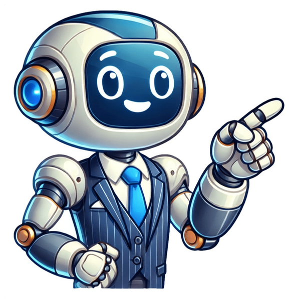

For a long time, it has been assumed that brain plasticity peaks at young age and then gradually decreases as one gets older. This is also underscored by the expression that one cannot teach an old dog new tricks, implying that people who have become used to doing things in a particular way will not easily abandon their habits and change their behavior. Interestingly, thanks to tremendous advances in medical imaging techniques for assessment of brain structure and function, mounting evidence for lifelong brain plasticity has been generated over the past years. Practice leads to improvement in and refinement of performance on motor - or any other -tasks and this dynamic behavioral process is associated with altered brain activity, occurring in a similar manner in young and older adults [1]. Besides functional brain changes, practice also induces structural changes, such as alterations in regional brain grey and white matter structures that are typically recruited during task performance [2]. In the context of practice-induced task learning, a key question is how brain plasticity can be optimized and this is an even more important consideration for older adults. Noninvasive brain stimulation techniques have been utilized to induce and optimize mechanisms of neuroplasticity during rest or during task practice via alteration of cortical excitability processes [3]. However, the effectiveness of such approaches has been questioned lately and we would argue that the gold standard to elicit brain plasticity is to practice new tasks intensively and to organize the training epochs in such a way that skill learning and retention are maximized. A critical requirement for neuroplasticity to emerge is to make the practice context sufficiently difficult for the learner. One way to challenge the environmental context is to confront learners with practicing more than one task within each practice session. More specifically, rather than performing subtasks in a sequential or blocked manner, one after the other (less challenging), one can also apply a more demanding random practice regime such that learners have to switch tasks from trial to trial during practice (more challenging). The latter condition has led to the apparent paradox that reduced performance levels are obtained during the training phase but better long-term retention and memory formation of the skill are observed at later stages as a result of more profound inter-task information processing strategies. This is generally known as 'contextual interference' (CI). Even though CI seemingly induces complication of the learning environment, it has been shown that older adults can equally cope with this increased contextual complexity as young adults do and that it benefits longer-term skill retention [4,5]. Using magnetic resonance spectroscopy (MRS), we explored the neurochemical basis of the CI effect via determination of the practice-induced modulation of gamma-aminobutyric acid (GABA), i.e. the chief inhibitory neurotransmitter that also plays a major role in brain plasticity [6]. Young and older participants were trained on 3 variations of a bimanual visuomotor tracking task over 3 days, according to either a blocked or random practice schedule. A retention test was carried out 6 days later. With MRS, we determined the GABA levels before and after training in primary sensorimotor cortex and occipital cortex, because our previous work showed considerable brain activity in both visual and motor processing regions, in agreement with the unique visuomotor signature of this tracking task [7]. We found that (1) the behavioral data confirmed the typical contextual interference effects, i.e., random as compared to blocked practice led to temporary inferior performance levels during the acquisition phase but superior skill retention in both age groups, (2) the MRS data demonstrated a training-induced decrease in occipital GABA level during random practice but an increased GABA level during blocked practice and this effect was even more pronounced in older adults [6]. These findings are critical for a better understanding of neuroplasticity in older adults. First, the data suggest that older adults can indeed cope with more complex random practice contexts (a form of contextual enrichment) that challenge their instantaneous performance but boost their learning potential and skill retention. Second, training-induced modulations in GABA appear to be a function of degree of contextual challenge and this effect is even amplified by aging. This modulatory capability is preserved in spite of the fact that initial GABA levels were lower in older as compared to young adults [6]. This is important because it is reflective of training-induced neuroplasticity in older adults. More specifically, a decrease in GABA levels is indicative of a release from inhibition to promote mechanisms of long-term potentiation, cortical plasticity and learning [6,8]. Whereas previous work reported this GABA modulation to occur in the motor cortex [Floyer-Lea A, et al. 2006] we observed it in the broader occipital territory, containing brain areas involved in processing of the stimulus structure and the movement-generated visual feedback, made available during practice (such as primary and secondary visual cortex and posterior precuneus), as exemplified by task-related functional magnetic resonance imaging [7]. Conversely, the less demanding blocked-practice schedule consisted of repeated presentation of stimuli, resulting in attenuated neuronal responses (repetition suppression) and enhanced GABA levels. These data provide additional confirmation for task-training induced lifelong plasticity. New motor and other skills can be acquired at any age even though the progress may be somewhat attenuated in older as compared to young populations. The importance of these results should not be underestimated. We live in a highly dynamic society in which dramatic technical developments are implemented at a rapid pace. This forces citizens to abandon their pre-existing/old habits and replace these by new ones, continuously challenging the adaptability and flexibility of their brains. In view of the demographic evolution of society, characterized by a steadily increasing proportion of older adults, the evidenced lifelong brain plasticity provides a critical foundation for a sustained role of older adults in society and for securing prolonged functional independence and quality of life. Society needs to provide the right context in which older adults remain challenged and encouraged to adapt to new contexts such that the negative consequences of age-related brain degeneration are reduced or even reversed and healthy brains are promoted. applied neurology neuro mentorship neuroscience Dec 16, 2024 But what if that belief is holding your clients—and even your practice—back? For years, it was widely believed that neuroplasticity—the brain's ability to adapt and rewire itself—declined sharply after adulthood. If you weren't fortunate enough to excel in your youth, conventional wisdom suggested you'd missed your opportunity to reach peak cognitive and physical function. However modern neuroscience has disproven these myths and proven that the brain retains its plasticity throughout life, even in the later years. This shift in understanding has an enormous upside for trainers, therapists, and health professionals working with aging clients. It provides a powerful toolkit for reversing cognitive decline, enhancing physical capabilities, and offering older adults a path to genuine transformation. Let's understand how neuroplasticity can be harnessed to support the aging population and why it should be central to your practice. The Science Behind Neuroplasticity and Aging In the early days of neuroplasticity research, scientists assumed that an older brain couldn't match the adaptability of a younger one. This perspective began to shift in the 2000s, with incremental studies suggesting that while neuroplasticity might decline with age, it didn't disappear. Fast forward to more recent findings (2017 onwards), and the landscape has changed dramatically: older brains can indeed adapt, but they require a specific approach. One pivotal study involved an experiment with octogenarians and 20-year-olds. Both groups underwent vision training exercises aimed at stimulating brain change. The surprising result? The 80-year-olds demonstrated the same rate of neuroplastic change as their younger counterparts—provided the stimulus was novel and engaging enough. Key Insight: Aging brains don't lose their plasticity; they simply need the right triggers to engage it. The challenge lies in moving beyond routine and introducing fresh, stimulating tasks. What does this mean for your practice? For aging clients, the focus should be on introducing new motor skills and complex, engaging tasks rather than routine physical activities. When you guide them through novel movement practices—like learning a new dance, playing a musical instrument, or engaging in context-rich drills—you ignite their brain's capacity to grow and adapt. Practical Ways to Boost Neuroplasticity in Aging Clients 1. Encourage Novel Movement Routine exercise isn't enough. To stimulate neuroplastic change, older clients need to engage in activities that are new and challenging. This might mean stepping out of the gym and trying something different: dancing, learning a musical instrument, or participating in a sport that requires coordination. 2. Contextual and Real-World Practice The brain responds best when training mimics real-life situations. For example, instead of static vision drills that don't align with daily activities, incorporate movement that reflects real-world needs, such as practicing visual tasks while walking. 3. Cognitive Load and Recovery Balance Learning and brain adaptation improve when tasks are slightly challenging and require focus. Introducing distractions or cognitive loads during exercises can increase long-term retention. For example, you can have clients practice coordination drills while engaging in simple conversation or auditory tasks. Equally important is recovery—neuroplasticity solidifies during rest periods, so ensuring a balance of work and recovery is crucial. 3 keys For Unlocking Neuroplasticity In Aging Clients If you have clients who feel stuck in their progress or experience chronic issues, applying these neuroplastic principles can be transformative. The three keys to unlocking neuroplasticity in aging clients include: The 4 Benefits of Neuroplasticity-Driven Training Engaging in neuroplasticity-focused practices provides older adults with: A Call to Action for Health Professionals The idea that neuroplasticity peaks and then fades with age is not just outdated—it's limiting. Your older clients have the potential for continued growth, recovery, and resilience. By integrating neuroplastic-focused training into your practice, you can help them defy expectations and achieve meaningful, lasting change. Are you ready to offer your clients something beyond the standard regimen? Explore the power of neuroplasticity and become a leader in progressive health practices. Would you like more insights on how to apply these methods or are you curious about specialized training approaches? Consider joining the Next Level Neuro Mentorship and gain the tools you need to lead your field. We hate SPAM. We will never sell your information, for any reason. A new study from the University of California, Davis has found that psychedelic drugs such as LSD and DMT promote neural plasticity and development, indicating a potential mechanism for their therapeutic benefits. Patients who suffer from depression and post-traumatic-stress-disorder tend to have impaired neurogenesis and neuroplasticity – their brain cells grow more slowly and are less adaptable. These structural changes can lead to atrophy of various brain regions, including the hippocampus (which is involved in learning and memory) and the prefrontal cortex (which mediates personality and decision-making). Counteracting this damage by promoting structural and functional neural plasticity has been suggested as novel way of treating psychiatric disorders. However, relatively few compounds that promote neuroplasticity – which the authors of the new study term 'psychoplastogens' – have been found capable of achieving this without drawbacks. Ketamine, a dissociative anaesthetic with hallucinogenic properties, is a notable exception. By activating pathways involved with forming neurone connections, it has been found to be an extremely effective therapeutic for treatment-resistant depression (figure 1b). Similarly, the Beckley/Imperial Psychedelic Research Programme have demonstrated significant and even longer-lasting benefits of psilocybin in the treatment of depression (figure 1a). Fig.1a – Psychedelics have a significant, long-lasting benefit in treating depression (adapted from Carhart-Harris et al. 2016). Fig.1b – Ketamine has similarly shown promise in treatment-resistant depression, though effects do not last as long as those observed with psilocybin (from Zarate et al. 2012). This new study hoped to elucidate the cellular mechanism by which psychedelics achieve their therapeutic effect by investigating whether and how they affect neural growth and plasticity. In the first experiment, scientists treated cultures of cortical neurones with psychedelics, and then observed how the neurones developed and increased in complexity (figure 2). Fig.2 – Psychedelics significantly increased complexity of cortical neurones compared to VEH – no drug. This was measured by analysing how often cell branches and offshoots (neurites) crossed over each other at various distances from the cell centre. The figure includes a representative tracing of a cell from each treatment (Ly et al. 2018) They found that LSD, DMT, and DOI – all serotonergic psychedelics – significantly increased the growth and complexity of neurones in a similar manner to ketamine, with LSD particularly potent. Interestingly, ibogaine was found to have no effect on neuroplasticity – but its metabolite noribogaine did, suggesting it was the active molecule in the anti-addictive properties of iboga. As a comparison, amphetamine and serotonin – which share structural similarities with psychedelics – were also tested, and were found to have no effect on measures of neurogenesis. These effects were observed not only in cell cultures, but also by testing the compounds on the brains of fly larvae and zebrafish, showing that they also have a tangible effect in living organisms. In a separate measure of neural plasticity, psychedelics were found to significantly increase the number of dendritic spines on cortical neurones, with LSD almost doubling their density (figure 3). These spines form synapses with other neurones and are a major site of molecular activity in the brain. Their functioning is closely related to higher cognition, and loss of these structures is a hallmark of depression and other neuropsychiatric disorders. Fig.3 – Effect of psychedelics on spinogenesis and synaptogenesis. The number of dendritic spines on cortical neurones – which act as gateways and connections to other neurones – was significantly increased following treatment with psychedelics (Ly et al. 2018) The positive effects were not only structural, but functional – electrophysiological recordings found that the frequency and strength of neural currents were increased for many hours after the psychedelic compounds had been removed. The study then further elucidated the molecular mechanisms involved. mTOR is a key component of internal neural signalling pathways, involved with recruiting receptors and forming synapses, and is also known to be the target through which ketamine achieves its antidepressant effect. When mTOR was blocked, the psychoplastogenic effects of psychedelics seen above were also inhibited, indicating that they achieve their effect through a similar mechanism to ketamine. This study builds on previous findings by the Beckley/Sant Pau Research Programme, which observed that components of the psychedelic brew ayahuasca promoted growth and maturation of neurones (figure 4). Ayahuasca has also been demonstrated to have significant anti-depressant effects, further suggesting neuroplasticity as a common mechanism for the actions of ketamine and psychedelics like LSD and ayahuasca. The Beckley Foundation is determined to investigate this further, both in vitro and in humans, as part of our Research Programmes with Brazil, Maastricht University and Imperial College London. Fig.4 – Components of the psychedelic brew ayahuasca promote growth and maturation of neurones, demonstrated by cell-cycle-specific staining of neurones in cell culture (from Morales-Garcia et al. 2017). These findings add yet more scientific evidence for the therapeutic benefit of psychedelic compounds. Not only have psychedelics been demonstrated to have ground-breaking potential in treating previously untreatable psychiatric disorders, but they are almost unique in their ability to promote neuroplasticity in a safe way, healing both mind and brain. Sadly, psilocybin, LSD, DMT, and other psychedelics remain on Schedule I of both the UK and the UN's global drug conventions, severely limiting both research and clinical application. The Beckley Foundation is actively campaigning to have psychedelics and other psychoactive medicines re-scheduled, so that doctors and psychotherapists may use them as a tool to help heal those who are not benefiting from currently available treatments. Despite restrictions, we continue to collaborate with research groups all over the world to better understand these compounds – together, we hope to create a paradigm shift in psychiatry which will benefit all of society. Nick Cherbanich Aging is associated with progressive losses in function across multiple systems, including sensation, cognition, memory, motor control, and affect. The traditional view has been that functional decline in aging is unavoidable because it is a direct consequence of brain machinery wearing down over time. In recent years, an alternative perspective has emerged, which elaborates on this traditional view of age-related functional decline. This new viewpoint--based upon decades of research in neuroscience, experimental psychology, and other related fields--argues that as people age, brain plasticity processes with negative consequences begin to dominate brain functioning. Four core factors--reduced schedules of brain activity, noisy processing, weakened neuromodulatory control, and negative learning--interact to create a self-reinforcing downward spiral of degraded brain function in older adults. This downward spiral might begin from reduced brain activity due to behavioral change, from a loss in brain function driven by aging brain machinery, or more likely from both. In aggregate, these interrelated factors promote plastic changes in the brain that result in age-related functional decline. This new viewpoint on the root causes of functional decline immediately suggests a remedial approach. Studies of adult brain plasticity have shown that substantial improvement in function and/or recovery from losses in sensation, cognition, memory, motor control, and affect should be possible, using appropriately designed behavioral training paradigms. Driving brain plasticity with positive outcomes requires engaging older adults in demanding sensory, cognitive, and motor activities on an intensive basis, in a behavioral context designed to re-engage and strengthen the neuromodulatory systems that control learning in adults, with the goal of increasing the fidelity, reliability, and power of cortical representations. Such a training program would serve a substantial unmet need in aging adults. Current treatments directed at age-related functional losses are limited in important ways. Pharmacological therapies can target only a limited number of the many changes believed to underlie functional decline. Behavioral approaches focus on teaching specific strategies to aid higher order cognitive functions, and do not usually aspire to fundamentally change brain function. A brain-plasticity-based training program would potentially be applicable to all aging adults with the promise of improving their operational capabilities. We have constructed such a brain-plasticity-based training program and conducted an initial randomized controlled pilot study to evaluate the feasibility of its use by older adults. A main objective of this initial study was to estimate the effect size on standardized neuropsychological measures of memory. We found that older adults could learn the training program quickly, and could use it entirely unsupervised for the majority of the time required. Pre- and posttesting documented a significant improvement in memory within the training group (effect size 0.41, p