes to faster cognitive and functional decline, greater caregiver stress, and increased healthcare costs. Despite these risks, there remains a noticeable gap in the provision of effective, tailored psychological treatments for this population. Traditional approaches to managing depression and anxiety in dementia have largely focused on pharmacological solutions. However, evidence for the efficacy of antidepressants in this population is mixed, and concerns about side effects and limited long-term benefits persist. As such, psychological interventions have gained growing attention as viable alternatives, especially those rooted in cognitive behavioural therapy (CBT), psychodynamic approaches, relaxation training, and supportive counselling.

Earlier reviews, such as Orgeta et al. (2015), suggested that psychological therapies could yield modest but meaningful improvements in depressive symptoms among people with dementia. However, those findings were limited by methodological weaknesses, including variability in intervention types, durations, and outcome measurements. Moreover, the literature lacked a systematic investigation into how such interventions could be adapted to suit the cognitive limitations inherent in dementia and MCI. To address these gaps, Robinson and Moghaddam (2022) conducted a comprehensive systematic review and meta-analysis of nine randomized controlled trials (RCTs) that evaluated psychological treatments for individuals with dementia and MCI experiencing psychological distress. Their analysis emphasized not only the effectiveness of these treatments but also the importance of specific therapy adaptations that enhance accessibility and acceptability. The included studies represented a diverse array of interventions, such as CBT, relaxation-based therapies, psychodynamic therapy, and cognitive rehabilitation, delivered across various settings and modalities. Overall, the results revealed a small but statistically significant reduction in depressive symptoms following psychological treatment, particularly when interventions were modified to accommodate cognitive limitations. However, the impact on anxiety was inconclusive due to limited and heterogeneous data. Importantly, the review highlighted several adaptations that appeared to enhance the effectiveness of psychological therapy in this population. These included simplifying language, incorporating repetition, using visual aids, slowing session pace, and tailoring examples to participants' life experiences. Therapies were also more successful when delivered in familiar environments and when caregivers were included in the treatment process. Despite encouraging outcomes, the authors noted limitations, including the small number of high-quality RCTs, lack of standardization in intervention protocols, and inconsistent follow-up periods. These limitations underscore the need for more robust, long-term studies with clearly defined outcome measures and treatment fidelity assessments.

In summary, the evidence supports the feasibility and potential effectiveness of psychological therapies for reducing depression and distress in individuals with dementia and MCI.

Therapy adaptations tailored to cognitive and sensory needs appear essential for maximizing therapeutic engagement and outcomes. The study by Robinson and Moghaddam (2022) marks a significant contribution to the field by systematically identifying which modifications improve therapy acceptability, and it reinforces the case for integrating adapted psychological interventions into standard dementia care.

G. Ahlskog et al. (2011)

Dementia, especially Alzheimer's disease (AD), presents one of the most pressing challenges to public health as populations age globally. Despite extensive research into pharmacological treatments targeting neurodegenerative processes, effective disease-modifying drugs remain elusive. In this context, a growing body of research has turned its attention toward lifestyle-based interventions—particularly physical exercise—as a potentially powerful strategy to delay or reduce the risk of dementia and cognitive decline. The literature increasingly supports the role of aerobic exercise in promoting brain health and cognitive function. Epidemiological studies suggest that individuals who engage in regular physical activity during midlife show a significantly reduced risk of developing dementia or mild cognitive impairment (MCI) in later years. Ahlskog et al. (2011) reviewed meta-analyses and large-scale prospective cohort studies that consistently found lower rates of cognitive decline among physically active individuals. For example, in one meta-analysis, midlife exercise was associated with approximately a 28% reduction in dementia risk. Clinical trials further reinforce the cognitive benefits of exercise. Randomized controlled trials (RCTs) involving individuals with MCI or early dementia showed that those participating in structured aerobic exercise programs exhibited better performance on cognitive assessments compared to sedentary controls. Improvements were noted in domains such as memory, executive functioning, and processing speed, even with relatively short interventions lasting six to twelve months.

Neuroimaging studies offer biological evidence for these outcomes. Longitudinal trials involving seniors have demonstrated increased hippocampal volume—a brain region essential for memory—following consistent aerobic exercise. Functional MRI (fMRI) studies also indicate enhanced connectivity in cognitive networks after regular physical activity. These structural and functional brain changes suggest that exercise may help counteract the neural deterioration typically associated with aging and dementia.

Animal research provides further support, identifying neuroplasticity as a key mechanism through which exercise exerts its protective effects. In rodents, physical activity boosts neurogenesis in the hippocampus, enhances dendritic complexity, and upregulates the expression of brain-derived neurotrophic factor (BDNF), insulin-like growth factor-1 (IGF-1), and other growth factors critical for cognitive function. These biochemical changes support learning and memory,

reinforcing the idea that exercise fosters a brain environment conducive to resilience against neurodegeneration.

In humans, correlations between fitness levels, serum BDNF concentrations, and cognitive performance also lend indirect support to the neurotrophic hypothesis. Some human trials have shown increased circulating BDNF following aerobic exercise, and these increases were often associated with better cognitive outcomes. However, results vary across studies, suggesting the need for further investigation into the specific conditions and populations in which these neurobiological effects are most pronounced. Importantly, exercise may also mitigate vascular contributions to cognitive decline. Vascular risk factors such as hypertension, diabetes, and obesity are known to exacerbate or even initiate dementing processes. Regular physical activity is known to improve vascular health, thereby potentially reducing the additive burden of cerebrovascular damage on the aging brain. While aerobic exercise is the most studied modality, emerging evidence also hints at benefits from resistance training, although findings are less consistent. Some trials suggest that strength-based exercise can also lead to cognitive improvement, particularly in executive function, but more research is needed to determine its efficacy and underlying mechanisms.

In conclusion, the literature reviewed by Ahlskog et al. highlights regular physical exercise—especially aerobic activity—as a promising intervention for both preventing and slowing the progression of dementia. The benefits appear to arise from both direct effects on brain plasticity and indirect improvements in vascular and metabolic health. Given its low cost, accessibility, and wide-ranging health benefits, exercise represents a critical and underutilized strategy in dementia prevention and care.

H. Cui et al. (2018)

Alzheimer's disease (AD), the most common form of dementia, represents a significant challenge in aging societies due to its progressive nature and limited treatment options. While pharmacological treatments such as cholinesterase inhibitors and memantine have been widely used to manage cognitive symptoms, their clinical effects are often modest and temporary, especially in later stages. This has intensified interest in non-pharmacological interventions, particularly exercise-based therapies, as potential strategies to mitigate cognitive decline across different stages of Alzheimer's and related conditions. Physical and cognitive exercise interventions have been increasingly explored for their capacity to promote neuroplasticity and cognitive resilience. Exercise has been associated with improvements in neurovascular function, reduced oxidative stress, decreased amyloid and tau pathology, and enhanced neurotrophic factors such as BDNF and IGF-1. These biological effects underpin the hypothesis that exercise can slow the progression of cognitive deterioration, not only in those with AD but also in individuals with mild cognitive impairment (MCI) or at risk of dementia.

Cognitive training (CT), cognitive stimulation (CS), and cognitive rehabilitation (CR) represent the primary categories of cognitive exercises. CT involves repeated practice of structured tasks aimed at enhancing specific cognitive skills, while CS typically employs less standardized, broader activities like puzzles and games. CR focuses on helping individuals maintain independence in daily activities through compensatory strategies. Although cognitive exercises can improve certain domains like reasoning and memory, their generalization to real-life tasks is sometimes limited, especially when tasks differ from training content. Physical exercise, both aerobic and resistance training, has demonstrated positive effects on cognitive and physical function in aging populations. Aerobic training in particular has been linked to increased hippocampal volume, improved white matter integrity, and elevated levels of neurotrophic factors. Resistance training contributes by modulating neural biomarkers such as IGF-1 and reducing harmful substances like homocysteine. Moreover, integrative approaches such as Tai Chi and multimodal training (combining strength and balance) show promise in enhancing executive functioning and postural stability. Research also supports the synergistic value of combined cognitive and physical training. Interventions like exergaming—which merges physical activity with cognitive challenges—have shown greater improvements in global cognition, executive function, and neuroplasticity compared to either modality alone. These interactive formats also appear to boost participant engagement, which is critical for long-term adherence. Other variables such as duration, intensity, and participant characteristics also influence the effectiveness of exercise interventions. Studies suggest that consistent, long-term activity yields better outcomes than short-term efforts. Interestingly, gender differences have been observed, with females often showing greater improvements in executive functions following aerobic training—possibly due to differences in hormonal or neurochemical responses. Exercise appears particularly effective in early stages of cognitive decline, such as MCI. Clinical trials have demonstrated significant improvements in memory, executive function, and daily living skills after exercise-based interventions. While results in moderate to severe AD are more variable, evidence supports that structured physical activity can maintain functional abilities and reduce behavioural symptoms. At the molecular level, exercise affects AD pathology by regulating the amyloid cascade, reducing oxidative damage, modulating inflammation, and enhancing cholinergic neurotransmission. Experimental studies on transgenic mouse models and neuroimaging in humans confirm reductions in amyloid load and improvements in brain metabolism following sustained physical activity.

In summary, the literature supports the role of exercise as a multi-dimensional intervention with the potential to delay cognitive decline, especially when introduced early in the disease process. While further research is needed to determine standardized protocols, exercise remains a low-cost, accessible,

and non-invasive strategy that complements existing therapeutic approaches for Alzheimer's disease.

I. Lautenschlager et al. (2012)

With the growing aging population worldwide, dementia has emerged as a major global health challenge, particularly Alzheimer's disease (AD), which remains the most common neurodegenerative condition in older adults. The lack of curative treatments has turned scientific attention toward lifestyle interventions that may delay or prevent cognitive decline. Among these, physical activity (PA) has gained increasing prominence as a potentially effective and accessible preventive strategy for brain aging and dementia. Early observational and epidemiological studies have consistently reported associations between regular physical activity and a reduced risk of cognitive impairment. Several large-scale prospective cohort studies have shown that both moderate and vigorous exercise are linked with better cognitive performance and lower incidence of dementia. For instance, a meta-analysis by Sofi et al. found that individuals engaging in high or moderate levels of physical activity had a 35–38% reduced risk of cognitive decline. Similarly, longitudinal studies like the National Long Term Care Survey have demonstrated that even light activities such as gardening and walking can protect against loss of cognitive function and help maintain independent living. Despite these promising trends, researchers have noted challenges in comparing studies due to inconsistencies in the definition, intensity, and duration of physical activity, as well as variability in cognitive assessment tools. Tools like the MMSE (Mini-Mental State Examination), while widely used, may lack the sensitivity needed to detect subtle cognitive improvements. Nonetheless, there is a general consensus that aerobic exercise, particularly when sustained over time, has the greatest cognitive benefit, especially in domains such as executive function, attention, and processing speed.

Randomized controlled trials (RCTs) have expanded the evidence base by evaluating the effects of structured exercise interventions in diverse older adult populations. These trials indicate that both aerobic and resistance training can positively impact cognitive outcomes, especially in individuals with mild cognitive impairment (MCI) or subjective memory complaints. For example, studies have shown that 6- to 12-month aerobic exercise programs can improve memory performance and increase hippocampal volume—a brain region critical for learning and memory. Research also suggests that the benefits of physical activity may differ based on individual characteristics such as gender, baseline cognitive status, and genetic factors like the presence of the APOE ϵ 4 allele. Moreover, activities that combine physical and cognitive engagement, such as Tai Chi or dance, may offer added benefits by stimulating motor planning and memory simultaneously. In addition to cognitive outcomes, physical activity has been found to enhance cerebral blood flow, promote neurogenesis, reduce oxidative stress, and modulate key biomarkers related to

Alzheimer's pathology, such as amyloid beta and brain-derived neurotrophic factor (BDNF). These biological mechanisms have been further supported by animal studies showing that exercise reduces amyloid deposits and enhances synaptic plasticity in rodent models of AD. In populations already diagnosed with dementia, exercise may still offer value. Studies have demonstrated improvements in mobility, balance, and activities of daily living, as well as potential slowing of cognitive deterioration. Though fewer in number, trials involving individuals with diagnosed dementia suggest that structured, multi-component physical training can help maintain functional independence and improve behavioural symptoms.

Despite promising results, more research is needed to determine the optimal type, duration, and intensity of exercise interventions for various stages of cognitive decline. Future studies should standardize measurement tools and explore long-term outcomes, including institutionalization rates and quality of life. Integrating physical activity into public health recommendations for aging populations represents a promising avenue to reduce the societal burden of dementia.

J. Karamacoska et al. (2023)

Cognitive decline, particularly in the form of mild cognitive impairment (MCI), vascular cognitive impairment (VCI), and subjective cognitive decline (SCD), represents an early warning stage for the development of dementia. As the global burden of dementia increases, there has been growing interest in non-pharmacological strategies for prevention and delay of cognitive deterioration. Among these, physical exercise has emerged as a promising intervention with the potential to influence both cognitive outcomes and underlying brain function. Numerous studies have demonstrated that exercise interventions—including aerobic, resistance, and mind-body practices—can have a beneficial impact on cognitive performance in aging individuals. Meta-analyses and randomized controlled trials (RCTs) have shown that regular physical activity can improve global cognition, executive functioning, and even delay the onset of Alzheimer's disease in at-risk populations. However, most of the earlier research has focused on behavioural and clinical outcomes, with relatively limited attention paid to the neurophysiological mechanisms underlying these improvements. Karamacoska et al. (2023) addressed this gap by systematically reviewing and meta-analysing studies that evaluated both cognitive and brain function changes following exercise interventions in adults with cognitive decline. Unlike earlier reviews, this work emphasized the integration of functional neuroimaging and electrophysiological data to explore the brain-level adaptations associated with exercise. Their review included 12 unique RCTs involving 646 participants with cognitive frailty, MCI, or VCI. The interventions typically involved structured exercise performed 2–3 times per week for at least four weeks, with modalities including aerobic activity (such as walking or cycling), resistance training, and mind-body exercises like

Badu Anjin. Cognitive performance was measured across domains such as global cognition, executive function, processing speed, and short-term memory, while brain function was assessed using tools like functional MRI (fMRI), EEG, event-related potentials (ERP), and cerebral blood flow imaging.

The meta-analysis revealed significant improvements in global cognition and executive function in those who participated in exercise interventions compared to control groups. However, gains in processing speed and short-term memory were not statistically significant. Importantly, neuroimaging data showed that exercise led to functional changes across multiple brain regions—including the frontal, temporal, and parietal lobes—as well as improved cerebral blood flow and neural efficiency. For example, fMRI studies found increased hemodynamic activity in brain regions responsible for memory and executive function following aerobic and resistance training. EEG and ERP data indicated enhanced neuronal complexity and faster processing, while studies using Doppler and near-infrared spectroscopy (NIRS) reported improved cerebral perfusion. These physiological changes were often correlated with better cognitive test performance, suggesting a functional link between exercise-induced brain plasticity and cognitive resilience. Despite the promising findings, the review also noted substantial variability in study design, intervention protocols, and outcome measures. Only a minority of studies were rated as high-quality, and publication bias or inconsistent methodologies may have influenced some results. Furthermore, there remains a lack of studies involving individuals with SCD, despite its potential as a critical intervention window.

In conclusion, the literature reviewed by Karamacoska et al. supports the notion that exercise not only enhances cognitive performance in individuals with cognitive decline but also induces measurable functional brain changes. This dual impact underscores the value of physical activity as a multidimensional intervention capable of targeting both behavioural and neural aspects of dementia risk. Future research should aim to standardize intervention protocols, include diverse populations, and explore long-term impacts on both cognition and brain health.

III. RATIONALE OF THE STUDY

Currently, there is no known treatment to cure dementia and related disorders completely. Also, only a little is known about such disorders, specially how the brain actually works during an episode of memory loss in a dementia patient. Overviewing the above studies have stated that brain exercises can actually help slow down the progression of early-onset dementia, but a specific kit has never been made which includes targeted mental exercises along with the combination of acupressure to assist in the process of slowing down the progression of early-onset dementia.

The aim of this exploratory research study is to track the mental and physiological progress of patients of dementia, specifically targeted towards Alzheimer's early-onset patients, with the periodic (weekly) use of newly developed K-MEPSE (Kamalpuria's Mental Exercise and Physiological Strength Enhancer) kit, in a specific period of six months. There would be weekly sessions involving conduction of the kit exercises along with blood pressure and sugar tests. The progress would be tracked monthly by the dementia screening test RUDAS (Rowland's Universal Dementia Assessment Scale).

This study also reviews the existing literature and understandings about dementia and the effect of therapeutic measures on helping slow down its progression. It is also very important to have a deep research and analysis on memory loss episodes of dementia patients because it is seriously traumatizing for the patients, which can question a person's safely and leave deep scars on their mind.

Despite the number of studies on this aspect being limited, the work carried out by researchers till date has provided great insights and valuable connections and relationships into how therapeutic interventions can be linked to dementia and its progression. Results from various survey studies have revealed a large number of different and divergent aspects of dementia, which can be linked to brain exercises. Many of these studies have efficiently used large sample sizes, which is a great strength and advantage to the work. However, there still exist large gaps in the studies that future studies need to assess, hereby, this study is just a block in the bridge of fillings these gaps significantly.

IV. METHODOLOGY

A. Aim

This research study seeks to observe the effect of the use of K-MEPSE kit (Kamalpuria's Mental Exercise and Physical Strength Enhancer Kit) in early-onset Dementia patients (specifically Alzheimer's patients) in a span of six months.

B. Objectives

1. To find out improvements in scores of RUDAS scale caused by the use of K-MEPSE kit.
2. To find out if K-MEPSE kit helps in keeping Blood pressure and Blood sugar levels in control.
3. To find out where the improvement stops while using the kit.

C. Research Methodology

- **Research Design:**

Quantitative and qualitative study to be conducted using face-to-face interviews and sessions.

- **Step 1:** Conduction of RUDAS test. This is a paper pencil test which gives us a specific score determining the current stage of dementia in the patient. This test was repeated every month on the same date to track the progress for a period of six months.
- **Step 2:** Checking Blood Pressure and Blood Sugar. These tests were repeated every month on the same date to track the progress for a period of six months.
- **Step 3:** The Kit exercises began after the first RUDAS, BP and Blood sugar test. Kit exercises takes place once a week regularly for a period pf 6 months and the progress was tracked through RUDAS test along with BP and Blood Sugar test.

K-MEPSE kit exercises may take up to 1 to 2 hours for conduction per person, depending upon the patient's current mental state.

D. Ethical Considerations

Given the nature of this project, there are minimal ethical considerations that need to be taken into consideration. All participants will be treated with respect and courtesy. A strategy of 'informed consent' will be adopted, with the aim and methods of the research being made clear to all participants. The confidentiality and anonymity of individual respondents will be assured, and consent will be sought for the sessions. There will be a caretaker (relative of the participant) present at all times with the participant and his consent will also be taken.

E. Sample

1. *Sampling Technique:* Homogeneous Purposive Sampling technique was used in this study. The sample comprised of Young adults (20-40 years) and middle age adults (41-60 years) with clinically diagnosed Dementia.
2. *Sampling Size & Nature:* Total 50 participants were taken, out of which 25 participants were below 60 years and 25 participants were of 60-65 years.
3. *Research Period:* 6 Months
4. *Research Instrument:* Kamalpuria's Mental Exercise and Physiological Strength Enhancer Kit (K-MEPSE Kit).

F. Sampling Criteria:

1. Inclusion criteria for the study:
 - Subjects below 65 years of age.
 - Subjects only from Mumbai.
 - Subjects either male or female.
 - Subjects those who have diagnosed Dementia.

2. Exclusion Criteria for the study:

- Subjects above 65 years of age.
- Subjects outside Mumbai.
- Subjects other than male or female.
- Subjects are those who do not have Dementia clinically diagnosed by a physician.

G. Tests/Tools

The tool used for data collection are stated below:

1. Rowland Universal Dementia Assessment Scale: RUDAS

- **Authors' name:** Joella E. Storey and Jeffrey T. J. Rowland et. al. (2004)

• Sub factors:

- Memory: Short-term recall of a newly learned four-item grocery list.
- Body Orientation: Ability to identify and cross-reference left and right body parts.
- Praxis: Motor planning and execution, tested by mimicking a multi-step hand movement.
- Visuospatial: Visuospatial skills, evaluated by drawing a cube.
- Language: Fluency and word generation, measured by naming as many animals as possible in 60 seconds.
- Judgment: Practical problem-solving and safety awareness, assessed using a standard traffic crossing scenario.

• Total number of items: 6

- **Scoring:** Each of the six tested areas carries a specific point value: memory recall is worth 2 points, body orientation is worth 1 point, praxis (motor skills) is worth 2 points, visuospatial drawing is worth 1 point, language/animal fluency is worth 2 points, and the judgment scenario is worth 2 points. Unlike many other cognitive assessments, the scoring of the language component is adjusted based on the individual's formal education level rather than their native language proficiency, ensuring the test remains culturally fair and education-neutral. A final score of 22 or lower is widely recognized as the clinical threshold warranting further diagnostic evaluation for dementia.

- **Norms:** A threshold score of 22 out of 30 is globally utilized as the optimal boundary for identifying cognitive impairment, yielding a sensitivity of 89% and a specificity of 98%. To maintain this equity without complex normative conversions, a single-point correction is added directly to the total score if an individual has completed less than six years of formal education. This built-in

adjustment effectively eliminates the need for separate demographic norms, making the instrument exceptionally reliable for multicultural clinical assessments.

- **Reliability and Validity:** In terms of reliability, the scale displays outstanding consistency across various evaluations, typically yielding test-retest and inter-rater reliability coefficients above 0.90, which confirms that different clinicians can achieve virtually identical results over time. Its construct and concurrent validity are reinforced by strong correlations with older, established cognitive tests like the Mini-Mental State Examination (MMSE) and the Montreal Cognitive Assessment (MoCA). Furthermore, diagnostic validity is exceptionally high; receiver operating characteristic (ROC) analyses frequently show an area under the curve (AUC) between 0.84 and 0.98. At its traditionally recommended cut-off score of 22, the tool achieves a sensitivity of roughly 89% and a specificity of up to 98% in identifying definite cognitive disorders, proving that it is both a highly stable and highly accurate diagnostic asset.

H. Data Collection

1. **Primary Data:** The Primary data comprises of one to one individual sessions with early-onset dementia patients (specifically Alzheimer's).
2. **Secondary Data:** The Secondary data was collected through research papers on internet and references from library.

V. DATA ANALYSIS

RUDAS was conducted at the beginning of the trial and their MEAN SCORES were noted. After the conduction of RUDAS, it was found that the patients scores differentiated age-wise in their ranges (Mild and Severe cognitive impairment). Since the study focusses on Early-onset Dementia (specifically Alzheimer's), Patients below the age of 65 with dementia were taken as sample. Below are the Mean Scores of the sample at the beginning of the trial, as compared to the end of the trial, i.e., after 6 months.

Beginning of the Trial		
AGE	RUDAS SCORE	RANGE
Below 60 Years	23	Mild Neuro-Cognitive Impairment
60-65 Years	18	Severe Neuro-Cognitive Impairment

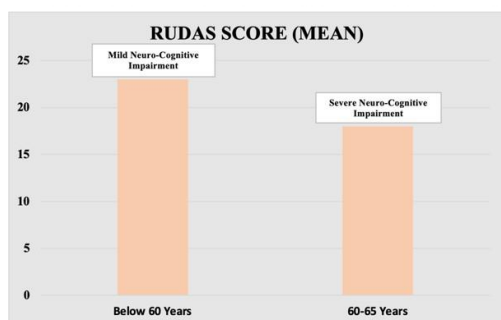


Figure 1: Graphical representation of RUDAS Mean Scores Before

End of the Trial (After 6 Months)		
AGE	RUDAS SCORE	RANGE
Below 60 Years	27	Mild Neuro-Cognitive Impairment
60-65 Years	21	Severe Neuro-Cognitive Impairment

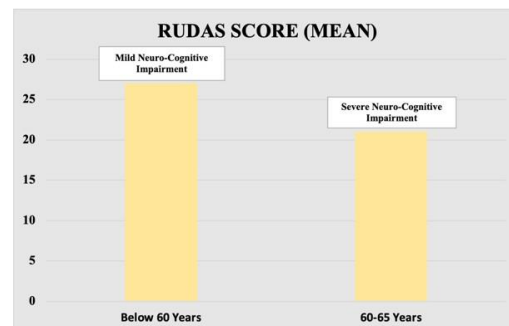


Figure 1: Graphical representation of RUDAS Mean Scores After

VI. FINDINGS & OBSERVATIONS

The following observations could be made after conduction of the trial:

- The RUDAS score of sample group below 60 years of age (25 people) showed an increase by 4 points, i.e., from 23 to 27. Although the range of neuro-cognitive impairment remained the same, it can be assumed that with further continuous practice, the range can also be changed.
- The RUDAS score of sample group 60-65 years of age (25 people) showed an increase by 3 points, i.e., from 18 to 21. Although the range of neuro-cognitive impairment remained the same, it can be assumed that with further continuous practice, the range can also be changed. For severe impairment, this change may take more time than for people with mild neuro-cognitive impairment.
- With continuous conduction of K-MEPSE Exercise for 6 months, there was minimal practice effect noticed in dementia patients. This may change if the scores keep increasing.
- The progress/improvement did not seem to stop at any stage within a period of 6 months. It may differ once the patient's RUDAS score has reached normal neuro-cognitive functioning (zero or minimal impairment), due to improvement in memory, leading to practice effect.
- The Blood Pressure (Post-meal weakly) and Blood Sugar (Post-meal monthly) levels were checked which also showed slight stability during the later stages of the trial as compared to initial fluctuations and imbalances.
- With the help of Projective depression screening tests, we were able to track symptoms of onset of depression or depressive traits in a few patients, which were immediately looked into and initiated the treatment for, by their Consultant psychiatrist.

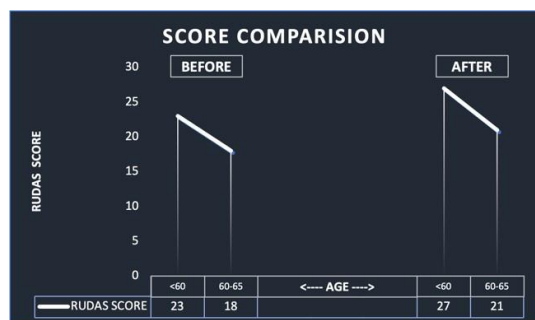


Figure 3: Comparison chart of RUDAS Mean Scores Before and After

VII. CONCLUSION

Backed up with detailed analysis of each case, it can be said that the aim of this exploratory research study, which was to track the mental and physiological progress of patients of dementia, specifically targeted towards Alzheimer's early-onset patients, with the periodic (weekly) use of newly developed K-MEPSE (Kamalpuria's Mental Exercise and Physiological Strength Enhancer) kit, in a specific period of six months, was fulfilled, showing positive results and increase in RUDAS scores of dementia patients. Improvement, although by 3-4 point scores, but was noted in the RUDAS scale, depressive traits were tracked and handled effectively, and the acupressure exercises were found to be effecting in stabilizing the overall physiological health of the patient. Therefore, the K-MEPSE kit can surely be used as a complete package of mental exercises and acupressure which can help in improving brain's cognitive functions significantly.

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APPENDICES

R U D A S	
The Rowland Universal Dementia Assessment Scale: A Multicultural Cognitive Assessment Scale. (Storey, Rowland, Basic, Conforti & Dickson, 2004). International Psychogeriatrics, 16 (1), 13-31	
Date: ____/____/____ Patient Name: _____	
Item	Max Score
Memory	
1. (Instructions) I want you to imagine that we are going shopping. Here is a list of grocery items. I would like you to remember the following items which we need to get from the shop. When we get to the shop in about 5 mins. time I will ask you what it is that we have to buy. You must remember the list for me. Tea, Cooking Oil, Eggs, Soap Please repeat this list for me (ask person to repeat the list 3 times). (If person did not repeat all four words, repeat the list until the person has learned them and can repeat them, or, up to a maximum of five times.)	
Visuospatial Orientation	
2. I am going to ask you to identify/show me different parts of the body. (Correct = 1). Once the person correctly answers 5 parts of this question, do not continue as the maximum score is 5.	
(1) show me your right foot	1
(2) show me your left hand	1
(3) with your right hand touch your left shoulder	1
(4) with your left hand touch your right ear	1
(5) which is (indicate/point to) my left knee	1
(6) which is (indicate/point to) my right elbow	1
(7) with your right hand indicate/point to my left eye	1
(8) with your left hand indicate/point to my left foot	1
.....5	
Praxis	
3. I am going to show you an action/exercise with my hands. I want you to watch me and copy what I do. Copy me when I do this . . . (One hand in fist, the other palm down on table - alternate simultaneously.) Now do it with me: Now I would like you to keep doing this action at this pace until I tell you to stop - approximately 10 seconds. (Demonstrate at moderate walking pace). Score as: Normal = 2 (very few if any errors; self-corrected, progressively better; good maintenance; only very slight lack of synchrony between hands) Partially Adequate = 1 (noticeable errors with some attempt to self-correct; some attempt at maintenance; poor synchrony) Failed = 0 (cannot do the task; no maintenance; no attempt whatsoever)	
.....2	
Visuoconstructional Drawing	
4. Please draw this picture exactly as it looks to you (Show cube on back of page). (Yes = 1)	
Score as:	
(1) Has person drawn a picture based on a square?	1
(2) Do all internal lines appear in person's drawing?	1
(3) Do all external lines appear in person's drawing?	1
.....3	
Judgment	
5. You are standing on the side of a busy street. There is no pedestrian crossing and no traffic lights. Tell me what you would do to get across to the other side of the road safely. (If person gives incomplete response that does not address both parts of answer, use prompt: "Is there anything else you would do?") Record exactly what patient says and circle all parts of response which were prompted.	
.....	
Score as:	
Did person indicate that they would look for traffic? (YES = 2; YES PROMPTED = 1; NO = 0)	2
Did person make any additional safety proposals? (YES = 2; YES PROMPTED = 1; NO = 0)	2
.....4	

Memory Recall	
1. (Recall) We have just arrived at the shop. Can you remember the list of groceries we need to buy? (Prompt: If person cannot recall any of the list, say "The first one was 'tea'." (Score 2 points each for any item recalled which was not prompted – use only 'tea' as a prompt.)	
Tea	2
Cooking Oil	2
Eggs	2
Soap	2
.....8	
Language	
6. I am going to time you for one minute. In that one minute, I would like you to tell me the names of as many different animals as you can. We'll see how many different animals you can name in one minute. (Repeat instructions if necessary). Maximum score for this item is 8. If person names 8 new animals in less than one minute there is no need to continue.	
1.	5.
2.	6.
3.	7.
4.	8.
TOTAL SCORE =	
.....8 /30	

