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Review

Post-learning micro- and macro-structural neuroplasticity changes with time and sleep

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ABSTRACT

Neuroplasticity refers to the fact that our brain can partially modify both structure and function to adequately respond to novel environmental stimulations. Neuroplasticity mechanisms are not only operating during the acquisition of novel information (i.e., online) but also during the offline periods that take place after the end of the actual learning episode. Structural brain changes as a consequence of learning have been consistently demonstrated on the long term using non-invasive neuroimaging methods, but short-term changes remained more elusive. Fortunately, the swift development of advanced MR methods over the last decade now allows tracking fine-grained cerebral changes on short timescales beyond gross volumetric modifications stretching over several days or weeks. Besides a mere effect of time, post-learning sleep mechanisms have been shown to play an important role in memory consolidation and promote long-lasting changes in neural networks. Sleep was shown to contribute to structural modifications over weeks of prolonged training, but studies evidencing more rapid post-training sleep structural effects linked to memory consolidation are still scarce in human. On the other hand, animal studies convincingly show how sleep might modulate synaptic microstructure. We aim here at reviewing the literature establishing a link between different types of training/learning and the resulting structural changes, with an emphasis on the role of post-training sleep and time in tuning these modifications. Open questions are raised such as the role of post-learning sleep in macrostructural changes, the links between different MR structural measurement-related modifications and the underlying microstructural brain processes, and bidirectional influences between structural and functional brain changes.

1. Introduction

Neuroplasticity refers to the fact that our brain can partially modify both structure and function to adequately respond to novel environmental stimulations [1,2]. Neuroplasticity mechanisms are not only taking place during the acquisition of novel information (i.e., online) but also during the offline periods taking place after the end of the actual learning episode. In the short term, synaptic plasticity involves cellular determinants of synaptic strength and persistence that are triggered within individual neurons in the minutes and hours after memory encoding [3]. Later on, memory consolidation gradually develops at the systems level [4] over extended periods (days to weeks) of time and sleep to shape and integrate novel memories into pre-existing brain networks [5,6]. In humans, many studies conducted over the past decades evidenced functional brain changes in response to learning (e.g., [4,7–10]). In comparison, evidence for structural brain changes remains narrow, a paucity partly due to the fact that non-invasive brain imaging techniques such as structural magnetic resonance imaging (MRI) have for long exhibited restricted sensitivity to subtle, short-term changes in neural architectonics, hence preventing the detection of rapid structural brain remodelling. We aim here at reviewing the available evidence establishing a link between different types of training/learning and the ensuing structural changes, with an emphasis on the role of post-training sleep and time in fine-tuning these modifications. Open questions will be raised such as the role of post-learning sleep in macrostructural changes, the link between different MR structural measurement-related modifications, the underlying microstructural brain processes, and bidirectional influences between structural

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and functional brain changes.

1.1. Learning-related macrostructural changes

Longitudinal MRI studies conducted in human participants have disclosed learning-related changes in cortical thickness and grey matter (GM) volume (using surface-based [SBM] and voxel-based [VBM] morphometry analyses, respectively) that take place within a timescale ranging weeks to months following training. In the motor skill domain, 3 months of juggling practice was initially shown to result in a bilateral GM expansion in the occipito-temporal junction (hMT/V5) as well as in the left posterior intraparietal sulcus in young adults [11,12] (Fig. 1), or in the left hippocampus and nucleus accumbens in elderly participants [13].

Noticeably, follow-up analyses evidenced structural changes already after 7 days practice in hMT/V5 as well as in the frontal and temporal lobes and in the cingulate cortex, bilaterally [14]. In the language domain, learning a second language was associated with increased cortical thickness in the left anterior cingulate cortex (ACC) [15], right [15] and left middle temporal (MTG), left superior temporal (STG) and left inferior frontal (IFG) [16] gyri. VBM also evidenced increased hippocampal volume [16] and GM density in the left IFG and anterior temporal lobe (ATL) [17]. Extending these findings onto other domains, GM modifications have been reported following cognitive [18,19], action observation [20], video game [21,22], golf [23], balance [24] and musical [25] training, as well as conceptual knowledge [12,26,27] and Morse code [28] learning. Similarly, white matter (WM) changes are observed using standard diffusion weighted imaging (DWI) measurements (e.g., using diffusion tensor imaging [DTI] analyses computing mean diffusivity [MD] and increased fractional anisotropy [FA] values that estimate tissue density and fibre organization/directionality, respectively) after various forms of exercise, including procedural [24,29,30], working memory [31,32] and intensive reading [33,34] training, and second language acquisition [35]. WM changes were often found to parallel GM alterations when both measurements were available [24,26,36,37]. Also, both correlated with behavioural improvement, indicating that both GM and WM are capable of structural plasticity and remodelling following the acquisition of novel information [11,15–17,20,24,25,33].

1.2. Cellular mechanisms underlying macrostructural changes

As shown above, neuroimaging techniques and especially MRI/DWI proved useful to evidence practice-related macrostructural brain remodelling in human, as they are non-invasive and can be repeatedly used to monitor changes in the cerebral architecture within a same individual. Notwithstanding, standard MRI/DWI measurements are inherently limited even if sensitive. Indeed, their outcomes lack specificity and does not provide information about the underlying cellular processes [38,39], leaving unclear the link between neuroimaging measurements and their underlying biology. The candidate mechanisms underlying GM, WM and extra-neuronal modifications in response to learning have been discussed (see e.g. [39] for a detailed review). Regarding GM changes, neurogenesis was amongst the first suggested underlying mechanisms, since adult-born neurons were found to develop [40–43] into fully functional neurons [44] in rodents in response to learning. However, this phenomenon is from small nature [40] and mostly evidenced in hippocampal area [41–44]; neocortical neurogenesis remains controversial to this day [45–47]. By contrast, rodent studies show that learning usually elicits synaptogenesis and

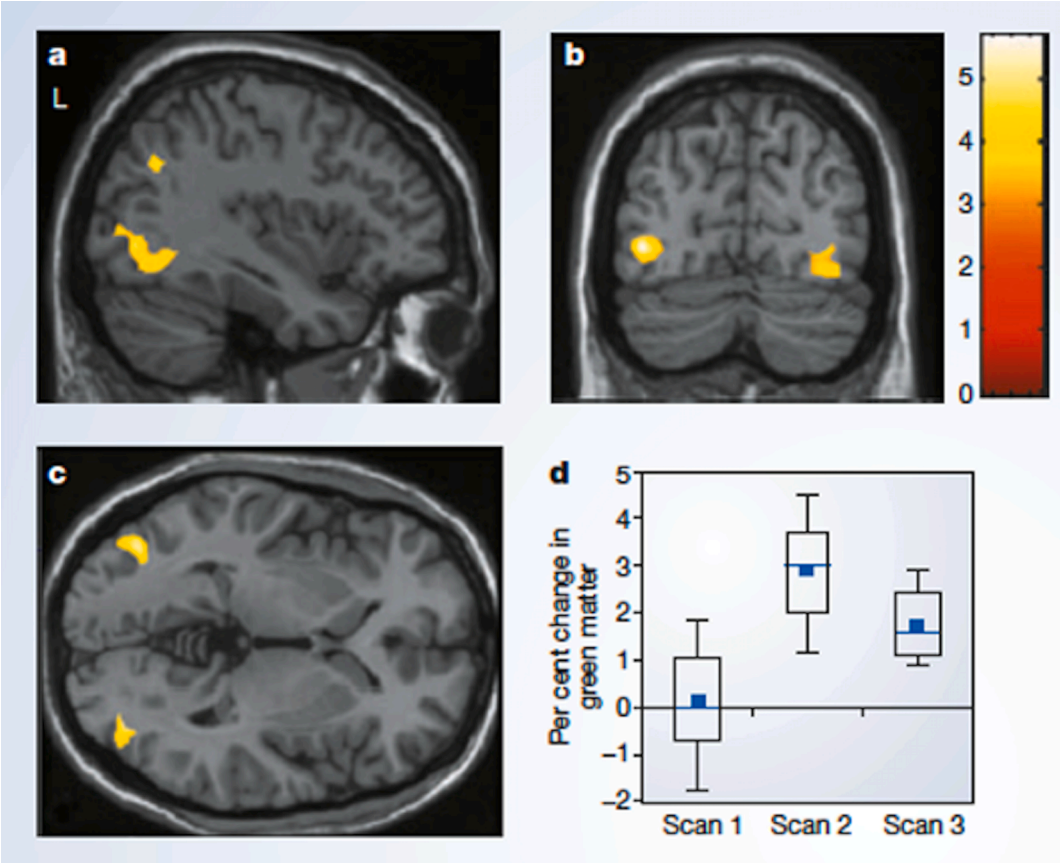

Fig. 1. Learning-induced brain structural changes. Statistical parametric maps evidencing a expansion in GM in the left posterior intraparietal sulcus (a) and bilaterally in the mid-temporal area (hMT/V5; b-c) following a 3-months juggling training in young adults [11]. These transient changes tended to decrease within the 3 next months as reflected by the boxplots showing the range, mean and standard deviation of GM change (d). Reprinted from [11], Fig. 1 page 311.

neuronal morphology changes on a larger scale, including neocortical areas such as the motor cortex [48–52] (Fig. 2).

Additionally, sprouting of new mossy fibre terminals was observed following spatial learning [53] or physical exercise [54] in the hippocampus. Gliogenesis also emerged as a promising underlying mechanism for modified MRI signals. Indeed, glial cells have a preserved capacity to divide in the adult brain [39,55], and post-learning neocortical gliogenesis was reported in several animal studies [47,56]. This hypothesis is further supported by the fact glial cells largely outnumber neurons in the GM, with an approximate 6:1 ratio depending on the brain region [39]. Lastly, vascular changes might also be responsible for GM modifications; blood vessels constitute about 5% of the GM volume [39], that was itself found to increase following physical exercise [56–59].

Besides GM and its underlying cellular components, WM also plays a key role in learning by optimizing information transmission through myelin remodelling [60,61]. There is growing evidence for an activity-dependent regulation of myelin playing a contributing role in learning processes [55,60–65]. For instance in rodents, 7 days of optogenetic stimulation in the premotor circuit resulted in increased myelin thickness 4 weeks later, associated with improved motor function in the contralateral limb [66]. Also in humans, an increased FA that might be attributable to increased myelination was observed in the white-matter tracts connecting task-related areas in the contralateral hemisphere after 4 weeks of unimanual motor training [30]. Learning-related changes may also be found in fibre bundles (axon number, diameter, density, organization, orientation, etc.) following the acquisition of new skills. In adult monkeys, tool-use training for 2 weeks eventually led to the development of novel neural connections. Albeit sparse connexions were found in the cortex of untrained animals, projections were far more dense and large-scaled in trained monkeys [67], supporting the notion that a pre-existing neural route was fine-tuned and reinforced according to task demands [68]. Furthermore, inactive axons are quickly eliminated in the context of competition with active axons, highlighting the role of neural activity in the formation of functional memory circuits delineated by the activity-dependent competition between axons after their development [69]. Finally, it must be stressed here that MRI/DWI signal changes likely do not result from a single cellular process, but rather from a combination between different processes mentioned above and their complex interaction to adequately cope with learning demands [39,56].

1.3. Sleep and memory consolidation: structural changes

Besides a mere effect of time, post-learning sleep mechanisms play an important role in memory consolidation by promoting long-lasting changes in neural networks subtending improved or stabilized performance. Beneficial effects of post-learning sleep on declarative and procedural memory have been repeatedly reported (for reviews, see [70–73]). Functional neuroimaging studies in human provided evidence that neural activity during sleep can be modulated by prior experience/learning and contribute to the consolidation of recently acquired memory traces, both during REM sleep (e.g. [74–80]) and SWS (e.g. [81–88]). As well in primates, cross-frequency coupling in the hippocampus during both REM and NREM sleep [89] is compatible with synaptic and systems memory consolidation processes, respectively. In rodents, invasive procedures evidenced neuronal correlates of memory reactivation in various contexts [90–93]. For instance, motor learning-related development of dendritic spines was observed during subsequent NREM sleep [47], and replay of task-related ensembles was linked to the coincidence of slow-wave events and bursts of spindle activity [94]. In the context of the focus of this review on experience-dependent structural modifications, it is noticeable that robust relationships have been evidenced between features of sleep oscillations reflecting brain plasticity processes (e.g., sleep spindles [95–97], slow-wave activity [98] or the slope of slow oscillations [99] during NREM sleep), and brain structural measures such as inter-individual differences in GM volume [95,96,98,100], GM cortical thickness [101] or WM diffusion [96,98]. These results indicate that besides reflecting the dynamics of neuronal networks, sleep oscillations may to some extent reflect, and be constrained by, the microstructural properties of their underlying localized brain sources. Indeed, specific white matter tracts located in the frontal temporal and subcortical regions exhibited higher axial diffusivity in individuals with a steeper rising slope of slow wave oscillations or increased sleep spindle power [95]. Additionally, data linking sleep spindles with learning-related projections and offline memory gains suggest that the way microstructural white matter fascicles characteristics influence memory consolidation is mediated through sleep spindles density [97]. Lastly, hippocampal GMV was found correlated with

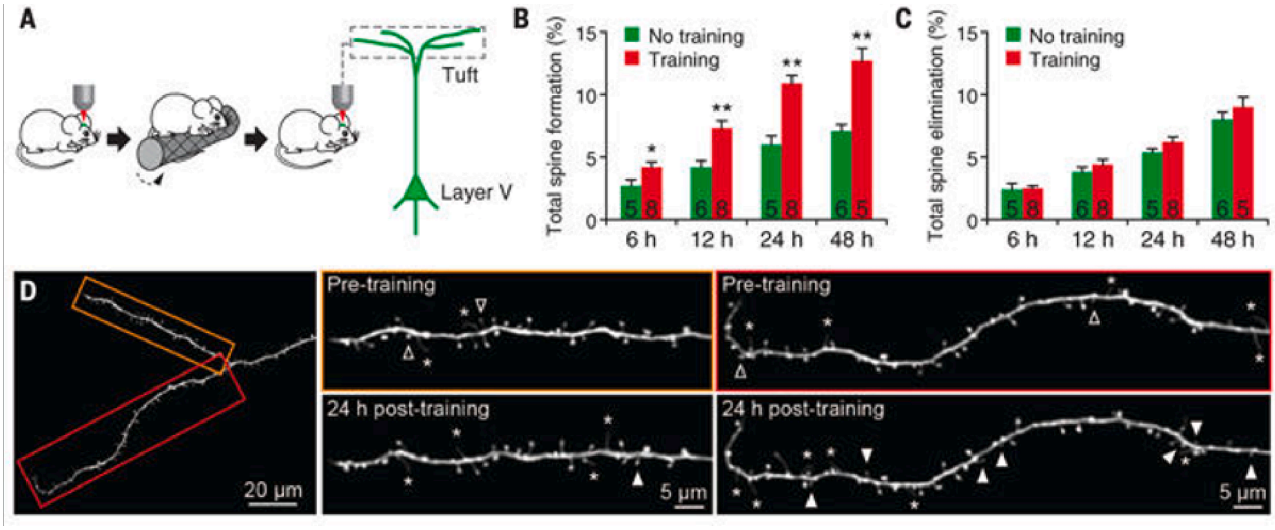

Fig. 2. Spine formation following motor learning. In this study [52], mice performed one session of rotarod motor training (20 trials) and underwent twice (before and after) in-vivo transcranial two-photon imaging (A). While the percentage of dendritic spine formation was significantly higher in the primary motor cortex of the trained group compared to the control group (B), the elimination rate did not differ between both groups over the 48 h following training (C). The sample size is mentioned within the columns (B & C). The last images illustrate the evolution of dendritic spines 24 h post-training. Filled arrowheads point towards newly formed dendritic spines while open ones indicate eliminated spines compared to the pre-training image. Asterisks indicate dendritic filopodia (D). Reprinted from [52], Fig. 1 page 1173.

differential spindle expression; whereas hypothalamic and medial prefrontal cortical GMV were associated with slow wave oscillations [100].

Notwithstanding reliable findings evidencing functional changes in brain response and behaviour after offline periods including and in relation to sleep (for reviews, see e.g. [70,71]), and in contrast with the widespread literature documenting GM/WM plasticity changes accompanying extended [11,13–17,19,23–28] or even restricted [102] online practice, concrete substantiation for sleep-dependent offline structural modifications in learning-related human brain networks remains scarce. To the best of our knowledge, one study investigated this topic using a sleep deprivation (SD) paradigm [103]. In this study, diffusion weighted imaging (DWI) structural scans were recorded in participants at baseline in the evening then every 12 h first after sleep then after extended wakefulness for 24 h spent awake, and finally after recovery sleep (i.e., 5 DWI measurements in total). The procedure was repeated two times (spaced two weeks apart) within individuals using a different cognitive task. During each of the 24-hours wakefulness periods, they completed 6 training sessions (2 h each) either playing a video driving stimulation (DS) game or being administered a set of executive functions (EF) tests. Based on a prior study, the authors defined regions of interest in visuomotor (DS task) and prefrontal cortex [104]. Results globally disclosed decreased mean diffusivity (MD) in the cortical GM after 12 h of training, magnified after an additional 12 h spent awake with practice, these effects being reverted after recovery sleep. Additional analyses in a distributed cortico-subcortical network disclosed time by condition interactions on MD in the superior temporal cortex and in the pars triangularis of the inferior frontal gyrus. In parallel, non-specific (i.e., independently of the task performed) volumetric effects were found after prolonged practice with decreased ventricular volume and increased GM and WM subcortical volumes, these changes being reverted after recovery sleep. At variance, cortical thickness did not change over the course of extended wakefulness but increased after recovery sleep. Finally, structural alterations partially predicted cognitive performance, although recovery sleep parameters were not associated with structural changes. These results were interpreted as support for the synaptic homeostasis hypothesis [105] that proposes that locally increased synaptic strength over learning at wake is downscaled to baseline level by slow NREM oscillations during subsequently sleep, restoring neural space for future learning while consolidating the recently acquired memory trace. The fact that no within-individual association was found between recovery sleep parameters and macrostructural changes, whereas inter-individual correlations were evidenced between sleep and structural parameters as discussed above [95–98,100,101] may suggest either a lack of sensitivity of DWI and volumetric measurements, or that these associations reflect hardwired individual traits more than modulated brain states.

1.4. Sleep deprivation-related macrostructural modifications

Besides a possible sleep deprivation (SD) effects on learning-related neural circuitries discussed hereabove, brain structural parameters can be modulated by SD on its own. For instance, one study identified increased GM volume in the left striatum and parts of the cingulate cortex after acute SD, paralleled by decreased GM volume in other areas such as the thalamus, cerebellum, insula and parietal cortex, and effect reinforced and spread to other brain regions with the extension of SD duration [106]. Likewise, decreased cortical thickness was observed in the parietal cortex after 23 h of SD [107]. Lastly, GM density increased in the right frontal pole, the right superior and the right middle frontal gyrus after 24 h of SD whereas the GM density modifications in the right middle frontal gyrus correlated positively with the sleepiness scores (measured by the Karolinska Sleepiness Scale [KSS]) [108]. One (amongst others) potential modulatory factor for SD-related volumetric changes in GM might be vascular, since regional cerebral blood flow modifications have been also observed after SD [109–111] and changes in vascular supply are partially responsible for modifications in GM volume [39]. DTI analyses also evidenced SD-related widespread decreases in FA, mainly in frontotemporal and parieto-occipital WM, corpus callosum, thalamus, and brainstem [112]. Notwithstanding, changes in WM [112] and GM [107] parameters were also observed over daytime in a 24-hours period, which emphasizes the importance to delineate time- and circadian-driven alterations [113–115] from specific SD-related changes.

1.5. Sleep and synaptic remodelling

Although evidence for a role of sleep in structural neuroplasticity remains scarce in human studies and is still awaiting robust substantiation, animal studies for their part provided outputs to explain how sleep might modulate synaptic structures, by affecting not only spine numbers and morphology [116] but also astrocytes [117,118] and myelin thickness [119].

In this context, dendritic spines have been scrutinized by the scientific community over the last decade and experimental data suggest that sleep may impact synaptic plasticity in different ways [116]. Spines are specialized postsynaptic membranous compartments, composed of a base emerging from the dendritic membrane and a neck overhung by a head containing signalling molecules and receptors crucial for synaptic transmission [116]. Based on ultrastructural indices such as (amongst others) shape, length and head volume, dendritic spines are classified into 4 categories: mushroom spines, stubby spines, thin spines, and filopodia. They can rapidly adapt to the environmental demands and therefore adjust their shape, eventually shifting over time from one category to the other [116].

According to the synaptic homeostasis hypothesis [105] mentioned earlier, one of the core functions of sleep would be to downscale the increased synaptic strength that took place during a precedent time episode spent awake. Accordingly, the axon-spine interface (ASI) considered as an ultrastructural marker of synaptic strength was shown to decrease by 33.9% on average in layer 2 of the primary motor cortex of 2-week-old mouse pups after sleep as compared to extended enforced wakefulness [120]. In this study, all synapses were affected independently of their size but in counterpart, sleep did not affect spine density. Likewise, ASI decreased by ~8% on average after sleep as compared to wakefulness or extended wakefulness [121]. Spine head volume also decreased by the same amount in these adolescent mice, with a trend to selectively spare larger synapses located on crowded dendritic branches or lacking endosomes. Another study showed increased ASI in perforated synapses in the CA1 region of the hippocampus after a waking episode and even more after prolonged wakefulness as compared to sleep. At variance in non-perforated synapses, ASI size significantly differed between sleep and extended wakefulness only [122]. Timescale also appears crucial in this process. Indeed, no differences were found when assessing synaptogenesis or pruning in the cortex after 2–3 h only of sleep or wakefulness [123], suggesting that a longer episode (about 7–8 h) is required to create or prune dendritic spines. Additionally, spine number in adult mice was not found modulated by sleep, a similar spine turnover (i.e., ratio between lost and gained spines) being found after sleep and wakefulness, which suggests that other mechanisms contribute to the modulation of synaptic strength in the cortex of young adults [123].

The synaptic homeostasis hypothesis [105] builds up on mechanisms taking place during NREM sleep and especially slow oscillations, but REM sleep also appear to be a privileged state for structural plasticity. In this respect, a complementary role for REM and NREM sleep was advocated [124] in the framework of the Synaptic Tagging and Capture hypothesis (STC) [3,125] that learning creates a possibility for long-term synaptic changes by setting ‘tags’ at remodelling synapses. The STC proposes that delayed reactivation within a few hours of the neuronal network surrounding tagged (in other words, “primed”) synapses will trigger the capture and translation of Plasticity Related Products (PRPs) eventually leading to the stabilization of changed

synaptic weights [3]. These tags can be positive or negative; therefore, they can prime synapses for further spinogenesis (strengthening) or pruning (weakening). In a nutshell, it was proposed that the reactivation of task-specific circuits during NREM oscillations following priming during wakefulness would allow the capture of PRPs, while REM sleep would support the translation mechanisms of PRPs transcripts eventually stabilizing synapse-specific structural plasticity [124]. Accordingly, after motor learning in adolescent mice, pruning of excessive dendritic spines in the primary motor cortex mostly takes place during REM sleep [126]. At the same time, the size of a fraction of spines recently formed in the learning-related network also selectively increased during REM sleep, and was predictive of the long-term survival 4 days later of these new spines [126]. Also, an experience-dependent elimination of dendritic spines in the mouse visual and frontal association cortices was recently evidenced during REM sleep [127]. Altogether, these findings further support the hypothesis that REM sleep is involved in experience-dependent synapse selection and memory consolidation.

Besides, experimental data suggest that sleep not only modulates dendritic spines formed during a previous wake or learning episode but that it might also promote spine formation [128]. Tracking formation and elimination of dendritic protrusions across sleep and wake in the primary somatosensory cortex of adolescent mice after motor learning, Yang et al. [128] found that elimination rate was higher during sleep as compared to wakefulness, but that the formation rate was similar between states, eventually leading both to a seemingly paradoxical net spine increase during wakefulness and a net decrease after sleep (Fig. 3).

This further suggests that sleep is involved in active remodelling following training, above a mere downscaling process. In a follow-up study investigating post-motor learning synaptic plasticity [52], dendritic spine formation was triggered within the 6 h after training and continued to expand over the next 24 h. Noticeably, post-training SD was associated with reduced spine formation, but this reduction could not be compensated by either additional training or subsequent sleep (Fig. 2). Likewise, the survival rate of newly formed spines was significantly higher when mice were allowed to sleep following training, and motor performance after 1 or 5 days was higher in sleeping than SD mice suggesting a role for sleep in spine formation and survival as well as the retention of motor skills. Finally, REM sleep deprivation did not impact learning-induced spine formation [52] (although it was found involved in the experience-dependent elimination of synapses, but not in the context of motor learning [127]). A net decrease in spine density after SD was similarly observed in the CA1 region of the hippocampus [6,129] and the dentate gyrus [130]. However, the effects of SD on spine density in the hippocampus were alleviated after 3 h of recovery sleep [6], again in support of the hypothesis that sleep promotes rapid spine formation above mere downscaling [116].

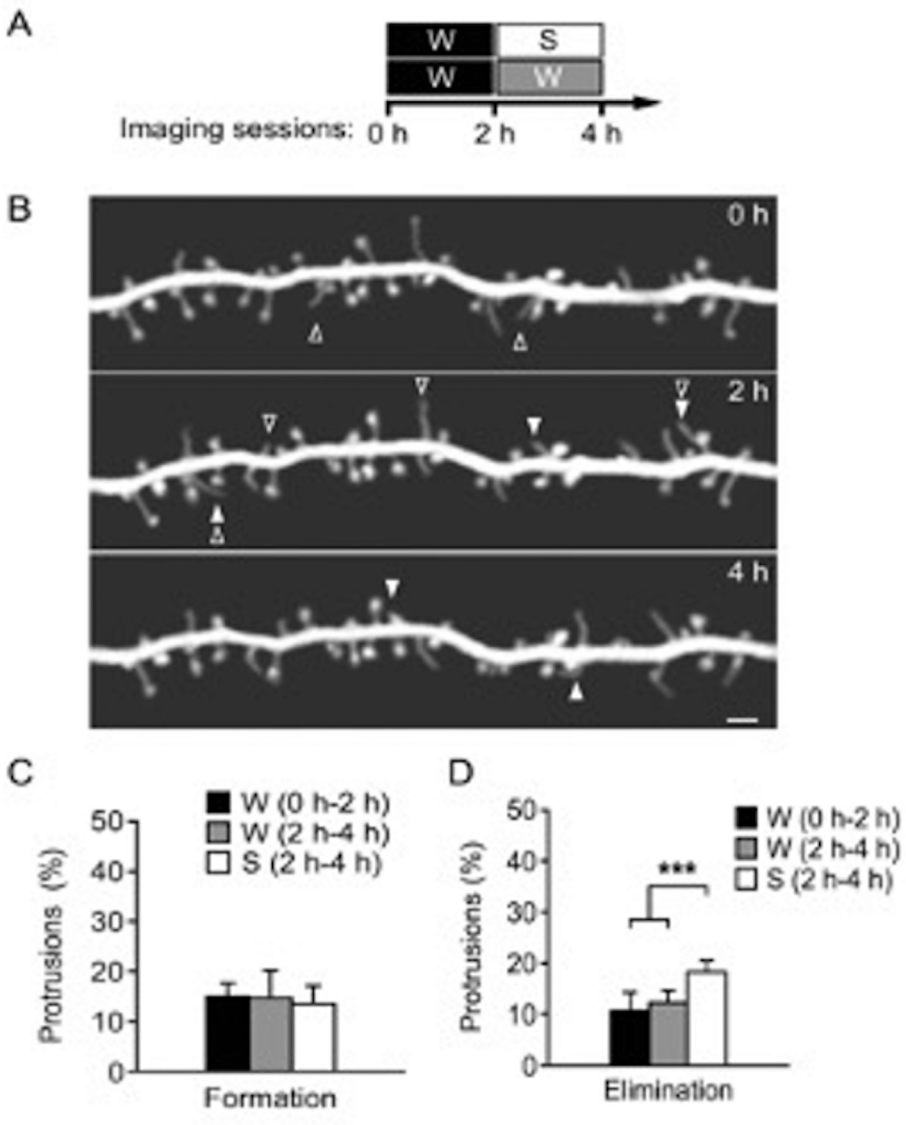

Fig. 3. Protrusion formation and elimination during both wakefulness and sleep. In this study [128], half of the mice were kept awake for 2 consecutive periods of 2 h while the other half was allowed to sleep during the second 2 h-period (A). The in-vivo imaging sessions took place at 0, 2 and 4 h. An example of the dendritic evolution of a segment every 2 h in the primary somatosensory cortex of a mouse that was awake during the first 2 h (from 0 to 2) and asleep during the 2 next hours (from 2 to 4). Filled arrowheads indicate newly formed dendritic protrusions while open arrowheads indicate dendritic protrusions that were eliminated (B; scale bar, 2 μm). While protrusion formation did not differ between sleep and wake, protrusion elimination on the other hand was significantly higher during sleep compared to wake (C & D). Reprinted from [128], Figs. 2 and 3 page 1391.

Another possibility for sleep to impact the brain's microstructure is the modulation of astrocytes and myelin coating. Time elapsed and sleep quality/quantity appear to be modulating factors to consider in this case. Indeed, chronic sleep restriction over 4.5 days (vs. regular sleep) was found to result in the thinning of myelin sheaths, whereas acute SD (i.e., 6–8 h) did not significantly differ both from chronic sleep restriction and regular sleep [119]. Furthermore, myelin thinning equally affected all axons independently of their size and 32 h of recovery sleep was not sufficient to restore the initial myelin envelop [119], which suggests that myelin plasticity is a gradual process that needs several days to occur. Astrocytes' proximity to the synaptic cleft in response to neuron's demands also seem to be modulated by wake and sleep episodes [117], although on shorter temporal dynamic. Indeed, astrocytes were found to move closer to the clefts after a short wakefulness episode (6 to 8 h), the effect being magnified with prolonged sleep restriction as the astrocytic coverage of the synapses increased [117]. This can be explained by the fact that active synapses require more housekeeping functions to operate efficiently. Accordingly, medium to large synapses are more subjected to astrocytic coverage. Further supporting a role for sleep in the modulation of astrocytes' movements, they seem to withdraw from the synaptic cleft during sleep [117].

1.6. Learning and sleep at the molecular level

Besides MRI techniques, the advancement and refining of other techniques over the years might also help to gain insight in the processes unfolding during learning. At the molecular level, transcriptomic methods allow the quantification of single gene's expression by accessing RNA levels, making them a prominent, reliable and unbiased tool for to identification of the cells and molecules participating in memory processes [131]. Two main approaches coexist to assess gene expression. The *in-situ* hybridization (ISH)-based requires identifying *a priori* RNA transcripts of interest to be able to hybridize them with a target-specific probe. The method has the advantage of hybridizing RNA in native tissue, hereby allowing for spatial registration. The second technique, the sequencing-based approach, requires cells to be dissociated from the native tissue to be efficiently sequenced, making the elaboration of spatial maps impossible unless working on single cells. However, it allows identifying at the whole-genome scale all RNA transcripts, hence allowing to measure gene expression levels in an unbiased and accurate way. The combination of both methods makes it possible to sequence the entire transcriptome, without losing the organizational pattern [131]. Using these methods, genes coding for particular rRNAs [132] and specific proteins regulating synaptic plasticity through the modulation of certain mRNA [133,134] could already be identified, and microRNAs were also found to intervene in synaptic plasticity and learning (for reviews, see [135,136]. Additionally, different hippocampal mRNAs and miRNAs crucial to spatial memory consolidation were found impacted by post-learning REM sleep deprivation [137] as well as several hippocampal and cortical genes [138,139]. Although these modifications offer clues concerning synaptic plasticity in response to learning and its modulation by sleep, much remains to be discovered to unravel the precise relationships between the molecular, microstructural and macrostructural levels and learning, memory and sleep.

2. Concluding remarks

With this contribution, we first attempted to review the extended literature demonstrating a robust link between various forms of experience (training/learning) and resultant structural changes at the brain level. We then summarized the microstructural brain processes that might subtend changes recorded in human using MRI/DWI and explored the potential links between post-training sleep and time and related macro structural and microstructural modifications, both in man and animal. Finally, we also discussed potential sleep deprivation related biases inherently linked to classically used experimental protocols. That experience-dependent structural and functional changes take place in the human brain and that sleep contributes memory consolidation are now well supported hypotheses, but how and whether sleep triggers observable brain structural changes in learning/practice networks, and what are the underlying microstructural processes, require further detailed investigation.

In human, one supportive DWI study evidenced increasing cortical MD after extensive training/wakefulness, that reverted after recovery sleep [103]. This observation paralleled microstructural data showing decreased ASI and spine head volume after sleep vs. wake in mice [121], in line with the proposed role of sleep (and in particular, NREM sleep) in homeostatic synaptic downscaling [105]. Still, other reports indicate that post-training sleep contributes to an active remodelling of the synaptic structures, more than a mere downscaling of synaptic weights. There is animal evidence both for spine formation during sleep [52,126,128–130] and decreased spine density after sleep [126–128]. More animal research is needed to track the evolution of spines (size, number, density, etc.) on dendritic branches in relation to sleep and especially of the different sleep states in mammals to explain their specific role in dendritic formation and pruning. At present, REM sleep seems to be a sleep state in which most of the pruning of excessive dendritic spines happens together with a selective increase in the size of some spines predictive of long-term survival [126]. It was proposed that NREM sleep might be the state in which spines are formed during the neuronal replay of prior learning experience [52,124], then stabilized through translation mechanisms during REM sleep [124]. Evidence for neuronal replay and synaptic alterations should be simultaneously obtained over time in the same neurons to ascertain this hypothesis [52]. Still, taken together, one might hypothesize that NREM is indeed the main state for spine formation while neuronal replay stimulates the previously activated networks, while the following REM episode prunes and stabilizes experience-relevant spines [124].

There are also methodological caveats to address before being able correctly interpreting the macrostructural changes in MRI signal measured in human studies, and how they link with microstructural modifications. As discussed above, a major limitation of e.g., cortical thickness, GM volume/density, MD and FA is that they provide little information about the underlying cellular processes. Indeed, different cellular mechanisms may give rise to seemingly similar changes in the MRI signal. There is hope in this respect as the last decade witnessed the speedy development of advanced MR methods aimed at more precisely linking brain macro- and micro-structural [140] modifications, and track fine-grained changes in the brain at shorter timescales up to hours or even tens of minutes [102,141–145]. Improved specificity is now possible using novel conceptual frameworks that better take into account the complexity of intra- and extra-cellular processes giving rise to changes in MRI information. For instance, CHARMED (Composite Hindered And Restricted Model of Diffusion; [146], AxCaliber [147], and ActiveAx [148] frameworks can be used to provide a more precise assessment of the axonal diameter [140] and conduction velocity [149] in WM. Another limitation in the analysis of DWI data is that most models loose specificity to characterize WM tracks in areas of complex fibre configuration. In this respect, the NODDI (Neurite Orientation Dispersion and Density Imaging; [38]) appears to more accurately model the dispersion/fanning of axonal fibres and dendrites in areas of crossing fibres, and disentangle the microstructural effects underlying FA (e.g. myelination, number of fibres, … [150]). As well, NODDI was found efficient to delineate the internal anatomy of the mouse hippocampus [151] and better characterize GM microstructural modifications in early Alzheimer's disease by evidencing specific alterations in orientation dispersion and neurite density separately [152]. Still, NODDI failed capturing selective dendritic alterations at the early stages of experimental multiple sclerosis in the mouse hippocampal subfields, whereas DTI parameters did so, suggesting reduced sensitivity in some configurations [151]. How and whether these advanced methods to

analyse MRI signal will contribute to better understand experience-dependent sleep- and time-related modifications in the brain structure remains to be investigated in upcoming studies.

Finally, little is known about the reciprocal interactions between functional connectivity and cerebral anatomical structure, and the direction of these relationships and causalities, in other words the chicken or the egg causality dilemma. Nowadays, there is accumulating evidence for an overlap between functional and structural brain networks (e.g. in GM [15,153] and WM [153–159]). It was also shown that momentary adaptations in functional connectivity alter structural connections, which in turn affect functional connectivity [160]. Indeed, correlations were evidenced between post-learning alterations in GM and functional connectivity between learning-related cortical areas [15,48,65,161,162], as well as between WM modifications and changes in functional networks [161]. Hence, there is supporting evidence that experience-based neuroplasticity usually meets structure-function correspondence, even if directionality and causality remain uncertain.

CRediT authorship contribution statement

Whitney Stee: Conceptualization, Investigation, Writing - original draft, Writing - review & editing, Visualization. **Philippe Peigneux:** Conceptualization, Writing - original draft, Writing - review & editing, Supervision.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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