Posterior parietal cortex contributions to cross-modal brain plasticity upon sensory loss

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Highlights

- The inferior posterior parietal cortex has a role in cross-modal plasticity
- Age, social circumstances, attention and stress affect the posterior parietal cortex and cross-modal plasticity
- Rehabilitation strategies for multimodal recovery or via functional implants should include the posterior parietal cortex

Running title: **Posterior parietal cortex and cross-modal plasticity**

Abstract

Sensory loss causes compensatory behavior, like echo localization upon vision loss or improved visual motion detection upon deafness. This is enabled by recruitment of the deprived cortical area by the intact senses. Such cross-modal plasticity can however hamper rehabilitation via sensory substitution devices. To steer rehabilitation towards the desired outcome for the patient, having control over the cross-modal takeover is essential. Evidence accumulates to support a role for the posterior parietal cortex (PPC) in multimodal plasticity. This area shows increased activity after sensory loss, keeping similar functions but driven by other senses. Patient specific factors like stress, social situation, age and attention, have a significant influence on the PPC and on cross-modal plasticity. We propose that understanding the response of the PPC to sensory loss and context is extremely important for determining the best possible implant-based therapies, and that mouse research holds potential to help unraveling the underlying anatomical, cellular and neuromodulatory mechanisms.

44 **Introduction**

45 Sensory deficits related to hearing loss, vision loss or limb amputation, impact millions of people
46 worldwide [1,2]. Whether congenital or acquired, the brain adapts to this disabling condition via
47 different plasticity processes. In cases of partial sensory loss, the brain reorganizes to boost
48 residual inputs to the deprived sense [3–5]. Ocular dominance plasticity in the visual cortex in
49 relation to monocular vision loss is a seminal example of such adaptation. Loss of all inputs, as
50 with blindness, leads to compensatory cross-modal plasticity: the intact senses take-over the
51 cortical territory of the deprived sense, and attention is a potential driver of this phenomenon [4,6].
52 Despite the reprogramming, the deprived brain area often retains its original function and
53 contributes to new or improved compensatory behavior. Examples are visual cortex contributions
54 to braille reading and echo localization in the blind [7,8]. The posterior parietal cortex (PPC) is
55 multisensory in nature and is gaining attention as an important player in cross-modal plasticity [9].
56 Anatomically located between cortical territory dedicated to audition, vision and somatosensation
57 (Fig. 1), it is involved in multisensory and sensory-motor integration, like spatial navigation, motor
58 planning, spatial attention, the use of egocentric and allocentric reference frames, as well as
59 working memory and action execution [10,11]. In this review we will discuss the latest research
60 concerning the role of the PPC in cross-modal plasticity, how this relates to environmental factors
61 such as social isolation, as well as its potential in developing more potent rehabilitation strategies
62 to compensate for sensory loss. We will argue that especially the inferior PPC is an essential brain
63 region to target for clinically relevant mediation of cross-modal plasticity, and how advancements
64 in mouse research can facilitate additional research, investigating the PPC as a key player in cross-
65 modal plasticity.

66 **Posterior parietal cortex and cross-modal plasticity**

67 The role of the PPC in mediating cross-modal plasticity between sensory modalities is emerging
68 from several observations, showing increased activity and preservation of its function in the
69 context of sensory loss [12–14]. The cross-modal take-over of a deprived sensory area by the
70 remaining senses is influenced by the brain's supramodality, keeping the original function of the
71 deprived area, yet based on inputs from a different sense [15,16]. A common example occurs after
72 vision loss, where the area used for normal reading will be activated during braille reading, albeit

73 driven by touch. Although such functional preservation is better described for sensory cortices
74 [17,18], it is also detected within the PPC for guided reaching [19,20].

75 Magnetoencephalography in a congenitally blind patient revealed how cross-modal touch
76 activation of primary visual cortex starts from primary somatosensory area 3b and runs via PPC
77 areas 7 and 40 and visual associative areas V5/MT and V3 [14]. Human area 7 belongs to the
78 superior PPC, divided by the intraparietal sulcus from the inferior PPC, which contains area 40
79 and 39 (Fig. 1). Other studies confirm this higher activity of the inferior and superior PPC in the
80 blind, as well as the dorsal extrastriate pathway and specifically area V5/MT [12,21–23]. In deaf
81 patients, the inferior PPC also displays higher activity. More specifically of the angular gyrus,
82 located in area 39, and the supra marginal gyrus, located in area 40 [24–26]. When processing
83 visual stimuli, deaf subjects display a stronger functional connection between auditory cortex and
84 area V5/MT [17]. In sum, in patients, inferior PPC and area V5/MT surface as common potential
85 mediators of cross-modal plasticity.

86 Primate research on cross-modal plasticity, and precise contributions of the PPC, is scarce. Cats
87 and rodents are preferred animal models to reveal fundamental mechanisms and principles.
88 Anatomically the primate PPC is organized as in humans, consisting of an inferior and superior
89 PPC. Primate inferior PPC, containing area 7a, is the homolog of human area 40 and 39, and of
90 posterior area 7 in cats (Fig. 1) [27–29]. In primates, binocular deprivation increases
91 somatosensory activation of area 7 by more than 100% [30]. Retinal lesion and auditory loss
92 studies in cat revealed an ensuing reactivation of area 7 and increased PMLS activity, the
93 homologue of primate V5/MT, and more connectional projections from area 7 towards auditory
94 cortex [31,32]. Such projections were not found upon acquired deafness, making it more likely
95 that developmentally established feedback connections from the PPC adapt instead of the creation
96 of new projections. This fits observations in the deaf in relation to critical period plasticity and the
97 brain's higher susceptibility for cochlear implants early in life [33]. Animal studies thus underscore
98 the main involvement of the inferior PPC and area V5/MT/PMLS, although other PPC areas could
99 also be involved depending on the type of sensory loss. In motor-sensory related injuries, there
100 might be a higher involvement of e.g. the primate parietal reach region than area 7 [34].

101 Hishida et al. (2019) recently indicated that pharmacological inhibition of PPC activity in mice,
102 can regulate primary visual cortex activity, enhancing its responsiveness to bottom-up visual

stimuli via excitatory neurons [35]. Suppression of the PPC feedback seems to block top-down sensory signals, supporting the potential of the PPC to mediate cross-modal plasticity. Local activation of Somatostatin interneurons in mouse primary visual cortex enhances the activity of excitatory neurons via disinhibition, controlling cross-modal plasticity [36]. Shifts in signal-to-noise between visual stimuli and inputs from other modalities within V1 could be key to the cross-modal plasticity outcome and are likely determined by local changes in excitation-inhibition (E-I) balance.

In adult mice, cross-modal plasticity can be induced by monocular enucleation, which, in time leads to an almost complete reactivation of the deprived visual cortex by the somatosensory modality (Fig. 2A) [4]. It remains unexplained why this reactivation takes several weeks, but it might relate to PPC involvement. Its individual neurons have different activity patterns over several weeks, but the global population activity remains stable, balancing plasticity and stability[37]. Currently, it is unknown if there are any changes in E-I balance within the PPC upon sensory loss, but parvalbumin interneurons can switch the dominance of sensory modalities within mouse PPC [38]. Recent delineations of mouse PPC subareas push the Rostro-Lateral area (RL) forward as a likely candidate. RL is involved in somatosensory-visual processing of nearby space and in global motion perception, as an area V5/MT homologue [39–41]. In-depth investigation of RL could significantly contribute to understanding the role of the PPC in cross-modal plasticity outcome.

Influence of patient specific factors / environmental factors

Age, social interaction, attention and stress are being controlled by the PPC or have an influence on its activity, possibly influencing the outcome of PPC-mediated neuroplasticity (Fig. 3). With regard to age, both congenital as well as sudden or gradual sensory loss can induce cross-modal plasticity [42,43]. Research in visually deprived mice has illustrated a difference in cross-modal plasticity occurrence between juvenile (P28), adolescent (P45) and adult (P120) mice; with adolescent mice having less plasticity (Fig. 2B and 2C) along with a deviating E/I balance in visual cortex [4,44,45]. In humans, information about sensory loss in adolescence is scarce. Yet the relation to age-at-onset matters since cross-modal plasticity negatively correlates to the behavioral outcome of implant-based rehabilitation [42,46]. The reason for reduced cross-modal plasticity during adolescence, as observed in mice, remains unclear, but could reflect still ongoing brain

maturation, including late critical periods for association areas like the PPC [47]. With age, cross-modal activity might rely more on association areas, compared to the presence of more direct sensory connectivity in the juvenile brain [48]. Ongoing maturation will cause changes in E-I, for example leading to more parvalbumin expression, plateauing in adulthood. Possible triggers are an upregulation of the dopamine system, BDNF or puberty hormones, which lower again in adulthood [47,49,50]. These changes can cause increased or decreased cortical plasticity, depending on domain, area and gender. Rodent research supports the involvement of the E-I balance, showing that a rejuvenation of the inhibitory system, either via dark exposure or suppressing inhibition pharmacologically, can reinstate juvenile like plasticity in adults, but not in adolescents [51,52].

Social isolation can have an important impact on brain plasticity [53,54]. When adult mice are put in social isolation right after monocular enucleation, cross-modal reactivation of the visual cortex is markedly reduced (Fig. 2D). From human research we know that the PPC is involved in social distance and status evaluation, personal space, nearby space attention, emotions and imitation of action execution from other individuals via its mirror cells [55,56], and is part of the parietal-frontal network, which is involved in social interaction and attention. Additionally, the PPC is a functional hub in the default network for its role in thinking about others and past and future simulations. The default network is also considered to be a hub for shifting between feelings of loneliness and purpose [57]. Loneliness and depression cause an increased integration and hyper-connectivity of these networks, leading to less modularity of the brain [57,58], as well as a negative correlation of white matter density with the inferior PPC [59]. Multiple studies indicate that especially the inferior PPC is affected by loneliness, social isolation and depression [59,60].

The observation that social isolation impairs cross-modal plasticity could also reflect changes in the attentional network. Social isolation decreases environmental spatial attention [61], a type of attention mediated by the PPC [62,63]. Lo et al. (2019) confirmed that the PPC is involved in orientating attention, but not in alerting or executive control. It seems that this type of attention, is very important to enable cross-modal plasticity. After hearing loss, a passive stimulus, like a light flash, does not activate the auditory cortex, while a visual attention task does [64]. This is further supported by tinnitus research in which activation of the attention network is observed. One could conclude that attention is very important during a patients rehabilitation therapy, but research by

Jiang et al. (2020), showed improved vision in the blind cortex, caused by stroke, by performing multisensory training under anesthesia [65]. It is unclear if this could also facilitate cross-modal plasticity upon peripheral sensory loss, or if this is specific for central cortical damage. Attention as well as cross-modal plasticity can also be influenced by neuromodulators like acetylcholine and serotonin [61,66–68]. Cholinergic signaling to the PPC mediates the attention necessary to learn new things, navigation in new environments and elevates the signal to noise of relevant sensory stimuli compared to background distractions [16,69,70].

Losing a sense or sensory input can be extremely stressful because it can be life altering, especially when there is a lack of support by peers or family, which can have an influence on plasticity and the inferior PPC. Post-traumatic stress disorder research indicates reduced volumes of the inferior PPC, involved network connections of the right PPC and deficits in difficult attention-driven tasks [71,72]. Recently Libovner et al. (2020) demonstrated that stressful situations can lead to significant loss of excitatory synapses in the PPC of adolescent mice. This decreased synaptic strength is allocated to the sensory cortices as well as retrosplenial cortex, which can disturb multi-sensory integration and thus cross-modal plasticity [73].

Potential target for rehabilitation

Boosting or supporting cross-modal plasticity can be beneficial for patients to learn new skills or improve residual sensing. Influencing the activity of the PPC via multisensory exercise, attention stimulation, or via implementation of meditation in the patients daily routine, could be excellent treatment options. Treating sensory disability should also include the choice of saving the residual function of the disabled sense or providing a relevant functional implant. Either way, it would be beneficial to be able to control cross-modal plasticity. In the context of vision or hearing loss, a functional implant providing bottom-up input to visual or auditory cortex, could benefit from combined modulation of PPC activity, modulating its E-I balance and leading to cross-modal inhibition and a proper integration of that implant (Fig. 4A). Time-wise this should be done as soon as possible post-insult, since the brain-state before injury or treatment can make a significant difference. Brain state manipulation in humans via high frequency transcranial magnetic stimulation, before stimulation of the intraparietal sulcus increased the duration of attention by more than 100% [74]. In mice, manipulation of brain state via activation of interneurons before

sensory loss had more effect on the inhibition of cross-modal plasticity than when performed 3 weeks after injury [36]. The best area to target seems to be the PPC inferior lobe or even V5/MT.

Loss of a limb or paralysis needs a different type of functional implant, a cognitive neural prosthetic, that can be controlled by the brain for external device control, like a robotic limb for reaching, grabbing or walking, or to control an effector like a computer cursor [34,75]. Although these type of prosthetics can function when placed in motor cortex, the PPC is a far better candidate because of its ability to drive the intention of a motor movement, prior to motor cortex activation (Fig. 4B) [76,77]. It allows the patient to have more control over planned movements. On top of that, the involved PPC sub-regions, like the parietal reach region, have the ability to direct both body sides upon single area stimulation [78,79]. Its capability of partially mixed encoding in which neurons respond to multiple variables only adds to its strength [80,81]. First studies in tetraplegic patients showed great potential for PPC implants, in line with previous animal studies [82–84]. PPC area 5, the parietal reach region, AIP and LIP, located in the intraparietal sulcus [34,79,83] or area MMA in mice show the most potential as implant targets [39]. However, just as for sensory type implants, a double approach could lead to the best outcome. Adding an implant in the somatosensory cortex, which is connected with touch sensors on the prosthetic device, can foresee in feedback to the PPC related to peripersonal space and body position, enabling a more precise control (Fig. 4B) [79,84–86]. This is also observed in hemiplegic upper limb paralysis due to stroke, where the amount of somatosensory feedback will contribute to the patients rehabilitation outcome via robot training [87].

Conclusions

The PPC deserves attention in the quest for new therapies for sensory loss. Its multisensory integration characteristics put the PPC forward as a facilitator of cross-modal plasticity, enabling intact sensory modalities to compensate for the deprived sense. Including this area in rehabilitation strategies, either to control cross-modal plasticity in combination with functional sensory implants, or as a driver for functional motor implants is becoming more important and necessary. Additional patient-specific factors, including age, social environment, attention and stress converge onto the PPC. These factors need to be taken into account in the context of sensory loss, especially in relation to changes in E-I within the PPC. Therapies like neuromodulation via the cholinergic

221 system, targeting of specific interneurons or boosting attention are only some examples that
222 deserve further investigation.

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Figures

Figure 1 – Schematic Posterior parietal cortex comparison of humans, primates and mice.

Left – Human posterior parietal cortex consists of area 5 and 7, making up the superior lobe, as well as area 40 and 39, making up the inferior lobe, and separated by the intraparietal sulcus.

Middle – Primate posterior parietal cortex consists of area 5, being the superior PPC, and area 7, being the inferior PPC, and separated by the intraparietal sulcus.

Right – Mouse posterior parietal cortex has no division in inferior or superior parietal lobes or an intraparietal sulcus. The most posterior lateral PPC area is the rostro-lateral area (RL) [39].

Figure 2 – Monocular enucleation mouse model for cross-modal plasticity of the visual cortex. **A.** Adult (P120) mice with normal binocular sight have high *zif268* levels (black signal) in the binocular (BZ) and monocular zones (MZ) of visual cortex. Removal of the right eye cause a clear drop in *zif268* levels in monocular visual cortex (white-gray signal). Until three weeks after injury the cortical territory of the remaining eye (BZ) will expand. After seven weeks, there is an almost complete reactivation of the visual monocular zone, indicated by high *zif268* levels, and driven by the whiskers of the somatosensory system [4]. **B-D.** *zif268* read-out of recovery of cortical activity seven weeks post monocular enucleation in **B.** a juvenile (P28) mouse: high *zif268* levels in the binocular and monocular zone, but mainly in upper layers; **C.** an adolescent (P45) mouse: expansion of the binocular zone, but *zif268* levels remain very low in the monocular zones; **D.** an adult (P120) socially isolated mouse: abnormal reactivation with a binocular zone that does not expand and limited *zif268* levels in the monocular zones.

Figure 3 – Environmental factors like age, social isolation, attention and stress that influence the PPC and brain plasticity. Relevant references for **Age**: [42,44,45,47,48]; **Social isolation**: [55–60]; **Attention**: [16,61–64,69] and **stress**: [71–73]

Sensory impairment

Motor - Sensory impairment

Figure 4 – The role of the posterior parietal cortex in functional prosthesis-driven rehabilitation upon sensory or motor-sensory loss. Left – Activity modulation of the PPC via transcranial magnetic stimulation could lead to better control of cross-modal plasticity, enabling better integration of the sensory substitution device. Right – The PPC is a perfect candidate to drive the movement of the functional prosthesis, while touch sensors provide feedback via the somatosensory cortex.

Annotated references

* Papers of significant importance

** Papers of outstanding importance

14. Ioannides AA, Liu L, Poghosyan V, Saridis GA, Gjedde A, Ptito M, Kupers R: **MEG reveals a fast pathway from somatosensory cortex to occipital areas via posterior parietal cortex in a blind subject.** *Front Hum Neurosci* 2013, doi:10.3389/fnhum.2013.00429.

Two sighted and one congenitally blind patient underwent magnetoencephalography recordings to understand which pathways were involved in non-visual tasks and cross-modal brain? activation. Upon somatosensory stimulation they observed activity in the visual cortex, originating from several somatosensory areas; 1, 2, 3a and most clearly area 3b, via posterior parietal areas 7 and 40 and visual association areas V5 and V3, before reaching V1.

16. Ljubojevic V, Luu P, Gill PR, Beckett LA, Takehara-Nishiuchi K, De Rosa E: **Cholinergic modulation of frontoparietal cortical network dynamics supporting supramodal attention.** *J Neurosci* 2018, doi:10.1523/JNEUROSCI.2350-17.2018.

Lesioning of the nucleus basalis of Meynaert, the primary cholinergic source for the frontoparietal network, reduces attention and alertness to incoming sensory stimuli. Beta oscillation synchronicity is reduced between prefrontal and parietal cortex and their strength within the PPC itself. This suggests a role for acetylcholine in regulating supramodal attention.

32. Burnat K, Hu TT, Kossut M, Eysel UT, Arckens L: **Plasticity beyond V1: Reinforcement of motion perception upon binocular central retinal lesions in adulthood.** *J Neurosci* 2017, doi:10.1523/JNEUROSCI.1231-17.2017.

Alterations in neuronal activity and motion discrimination of several cortical areas were tested after inducing central retinal lesions in adult cats. Noteworthy is the increased expression of neuronal activity marker *zif/268* within area PMLS (V5/MT homologue) and area 7 (PPC), and correlating with improved perception of moving stimuli, as tested with

random dot kinematics.

35. Hishida R, Horie M, Tsukano H, Tohmi M, Yoshitake K, Meguro R, Takebayashi H, Yanagawa Y, Shibuki K: **Feedback inhibition derived from the posterior parietal cortex regulates the neural properties of the mouse visual cortex.** *Eur J Neurosci* 2019, doi:10.1111/ejn.14424.

The mouse posterior parietal cortex can regulate activity of excitatory neurons within the visual cortex. Pharmacological inhibition of this region increases activity of the primary visual cortex, while transcranial electrical stimulation, suppressed visual responses in V1, leading to short-term depression.

36. Scheyltjens I, Vreysen S, Van den Haute C, Sabanov V, Balschun D, Baekelandt V, Arckens L: **Transient and localized optogenetic activation of somatostatin-interneurons in mouse visual cortex abolishes long-term cortical plasticity due to vision loss.** *Brain Struct Funct* 2018, doi:10.1007/s00429-018-1611-7.

Cross-modal plasticity takes place upon vision loss, where the somatosensory modality will reactivate the deprived visual cortex. This paper reveals a role for somatostatin positive interneurons in controlling the occurrence of cross-modal plasticity. Optogenetic activation of somatostatin interneurons reduces cross-modal take-over, with a considerable larger effect when optogenetic stimulation takes place before vision loss, instead of 3 weeks post monocular enucleation.

37. Driscoll LN, Pettit NL, Minderer M, Chettih SN, Harvey CD: **Dynamic Reorganization of Neuronal Activity Patterns in Parietal Cortex.** *Cell* 2017, doi:10.1016/j.cell.2017.07.021.

Activity patterns within layer 2/3 of the PPC were tracked over a period of several weeks via chronic imaging during a virtual navigation task. The initial single neuron activity peaks changed over different trials, shifting to activity peaks in different neurons, but resulting in a stable overall activity. This could be a necessary characteristic for association areas to balance flexibility and stability.

38. Song YH, Kim JH, Jeong HW, Choi I, Jeong D, Kim K, Lee SH: **A Neural Circuit for**

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In mice that perform an auditory – visual conflict task, there is a dominance for the auditory stimulus. This dominance is evoked by an auditory – PPC – visual circuit where an auditory stimulus will activate Parvalbumin positive interneurons, which in turn will mediate feedforward inhibition over the visual stimulus.

41. La Chioma A, Bonhoeffer T, Hübener M: **Area-Specific Mapping of Binocular Disparity across Mouse Visual Cortex.** *Curr Biol* 2019, **29**:2954-2960.e5.

Binocular disparity of higher order visual areas RL, LM and V1 was measured via two-photon calcium imaging. Dichoptic gratings revealed increased tuning in RL for nearby disparity. Neurons that respond to nearby disparity are found to be more correlated with RF of the lower visual field, which mainly dominates the visual field of RL and coincides with the whiskers [88]

51. Huang S, Gu Y, Quinlan EM, Kirkwood A: **A refractory period for rejuvenating GABAergic synaptic transmission and ocular dominance plasticity with dark exposure.** *J Neurosci* 2010, doi:10.1523/JNEUROSCI.4384-10.2010.

Dark exposure, where rats are put in the dark for two consecutive days, can lead to a rejuvenation of the inhibitory system. In adulthood this leads to the reactivation of ocular dominance plasticity and reexpression of endocannabinoid-dependant inhibitory LTD. Dark exposure had no effect when performed in adolescence, demonstrating this is a refractory period in life that limits the regulation of synaptic transmission via endocannabinoid receptors.

65. Jiang H, Rowland BA, Stein BE: **Reversing Hemianopia by Multisensory Training Under Anesthesia.** *Front Syst Neurosci* 2020, doi:10.3389/fnsys.2020.00004.

In this article cats underwent a cortical lesion to mimic hemianopia. Electrophysiological recordings of superior colliculus deep layer neurons, which receive direct retinal input, was performed. A remarkable recovery of their activity was induced by presenting spatiotemporal congruent auditory-visual exposure during anesthesia, which was not

possible with modality specific or incongruent stimuli, according to previous studies.

73. Libovner Y, Fariborzi M, Tabbà D, Ozgur A, Jafar T, Lur G: **Repeated exposure to multiple concurrent stresses induce circuit specific loss of inputs to the posterior parietal cortex.** *J Neurosci* 2020, doi:10.1523/JNEUROSCI.1838-19.2020.

Repeated stress over a 10 day period, induced by excessive light, noise, reduced space and increased movements, affects the function and inputs to the PPC during adolescence. Disruption of spatial navigation and an increase of c-Fos expression, a stress induced neuronal activity marker, were observed in the PPC. Loss of excitatory synapses in L5/6 of the PPC were observed, but no loss of neurons, contrary to auditory, visual and retrosplenial cortex, which had a significant decrease in neurons that project to the PPC after repeated stress.

74. Edwards G, Contò F, Bucci LK, Battelli L: **Controlling brain state prior to stimulation of parietal cortex prevents deterioration of sustained attention.** *bioRxiv* 2020, doi:10.1101/2020.01.06.896357.

In this study they used low-frequency transcranial magnetic stimulation of the intraparietal sulcus to sustain attention longer, without any effect. However, controlling neural excitability by using high-frequency transcranial random noise stimulation before the low-frequency stimulation led to a doubling in sustained attention.

80. Zhang CY, Aflalo T, Revechikis B, Rosario ER, Ouellette D, Pouratian N, Andersen RA: **Partially Mixed Selectivity in Human Posterior Parietal Association Cortex.** *Neuron* 2017, doi:10.1016/j.neuron.2017.06.040.

Physiological recordings of a tetraplegic patient in area AIP of the PPC revealed activity upon hand en shoulder movements, imagined or attempted, with either side of the body. There was no specific subpopulation for the type of body movement, but there was however a correlation between neurons encoding for body side as well as for cognitive strategy. These correlations were specific within bodyparts and not between, meaning the body part is the first variable that determines how the other variables are encoded, leading to a partial mixed selectivity.

81. Zhang CY, Aflalo T, Revechkis B, Rosario E, Ouellette D, Pouratian N, Andersen RA:
**Preservation of partially mixed selectivity in human posterior parietal cortex across
changes in task context.** *eneuro* 2020, doi:10.1523/eneuro.0222-19.2019.

Building up on their previous publication (Zhang et al., 2017), this study provides
evidences that a 4x4 mm array patch in the human PPC of a tetraplegic patient can drive
body side and cognitive strategy independent of task context during a brain machine
interface control.

Declaration of interest

None.

Author contributions

Sara Gilissen: Conceptualization, Writing

Lutgarde Arckens: Conceptualization, Resources, Writing, Supervision, Funding acquisition

Role of the funding source

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