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Advances in understanding the mechanisms and management of persistent pain in older adults†

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Older adults with persistent pain are not simply a chronologically older version of younger pain patients. Pain-related disability in older adults may be driven by pain 'homeostenosis', that is, diminished ability to effectively respond to the stress of persistent pain. Some of the comorbidities of ageing that can contribute to pain homeostenosis include cognitive and physical impairments, increased sensitivity to suprathreshold pain stimuli, medical and psychological comorbidities, altered pharmacokinetics and pharmacodynamics, and social isolation. A key distinction between older and younger individuals with persistent pain is the normal and pathological ageing-associated brain changes. These may alter the expression and experience of pain with impaired descending inhibition and dysfunction of pain gating mechanisms. Cognizance of these brain changes is needed to guide appropriate evaluation and treatment approaches. This paper reviews data that support these ageing-associated phenomena. Specifically, we discuss age-related changes in the brain (both normal and pathological) and in pain physiology; changes in experience and expression of pain that occur with dementia and contribute to pain homeostenosis; and unique aspects of age and pain-associated psychological function and their contribution to disability. We also present data demonstrating changes in brain morphology and neuropsychological performance that accompany persistent non-malignant pain in older adults and the treatment implications of these brain changes. Finally, preliminary data are presented on the efficacy of mindfulness meditation, a treatment that has been examined explicitly in older adults and targets optimizing brain function and descending inhibition.

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As the population of developed countries ages, there has been an increase in the prevalence of conditions associated with persistent pain across settings of care.[24] In the USA and Canada, 25–50% of community-dwelling older adults and 49–83% of nursing home residents report pain.[21 34 36 95] Data from Europe echo these prevalence estimates.[26 100 108 121] The prevalence of persistently painful conditions among older adults is particularly noteworthy in light of their association with functional impairment, sleep disturbance, depression and anxiety, and decreased socialization.[1]

The physiological, psychological, and environmental changes that accompany ageing and restrict homeostasis may further exacerbate the consequences of persistent pain. Allostasis or homeostasis (i.e. 'maintaining stability through change')[70] is the response of the body to stress by activation of physiological reserves. These reserves include cognitive and emotional resilience to stress and activation of the neuroendocrine system, the autonomic nervous system, and the immune system. In contrast to the homeostasis of maintained internal equilibrium through the adjustment of physiological processes, *homeostenosis* is the constriction of an ageing organism's ability to effectively respond to stress because of diminished biological, psychological, and social reserves.[5 89 109] When the inherent reserve capacity is exceeded, this may result in disability or death.

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We use the term *pain homeostenosis* to describe the diminished ability of an organism to effectively respond to the stress of persistent pain. A number of factors may contribute to this diminished ability in older adults. These include decreased cognitive reserves, decreased density of opioid receptors; altered pharmacokinetics and pharmacodynamics of the ageing body; polypharmacy, high medical comorbidity, the frequent social isolation, loneliness, and depression of old age; and impairments in activities of daily living.

A key distinction between older and younger individuals with persistent pain is the normal and pathological ageing-associated brain changes that may contribute to pain homeostenosis. That is, the older adult's experience of pain may be altered because of dysfunctional brain changes that cause impaired descending inhibition. Intact descending inhibition is a key component of modulation of the barrage of sensory input from the periphery that ascends to the brain, as described in the Gate Control Theory of Pain (GCT).[73] Impaired descending inhibition caused by fear, dysfunctional coping, depression, and anxiety has been found to play a major role in driving disability in younger patients with persistent pain. This paper focuses on changes in the brain and other contributors to pain homeostenosis and the implications that these changes have for the evaluation and management of persistent pain in older adults. Specifically, we discuss the evidence that demonstrates (i) age-associated changes in pain processing, (ii) changes in pain processing and expression that occur in older adults with dementia and the treatment implications of these changes, and (iii) the role of age and pain-associated changes in psychological function in contributing to disability. We also present data demonstrating pain-associated brain changes in cognitively intact older adults with persistent non-malignant pain. Finally, preliminary data are presented on the efficacy in older adults with persistent pain of mindfulness meditation, a treatment designed to optimize descending inhibition.

Age-associated changes in pain processing

The density of both myelinated and unmyelinated peripheral fibres has been found to decrease with age.[81 82 87] The number of sensory fibres, both myelinated and unmyelinated, with signs of damage or degeneration (e.g. axonal involution, Wallerian degeneration) also shows a marked increase with advancing age, and peripheral nerve conduction velocity may slow somewhat.[27 51 59] Selective age-related impairment of myelinated nociceptive fibre function and consequent impairment in the early warning functions of nociceptive A-delta fibres have also been observed.[14 38]

The effects of age on pain threshold are contradictory. It has been reported that somatosensory thresholds for non-noxious stimuli increase with age, whereas pressure pain thresholds have been reported to both increase and decrease and heat pain thresholds may show no age-related changes.[38 39] Threshold and tolerance of experimentally induced ischaemic pain is significantly less in older than in younger adults.[28] Apart from an enhanced temporal summation of heat pain, pain summation may not be critically affected by age.[62] However, recent work has suggested that the nociceptive system of older subjects may indeed have a reduced capacity to down-regulate subsequent to sensitization.[32 37] The relationship between these physiological changes observed in the laboratory and clinical pain states is unknown,[37] although data suggest that older adults with persistent pain may function at a higher psychological and physical level than their younger counterparts.[118]

Age-associated brain changes

The GCT and modern pain theory have relevance across the lifespan, although age-related changes within the nervous system (e.g. peripheral nociceptors, spinal cord, and brain) may affect the pain experience in older adults. In the brains of most older adults, some evidence of pathological changes are evident.[7 47] Brain morphology and function [i.e. neuropsychological performance (NP)] change as a result of normal ageing.[19 31 35 88 115] Alterations in brain morphology are associated with decrements in NP in the absence of dementia.[18 124] Loss of brain volume, senile neuritic plaques, and neurofibrillary tangles may also be observed with no evidence of cognitive impairment.[58] It is unknown if these non-clinical brain changes which affect the medial pain tract (e.g. frontal cortex, anterior cingulate cortex, insula cortex, and hypothalamus) affect the experience of the older adult living with persistent pain. It is the medial pain system that is thought to be involved in the motivational–affective, cognitive–evaluative, and autonomic–neuroendocrine components of pain perception. It is known, however, that when plaques and tangles of sufficient density have spread to the neocortex and destroyed a critical number of neurons, cognitive impairment develops.[76]

The pathophysiology of both mild age-related changes and more severe changes associated with dementia is neuronal death and gliosis. Areas of the brain involved with pain perception and analgesia are susceptible to these pathological changes. Functionally, neuronal death and gliosis may directly interrupt neuronal tracts involved in descending inhibition, especially those involved with the periaqueductal gray, locus coeruleus, and nucleus raphe magnus, areas rich in opioid and monoamine receptors.[122 123]

In addition to functional changes, another result of ageing and disease is changes in behaviour. Many older adults have excellent coping skills and live with persistent pain that is not disabling.[118] Some individuals such as

those who suffer from comorbid dementia and/or depression, however, may experience behavioural changes including decreased ability to cope with pain, impaired ability to effectively express needs and distress, and difficulty with adhering to an analgesic or other somatic regimen.

Age-associated central changes in significant neurotransmitters

Areas of central pain regulation have been identified in the midbrain, pons, and the medulla, especially around the cerebral aqueduct (i.e. the periaqueductal gray matter).[8] These areas of the brain are rich in endogenous opioids and opioid receptors and they also give rise to fibre tracts that project to the dorsal horn of the spinal cord, where serotonin (in the raphe nucleus), norepinephrine (in the locus coeruleus), and acetylcholine are released. The action of these tracts is to inhibit nociceptive input from afferents and/or output by nociceptive second-order neurones.[53] These neurotransmitters, especially serotonin, result in inhibition of dorsal horn nociceptive structures, which are mediated by the activation of opioid-releasing interneurones.

There is evidence of a progressive age-related loss of serotonergic and noradrenergic neurones in the dorsal horn.[48 60] Within the limbic system, there is a decline in the concentration and turnover of catecholamines,[4] GABA[103] and opioid receptors,[2] and a reduction in serotonin receptor density,[52] particularly within the anterior cingulate and prefrontal cortex, brain areas involved in descending inhibition.[99] The cerebral cortex displays similar age-dependent reductions in dopamine, noradrenaline, GABA, and acetylcholine neurotransmission[23 40 42 71 90 91 116] and a reduction in the density of serotonin[68 120] and glutamate receptors.[96] Age-related changes in glutamate and GABA are also noticeable in the prefrontal cortex and may result in abnormal pain summation.[41] These age-related decreases in monoamines and other significant neurotransmitters may contribute to pain homeostenosis. In other words, the neurochemicals necessary for pain modulation may not be sufficiently available in older adults.

Additional factors that may contribute to pain homeostenosis

Normal ageing may be associated with homeostenosis of a number of biological, psychological, and social systems that restrict the ability to respond to the stress of persistent pain, that is, pain homeostenosis. Social losses include status, independence, spouse/partner, friends, and financial income. Facing these losses, one of the main challenges in late life is to maintain mental activity and social engagement, and avoid isolation, marginalization, depression, and stigmatization. When these losses overwhelm psychological, cognitive, and physical resources, descending inhibition of persistent pain conditions may be compromised. For example, in our work in geriatric medical and mental health treatment settings, we encounter patients with worsening pain conditions such as chronic low back pain (CLBP), osteoarthritis pain, and fibromyalgia consequent to (i) the loss of a spouse, (ii) worsening frailty and fears about independence, and (iii) exacerbation of other medical problems such as cardiac and pulmonary disease. For all patients with persistent pain, but older adults in particular, attention to comorbid social, medical, and cognitive losses is crucial in an effort to optimize descending inhibition and, therefore, pain management.

Physiological homeostenosis is a common part of ageing. Significant changes occur in transporting an appropriate concentration of drug to its site of action and the ability of membrane receptors to respond appropriately (e.g. to opioids, NSAIDS, and antidepressants). In particular, pharmacokinetics (absorption, distribution, metabolism, and excretion) and pharmacodynamics (altered receptor sensitivity, homeostasis, half-life, and steady state) all change to various degrees with age.[25 44] These biological changes frequently affect treatment response and sensitivity to side-effects, making it more challenging to achieve effective analgesia with medications in many older adults.

Compounding these changes in the physiological milieu, it is common for older adults to incorrectly take prescribed or over the counter medications. Reasons for problems with adherence include: (i) misunderstanding of prescribing instructions; (ii) problems with vision and hearing; (iii) confusion and cognitive impairment; or (iv) simply lack of interest.[67] Using common sense, the assistance of caregivers and safe prescribing habits can minimize the variance in treatment outcomes that can result from these potential contributors to pain homeostenosis in late life.

Pathological brain changes and pain processing

Alzheimer's disease (AD) is the most common dementing illness among older adults, with an estimated 8–15% affected who are 65 yr of age and older. AD primarily affects the medial pain system, with corresponding effects on the motivational–affective, cognitive–evaluative, and autonomic–neuroendocrine components of pain. The lateral pain system, involved with the sensory-discriminative elements of pain, only becomes involved relatively late in the disease. Patients with AD may have less affective response to pain[92] while maintaining a comparable pain threshold to cognitively intact patients.[6]

Support also exists for similar and exaggerated affective pain responses in AD. Porter and colleagues[86] demonstrated exaggerated facial expressions in response to venepuncture in older adults with AD when compared with cognitively intact older adults. One functional magnetic resonance imaging study demonstrated activation of the medial and lateral pain pathways in both AD and control subjects and comparable unpleasantness ratings in response to mechanical pressure stimuli.[17] Those with AD demonstrated greater amplitude and duration of pain-related activity in sensory, affective, and cognitive processing regions, interpreted as greater attention to noxious stimuli. For a discussion of the impact of other types of dementia on pain processing, the reader is referred to a review by Scherder and colleagues.[94]

Pathological brain changes and pain expression

For cognitively intact older adults, pain assessment relies on the reliability and validity of self-report[1] and behavioural observation.[56] Pain expression in those with dementia, however, may pose threats to the validity of traditional approaches to pain assessment. For example, patients with cognitive impairment generally report less pain than cognitively intact older adults even though there is no evidence that cognitive impairment reduces the ability to feel painful stimuli.[84 112] On the other hand, older adults with dementia may display behavioural indicators of pain (e.g. bracing) in the absence of self-reported pain.[97] When evaluating pain in older adults with dementia, experts recommend incorporating several methods: self-report, proxy report, and behavioural scales.[1]

Pain measurement in older adults with dementia should take into account the severity of cognitive loss.[93] Evidence for the reliability of current pain self-report in those with mild to moderate dementia is strong.[15 105 106] The reliability of historical pain reporting has not been evaluated in those with dementia. As cognitive decline progresses to more advanced stages that are associated with further deterioration of memory and verbal abilities, the utility of self-report scales becomes more limited and proxy pain assessments are increasingly relied upon.[1]

Professional caregivers (nurses or nursing assistants) tend to underestimate the presence of pain,[30] but not at higher levels of pain intensity.[98] In contrast, family caregivers tend to report more pain than that reported by the cognitively impaired individual. The accuracy of proxy pain assessment also may be impacted by dementia-associated behavioural changes. For example, high levels of agitation are associated with increased likelihood of pain rating disagreement between patients and caregivers.[98]

Formal pain behaviour observation instruments have been developed to assist with pain assessment in those unable to report pain.[43] Domains frequently included are changes in facial expression, verbalizations or vocalizations, body movements (guarding, pacing, rocking, and rigid tense body posture), changes in interpersonal interactions (withdrawn, disruptive, aggressive), changes in activity patterns or routines (changes in appetite, sleep, or routines), and mental status changes (confusion and crying).[1] No one instrument has sufficiently developed psychometric properties to be recommended for routine clinical use.[43 45] Importantly, the specificity of the behaviours identified using these scales for identifying pain has not been evaluated. For example, psychological symptoms (e.g. depression, anxiety, and fear) and unmet physical needs (e.g. hunger, social isolation, and soiled diaper) could cause behavioural expressions that might be misconstrued as pain.

Although behavioural changes such as altered movement patterns (e.g. bracing, guarding) are often relied upon as indicators of pain, these changes may occur as part of dementia itself. For example, progressive AD may be accompanied by parkinsonian rigidity, spasticity, and spontaneous non-startle myoclonic jerks.[117] Rigidity is also a core feature of Lewy body dementia.[72] Vascular dementia may be accompanied by a variety of cognitive, behavioural, and neuromuscular changes depending on the location and degree of neuronal injury or loss.[9] It is not difficult to imagine, therefore, that these changes might impact the specificity of behavioural pain ratings.

We recently evaluated the validity of traditional pain behaviours (guarding, bracing, rubbing, grimacing, and sighing) by examining if pain status and/or cognitive status were independently associated with the frequency of observed behaviours. The number of pain behaviours was recorded as participants completed a structured protocol that simulated activities of daily living.[111] The two pain groups were CLBP and pain-free and the two cognitive status groups were mild to moderate dementia and cognitively intact. Participants with CLBP, independent of cognitive status, displayed significantly more grimacing and guarding behaviours than the pain-free group. Participants with dementia, independent of their pain status, displayed significantly more guarding, bracing, and rubbing behaviours than cognitively intact participants. It is noteworthy that rubbing is considered a stereotypical movement in persons with dementia.[80] Although this behaviour is more common in persons with frontotemporal dementia, it does occur in other more common neurodegenerative diseases such as AD.[74] Thus persons with dementia may exhibit behaviours that could just as easily be related to pain as to the underlying neurodegenerative disease itself.

Dementia and pain treatment implications

Because of the non-specificity of some pain behaviours in older adults with dementia, pain assessment in these

individuals requires a broad and thoughtful approach.[46] When behaviours indicate the possibility of underlying pain, a comprehensive search for other potential contributors should ensue before pain-specific treatment is initiated. The contribution of psychological factors must be carefully considered. Older adults with dementia may have exaggerated fear avoidance and catastrophize excessively when faced with pain. Impaired coping may be the primary driver of the patient's disability, as illustrated by the following case.

An 82-yr-old woman presented with a 2 yr history of low back and right leg pain and a diagnosis of lumbar spinal stenosis based upon magnetic resonance imaging. She had been very active, working full time in a dress shop until 2 yr earlier when she was forced to retire because the company was downsizing. She lived alone and her pain started after she retired. She reported increased pain intensity with prolonged standing and walking and improvement with application of heat. She denied fever, chills, weight loss, paresthesias, lower extremity weakness, or change in function of her bowels or bladder. She denied nocturnal symptoms. It had become increasingly difficult for her to do heavy housework. She reported frequent near-falls, passive suicidal ideations, and fear of going on the bus alone, so she was spending more time at home alone. Her medications included gabapentin, oxycodone CR, celecoxib, tramadol, acetaminophen, olanzapine, escitalopram, and lorazepam. Physical examination was notable for very impaired righting reflexes (i.e. inability to right herself in response to a gentle backwards tug at the waist) and performance on the clock drawing test, marked kyphoscoliosis, and tenderness on palpation of the right sacroiliac joint, tensor fascia lata (TFL), and erector spinae. Strength testing was limited by extreme guarding behaviour.

She was admitted to a nursing home for detoxification. All of her medications were discontinued with the exception of regularly scheduled acetaminophen and p.r.n. tramadol and she was prescribed physical therapy for gait training and for treatment of her TFL myofascial pain and dysfunction. Her balance and cognitive function improved markedly and her pain complaints became infrequent. Assisted living facility placement was recommended, as social isolation and mild dementia were felt to have significantly contributed to her pain complaints, but the patient and her family refused. Within 24 h of discharge, the patient's pain complaints escalated. She began calling frequently, asking for more pain medication. Because the physician (D.K.W.) remained firm in her conviction that the patient's social situation was driving her pain behaviour, the analgesic regimen was not changed. She, therefore, sought another pain provider who escalated her pain medication, culminating in an unsuccessful morphine pump trial. Ultimately, the patient was admitted to an assisted living facility, where she did well.

In this case, social isolation, fear, and dementia together caused pain homeostenosis and the patient's disability. When social isolation was removed by assisted living facility placement, her pain homeostenosis was manageable and no longer disabling. Pain treatment *per se* played a very minor role in the patient's improvement. In older adults with dementia and disability, therefore, it is critical that factors other than pain are managed aggressively so as to optimize quality of life and avoid morbidities that can result from treatment focused exclusively on pain.

Age and pain-associated changes in mood and coping: treatment implications

Depression and anxiety disorders are common in older adults and, like dementia, can contribute to pain homeostenosis. Depressive symptoms that cause distress and interfere with day-to-day functioning occur in approximately 15% of community-dwelling older adults.[78] Rates are higher in medically hospitalized and nursing home residents.[16 49 65 66] Anxiety disorders are frequently comorbid with depression in older adults, and the point prevalence of anxiety in late life is estimated to be as high as 65% in treatment-seeking samples.[64 79 83] As described earlier, affective and anxiety disorders share brain areas and neurotransmitters involved with persistent pain. Commonalities include high comorbidity, a recurrent and chronic natural history, mutual exacerbation, and flaring of symptom levels in response to external stimuli such as physical or emotional stress. Treating symptoms of mood and anxiety and addressing passive and ineffective pain coping strategies is critical to optimize analgesia.

When comorbid, pain has been shown to slow treatment for major depressive disorders among older adults receiving treatment with paroxetine and interpersonal psychotherapy.[55] In a large treatment study of older adults (n=524), interference from pain was found to hinder recovery from depression.[69] While affecting treatment outcomes in late life, pain and depression have also both been found to be risk factors for each other. For example, in a study of community dwelling adults aged 70 and older who were independent in bathing, walking, dressing, and transferring at baseline, Reid and colleagues[12] found that after adjusting for potential confounders, the presence of depressive symptoms was independently associated with the occurrence of disabling back pain of at least 3 months duration (adjusted odds ratio=7.8). These findings are supported by a large survey of community-dwelling older adults (n=55 690 at follow-up) which found baseline depression symptoms increased the odds of disabling low back pain after 2 yr independent of sociodemographic characteristics, medical, and functional status. In this report, disabling low back pain at baseline also increased the odds of depressive symptoms after 2 yr to a similar degree.[75] These studies reinforce the overlap between depression and pain in late life and support the significance of simultaneous treatment of both affective illness and pain.

Although no treatment studies for anxiety in older adults are available for direct comparison, data from cross-sectional observational studies suggest a similar interaction. For example, anxiety was the only significant predictor of pain in a sample of patients over the age of 65 receiving inpatient rehabilitation after orthopaedic surgery (e.g. knee or hip replacement surgery).[33] In another study of assisted living and skilled nursing home residents, anxious effect was found to exhibit a moderately stronger relationship to number of localized pain complaints than did depression.[13] As with younger adults, to optimize descending inhibition and analgesia, comorbid depression and anxiety should be aggressively treated to remission.

Catastrophizing is a maladaptive pain coping style (e.g. characterizing pain as awful and unbearable, magnifying and ruminating about painful stimuli)[20 101 104] that is associated with increased pain intensity and disability,[85 119] low self-efficacy,[57] external locus of control,[102] depression,[61] and suicidal ideation.[29] Based upon our clinical experience, older adults who catastrophize often experience higher rates of fear avoidance. An example of this thought sequence is the older adult who is convinced that if they go for a walk or participate in physical therapy, they will experience either a pain flare and/or physical reinjury. Fear avoidance beliefs are associated with reduced activity and increased disability,[110] and catastrophic thinking leads to avoidance of behaviours that they perceive as potentially dangerous to their physical or emotional well-being. These fear avoidance behaviours usually manifest as overuse of analgesics, avoidance of physical activity, and social isolation.

Pain-associated brain changes in older adults: preliminary evidence and speculation regarding treatment implications

Multiple lines of evidence, discussed in this paper, indicate that normal and pathological ageing-associated changes in the brain (i.e. dementia, depression, and anxiety) impact pain processing and pain treatment and support that older adults with persistent pain are not simply a chronologically older version of younger pain patients. Evidence also indicates that pain itself impacts the brain and may have important treatment implications.

We have gathered preliminary data demonstrating that older adults with CLBP have brain morphology differences from pain-free individuals.[10] Specifically, we found decreased gray matter volume in the posterior parietal cortex and middle cingulate white matter volume of the left hemisphere. These changes are distinct from those observed in younger patients with CLBP.[3] We have also found that in community-dwelling older adult patients with heterogeneous persistent non-malignant pain disorders, pain severity was associated with diminished mental flexibility.[54] In older adults with CLBP, we have found decrements in multiple domains of NP when compared with pain-free age-matched controls, specifically immediate and delayed memory, language, mental flexibility, and manual dexterity.[114] As anticipated, significant inverse relationships between pain severity and NP and between pain severity and physical performance were demonstrated. Perhaps the most noteworthy finding was that NP mediated the relationship between pain severity and physical performance. That is, the significant relationship between pain severity and physical performance no longer existed after controlling for NP.

Traditional physical therapy approaches to persistent pain rehabilitation are often generically prescribed and focus on trying to ameliorate the direct effects of pain on the body (e.g. for CLBP, optimizing posture, body mechanics, and spinal flexibility). When applied to older adults, these approaches do not appear to provide functional gains above and beyond that of analgesia alone.[113] If, indeed, NP mediates the relationship between pain and disability, perhaps persistent pain-associated disability and the approach to its rehabilitation should be reconceptualized for older adults. For example, cognitive retraining (i.e. improving or restoring a person's skills in the areas of paying attention, remembering, organizing, reasoning and understanding, problem-solving, decision-making, and higher level cognitive abilities) should be incorporated as one component of efforts to rehabilitate older adults with persistent pain.

At a minimum, the potential deleterious impact of persistent pain on NP and, therefore, treatment compliance should be recognized. This may be especially important for older adults with comorbid minimal cognitive impairment (MCI) or dementia. For example, if memory is further impaired by pain in an older adult living with a condition whose cardinal feature is memory loss (e.g. dementia), they may forget: (i) to take their pain medications at the prescribed times if at all, (ii) to follow-up with their physician and prescribed non-pharmacological pain treatments such as physical therapy and exercise, or (iii) to consistently use an ambulatory assistive device such as a cane or walker to improve mobility and reduce the risk of falls and further injury. Patients living with MCI or dementia whose executive functioning is further impaired by the presence of pain may not be able to effectively problem solve solutions to minimizing pain and disability and maximizing function. For example, the mental flexibility required to (i) relate persistent pain with the need to change their analgesic schedule or (ii) incorporate more adaptive coping strategies such as behavioural activation, prayer, or distraction may not be available to these patients.

Meditation: an innovative age-specific management targeting enhanced descending inhibition

The important role of descending inhibition in pain processing and the limitations of pain treatments focused

purely on the body have stimulated an interest in complementary mind–body techniques for pain reduction in the older adult. We performed a pilot study investigating mindfulness meditation for the treatment of CLBP in adults 65 yr of age and older.[77] This form of meditation has it roots in Asian meditation traditions. Kabat-Zinn[50] conceptualized mindfulness meditation for a Western audience, divorcing it from its religious roots, but keeping the intent and format of the meditation intact. Thus, he pioneered the mindfulness-based stress reduction (MBSR) programme that has been successfully taught for more than 25 yr in the USA and worldwide. Because it has been operationalized, it has been studied in numerous clinical trials. Mindfulness meditation utilizes everyday activities such as sitting and walking and transforms them into a meditation through focused, non-judgemental attention to body sensation, thoughts, or emotion.

We wanted to study the MBSR programme in older adults, since little research had been done in this population, and because of its promising effects on depression and anxiety, both known to influence the experience of pain.[11 107] We found in our pilot study that mindfulness meditation significantly improved self-reported physical function and improved coping with pain as measured through greater ability to accept pain and engage in daily activities. Additionally, we found older adults enthusiastic and eager to learn the meditation.

Although the mechanism of mindfulness meditation has not been fully elucidated, there are several neuroimaging studies that offer clues. Of particular note is the finding of increased cortical thickness in the prefrontal cortex and right anterior insula among long-time meditators compared with controls. Although the prefrontal cortex and occipitotemporal region showed a typical decline with age in non-meditators, meditators aged 40–50 maintained their cortical thickness.[63] This is an exciting finding in light of the generalized neuronal loss and cortical thinning that occurs with ageing, and the potential that meditation may offer to slow the process. However, a possible link with improved NP and consequent pain reduction resulting from meditation remains to be determined. Another study found that mindfulness meditation caused asymmetric left-sided anterior cortical activation on EEG after healthy adults had participated in the MBSR programme. This indicates a shift to a more positive effect.[22] These studies offer preliminary evidence that meditation can have direct effects on the brain. Mindfulness meditation thus offers a complementary therapy for pain that is particularly useful in the older adult, with potential positive central effects and measurable effects on function and coping.

Conclusions and future directions

Effective pain treatment for older adults requires practitioners to acknowledge that older adults with persistent pain are not simply a chronologically older version of younger patients with persistent pain. Evaluation and treatment must consider the multiple factors that contribute to pain homeostenosis and, in turn, drive pain-associated disability. Individualized pain care for older adults should account for age-related and pathological cognitive impairments, the unique pharmacokinetic and pharmacodynamic milieu of the ageing body, caregiving burden and distress, and other issues pertinent to the care of older adults such as access to transportation, financial concerns, social isolation, and increased risk of falls and delirium. Clearly, additional research is needed that may require challenging traditional pain paradigms. Investigations should be designed that attempt to understand the interaction of ageing physiology and pain and ultimately optimizes rehabilitation and ameliorates the risk of disability for our vulnerable older patients.

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