

Repetition Suppression and Related Effects

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Abstract. We present an extensive review of the repetition effect aiming to understand a simple experimental fact: why the repetition of a stimulus leads to reduced neural activity as compared to unrepeatable stimuli. Previous attempts to understand this fact were connected mainly with the application of the mechanisms of local models (synchrony, facilitation and sharpening). We show that the Neurolocator model earlier proposed for the explanation of neural memory, focused attention, and Pavlovian conditioning can also successfully explain most difficult cases of repetition effects. The Supplement Material includes wide range of related effects such as habituation and sensitization, negative priming, inhibition of return, whole brain segregation and integration. All of them need an additional works to build specific models in each case, but we believe that their successful modeling will be closely connected with the Neurolocator model.

Key words: *repetition suppression, repetition enhancing, habituation, oscillatory neural networks, phase oscillators, neurolocator model.*

1. INTRODUCTION

Repetition suppression (RS) effects are ubiquitous in nervous system and RS has remained one of the most widely studied experimental finding in primates and humans, but many researchers point to slow progress in RS mechanism understanding as can be seen from the following.

“In spite of this enormous research effort, there is still no single theory that can account for the available experimental findings on RS... It is perhaps puzzling that the mechanisms behind such a simple phenomenon as the reduction of the neural activity to repeated compared to unrepeatable stimuli remain insufficiently explained today, after almost fifty years of study” [1]. “RS is not a unitary phenomenon, but likely subsumes multiple mechanisms that operate under different conditions. These mechanisms of course need to relate to single-cell data and known physiological mechanisms; but to make further progress, I believe we need dynamical neural network models that relate these mechanisms to the properties of neural populations that are measured by fMRI and EEG data” [2].

The most important problem of cognitive neurobiology is the understanding of a mechanism that relates repetition suppression and repetition priming (RP) as can be seen from the following quotations.

“Many studies of the neural bases for behavioral priming have encountered an interesting paradox: in spite of faster behavioral responses, repeated stimuli usually elicit weaker neural responses” [3]. “How can reduced neural activity lead to improved behavioral performance?” [4]. “The neuronal mechanisms that enhance performance in the face of reduced brain activity are unknown” [5]. “A central goal of cognitive neuroscience is to understand the relationship between repetition suppression and priming” [6].

In this paper after reviewing many works on RS and RP we present a dynamical model that can satisfy the above requirement of Henson [2] and explain the paradoxical relationship between RS and RP.

2. EXPERIMENTAL DATA

2.1. Basic characteristics of the repetition suppression

1) *Initial activation.* The magnitude of RS was greater for stimuli that initially elicited the largest activation in the neocortex [6, 8, 9] and hippocampus [10–12].

2) *Time parameters.* RS appears after the stimulus onset starting between 100 and 250 msec in the visual modality and between 40 and 60 msec in the auditory modality [13]. RS is maximal after the peak of the response and then reduces. RS in IT is still present for ISIs of at least 900 msec [8]. Depending on cortical region it increases with prime duration initially presented up to 200–250 msec [14, 15], but has no effect in 300–3000 msec [16] and declines for larger duration [15, 17]. RS is maximal for short ISI and decayed with increasing ISI [18]. The upper limit for the fMRI repetition time lag is typically of the order of seconds [19]. RS effects occur early within initial stage of stimulus processing and persist for the entire stimulus duration [20].

3) *Recovery.* RS recovers with increasing the interstimulus interval [17, 19, 21, 22] or the number of presentations of the adaptor [17, 23] even if test stimuli are presented in a different format [24].

4) *Multiple time-scales.* RS can be observed on at least three-time scales:

(a) on the scale of tens of milliseconds peaking at ~100 msec [25, 26]; (b) on the time-scale from hundreds msec to min [19, 27–30]; (c) on the time scale from min to days [31–33]. These effects most likely relay on different mechanisms [2, 25, 34], though repetition effects with novel stimuli can occur over long-time scales [35]. There exists time-scale gradient along anterior-posterior axis [36–40] with long time-scale at higher hierarchical levels (but see [29]). Multiple time scales might be due to the power-law behavior of adaptation [41].

5) *Stimulus generalization.* RS occurs despite negligible visual similarity (in orientation, size, position, expectation, etc.) between the initial and repeated object [4, 42–48] and even in multimodal and cross-modal tasks [49–51]. The category-specific regions showed RS for more than one stimulus category [52, 53]. RS is larger for non preferred stimulus category [6, 20].

6) *Stimulus selectivity.* The dependence of RS on the degree of similarity between the adaptor and test stimuli was limited mainly to the early phase of the neural response [46] or with short adaptation duration [45]. RS was stimulus selective only on a time scale of ~100 msec [25].

7) *Attentional modulation.* RS was modulated by spatial and feature attention [31, 39, 54–57]. Some forms of RS can be preserved in the absence of attention [31, 58, 59]. RS comprises both attention independent and attention dependent components [31, 60]. RS is less viewpoint-dependent at long intervals [34, 45].

8) *Theta/gamma synchronization.*

(a) RS decreases gamma-power [7, 58, 61–71]. RS occurs in high frequency broad band, but not lower frequency band in many cortical regions [20, 58, 71] and the hippocampus [20, 71]. Sometimes postadaptation shows an increase in gamma synchrony [66, 72–75].

(b) RS shows specific phase locking effects in the theta range [4, 68, 76–81], including an anticorrelated relationship between local/gamma and global/theta synchrony [6, 62, 82] and increase in cross-frequency coupling [70].

(c) relationship between the gamma and other frequency bands becomes strongest during the first-stimulus (i.e., the novel stimulus) presentation and decreases with repeated presentations of stimuli [83], e.g., reduced theta-gamma phase amplitude coupling (PAC) around the third presentation [81].

9) *Emotional modulation.* RS effects decrease linearly with negative valence [84, 85], but opposite effects were described [51, 86–89] as possible attentional influence [90]. More suppression was caused by cues associated with a higher reward than with a lower reward [91].

10) *Nonlocal adaptation*. Usually, adaptation is thought to involve modified synaptic plasticity, but adaptation of motion vision of vertebrate and invertebrate occurs without any change in the parameters [92, 93]. RS in IT has transsynaptic origin [94].

11) *Self-organized criticality*. Strong visual stimulation initially causes the overall excitability. But repetition of visual input drives the cortical network dynamics to a critical state similar to physical phase transitions [95, 96]. Such adaptation self-organizes cortical circuits towards a balanced regime that maintains criticality in the cortex during sensory information processing [95, 97].

Theories and models. RS was initially considered as resulting from low-level and local adaptation mechanisms by which the response of a neuronal population would be reduced when a stimulus appears for the second time: the fatigue model entails, facilitation model, sharpening model. However, local models cannot explain the RS plateau which is reached within 5–10 repetitions [98]. Recent years have seen the development of an alternative view linking RS to top-down expectations within a predictive coding scheme [99]. According to this view, the brain constantly predicts forthcoming inputs and computes the discrepancy between actual and predicted sensory states to update its predictions. However, more careful experiments show that RS dissociates from top-down stimulus expectation effects [8, 13, 30, 100–107]. Henson [108] and many others suggest that RS is not a unitary phenomenon and likely includes multiple mechanisms that operate in different conditions, e.g., short (within trials) and long (between trials) intervals may activate different/dissociable RS mechanisms that exist in overlapping regions [34]. Traditionally, adaptation is attributed to synaptic plasticity, but recently Quiroga [109] proposed a model which does not require such plasticity. Previously Johnson et al. [110] built a model of short-term memory with metastable states with no need of synaptic learning. Similarly, Kryukov [111, 112] also proposed a neural model of short-term memory as metastable state without synaptic modification which is able to explain RS in a unified manner for short and long intervals due to criticality.

2.2. Repetition enhancing (RE) and repetition suppression

RS and RE commonalities. RS co-occurs with the converse phenomenon of repetition enhancement (RE):

- (a) Both occur in a single cortical region [113] or simultaneously in different brain regions [9, 11, 55, 114–116].
- (b) Both are influenced by a modulation signal from the hippocampus [10].
- (c) Both decrease with a lag between successive stimulus presentations [117].
- (d) Both are sensitive to multiple factors including preexisting stimulus representation, cognitive task, a lag between repetitions, and spatial attention [2].
- (e) Both are region specific and time scale dependent [78].
- (f) The duration of both depends on stimulus familiarity (seconds) and novelty (hours) [35].
- (g) The direction of repetition effects (suppression vs enhancement) is affected by visibility [118], the number of repetitions [119–122], and preexperimental stimuli familiarity [21, 23, 66, 123, 124].

RS and RE contrasts. RS and RE occur in different conditions, cognitive task, and temporal aspects (see Table 1).

2.3. Repetition priming and repetition suppression

The repetition priming (RP) is a form of positive priming meaning that later experiences of a stimulus will be processed more quickly and refers to improvements in accuracy and RT in behavior response with repeated stimulus presentation.

Table 1

RS	RE	References
high-contrast scene	low-contrast scene	[118]
stimulus moving in a certain direction	stimulus moving in a opposite direction	[25, 125]
6–9 repetitions of low-visible pictures	1–5 repetitions of low-visible pictures	[119]
unmasking	backward masking	[126]
repulsive tuning shifts	attractive tuning shifts	[115]
task-dependent	task-general	[127]
repulsive orientation shifts	attractive orientation shifts	[128]
unrelated semantic conditions	related semantic conditions	[68]
familiar object	unfamiliar or novel object	[21, 24, 66, 123, 124]
larger visual grating	smaller visual grating	[115];
inhibitory engram	excitatory engram	[129, 130]
massed learning	spaced learning	[88, 131–133]
recognition compromised	recognition improved	[131–136]
implicit learning	explicit learning	[137]
suppressed gamma synchrony	enhanced alpha synchrony	[61]
gamma power decrease	gamma power increase	[66, 72]
smaller latency	greater latency	[2, 113]
95–150msec	230–270ms	[138]
time window	time window	
100–250msec	> 300 msec	[13]
at early times	at latter times	[128, 139]

RS and RP similarity.

- (a) Both are reduced as the lag is increased [19, 21], but see [131].
- (b) Both occur on time scales from a second to days [31–33, 140].
- (c) Both are reduced with the prime duration or the number of presentations of repeated objects [15, 141]).
- (d) Both can be largely determined by previously learned stimulus-response associations [42].
- (e) Both are enhanced by grater neural synchrony between prefrontal and temporal cortices [142].
- (f) Both can have the same neuronal mechanism [1, 35, 143, 144], but others consider this question still open [6, 36, 52, 145]. Even independent mechanisms are possible.

RS and RP contrasts.

- (a) RS relatively reduces/derails encoding [78, 146], but RP makes more accurate encoding and stimulus classification [62, 147] by forming so called soft-assembly with variable topological dimensionality depending on task difficulty [148].
- (b) RS decreases classification accuracy [147], but RP improve target recognition [55, 35, 134–136, 149].
- (c) RS frequently causes tuning preferences to shift away from the adaptor [115, 139, 146, 150–152], but RP leads to shifts in tuning toward the adaptor [115]. Shorter adaptation in most cases produces repulsive shifts whereas adaptation exceeding nine minutes results in attractive shifts in the same visual unit [122, 128]. Attractive shifts are band-limited and are around test stimulus; the flanked responses remain unmodified [153, 154].
- (d) RS decreases gamma band ranges and phase synchrony but RP increases them both [65].

(e) RS and RP do not vary in a significantly correlated fashion across trials, although RS accompanies RP [155, but see 156].

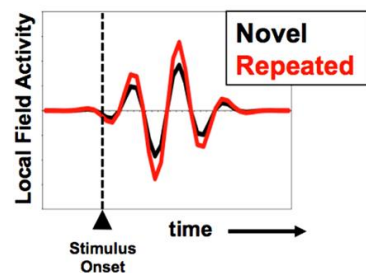
(f) Many data suggest that RS and RP have independent mechanisms [2, 123, 138, 157–161]. In particular, RS and increasing coupling in repetition priming varied independently, which requires modifications of all current models [162].

RS, RP and memory. RS and RP are implicit or “unconscious” memory [163] (but see [137]) with no relationship between hippocampal RS and subsequent memory [10, 162] or between RP and episodic memory [131] (but see [164]). However, the meta-analysis of neuroimaging experiments revealed that global topography of RS and RE were similar to that of the subsequent memory effects indicating that the mechanism of RS is a reduced version of the retrieval success, i.e. involvement in explicit memory [52]. Accordingly, perirhinal cortex supports both conceptual priming and the memory of conceptual information and predicts better subsequent memory [165, 166]. The data in the visual search suggest that implicit and explicit memory processes are linked in MTL and expressed via RS and RE mechanisms correspondingly [137] and driven by single underlying memory system [156] or at least they are functionally connected [167] (but see [168, 169]). RP can result from S-R binding memory [170] by repeating S and producing R more rapidly without necessary recapitulating RS and RE initially used to generate R.

Theories and models. Multiple theoretical models have been proposed to account for simultaneous observations of both neural repetition suppression and behavioral priming (reviewed in [78]; see Fig. 1.)

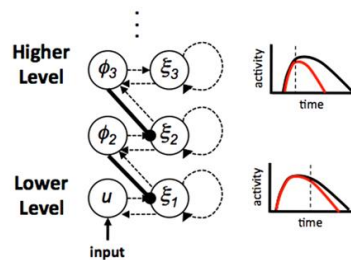
Synchrony

(Gotts, Chow, & Martin, 2012; Gilbert et al., 2010)



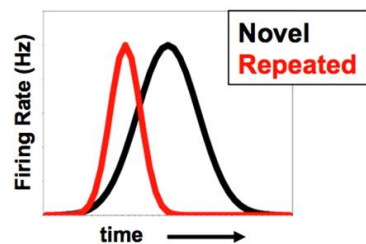
Predictive Coding

(Friston, 2005; Friston & Kiebel, 2009)



Facilitation

(Henson, 2003; James et al., 2000)



Sharpening

(Desimone, 1996; Wiggs & Martin, 1998)

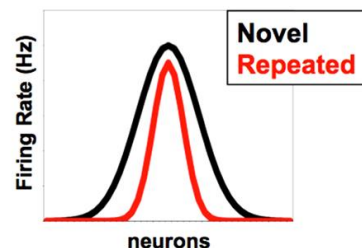


Fig. 1. Neural models of repetition suppression and priming. Four prominent models of repetition priming and repetition suppression are considered. The Synchrony model (upper left) holds that neural activity becomes more synchronized with repetition, permitting more coordinated propagation of activity at lower overall activity levels. The Predictive Coding model (upper right) holds that top-down causal influences are more strongly negative, leading to repetition suppression in the receiving region, along with a gain enhancement that leads to more rapid onset and offset of responses. The Facilitation model (lower left) claims that neural activity onset and offset are advanced in time, with earlier peak responses and a reduction in overall activity. The Sharpening model (lower right) holds that weakly tuned, poorly responsive cells are the ones driving repetition suppression, reducing downstream support for competing stimulus identities and speeding downstream stimulus-selective responses. The figures are reproduced with permission from Gotts et al. [78].

Tartaglia et al. [35] suggested RE as viable candidate for neuronal substrate of RP. Wikipedia describes six theories of RP, considering the synchrony theory as being the most probable mechanism [78, 171]. Gotts et al. [171] pose two challenges to existing models of RS and RP: 1) after relatively small number of stimulus repetitions the activity decrease occurs in occipitotemporal cortex without much concomitant change in neural tuning; 2) PFC exhibits reduced rather than enhanced stimulus selectivity, undermining popular joint explanation of RS and RP. Weiner and Grill-Spector [6] have added two more challenges: 3) anticorrelated relationship between local (gamma) and global (theta) interareal synchrony (which is at odds with Gotts's model); 4) the synchrony alone can not explain a multitude of RS effects.

Nonlinear dependence of activity of single neurons on the (total) activity of local and distal neurons is ubiquitous and is termed as divisive normalization (DN). A very successful explanation of such dependences is termed as canonical computation [172]. However, the neuronal mechanism of DN is not well understood. One direction is that normalization and attention might use a common mechanism [173–177] (but see Sanayei et al. [178]). Another attempt is to connect DN with Hebbian learning, which provides the best account of physiological data [179]. Also there exist several attempts to explain adaptation without plasticity [109, 110]. They have two deficiencies: a) no neurobiological explanation and b) the need to extend to adaptation in time [180, 181].

3. NEUROLOCATOR MODEL CAN UNIFORMLY EXPLAIN MOST OF THE REPETITION EFFECTS

3.1. The Assumptions of the NeurolocatorModel [182, 183]

1. The medial septum is a global pacemaker or a central oscillator.
2. The CA3 field of the hippocampus is a comparator and novelty detector.
3. The limbic circle, which includes the hippocampal CA1 field, performs the function of a delay line and connection between the hippocampus and the neocortex.
4. The theta-rhythm plays a critical role in conscious and unconscious perception, attention, memory and adaptation.
5. The functional unit of the model is a unified submodule resembling a cortical microcolumn and a cortical “barrel”.
6. A star architecture consisting of a single central oscillator and many cortical oscillators, operating at a theta rhythm frequency that is automatically regulated by a phase-frequency tracking system.
7. Learning in the model occurs as follows: when the number of synchronously working cortical oscillators reaches a critical value, their natural frequencies converge and get modified.
8. All submodules and the entire system can operate near self-organized criticality based on the principle of physical phase transitions both at the local level of microcolumns and at the global level of synchronization.

The details of the model formulation see in Supplement 1.

3.2. Experimental support/confirmation

Each of these assumptions has now the following experimental support/confirmation, correspondingly.

1. The medial septum operates as a global theta pacemaker [184–186], and the generation of theta activity depends significantly on its bilateral connections with the hippocampus [187–189].
2. The hippocampus, integrating spatiotemporal submodules [190], acts as a comparator [191–196], which ensures its central role in information processing [197, 198] and adaptation [10, 71, 199–201].

3. Anatomical [202] and functional [203–205] studies have shown connections between the hippocampus and the thalamus, and the reversible inactivation of the thalamic basal nucleus strongly suppresses the Global Signal coming from the hippocampus to the neocortex [206]. Thus, the thalamocortical system together with the limbic system can work like a radar transmitter of the request or probing signal from the hippocampus to the cortico-thalamic workspace [207], where the response signal is formed, reflecting information selection and integration [208].

4. The request or probing signal is mostly transmitted from the hippocampus to the neocortex on theta frequency, but the reflected or response signal is transmitted at gamma frequency [209–214].

5. It has been experimentally proved that cortical columns are building blocks or structural units of cortical functions [215].

6. Many data on Global Signal, especially Ma et al. [216] and Gutierrez-Barragan et al. [217], suggest that the Global Signal is the cortical response on the output signal of the central oscillator, which gives rise to all properties of the cortical functional connections, so the star architecture is supported by many experiments.

7. Long-term memory is not stored in the hippocampus [218–220], but in the neocortex [221–225] and is encoded not by synaptic weights [226–228], but by the neuronal populations [229, 230] or engrams [231, 232], composed of cortical columns [215]. “Engram cells that fire together wire together” [233]. Integrative “gluing” of memory and attention elements was discovered in self-citation (self-loving) activities [234].

8. Critical mode and phase transitions exist in the brain, providing high sensitivity to external changes, huge dynamic range of input signals and the high speed of adaptive processes [235, 236].

The details of experimental support see in Supplement 2.

Selected properties to be explained

To explain how our model works in adaptation, we select from very many repetition data only those that are most difficult to explain in terms of existing models requiring full strength of the Neurolocator model.

1. The magnitude of RS was greater for stimuli that initially elicited the largest activation in the neocortex and hippocampus (see basic characteristics of RS).

2. RS is larger for non preferred stimulus category [6, 20].

3. RS is maximal for short ISI and decayed with increasing ISI [18].

4. The direction of repetition effects (suppression vs enhancement) is affected by visibility [118], the number of repetitions [119–122], and preexperimental stimuli familiarity [21, 23, 66, 123, 124].

5. Stimulus repetition drives the cortical network dynamics to a critical state similar to physical phase transitions [95, 96].

6. RS reduce θ/γ phase amplitude coupling (PAC) [81, 83].

7. RS occurs in high frequency broad band but not lower frequency band in many cortical regions [20, 58, 71] and the hippocampus [20, 71].

8. RS and increasing coupling in RP varied independently which requires modifications of all current models [162].

Short explanation of selected properties

To explain these properties in terms of the Neurolocator model, let us remind that our model represents the multi-input phase-locked loop (PLL) (see Equation (1) in [183]), in which RP and RS appear as a result of global theta/gamma synchronization and desynchronization, correspondingly. In more details, the above properties 1–3 correspond to modulation of PLL by the parameters AK , Λ_0 and the function $g(t)$, correspondingly. The function $g(t)$ representing the cross correlation between septal and cortical spike activities

relates RS and RP (as measured by EEG and fMRI) to single neural activity (as registered by microelectrodes); the parameter Λ_0 regulates the degree of synchronization in the preferred cortical region: it improves the behavior performance at $\Lambda_0 \approx 0$ and desynchronizes neural activity at larger Λ_0 ; AK regulates the influence of the arousal, attentional enhancement and emotional influence ($A = \sum_{j=1}^n A_{0j}$). The property 4 is determined by the same mechanism for

RS and RE at different initial conditions. The property 5 follows from the critical state in microcolumns and also from global brain synchronization that allows the repetition time lags of the order of seconds and minutes. The property 6 corresponds to reducing θ/γ PAC during theta desynchronization. The property 7 follows from the above experimental fact 4 that cortical signals are transmitted to the hippocampus not only on theta but also on gamma frequency. The property 8 can be explained by integration of microcolumns in preferred cortical regions during synchronization and their independent dissociation in nonpreferred regions during desynchronizations. The computer solution for the RS and RE relation is given in Supplement 1.

The strength of our model can be appreciated by its capability to respond to the following outstanding question.

“Why wasn’t RS to long-lagged stimulus repetitions observed in all electrodes? ... Why was the shift in peak timing for face repetitions not observed in all electrodes? How does the neural response magnitude to preferred stimuli affect RS?” [20, p. 600]).

In our model, RS to long-lagged stimulus repetition is possible only if microcolumns are in a critical state of phase transition, which is possible to observe not in all electrodes. Analogously, not all electrodes are placed in preferred cortical regions in which a shift in peak timing for face repetition occurs due to isolable learning. The preferred stimulus (e.g., a face) by definition elicits maximal response in the corresponding cortical region (e.g., FFA) which is possible only if the frequency of the hippocampal theta rhythm is very near the natural frequency of cortical oscillators in this region and therefore most of other cortical oscillators will be in a desynchronized state and therefore RS will be near maximum. *“What is the relationship between adaptation of single neurons and populations?”* [237].

The relation between adaptation of single neurons and populations is very complex, so far unknown in details [238], and usually approximated by the so-called divisive normalization (DN) [239, 240]. DN refers to a non-linear computation whereby the response of a given neural unit is divided by the activity of another neural unit. One would need to extend such model to incorporate adaptation in time [180, 181]. One of the late successful variants of DN as presented by Burg et al. [241] can be related with our model in that their “normalization weights” can be collated with our spike crosscorrelation $g(t)$ and their so-called “restricted sets” of preferential stimuli is comparable with our assumption of preferred cortical regions with restricted Λ_0 . Besides our model, unlike all existing normalization models, have no unresolved problems of extending processing in time [242], attention [183] and consciousness [182].

4. CONCLUSIONS

We present a unified model of major available experimental data on RS and RE that has not been possible for more than fifty-five years of intensive study [1].

RS is not a unitary phenomenon, but as predicted by Henson [2], incorporates different mechanisms under different conditions.

The key point of the unified model is the central role of the medial septum as a global pacemaker and the hippocampal CA3 field as the phase comparator to regulate global theta frequency and to synchronize a number of microcolumns. These are the most important assumptions for the current task performance.

The Neurolocator model as the main mechanism of numerous repetition effects was not designed especially for RS but is closely related with global neural mechanisms of brain functioning based on three basic independent foundations: the principle of the dominant, the principle of phase transition, and the principle of the radar [182].

Our model by explaining many data provides a simple explanation of paradoxical relationship between RS and RP: in spite of faster behavior responses (i.e., RP), repeated stimuli also elicit weaker neural response (RS) because theta frequency synchronization in RP may independently cause θ/γ desynchronization (RS) in other parts of the neocortex and this RS-RP independence was confirmed by Gotts et al. [160].

SUPPLEMENT 1.

COMPUTER SOLUTION FOR RS AND RE RELATION

Kryukov et al. [112] derived the follows differential equation, which describes Neurolocator model as a multiple phase-locked loop (PLL)¹:

$$\frac{d\varphi_i}{dt} = \Lambda_{0i} - \left[\sum_{j=1}^n A_{0i} g(\varphi_i) + N_i(t) \right] F(p) - B_{0i} g(\varphi_i), \quad i=1, \dots, n, \quad (S1)$$

where φ_i is the phase difference between septal and cortical oscillators; Λ_{0i} is their natural frequencies detuning $\Lambda_{0i} = \omega_i - \omega_0$; A_{0i} is the strength of the i -th cortical oscillator's action on the central oscillator; B_{0i} – is the strength of central oscillator action on the i -th cortical oscillator, $g(\varphi)$ is a non-linear discrimination function, $N_i(t)$ is the “white” noise of the j -th hippocampal lamellae; $F(p)$ is the transfer function of the low pass filter of the midbrain Reticular Formation.

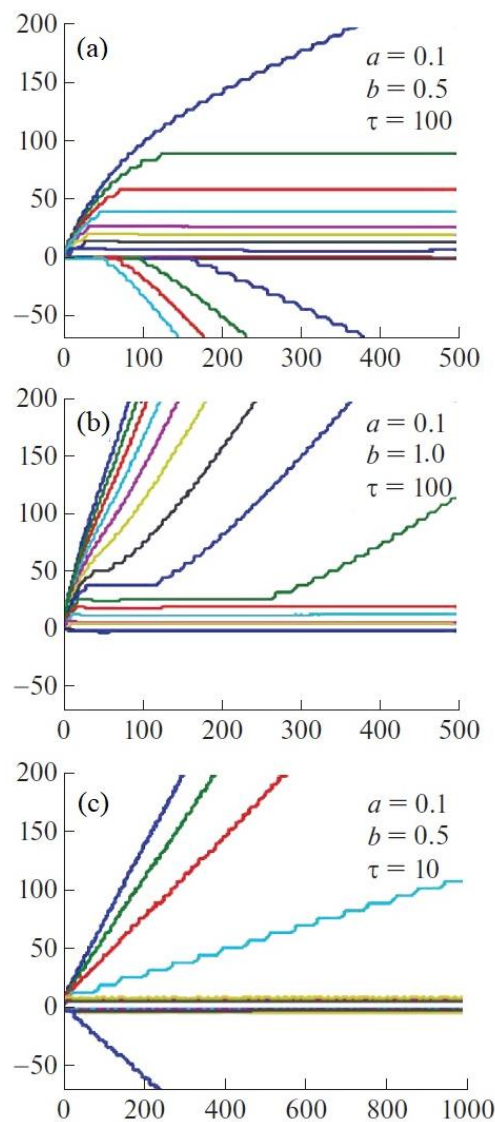


Fig. S1. Dynamics of synchronization (asymptotically horizontal curves) and desynchronization (all other curves). The vertical axis is the phase difference and the horizontal axis is time. Different oscillators with different w_i are presented by the curves of different colors.

¹ The term B_{0i} was originally erroneously omitted in Kryukov et al. [112], but later in [181] the error was corrected

To make clear how Equation (S1) can differentiate between RS and RE, let us simplify it in the following way:

- 1) all cortical oscillator are made identical $A_{0j} = a$ ($j = 1, \dots, n$) and $B_{0i} = b$ ($i = 1, \dots, n$);
- 2) the noise term $N_i(t)$ is eliminated;
- 3) $F(p) = (p\tau + 1)^{-1}$;
- 4) $g(\varphi) = \sin \varphi$;
- 5) $\varphi(t) = 0$ at $t = 0$.

As a result Equation (S1) will be transformed into Equation (S2):

$$\frac{\tau d^2 \varphi_i}{(dt)^2} + \frac{d\varphi_i}{dt} = \Lambda_{0i} - a \sum_{j=1}^n \sin \varphi_j - b \sin \varphi_i. \quad (S2)$$

The results of computations based on Equation (S2) for $n = 15$, $\omega_0 = 1$, $\omega_i = 2 - 0.1i$, $i = 0, \dots, 14$ and different parameters a , b , τ are presented in Figure S1. The same Equation (S2) at different a , b , τ is able to explain different degree of synchronization (RE) and desynchronization (RS), depending of parameter values.

SUPPLEMENT 2

S2.1. Habituation and sensitization

Habituation is defined as a behavioral response decrement that results from repeated stimulation and that does not involve sensory adaptation/sensory fatigue or motor fatigue. RS and habituation are two analogous concepts but refer to neural and behavioral levels, correspondingly. The habituation may be described as the ability to ignore familiar, predictable and inconsequential information. Although some researches use terms “repetition suppression” synonymously with the term “habituation”, RS alone is not sufficient to explain behavior habituation and vice versa.

The neural adaptation is the neural correlate of habituation [243] and constitutes habituation [244], but habituation has been distinguished from sensory adaptation or motor fatigue by the process of dishabituation [245].

According to Rankin et al. [245], the habituation after collapsing and correcting many experimental results has the following characteristics.

Decreased response. Repeated application of a stimulus results in a progressive decrease in some parameter of a response to an asymptotic level. A response may show facilitation prior to decrementing because of (or presumably derived from) a simultaneous process of sensitization.

Spontaneous recovery. If the stimulus is withheld after response decrement, the response recovers at least partially over the observation time.

Potentiation of habituation. After multiple series of stimulus repetitions and spontaneous recoveries the response decrement becomes successively more rapid and/or more pronounced.

Stimulus rate effect. Other things being equal, more frequent stimulation results in more rapid and/or more pronounced response decrement, and more rapid spontaneous recovery.

Intensity dependence. Within a stimulus modality, the less intense the stimulus, the more rapid and/or more pronounced the behavioral response decrement.

Below zero effect. The effects of repeated stimulation may continue to accumulate even after the response has reached an asymptotic level. This effect of stimulation beyond asymptotic levels can alter subsequent behavior, for example, by delaying the onset of spontaneous recovery.

Stimulus specificity. Within the same stimulus modality, the response decrement shows some stimulus specificity.

Dishabituation. Presentation of a different stimulus results in an increase of the decremented response to the original stimulus.

Habituation of dishabituation. Upon repeated application of the dishabituating stimulus, the amount of dishabituation produced decreases.

Long-term habituation. Some stimulus repetition protocols may result in the properties of the response decrement that last hours, days or weeks.

The habituation is frequently considered as an inherent aspect of physiological novelty detection mechanism [43, 59, 197, 246–253], showing also dishabituation with any changing stimulus parameter [197, 246]. In some conditions habituation can be reversed into priming or vice versa [254].

McDiarmid et al. [255] presented an extensive review of reported abnormalities in habituation (mostly reduced) in a variety of neuropsychiatric disorders: Autism Spectrum Disorder, Fragile X syndrome, Schizophrenia, Parkinson's disease, Huntington's disease, Attention Deficit Hyperactivity Disorder, Tourette's syndrome, and Migraine. Decreased habituation often correlates with symptom severity across underlying pathology of disorder.

Theories and models. The mechanism of behavioral habituation has not been identified [245, 256], but two following theories have been relatively successful and are prominent today. Stimulus-model comparator theory [257, 258] states that as the same stimulus is repeated, the stimulus-model develops and exerts increasing inhibition on the arousal system,

thus yielding habituation. If a new or altered stimulus occurs which does not match the stimulus model, then the inhibition is released and response strength recovers.

Dual-process theory [259] assumes that two independent processes take part in behavioral outcome: one decremental and one incremental that interact in the stimulus-response (S-R) pathways resulting habituation or sensitization, depending on their relative strength. Both theories are strongly supported by the experimental evidence – the former from hippocampal studies [197], the latter from invertebrate preparations [245, 256]. Both theories compliment each other in that single-trace and dual-trace mechanisms co-exist in higher and lower regions of nervous system and awaiting integration in unified theory. The hippocampal studies suggest a single unified system mechanism [112, 197, 260], while the dual-trace theory admits a multitude of different mechanisms as the cause of habituation [256] with the hope in future to extend the dual-trace hypotheses “to identify the mediatory mechanisms for habituation and determine when, where and how these mechanisms are activated” [245].

S2.2. Negative priming

Negative priming (NP) refers to the finding that people’s responses to probe target previously presented as prime destructor are usually slower and more error prone than to unrepeatd stimuli. NP was originally developed to measure attentional selection but in fact more than one process contribute to it [261]. Below we list only some NP effects that are analogous to that of RS.

a) NP reliably obtained with repeated items, not with novel items [262]; cf. [21]. A novel stimulus leads to positive priming causing faster than usual reactions [263, 264].

b) NP occurred as a function of target repetition: priming was initially positive and becomes negative as the number of repetitions increased [264]; cf. [119].

c) NP effects are multimodal: they can be observed in visual, auditory, tactile and olfactory, and bilingual stimuli [261].

d) In typical NP experiments, each probe target is accompanied by a destructor and without probe destructor NP diminishes. But it increases by increasing perceptual or conceptual difficulty [265].

e) NP is modulated by attention: the ignored repetition causes NP while attended repetition results in positive priming [266]. Similarly, NP under high control load was eliminated and could even be reversed to positive priming [267].

f) The stimulus-response binding contributes to NP effects [268]; cf. [269].

“The complexity of the NP phenomenon has not decreased despite several decades of intensive research... it is important to integrate NP research into larger cognitive theories or architectures” [261].

S2.3. Inhibition of return

Inhibition of return (IOR) refers to the relative suppression of processing of (detection of, orienting towards, responding to) stimuli that had recently been the focus of attention. By suppressing orienting toward previously inspected locations and objects IOR encourages orienting toward novelty. Many characteristics of IOR are similar to that of RS.

(a) IOR appearing approximately 250 msec following the presentation of the single cue [270] and dissipating within 3 sec [271]. Responses are faster when SOA is shorter than 250 msec [272].

(b) Like the RS property of stimulus generalization, the concept of IOR has been extended to include nonspatial dimensions [273, 274].

(c) Spatial and nonspatial IOR have both shared domain-specific neural mechanism [275].

(d) Like RS, the greater IOR occurs with increasing feature overlap of repeated stimuli [276].

(e) Different electrophysiological components have been associated with behavioral facilitation and IOR. There is no single marker of facilitation or IOR [277].

(f) Like RS, the IOR was observed with neutral and happy face targets, but disappeared with angry face targets [278]; cf. [84].

(g) Like RS, the IOR is modulated by attention [279, 280], but can be observed in the absence of attention [279] and even can be triggered by perceptual but not attentional processing [281].

Sensory adaptation may be the cause of at least some part of IOR effects [240]. Many findings suggest that habituation may account for the IOR effects as location-based manifestation of habituation [251]. It is argued that there is no process specifically designed to inhibit returns [276].

S2.4. Whole brain segregation and integration

The brain is able to separate the information into distinct modules which perform special local computations and integrate them into a meaningful internal representation. Segregation as partitioning between functional networks formally can be defined as the difference within the versus between community connections [282]. Integration refers to the ability of the brain to share and broadcast information across brain regions [283]. The integration decreases the segregation and vice versa. The balance between them is critical for healthy brain functioning [284, 285] and optimal functioning [286, 287]. How integration takes place over segregated modules remains elusive [213, 288], unclear [289, 290], or at least not fully understood [291]. Below it is shown that many properties of segregation and integration are parallel to that of repetition suppression and repetition enhancement, but also have more general meaning and importance being, e.g., a quantitative index of creativity [292] and neural correlates of consciousness [293].

Basic characteristics of segregation and integration are the following.

Baseline activity. During the baseline state the functionally connected network comprising mPFC, hippocampus, PCC, precuneus was observed to have a significant activation which deactivated with task performance and was defined as the default mode network (DMN) [294, 295]. Deactivation in DMN is associated with activation of some opposing network [296], called the task positive network (TPN) so that DMN and TPN become anticorrelated during performance of a task as well as in a resting state [296–301]. Critically, high levels of activation in DMN or TPN are associated with deactivations of opposing networks [302, 303] though in many cases these regions can be also positively correlated [304–308]. A greater initial contrast between the task state and resting state leads to better cognitive performance due to functional connections [309].

Multiple timescales. Segregation and integration processes occur over wide range of timescales. The change of brain connectivity between functional systems was observed on at least three timescales: a) from milliseconds to hundreds of milliseconds [310–313]; b) from seconds to minutes [314–316]; c) from hours to months [312, 317–319], and even to entire lifespan [320, 321]. A hierarchy of time scales reflects the anatomical distance from primary sensory cortex [322], faster dynamics in early auditory cortex, and slower dynamics in higher order brain regions [37, 316, 323–325], forming gradient time scales from early rapid sensory integration to long-term representations in the high level cortex [326]. Ultra slow timescale reflects the transient formation and dissociation of multiple communities of brain regions in the resting state [327], while cognition requires rapid transient and frequency-specific reorganization of the network topology [328]. Fast local and slow global cross frequency coupling (CFC) integrates functional systems across multiple spatiotemporal scales [329].

The segregation decreases in the elderly with age [282, 330–333], learning [318], sleep deprivation [334] or during awaking from deep sleep [335], greater task demands [308], emotions and motivation [336], awareness [335, 337], episodic memory retrieval [338]. The decrease in segregation leads to misses of the target sound in 50 % of trials [339] and is

associated with neurodegeneration [330] or some forms of psychiatry illness [340, 341] and late-life depression [342].

The segregation increases before and after task performance [343] with age of youth [344], in deep sleep [335], resting motor network activity [345], simple motor task [346, 347], task automatization [318, 348], in patients with hippocampal lesions [2], sometimes is associated with superiors' cognitive ability independently of age [282] and greater training improvements in older adults in complex tasks [349]. The increase in segregation benefits to lower metabolic costs [346, 350, 351]) and to effective performance as a function of learning [318].

Integration enables faster or more accurate performance on cognitive tasks [289, 346]. It is important for a range of cognitive functions including working memory and cognitive control [346], including task performance [318, 346], episodic memory retrieval [338], and conscious awareness [328, 337, 352], but integration can also occur without awareness [353, 354] only in the case of prior conscious training of the task [355]. Consciousness may be needed for integration of novel information [291]. A key role in the integration belongs to the hippocampus [189, 197, 288, 338, 356, 357] and PFC [288, 358–361]. Also, basal ganglia and thalamus form a part of the core circuit that supports large-scale integration of functionally diverse neural signals [207, 362, 363] through hippocampal-thalamic [201], thalamo-cortical, and cortico-thalamic connectivity. However, the electro physiological mechanisms underlying memory integration remain elusive [288].

Segregation and integration cooperation occur in complex tasks [349, 364, 365], metastable balance [291, 366], context dependent soft assemblies [148] changing cognitive demands [347, 367], control functioning [368], resting state metastability [283], dynamical connectivity [369], working memory [370], cognitive reserve [285], conscious states [236, 371, 372], and when DMN cooperates with TPN [296, 304, 305, 307, 348, 373, 374]. Note nevertheless that normally DMN and TPN inhibit each other [302, 303].

Table S2

Segregation	Integration	References
dominant low modularity	dominant high modularity	[375]
reduced metastable dynamics	normal metastable dynamics	[376]
task related deactivation	task related activation	[296]
decreased executive attention	executive attention	[377, 378]
negative cross correlations between regions	positive cross correlations between regions	[362]
operation at different frequency than integration	operation at different frequency than separation	[379]
no PAC, low correlations between regions	PAC, high correlations between regions	[380]
DMN – DAN anticorrelations	DMN – DAN correlations	[304, 305, 381]
stronger anticorrelations after sleep deprivation	weaker anticorrelations after sleep deprivation	[334]
SWS	REM	[382]
complex task, decreased modularity, forgotten items	simple task, increased modularity, remembered items	[357, 383; but see 343]
task-negative network	task-positive network	[295, 296, 384, but see 373]
complex task, negative correlations between modularity and performance	simple task, positive correlations between modularity and performance	[378]
simple motor task	complex WM task	[346]
motor execution	WM	[347]
resting state	complex reasoning	[343]
partial conscious state	conscious state	[347, 371, 385, 386]
more early (200–1400 msec) in WM task	more later (800–2200 msec) in WM task	[370]

Segregation and integration competition/opposition occurs in different brain states, cognitive tasks, and the time of appearance as shown in details in Table S2.

Attentional modulation. Attention effects on both segregation and integration in flexible manner usually but not always degrading the ability to temporally segregate stimuli and facilitating the integration, depending on goal task and stimulation [387]. Attentional control systems facilitate the integration or segregation of different cognitive systems [321]. The dorsal attention network (DAN) increased large-scale functional brain connectivity with a visual node, while decreasing connectivity with motor and sensory nodes [388]. Functional hubs underlying visuo-spatial attentional state are segregated not only anatomically but also by oscillatory frequencies [389], while conscious states are segregated by [390–393]. Too much focus can actually harm performance of creative and insightful problem-solving tasks [394].

Emotional modulation. Each of the emotions such as fear, anger, disgust, sadness, and happiness were characterized by reliable correlations with regional multimodal brain activations [395, 396]. One way in which emotions and motivation processing affect brain responses is by increasing global efficiency and decreasing separation [336]. However, the feeling of well-being is connected with optimal metastability in the brain [397]. Positive mood enhances verbal and impairs spatial working memory, whereas negative mood enhances spatial and impairs verbal working memory [398]. The socioemotional reasoning suppresses analytical form of reasoning and vice versa [302].

Outstanding questions. “The mechanism by which modules interact with one another over time remain elusive” [399]. “Mechanisms for information segregation and integration are not fully understood” [291, p. 9]. “The biological mechanisms responsible for driving fluctuations between integration and segregation remain unclear” [289, p. 2]. “How integration takes place over segregated modules remains elusive” [212, p. 1]).

REFERENCES

1. Kovács G., Schweinberger S.R. Repetition suppression - An integrative view. *Cortex*. 2016. V. 80. P. 1–4.
2. Henson R.N. Repetition suppression to faces in the fusiform face area: A personal and dynamic journey. *Cortex*. 2016. V. 80. P. 174–184.
3. Korzeniewska A., Wang Y., Benz H.L., Fifer M.S., Collard M., Milsap G., Cervenka M.C., Martin A., Gotts S.J., Crone N.E. Changes in human brain dynamics during behavioral priming and repetition suppression. *Progr. Neurobiol.* 2020. V. 189. P. 101788.
4. Gotts S.J. Incremental learning of perceptual and conceptual representations and the puzzle of neural repetition suppression. *Psychon. Bull. Rev.* 2016. V. 23. P. 1055–1071.
5. Westerberg J.A., Cox M.A., Dougherty K., Maier A. Repetitive stimulation enhances V1 encoding efficiency. *J. of Vision*. 2018. V. 18. P. 966.
6. Weiner K.S., Grill-Spector K. Synchrony upon repetition: One or multiple neural mechanisms? *Cogn. Neurosci.* 2012. V. 3. P. 243–244.
7. Merzagora A., Coffey T.J., Sperling M.R., Sharan A., Litt B., Baltuch G., Jacobs J. Repeated stimuli elicit diminished high-gamma electrocorticographic responses. *Neuroimage*. 2014. V. 85. P. 844–852.
8. Vogels R. Sources of adaptation of inferior temporal cortical responses. *Cortex*. 2016. V. 80. P. 185–195.
9. Vannini P., Hedden T., Sullivan C., Sperling R.A. Differential functional response in the posteromedial cortices and hippocampus to stimulus repetition during successful memory encoding. *Hum. Brain Mapp.* 2013. V. 34. P. 1568–1578.
10. Kremers N.A., Deuker L., Kranz T.A., Oehrns C., Fell J., Axmacher N. Hippocampal control of repetition effects for associative stimuli. *Hippocampus*. 2014. V. 24. P. 892–902.
11. Brodt S., Pöhlchen D., Flanagan V.L., Glasauer S., Gais S., Schönauer M. Rapid and independent memory formation in the parietal cortex. *Proc. Natl. Acad. Sci. USA*. 2016. V. 113. P. 13251–13256.
12. Gonzalez Andino S.L., Michel C.M., Thut G., Landis T., Grave de Peralta R. Prediction of response speed by anticipatory high-frequency (gamma band) oscillations in the human brain. *Hum. Brain Mapp.* 2005. V. 24. P. 50–58.
13. Grotheer M., Kovács G. Can predictive coding explain repetition suppression? *Cortex*. 2016. V. 80. P. 113–124.
14. Lin Z., Zhong S., Grierson D. Recent advances in ethylene research. *J. Exp. Bot.* 2009. V. 60. P. 3311–3336.
15. Zago L., Fenske M.J., Aminoff E., Bar M. The rise and fall of priming: how visual exposure shapes cortical representations of objects. *Cerebr. Cortex*. 2005. V. 15. P. 1655–1665.
16. Kuravi P., Vogels R. Effect of adapter duration on repetition suppression in inferior temporal cortex. *Sci. Rep.* 2017. V. 7. Article No. 3162.
17. Lanting C.P., Briley P.M., Sumner C.J., Krumbholz K. Mechanisms of adaptation in human auditory cortex. *J. Neurophysiol.* 2013. V. 110. P. 973–983.
18. Fritsche M., Spaak E., de Lange F.P. A Bayesian and efficient observer model explains concurrent attractive and repulsive history biases in visual perception. *Elife*. 2020. V. 9. Article No. e55389.
19. Henson R.N., Rylands A., Ross E., Vuilleumier P., Rugg MD. The effect of repetition lag on electrophysiological and haemodynamic correlates of visual object priming. *Neuroimage*. 2004. V. 21. P. 1674–1689.
20. Rangarajan V., Jacques C., Knight R.T., Weiner K.S., Grill-Spector K. Diverse temporal dynamics of repetition suppression revealed by intracranial recordings in the human ventral temporal cortex. *Cerebr. Cortex*. 2020. V. 30. P. 5988–6003.

21. Henson R.N., Shallice T., Dolan R. Neuroimaging evidence for dissociable forms of repetition priming. *Science*. 2000. V. 287. P. 1269–1272.
22. Brozinsky C.J., Yonelinas A.P., Kroll N.E., Ranganath C. Lag-sensitive repetition suppression effects in the anterior parahippocampal gyrus. *Hippocampus*. 2005. V. 15. P. 557–561.
23. Suzuki A., Goh J.O., Hebrank A., Sutton B.P., Jenkins L., Flicker B.A., Park D.C. Sustained happiness? Lack of repetition suppression in right-ventral visual cortex for happy faces. *Soc. Cogn. Affect. Neurosci.* 2011. V. 6. P. 434–441.
24. Piazza M., Pinel P., Le Bihan D., Dehaene S. A magnitude code common to numerosities and number symbols in human intraparietal cortex. *Neuron*. 2007. V. 53. P. 293–305.
25. Kar K., Krekelberg B. Testing the assumptions underlying fMRI adaptation using intracortical recordings in area MT. *Cortex*. 2016. V. 80. P. 21–34.
26. Whitmire C.J., Stanley G.B. Rapid sensory adaptation redux: a circuit perspective. *Neuron*. 2016. V. 92. P. 298–315.
27. Miller E.K., Desimone R. A neural mechanism for working and recognition memory in inferior temporal cortex. *Science*. 1991. V. 254. P. 1377–1379.
28. Liu Y., Murray S.O., Jagadeesh B. Time course and stimulus dependence of repetition-induced response suppression in inferotemporal cortex. *J. Neurophysiol.* 2009. V. 101. P. 418–436.
29. Fang P.C., Stepniewska I., Kaas J.H. Ipsilateral cortical connections of motor, premotor, frontal eye, and posterior parietal fields in a prosimian primate, *Otolemur garnetti*. *J. Compar. Neurol.* 2005. V. 490. P. 305–333.
30. Costa-Faidella J., Baldeweg T., Grimm S., Escera C. Interactions between “What” and “When” in the auditory system: Temporal predictability enhances repetition suppression. *J. Neurosci.* 2011. V. 31. P. 18590–18597.
31. Dong X., Gao Y., Lv L., Bao M. Habituation of visual adaptation. *Sci Rep.* 2016. V. 7. Article No. 19152.
32. van Turennout M., Ellmore T., Martin A. Long-lasting cortical plasticity in the object naming system. *Nat. Neurosci.* 2000. V. 3. P. 1329–1334.
33. Carbon C.C., Ditye T.J. Sustained effects of adaptation on the perception of familiar faces. *Exp Psychol. Hum. Percept. Perform.* 2011. V. 37. P. 615–625.
34. Epstein R.A., Parker W.E., Feiler A.M. Two kinds of fMRI repetition suppression? Evidence for dissociable neural mechanisms. *J. Neurophysiol.* 2008. V. 99. P. 2877–2886.
35. Tartaglia E.M., Mongillo G., Brunel N. On the relationship between persistent delay activity, repetition enhancement and priming. *Front. Psychol.* 2015. V. 5. Article No. 1590.
36. Weiner K.S., Sayres R., Vinberg J., Grill-Spector K. fMRI-adaptation and category selectivity in human ventral temporal cortex: regional differences across time scales. *J. Neurophysiol.* 2010. V. 103. P. 3349–3365.
37. Murray J.D., Bernacchia A., Freedman D.J., Romo R., Wallis J.D., Cai X, Padoa-Schioppa C., Pasternak T., Seo H., Lee D., Wang X.J. A hierarchy of intrinsic timescales across primate cortex. *Nat Neurosci.* 2014. V. 17. P. 1661–1663.
38. Sawamura H., Georgieva S., Vogels R., Vanduffel W., Orban G.A. Using functional magnetic resonance imaging to assess adaptation and size invariance of shape processing by humans and monkeys. *J. Neurosci.* 2005. V. 25. P. 4294–4306.
39. Gauthier B., Eger E., Hesselmann G., Giraud A.L., Kleinschmidt A. Temporal tuning properties along the human ventral visual stream. *J. Neurosci.* 2012. V. 32. P. 14433–14441.
40. Chaudhuri R., Bernacchia A., Wang X.J. A diversity of localized timescales in network activity. *Elife*. 2014. V. 3. Article No. e01239.

41. La Camera G., Rauch A., Thurbon D., Lüscher H.R., Senn W., Fusi S. Effect of adapter duration on repetition suppression in inferior temporal cortex. *Sci. Rep.* 2006. V. 7. Article No. 3162.
42. Horner A.J., Henson R.N. Repetition suppression in occipitotemporal cortex despite negligible visual similarity: evidence for postperceptual processing? *Hum. Brain Mapp.* 2011. V. 32. P. 1519–1534.
43. Murty V.P., Ballard I.C., Macduffie K.E., Krebs R.M., Adcock R.A. Hippocampal networks habituate as novelty accumulates. *Learn. Mem.* 2013. V. 20. P. 229–235.
44. Fang F., Murray S.O., He S. Duration-dependent fMRI adaptation and distributed viewer-centered face representation in human visual cortex. *Cerebr. Cortex.* 2007. V. 17. P. 1402–1411.
45. Verhoef B.E., Kayaert G., Franko E., Vangeneugden J., Vogels R. Stimulus similarity-contingent neural adaptation can be time and cortical area dependent. *J. Neurosci.* 2008. V. 28. P. 10631–10640.
46. Li B., Xiao L., Yin H., Liu P., Huang X. Duration aftereffect depends on the duration of adaptation. *Front. Psychol.* 2017. V. 8. Article No. 491.
47. Summerfield C., Trittschuh E.H., Monti J.M., Mesulam M.M., Egner T. Neural repetition suppression reflects fulfilled perceptual expectations. *Nat. Neurosci.* 2008. V. 11. P. 1004–1006.
48. Möhring W., Newcombe N.S., Frick A. Zooming in on spatial scaling: preschool children and adults use mental transformations to scale spaces. *Dev. Psychol.* 2014. V. 50. P. 1614–1619.
49. Tal N., Amedi A. Multisensory visual-tactile object related network in humans: insights gained using a novel crossmodal adaptation approach. *Exp. Brain Res.* 2009. V. 198. P. 165–182.
50. Wang X., Guo X., Chen L., Liu Y., Goldberg M.E., Xu H. Auditory to visual cross-modal adaptation for emotion: Psychophysical and neural correlates. *Cerebr. Cortex.* 2017. V. 27. P. 1337–1346.
51. Kim H. Brain regions that show repetition suppression and enhancement: A meta-analysis of 137 neuroimaging experiments. *Hum. Brain Mapp.* 2017. V. 38. P. 1894–1913.
52. Poppenk J., McIntosh A.R., Moscovitch M. fMRI evidence of equivalent neural suppression by repetition and prior knowledge. *Neuropsychologia.* 2016. V. 90. P. 159–169.
53. Moore K.S., Yi D.J., Chun M. The effect of attention on repetition suppression and multivoxel pattern similarity. *J. Cogn. Neurosci.* 2013. V. 25. P. 1305–1314.
54. Greene C.M., Soto D. Neural repetition effects in the medial temporal lobe complex are modulated by previous encoding experience. *PLoS One.* 2012. V. 7. Article No. e40870.
55. Chouinard P.A., Morrissey B.F., Kohler S., Goodale M.A. Repetition suppression in occipital-temporal visual areas is modulated by physical rather than semantic features of objects. *Neuroimage.* 2008. V. 41. P. 130–144.
56. Yi D.J., Kelley T.A., Marois R., Chun M.M. Attentional modulation of repetition attenuation is anatomically dissociable for scenes and faces. *Brain Res.* 2006. V. 1080. P. 53–62.
57. De Baene W., Vogels R. Effects of adaptation on the stimulus selectivity of macaque inferior temporal spiking activity and local field potentials. *Cerebr. Cortex.* 2010. V. 20. P. 2145–2165.
58. Yamaguchi S., Hale L.A., D'Esposito M., Knight R.T. Rapid prefrontal-hippocampal habituation to novel events. *J. Neurosci.* 2004. V. 24. P. 5356–5363.
59. Hsu Y.F., Hämäläinen J.A., Waszak F. Repetition suppression comprises both attention-independent and attention-dependent processes. *Neuroimage.* 2014. V. 98. P. 168–175.

60. Engel A.D. and McCharty G. Repetition suppression of face-selective evoked and induced EEG recorded from human cortex. *Hum. Brain Mapp.* 2014. V. 35. P. 4155–4162.
61. Kaliukhovich D.A., Vogels R. Stimulus repetition affects both strength and synchrony of macaque inferior temporal cortical activity. *J. Neurophysiol.* 2012. V. 107. P. 3509–3527.
62. Todorovic A., van Ede F., Maris E., de Lange FP. Prior expectation mediates neural adaptation to repeated sounds in the auditory cortex: an MEG study. *J. Neurosci.* 2011. V. 31. P. 9118–9123.
63. Gruber T., Muller M. Effects of picture repetition on induced gamma band responses, evoked potentials, and phase synchrony in the human EEG. *Brain Res.* 2002. V. 13. P. 377–392.
64. Gruber T., Muller M. Oscillatory brain activity dissociates between associative stimulus content in a repetition priming task in the human EEG. *Cerebr. Cortex.* 2005. V. 15. P. 109–116.
65. Fiebach C.J., Gruber T., Supp G.G. Neuronal mechanisms of repetition priming in occipitotemporal cortex: spatiotemporal evidence from functional magnetic resonance imaging and electroencephalography. *J. Neurosci.* 2005. V. 25. P. 3414–3422.
66. McDonald C.R., Thesen T., Carlson C., Blumberg M., Girard H.M., Trongnetrpunya A., Sherfey J.S., Devinsky O., Kuzniecky R., Dolye W.K., et al. Multimodal imaging of repetition priming: Using fMRI, MEG, and intracranial EEG to reveal spatiotemporal profiles of word processing. *Neuroimage.* 2010. V. 53. P. 707–717.
67. Mellem M.S., Friedman R.B., Medvedev A.V. Gamma- and theta-band synchronization during semantic priming reflect local and long-range lexical-semantic networks. *Brain Lang.* 2013. V. 127. P. 440–451.
68. Eliades S.J., Crone N.E., Anderson W.S., Ramadoss D., Lenz F.A., Boatman-Reich D.J. Adaptation of high-gamma responses in human auditory association cortex. *Neurophysiol.* 2014. V. 112. P. 2147–2163.
69. Malinowska U., Crone N.E., Lenz F.A., Cervenka M., Boatman-Reich D. Multi-regional adaptation in human auditory association cortex. *Front. Hum Neurosci.* 2017. V. 11. P. 247.
70. Kim K., Hsieh L.T., Parvizi J., Ranganath C. Neural repetition suppression effects in the human hippocampus. *Neurobiol. Learn. Mem.* 2020. V. 173. Article No. 107269.
71. Conrad C.D., McLaughlin K.J., Harman J.S., Foltz C., Wiecezorek L., Lightner E., Wright R.L. Chronic glucocorticoids increase hippocampal vulnerability to neurotoxicity under conditions that produce ca3 dendritic retraction but fail to impair spatial recognition memory. *J. Neurosci.* 2007. V. 27. P. 8278–8285.
72. Wang Y., Iliescu B.F., Ma J., Josić K., Dragoi V. Adaptive changes in neuronal synchronization in macaque V4. *J. Neurosci.* 2011. V. 31. P. 13204–13213.
73. Brunet N.M., Bosman C.A., Vinck M., Roberts M., Oostenveld R., Desimone R., De Weerd P., Fries P. Stimulus repetition modulates gamma-band Synchronization in primate visual cortex. *Proc. Natl. Acad. Sci USA.* 2014. V. 111. P. 3626–3631.
74. Hansen B.J., Dragoi V. Adaptation-induced synchronization in laminar cortical circuits. *Proc. Natl. Acad. Sci USA.* 2010. V. 108. P. 10720–10725.
75. Gilbert S.J., Henson R.N., Simons J.S. The scale of functional specialization within human prefrontal cortex. *J. Neurosci.* 2010. V. 30. P. 1233–1237.
76. Freunberger R., Klimesch W., Sauseng P., Griesmayr B., Höller Y., Pecherstorfer T., Hanslmayr S. Gamma oscillatory activity in a visual discrimination task. *Brain Res. Bull.* 2007. V. 71. P. 593–600.
77. Gotts S.J., Chow C.C., Martin A. Repetition priming and repetition suppression: A case for enhanced efficiency through neural synchronization. *Cogn. Neurosci.* 2012. V. 3. P. 227–237.

78. Hinman J.R., Penley S.C., Long L.L., Escabí M.A., Chrobak J.J. Septotemporal variation in dynamics of theta: speed and habituation. *J. Neurophysiol.* 2011. V. 105. P. 2675–2686.
79. Salisbury D.F., Taylor G. Semantic priming increases left hemisphere theta power and intertrial phase synchrony. *Psychophysiology.* 2012. V. 49. P. 305–311.
80. Graetz C., Ostermann F., Woeste S., Sälzer S., Dörfer C.E., Schwendicke F. Long-term survival and maintenance efforts of splinted teeth in periodontitis patients. *J. Dent.* 2019. V. 80. P. 49–54.
81. Park H.D., Correia S., Ducorps A., Tallon-Baudry C. Spontaneous fluctuations in neural responses to heartbeats predict visual detection. *Nat. Neurosci.* 2014. V. 17. P. 612–618.
82. Tsunada J., Baker A.E., Christison-Lagay K.L., Davis S.J., Cohen Y.E. Modulation of cross-frequency coupling by novel and repeated stimuli in the primate ventrolateral prefrontal cortex. *Front. Psychol.* 2011. V. 2. Article No. 217.
83. Gerlicher A.M., van Loon A.M., Scholte H.S., Lamme V.A., van der Leij A.R. Emotional facial expressions reduce neural adaptation to face identity. *Soc. Cogn. Affect. Neurosci.* 2014. V. 9. P. 610–614.
84. Rotshtein P., Malach R., Hadar U., Graif M., Hendler T. Feeling or features: different sensitivity to emotion in high-order visual cortex and amygdala. *Neuron.* 2001. V. 32. P. 747–757.
85. Ishai A., Pessoa L., Bikle P.C., Ungerleider L.G. Repetition suppression of faces is modulated by emotion. *Proc. Natl. Acad. Sci. USA.* 2004. V. 101. P. 9827–9832.
86. Ethofer T., Van De Ville D., Scherer K., Vuilleumier P. Decoding of emotional information in voice-sensitive cortices. *Curr. Biol.* 2009. V. 19. P. 1028–1033.
87. Ferrari V., Codispoti M., Bradley M.M. Repetition and ERPs during emotional scene processing: A selective review. *Int. J. Psychophysiol.* 2017. V. 111. P. 170–177.
88. Holt D.J., Weiss A.P., Rauch S.L., Wright C.I., Zalesak M., Goff D.C., Ditman T., Welsh R.C., Heckers S. Comparative Study. *Biol Psychiatry.* 2005. V. 57. P. 1011–1019.
89. Trapp S., Kotz S.A. Predicting affective information - An evaluation of repetition suppression effect. *Front. Psychol.* 2016. V. 7. Article No. 1365.
90. Shapcott K.A., Schmiedt J.T., Kouroupaki K., Kienitz R., Lazar A., Singer W., Schmid M.C. Reward-related suppression of neural activity in macaque visual area V4. *Cerebr. Cortex.* 2020. V. 30. P. 4871–4881.
91. Borst A., Flanagan V.L., Sompolinsky H. Adaptation without parameter change: Dynamic gain control in motion detection. *Proc. Natl. Acad. Sci. USA.* 2005. V. 102. P. 6172–6176.
92. Neri P. Fast-scale adaptive changes of directional tuning in fly tangential cells are explained by a static nonlinearity. *J. Exp. Biol.* 2007. V. 210. P. 3199–3208.
93. Fabbrini F., Van den Haute C., De Vitis M., Baekelandt V., Vanduffel W., Vogels R. Probing the mechanisms of repetition suppression in inferior temporal cortex with optogenetics. *Curr. Biol.* 2019. V. 29. P. 1988–1998.
94. Shew W., Clawson W., Pobs J., Karimipanih Y., Wright N.C., Wessel R. Adaptation to sensory input tunes visual cortex to criticality. *Nature Phys.* 2015. V. 11. P. 659–663.
95. Clawson W.P., Wright N.C., Wessel R., Shew W.L. Adaptation towards scale-free dynamics improves cortical stimulus discrimination at the cost of reduced detection. *PLoS Comput. Biol.* 2017. V. 13. Article No. e1005574
96. Wright N.C., Hoseini M.S., Wessel R. Adaptation modulates correlated subthreshold response variability in visual cortex. *J. Neurophysiol.* 2017. V. 118. P. 1257–1269.
97. Spigler G., Wilson S.P. Familiarization: A theory of repetition suppression predicts interference between overlapping cortical representations. *PLoS One.* 2017. V. 12. Article No. e0179306.

98. Aukstulewicz R., Friston K. Repetition suppression and its contextual determinants in predictive coding. *Cortex*. 2016. V. 80. P. 125–140
99. Larsson J., Smith A.T. fMRI repetition suppression: neuronal adaptation or stimulus expectation? *Cerebr. Cortex*. 2012. V. 22. P. 567–576.
100. Feuerriegel D., Churches O., Coussens S., Keage H.A.D. Evidence for spatiotemporally distinct effects of image repetition and perceptual expectations as measured by event-related potentials. *Neuroimage*. 2018. V. 169. P. 94–105.
101. Grotheer M., Kovacs G. Repetition probability effects depend on prior experiences. *J. Neurosci.* 2014. V. 34. P. 6640–6646.
102. Grotheer M., Kovacs G. The relationship between stimulus repetitions and fulfilled expectations. *Neuropsychologia*. 2015. V. 67. P. 175–182.
103. Kovács G., Kaiser D.A., Kaliukhovich Z., Vidnyánszky R., Vogels. Repetition probability does not affect fMRI repetition suppression for objects. *J. Neurosci.* 2013. V. 33. P. 9805–9812
104. Todorovic A., de Lange F.P. Repetition suppression and expectation suppression are dissociable in time in early auditory evoked fields. *J. Neurosci.* 2012. V. 32. P. 13389–13395.
105. Pajani A., Kouider S., Roux P., de Gardelle V. Unsuppressible repetition suppression and exemplar-specific expectation suppression in the fusiform face area. *Sci. Rep.* 2017. V. 7. Article No. 160.
106. Symonds R.M., Lee W.W., Kohn A., Schwartz O., Witkowski S., Sussman E.S. Distinguishing neural adaptation and predictive coding hypotheses in auditory change detection. *Brain Topogr.* 2017. V. 30. P. 136–148.
107. Henson R.N., Campbell K.L., Davis S.W., Taylor J.R., Emery T., Erzincliglu S., CamCAN, Kievit R.A. Multiple determinants of lifespan memory differences. *Sci. Rep.* 2016. V. 7. Article No. 32527.
108. Quiroga M., Morris A.P., Krekelberg B. Adaptation without plasticity. *Cell Rep.* 2016. V. 17. P. 58–68.
109. Johnson S., Marro J., Torres J.J. Robust short-term memory without synaptic learning. *PLoS One*. 2013. V. 8. Article No. e50276.
110. Kryukov V.I. Short-term memory as a metastable state. I. Master equation approach. In: *Cybernetics and system research*, v. 2, ed. R.Trapp, Elsevier. 1984. P. 261–265.
111. Kryukov V.I., Borisyuk R.M., Borisyuk G.N., Kirillov A.B., Kovalenko Y.E.I. Metastable and unstable states in the brain. In: *Nonlinear Science: Theory and Applications*. Ed. Dobrushin R.L., Kryukov V.I. and Toom A.L. Manchester-New York: Manchester University Press, 1990. P. 225–358.
112. de Gardelle V., Waszczuk M., Egner T., Summerfield C. Concurrent repetition enhancement and suppression responses in extrastriate visual cortex. *Cerebr. Cortex*. 2013. V. 23. P. 2235–2244.
113. Yassa M.A., Stark C.E. Multiple signals of recognition memory in the medial temporal lobe. *Hippocampus*. 2008. V. 18. P. 945–954.
114. Wissig S.C., Kohn A. The influence of surround suppression on adaptation effects in primary visual cortex. *J. Neurophysiol.* 2012. V. 107. P. 3370–3384.
115. Barraclough N.E., Page S.A., Keefe B.D. Visual adaptation enhances action sound discrimination. *Atten. Percept. Psychophys.* 2017. V. 79. P. 320–332.
116. Henson R.N., Greve A., Cooper E., Gregori M., Simons J.S., Geerligs L., Erzincliglu S., Kapur N., Browne G. The effects of hippocampal lesions on MRI measures of structural and functional connectivity. *Hippocampus*. 2016. V. 26. P. 1447–1463.
117. Turk-Browne N.B., Yi D.J., Leber A.B., Chun MM. Visual quality determines the direction of neural repetition effects. *Cerebr. Cortex*. 2007. V. 17. P. 425–433.
118. Muller N.G., Strumpf H., Scholz M., Baier B., Melloni L. Repetition suppression versus enhancement-it's quantity that matters. *Cerebr. Cortex*. 2013. V. 23. P. 315–322.

119. Bao M., Engel S.A. Distinct mechanism for long-term contrast adaptation. *Proc. Natl. Acad. Sci. USA*. 2012. V. 109. P. 5898–5903.
120. Stoelzel C.R., Huff J.M., Bereshpolova Y., Zhuang J., Hei X., Alonso J.M., Swadlow H.A. Hour-long adaptation in the awake early visual system. *J. Neurophysiol.* 2015. V. 114. P. 1172–1182.
121. Ghisovan N., Nemri A., Shumikhina S., Molotchnikoff S. Synchrony between orientation-selective neurons is modulated during adaptation-induced plasticity in cat visual cortex. *BMC Neurosci.* 2008. V. 9. Article No. 60.
122. Soldan A., Habeck C., Gazes Y., Stern Y. Neural mechanisms of repetition priming of familiar and globally unfamiliar visual objects. *Brain Res.* 2010. V. 1343. P. 122–134.
123. Liu T.T., Nalci A., Falahpour M. The global signal in fMRI: Nuisance or Information? *Neuroimage.* 2017. V. 150. P. 213–229.
124. Priebe N.J., Lampl I., Ferster D. Mechanisms of direction selectivity in cat primary visual cortex as revealed by visual adaptation. *J. Neurophysiol.* 2010. V. 104. P. 2615–2623.
125. James T.W., Gauthier I. Repetition-induced changes in BOLD response reflect accumulation of neural activity. *Hum. Brain Mapp.* 2006. V. 27. P. 37–46.
126. Gilmore A.W., Nelson S.M., Laumann T.O., Gordon E.M., Berg J.J., Greene D.J., Gratton C., Nguyen A.L., Ortega M., Hoyt C.R., Coalson R.S., Schlaggar B.L., Petersen S.E., Dosenbach N.U.F., McDermott K.B. High-fidelity mapping of repetition-related changes in the parietal memory network. *Neuroimage.* 2019. V. 199. P. 427–439.
127. Ghisovan N., Nemri A., Shumikhina S., Molotchnikoff S. Long adaptation reveals mostly attractive shifts of orientation tuning in cat primary visual cortex. *Neuroscience.* 2009. V. 164. P. 1274–1283.
128. Barron H.C., Garvert M.M., Behrens T.E. Repetition suppression: a means to index neural representations using BOLD? *Philos Trans R Soc Lond B Biol Sci.* 2016. V. 371. Article No. 20150355.
129. Barron H.C., Vogels T.P., Behrens T.E., Ramaswami M. Inhibitory engrams in perception and memory. *Proc. Natl. Acad. Sci. USA.* 2017. V. 114. P. 6666–6674.
130. Zhao X., Wang C., Liu Q., Xiao X., Jiang T., Chen C., Xue G. Neural mechanisms of the spacing effect in episodic memory: A parallel EEG and fMRI study. *Cortex.* 2015. V. 69. P. 76–92.
131. Xue G., Mei L., Chen C., Lu Z.L., Poldrack R.A., Dong Q. Facilitating memory for novel characters by reducing neural repetition suppression in the left fusiform cortex. *PLoS One.* 2010. V. 5. Article No. e13204.
132. Xue G., Mei L., Chen C., Lu Z.L., Poldrack R., Dong Q. Spaced learning enhances subsequent recognition memory by reducing neural repetition suppression. *J. Cogn. Neurosci.* 2011. V. 23. P. 1624–1633.
133. Lafontaine M.P., Théoret H., Gosselin F., Lippé S. Transcranial direct current stimulation of the dorsolateral prefrontal cortex modulates repetition suppression to unfamiliar faces: an ERP study. *PLoS One.* 2013. V. 8. Article No. e81721.
134. Reagh Z.M., Yassa M.A. Repetition strengthens target recognition but impairs similar lure discrimination: evidence for trace competition. *Learn. Mem.* 2014. V. 21. P. 342–346.
135. Miyoshi K., Minamoto T., Ashida H. Relationships between priming and subsequent recognition memory. *Springerplus.* 2014. V. 3. Article No. 546.
136. Geyer T., Baumgartner F., Müller H.J., Pollmann S. Medial temporal lobe-dependent repetition suppression and enhancement due to implicit vs. explicit processing of individual repeated search displays. *Front. Hum. Neurosci.* 2012. V. 6. Article No. 272.
137. Recasens M., Leung S., Grimm S., Nowak R., Escera C. Repetition suppression and repetition enhancement underlie auditory memory-trace formation in the human brain: an MEG study. *Neuroimage.* 2015. V. 108. P. 75–86.

138. Patterson C.A., Wissig S.C., Kohn A. Adaptation improves performance on a visual search task. *J. of Vision*. 2013. V. 13. Article No. 6.
139. McMahon D.B. All in the timing: Priming, repetition suppression, and synchrony. *Cogn. Neurosci.* 2012. V. 3. P. 244–246.
140. Miyoshi K., Kimura Y., Ashida H. Longer prime presentation decreases picture-word cross-domain priming. *Front. Psychol.* 2015. V. 21. Article No. 1040.
141. Ghuman A.S., Bar M., Dobbins I.G., Schnyer D.M. The effects of priming on frontal-temporal communication. *Proc. Natl. Acad. Sci. USA*. 2008. V. 105. P. 8405–2409.
142. Hawco C., Lepage M. Overlapping patterns of neural activity for different forms of novelty in fMRI. *Front. Hum. Neurosci.* 2014. V. 8. Article No. 699.
143. Berry C.J., Kessels R.P., Wester A.J., Shanks D.R. A single-system model predicts recognition memory and repetition priming in amnesia. *J. Neuroscience*. 2014. V. 34. P. 10963–10974.
144. Grill-Spector K., Henson R., Martin A. Repetition and the brain: neural models of stimulus-specific effects. *Trends Cogn. Sci.* 2006. V. 10. P. 14–23.
145. Patterson C.A., Wissig S.C., Kohn A. Adaptation disrupts motion integration in the primate dorsal stream. *Neuron*. 2014. V. 81. P. 674–686.
146. Kaliukhovich D.A., Vogels R. Decoding of repeated objects from local field potentials in macaque inferior temporal cortex. *PLoS One*. 2013. V. 8. Article No. e74665.
147. Wiltshire T.J., Euler M.J., McKinney T.L., Butner J.E. Changes in dimensionality and fractal scaling suggest soft-assembled dynamics in human EEG. *Front. Physiol.* 2017. V. 8. Article No. 633.
148. Segaert K., Weber K., de Lange F.P., Petersson K.M., Hagoort P. The suppression of repetition enhancement: a review of fMRI studies. *Neuropsychologia*. 2013. V. 51. P. 59–66.
149. Felsen G., Shen Y.S., Yao H., Spor G., Li C., Dan Y. Dynamic modification of cortical orientation tuning mediated by recurrent connections. *Neuron*. 2002. V. 36.
150. Kovacs G., Zimmer M., Bankó E., Harza I., Antal A., Vidnyánszky Z. Electrophysiological correlates of visual adaptation to faces and body parts in humans. *Cerebr. Cortex*. 2006. V. 16. P. 742–753.
151. Webster M.A., Kaping D., Mizokami Y., Duhamel P. Adaptation to natural facial categories. *Nature*. 2004. V. 428. P. 557–561.
152. Bouchard M., Gillet P.C., Shumikhina S., Molotchnikoff S. Adaptation changes the spatial frequency tuning of adult cat visual cortex neurons. *Exp. Brain Res.* 2008. V. 188. P. 289–303.
153. Marshansky S., Shumikhina S., Molotchnikoff S. Repetitive adaptation induces plasticity of spatial frequency tuning in cat primary visual cortex. *Neuroscience*. 2011. V. 172. P. 355–365.
154. McMahon D.B., Olson C.R. Repetition suppression in monkey inferotemporal cortex: relation to behavioral priming. *J. Neurophysiol.* 2007. V. 97. P. 3532–3543.
155. Ward E.V., Berry C.J., Shanks D.R. Age effects on explicit and implicit memory. *Front. Psychol.* 2013. V. 4. Article No. 639.
156. Ganel T., Gonzalez C.L., Valyear K.F., Culham J.C., Goodale M.A., Köhler S. The relationship between fMRI adaptation and repetition priming. *Neuroimage*. 2006. V. 32. P. 1432–1440.
157. Bunzeck N., Thiel C. Neurochemical modulation of repetition suppression and novelty signals in the human brain. *Cortex*. 2016. V. 80. P. 161–173.
158. Kaiser D., Walther C., Schweinberger S.R., Kovács G. Dissociating the neural bases of repetition-priming and adaptation in the human brain for faces. *J. Neurophysiol.* 2013. V. 110. P. 2727–2738.
159. Schacter D.L., Addis D.R., Buckner R.L. Remembering the past to imagine the future: the prospective brain. *Nat. Rev. Neurosci.* 2007. V. 8. P. 657–661.

160. Walther C., Schweinberger S.R., Kaiser D., Kovács G. Neural correlates of priming and adaptation in familiar face perception. *Cortex*. 2013. V. 49. P. 1963–1977.
161. Gotts S.J., Milleville S.C., Martin A. Enhanced inter-regional coupling of neural responses and repetition suppression provide separate contributions to long-term behavioral priming. *Commun. Biol.* 2021. V. 4. Article No. 487.
162. Henson R.N. Neuroimaging studies of priming. *Prog. Neurobiol.* 2003. V. 70. P. 53–81
163. Ferrari V., Bradley M.M., Codispoti M., Lang P.J. Massed and distributed repetition of natural scenes: Brain potentials and oscillatory activity. *Psychophysiology*. 2015. V. 52. P. 865–872.
164. Heusser A.C., Awipi T., Davachi L. The ups and downs of repetition: modulation of the perirhinal cortex by conceptual repetition predicts priming and long-term memory. *Neuropsychologia*. 2013. V. 51. P. 2333–2343.
165. Wang Y., Romani S., Lustig B., Leonardo A., Pastalkova E. Theta sequences are essential for internally generated hippocampal firing fields. *Nat. Neurosci.* 2015. V. 18. P. 282–288.
166. Park J.L., Donaldson D.I. Investigating the relationship between implicit and explicit memory: Evidence that masked repetition priming speeds the onset of recollection. *Neuroimage*. 2016. V. 139. P. 8–16.
167. Voss J.L., Reber P.J., Mesulam M.M., Parrish T.B., Paller K.A. Familiarity and conceptual priming engage distinct cortical networks. *Cerebr. Cortex*. 2008. V. 18. P. 1712–1719.
168. Voss J.L., Gonsalves B.D. Time to go our separate ways: opposite effects of study duration on priming and recognition reveal distinct neural substrates. *Front Hum Neurosci.* 2010. V. 4. Article No. 227.
169. Horner A.J., Henson R.N. Priming, response learning and repetition suppression. *Neuropsychologia*. 2008. V. 46. P. 1979–1991.
170. Gotts S.J., Milleville S.C., Martin A. Object identification leads to a conceptual broadening of object representations in lateral prefrontal cortex. *Neuropsychologia*. 2015. V. 76. P. 62–78.
171. Carandini M., Heeger D.J. Normalization as a canonical neural computation. *Nat. Rev. Neurosci.* 2011. V. 13. № 1. P. 51–62.
172. Reynolds J.H., Heeger D.J. The normalization model of attention. *Neuron*. 2009. V. 61. P. 168–185.
173. Lee J., Maunsell J.H. A normalization model of attentional modulation of single unit responses. *PLoS One*. 2009. V. 4. Article No. e4651.
174. Ni A.M., Ray S., Maunsell J.H. Tuned normalization explains the size of attention modulations. *Neuron*. 2012. V. 73. P. 803–813.
175. Montijn J.S., Klink P.C., van Wezel R.J. Divisive normalization and neuronal oscillations in a single hierarchical framework of selective visual attention. *Front. Neur. Circuits*. 2012. V. 6. P. 22.
176. Ray S., Ni A.M., Maunsell J.H. Strength of gamma rhythm depends on normalization. *PLoS Biol.* 2013. V. 11. Article No. e1001477.
177. Sanayei M., Herrero J.L., Distler C., Thiele A. Attention and normalization circuits in macaque V1. *Eur. J. Neurosci.* 2015. V. 41. P. 949–964.
178. Westrick Z.M., Heeger D.J., Landy M.S. Pattern adaptation and normalization reweighting. *J. Neurosci.* 2016. V. 36. P. 9805–9816.
179. Snow M., Coen-Cagli R., Schwartz O. Adaptation in the visual cortex: a case for probing neuronal populations with natural stimuli. *F1000Res*. 2017. V. 6. Article No. 1246.
180. Sinz F.H., Bethge M. What is the limit of redundancy reduction with divisive normalization? *Neural Comput.* 2013. V. 25. P. 2809–2814.
181. Hegumen Theophan (V.I. Kryukov). A potential solution to the “difficult problem” of consciousness. *Journal of Higher Nervous Activity*. 2020. V. 70. P. 434–467 (in Russ.).

182. Kryukov V.I. (Igumen Feofan). A model of attention and memory based on the principle of the dominant and the comparator function of the hippocampus. *Neurosci. Behav. Physiol.* 2005. V. 35. P. 235–252.
183. Sanchez-Alavez M., Robledo P., Wills D.N., Havstad J., Ehlers C.L. Cholinergic modulation of event-related oscillations (ERO). *Brain Res.* 2014. V. 1559. P. 11–25.
184. Amaral D.G., Witter M. Hippocampal formation. In: *The rat nervous system*. Ed. Paxinos G. New York: Academic Press, 2004. P. 443–493.
185. Hangya B., Borhegyi Z., Szilágyi N., Freund T.F., Varga V. GABAergic neurons of the medial septum lead the hippocampal network during theta activity. *J. Neurosci.* 2009. V. 29. P. 8094–8102.
186. Kang D., Ding M., Topchiiy I., Kocsis B. Reciprocal interactions between medial septum and hippocampus in theta generation: granger causality decomposition of mixed spike-field recordings. *Front. Neuroanat.* 2017. V. 11. Article No. 120.
187. Long L.L., Bunce J.G., Chrobak J.J. Theta variation and spatiotemporal scaling along the septotemporal axis of the hippocampus. *Front. Syst. Neurosci.* 2015. V. 9. Article No. 37.
188. Tsanov M. Septo-hippocampal signal processing: breaking the code. *Progr. Brain Res.* 2015. V. 219. P. 103–120.
189. Schedlbauer A.M., Copara M.S., Watrous A.J., Ekstrom A.D. Multiple interacting brain areas underlie successful spatiotemporal memory retrieval in humans. *Sci. Rep.* 2014. V. 4. Article No. 6431
190. Hunsaker M.R., Mooy G.G., Swift J.S., Kesner R.P. Dissociations of the medial and lateral perforant path projections into dorsal DG, CA3, and CA1 for spatial and nonspatial (visual object) information processing. *Behav. Neurosci.* 2007. V. 121. P. 742–750
191. Lee I., Hunsaker M.R., Kesner R.P. The role of hippocampal subregions in detecting spatial novelty. *Behav. Neurosci.* 2005. V. 119. P. 145–153.
192. Hasselmo M.E. What is the function of hippocampal theta rhythm? —Linking behavioral data to phasic properties of field potential and unit recording data. *Hippocampus.* 2005. V. 15. P. 936–949.
193. Forcato C., Bavassi L., De Pino G., Fernández R.S., Villarreal M.F., Pedreira M.E. Differential left hippocampal activation during retrieval with different types of reminders: An fMRI study of the reconsolidation process. *PLoS One.* 2016. V. 11. Article No. e0151381.
194. Bannerman D.M., Sprengel R., Sanderson D.J., McHugh S.B., Rawlins J.N., Monyer H., Seeburg P.H. Hippocampal synaptic plasticity, spatial memory and anxiety. *Nat. Rev. Neurosci.* 2014. V. 15. P. 181–192.
195. Taylor A.M., Bus T., Sprengel R., Seeburg P.H., Rawlins J.N., Bannerman D.M. Hippocampal NMDA receptors are important for behavioural inhibition but not for encoding associative spatial memories. *Phil. Trans. R Soc. London, B Biol. Sci.* 2013. V. 369. Article No. 20130149.
196. Vinogradova O.S. Hippocampus as comparator: role of the two input and two output systems of the hippocampus in selection and registration of information. *Hippocampus.* 2001. V. 11. P. 578–598 (in Russ.).
197. Misić B., Goñi J., Betzel R.F., Sporns O., McIntosh A.R. A network convergence zone in the hippocampus. *PLoS Comput. Biol.* 2014. V. 10. Article No. e1003982.
198. Gurguryan L., Rioux M., Sheldon S. Reduced anterior hippocampal and ventromedial prefrontal activity when repeatedly retrieving autobiographical memories. *Hippocampus.* 2021. V. 31. P. 869–880.
199. Ahn J.R., Lee H.W., Lee I. Rhythmic pruning of perceptual noise for object representation in the hippocampus and perirhinal cortex in rats. *Cell Rep.* 2019. V. 26. P. 2362–2376.

200. Zhang H., Watrous A.J., Patel A., Jacobs J. Theta and alpha oscillations are traveling waves in the human neocortex. *Neuron*. 2018. V. 98. P. 1269–1281.e4.
201. Sweeney-Reed C.M., Zaehle T., Voges J., Schmitt F.C., Buentjen L., Kopitzki K., Richardson-Klavehn A., Hinrichs H., Heinze H.J., Knight R.T., Rugg M.D. Clinical, neuropsychological, and pre-stimulus dorsomedial thalamic nucleus electrophysiological data in deep brain stimulation patients. *Data Brief*. 2016. V. 15. P. 557–561.
202. De Lima M.C., Douglas Corrêa Pereira P., Pereira Henrique E., Augusto de Oliveira M., Carvalho Paulo D, Silva de Siqueira L., Guerreiro Diniz D., Almeida Miranda D., André Damasceno de Melo M., Gyzely de Moraes Magalhães N. et al. Differential change in hippocampal radial astrocytes and neurogenesis in shorebirds with contrasting migratory routes. *Front. Neuroanat*. 2019. V. 13. Article No. 82.
203. Klein M.M., Cholvin T., Cosquer B., Salvadori A., Le Mero J., Kourouma L., Boutillier A.L., Pereira de Vasconcelos A., Cassel J.C. Ventral midline thalamus lesion prevents persistence of new (learning-triggered) hippocampal spines, delayed neocortical spinogenesis, and spatial memory durability. *Brain Struct. Funct*. 2019. V. 224. P. 1659–1676.
204. Halassa M.M., Kastner S. Thalamic functions in distributed cognitive control. *Nat. Neurosci*. 2017. V. 20. P. 1669–1679.
205. Turchi J., Chang C., Ye F.Q., Russ B.E., Yu D.K., Cortes C.R., Monosov I.E., Duyn J.H., Leopold D.A. The basal forebrain regulates global resting-state fmri fluctuations. *Neuron*. 2018. V. 97. P. 940–952.
206. Baars B.J., Franklin S., Ramsoy T.Z. Global workspace dynamics: cortical "binding and propagation" enables conscious contents. *Front. Psychol*. 2013. V. 4. Article No. 200.
207. Bell P.T., Shine J.M. Subcortical contributions to large-scale network communication. *Neurosci. Biobehav. Rev*. 2016. V. 71. P. 313–322.
208. Headley D.B., Paré D. Common oscillatory mechanisms across multiple memory systems. *NPJ Sci. Learn*. 2017. V. 2.
209. Malekmohammadi M., Elias W.J., Pouratian N. Human thalamus regulates cortical activity via spatially specific and structurally constrained phase-amplitude coupling. *Cerebr. Cortex*. 2015. V. 25. P. 1618–1628.
210. Jiang H., Bahramisharif A., van Gerven M.A., Jensen O. Measuring directionality between neuronal oscillations of different frequencies. *Neuroimage*. 2015. V. 118. P. 359–367.
211. Spaak E., Bonnefond M., Maier A., Leopold D.A., Jensen O. Layer-specific entrainment of γ -band neural activity by the α rhythm in monkey visual cortex. *Curr. Biol*. 2012. V. 22. P. 2313–2318.
212. Tang W., Liu H., Douw L., Kramer M.A., Eden U.T., Hämäläinen M.S., Stufflebeam S.M. Dynamic connectivity modulates local activity in the core regions of the default-mode network. *Proc. Natl. Acad. Sci. USA*. 2017. V. 114. P. 9713–9718.
213. Weaver K.E., Wander J.D., Ko A.L., Casimo K., Grabowski T.J., Ojemann J.G., Darvas F. Directional patterns of cross frequency phase and amplitude coupling within the resting state mimic patterns of fMRI functional connectivity. *Neuroimage*. 2016. V. 128. P. 238–251.
214. Chen J.E., Glover G.H., Greicius M.D., Chang C. Dissociated patterns of anticorrelations with dorsal and ventral default-mode networks at rest. *Hum. Brain Mapp*. 2017. V. 38. P. 2454–2465.
215. Ma Y., Ma Z., Liang Z., Neuberger T., Zhang N. Global brain signal in awake rats. *Brain Struct Funct*. 2020. V. 225. P. 227–240.
216. Gutierrez-Barragan D., Basson M., Panzeri S., Gozzi A. Ultraslow state fluctuations govern spontaneous fMRI network dynamics. 2018. *Curr. Biol*. 2019. V. 29. P. 2295–2306.

217. Draguhn A. Making room for new memories. *Neuroscience*. 2018. V. 359. P. 1461–1462.
218. Lehmann H., McNamara K.C. Repeatedly reactivated memories become more resistant to hippocampal damage. *Learn. Mem.* 2011. V. 18. P. 132–135.
219. Toropova K.A., Troshev D.V., Ivashkina O.I., Anokhin K.V. Activation of c-fos expression in the retrosplenial cortex, but not the hippocampus, accompanies the formation of an association between an installation and an unconditional stimulus and its subsequent extraction in mice. *Journal of Higher Nervous Activity*. 2018. V. 68. P. 756–770 (in Russ.).
220. Girardeau G., Benchenane K., Wiener S.I., Buzsáki G., Zugaro M.B. Selective suppression of hippocampal ripples impairs spatial memory. *Nat. Neurosci.* 2009. V. 12. P. 1222–1223.
221. Hunsaker M.R., Kesner R.P. The operation of pattern separation and pattern completion processes associated with different attributes or domains of memory. *Neurosci. Biobehav. Rev.* 2013. V. 37. P. 36–58.
222. Shema R., Kulicke R., Cowley G.S., Stein R., Root D.E., Heiman M. Synthetic lethal screening in the mammalian central nervous system identifies Gpx6 as a modulator of Huntington's disease. *Proc. Natl. Acad. Sci. USA*. 2015. V. 112. P. 268–272.
223. Makino Y., Polygalov D., Bolaños F., Benucci A., McHugh T.J. Physiological signature of memory age in the prefrontal-hippocampal circuit. *Cell Rep.* 2019. V. 29. P. 3835–3846.
224. Wirt R.A., Hyman J.M. ACC Theta improves hippocampal contextual processing during remote recall. *Cell Rep.* 2019. V. 27. P. 2313–2327.
225. Titley H.K., Brunel N., Hansel C. Toward a neurocentric view of learning. *Neuron*. 2017. V. 95. P. 19–32.
226. Attardo A., Fitzgerald J.E., Schnitzer M.J. Impermanence of dendritic spines in live adult CA1 hippocampus. *Nature*. 2015. V. 523. P. 592–596.
227. Chen H., Wang Y.J., Yang L., Hu C., Ke X.F., Fan Z.L., Sui J.F., Hu B. Predictive nature of prefrontal theta oscillation on the performance of trace conditioned eyeblink responses in guinea pigs. *Behav. Brain Res.* 2014. V. 265. P. 121–131.
228. Montijn J.S., Meijer G.T., Lansink C.S., Pennartz C.M. Population-level neural codes are robust to single-neuron variability from a multidimensional coding perspective. *Cell Reports*. 2016. V. 16. P. 2486–2498.
229. Elias G.A., Bieszczad K.M., Weinberger N.M. Learning strategy refinement reverses early sensory cortical map expansion but not behavior: Support for a theory of directed cortical substrates of learning and memory. *Neurobiol. Learn. Mem.* 2015. V. 126. P. 39–55.
230. Ryan T.J., Roy D.S., Pignatelli M., Arons A., Tonegawa S. Engram cells retain memory under retrograde amnesia. *Science*. 2015. V. 348. P. 1007–1013.
231. Poo M.M., Pignatelli M., Ryan T.J., Tonegawa S., Bonhoeffer T., Martin K.C., Rudenko A., Tsai L.H., Tsien R.W., Fishell G., et al. What is memory? The present state of the engram. *BMC Biol.* 2016. V. 14. Article No. 40.
232. Josselyn S., Tanegawa S. Memory engrams: Recalling the past and imagining the future. *Science*. 2020. V. 367. Article No. eaaw4325.
233. Sui J., Humphreys G.W. The Integrative Self: How self-reference integrates perception and memory. *Trends Cogn. Sci.* 2015. V. 19. P. 719–728.
234. Shew W.L., Plenz D. The functional benefits of criticality in the cortex. *Neuroscientist*. 2013. V. 19. P. 88–100.
235. Tagliazucchi E., Chialvo D.R., Siniatchkin M., Amico E., Brichant J.F., Bonhomme V., Noirhomme Q., Laufs H., Laureys S. Large-scale signatures of unconsciousness are consistent with a departure from critical dynamics. *J. R. Soc. Interface*. 2016. V. 13. Article No. 20151027.

236. Larsson J., Solomon S.G., Kohn A. fMRI adaptation revisited. *Cortex*. 2016. V. 80. P. 154–160.
237. Ferguson K.A., Cardin J.A. Mechanisms underlying gain modulation in the cortex. *Nat. Rev. Neurosci.* 2020. V. 21. P. 80–92.
238. Solomon S.G., Kohn A. Moving sensory adaptation beyond suppressive effects in single neurons. *Curr. Biol.* 2014. V. 24. P. 1012–1022.
239. Kaliukhovich D.A., Vogels R. Divisive normalization predicts adaptation-induced response changes in macaque inferior temporal cortex. *J. Neurosci.* 2016. V. 36. P. 6116–6128.
240. Burg M.F., Cadena S.A., Denfield G.H., Walker E.Y., Tolias A.S., Bethge M., Ecker A.S. Learning divisive normalization in primary visual cortex. *PLoS Comput. Biol.* 2021. V. 17. Article No. e1009028.
241. Kryukov V.I. Towards a unified model of pavlovian conditioning: short review of trace conditioning models. *Cogn. Neurodyn.* 2012. V. 6. P. 377–398.
242. Netser S., Zahar Y., Gutfreund Y. Stimulus-specific adaptation: can it be a neural correlate of behavioral habituation? *J. Neurosci.* 2011. V. 31. P. 17811–17820.
243. Pellegrino R., Sinding C., de Wijk R.A., Hummel T. Habituation and adaptation to odors in humans. *Physiol. Behav.* 2017. V. 177. P. 13–19.
244. Rankin C.H., Abrams T., Barry R.J., Bhatnagar S., Clayton D.F., Colombo J., Coppola G., Geyer M.A., Glanzman D.L., Marsland S., et al. Habituation revisited: an updated and revised description of the behavioral characteristics of habituation. *Neurobiol. Learn. Mem.* 2009. V. 92. P. 135–138.
245. Bragin A.G., Vinogradova O.S., Emel'ianov V.V. Influence of the dentate fascia on the sensory responses of neurons in hippocampal field CA3. *Journal of Higher Nervous Activity*. 1976. V. 26. P. 605–611 (in Russ.).
246. Vinogradova O.S., Dudaeva K.I. Comparator function of the hippocampus. *Dokl. Akad. Nauk SSSR*. 1972. V. 202. P. 486–489 (in Russ.).
247. McNaughton N. The role of the subiculum within the behavioural inhibition system. *Behavioural Brain Research*. 2006. V. 174. P. 232–250.
248. Fischer M.H., Castel A.D., Dodd M.D., Pratt J. Perceiving numbers causes spatial shifts of attention. *Nat. Neurosci.* 2003. V. 6. P. 555–556.
249. Moore D. 2003. Cortical neurons signal sound novelty. *Nat. Neurosci.* V. 6. P. 330–332.
250. Dukewich K.R. Reconceptualizing inhibition of return as habituation of the orienting response. *Psychon. Bull. Rev.* 2009. V. 16. P. 238–251.
251. Strange B.A., Dolan R.J. Adaptive anterior hippocampal responses to oddball stimuli. *Hippocampus*. 2001. V. 11. P. 690–698.
252. Kitchigina V.F. Theta oscillations and reactivity of hippocampal stratum oriens neurons. *Scientific World Journal*. 2010. V. 10. P. 30–43.
253. Faivre N., Kouider S. Increased sensory evidence reverses nonconscious priming during crowding. *J. Vis.* 2011. V. 11. Article No. 16.
254. McDiarmid T.A., Bernardos AC, Rankin CH. Habituation is altered in neuropsychiatric disorders-A comprehensive review with recommendations for experimental design and analysis. *Neurosci. Biobehav. Rev.* 2017. V. 80. P. 286–305.
255. Schmid S., Wilson D.A., Rankin C.H. Habituation mechanisms and their importance for cognitive function. *Front. Integr. Neurosci.* 2015. V. 8. Article No. 97.
256. Sokolov A.V. Specific antigens of the central nervous system. *Patol. Fiziol. Eksp. Ter.* 1960. V. 4. P. 23–26 (in Russ.).
257. Sokolov A.V. On specific antigens of the central nervous system. *Patol. Fiziol. Eksp. Ter.* 1963. V. 7. P. 79–81 (in Russ.).
258. Groves P.M., Thompson R.F. Habituation: a dual-process theory. *Psychol. Rev.* 1970. V. 77. P. 419–450.

259. Borisyuk R., Denham M., Hoppensteadt F., Kazanovich Y., Vinogradova O. Oscillatory model of novelty detection. *Network*. 2001. V. 12. P. 1–20.
260. Frings C., Schneider K.K., Fox E. The negative priming paradigm: An update and implications for selective attention. *Psychon. Bull. Rev.* 2015. V. 22. P. 1577–1597.
261. Strayer D.L., Drews F.A., Albert R.W. Negative priming and stimulus repetition: A reply to Neill and Joordens. *Percept. Psychophys.* 2002. V. 64. P. 861–865.
262. Grison S., Strayer D.L. Negative priming and perceptual fluency: more than what meets the eye. *Percept. Psychophys.* 2001. V. 63. P. 1063–1071.
263. Strayer D.L., Grison S. Negative identity priming is contingent on stimulus repetition. *J. Exp. Psychol. Hum. Percept. Perform.* 1999. V. 25. P. 24–38.
264. Frings C., Spence C. Increased perceptual and conceptual processing difficulty makes the immeasurable measurable: negative priming in the absence of probe distractors. *J. Exp. Psychol. Hum. Percept. Perform.* 2011. V. 37. P. 72–84.
265. Malley G.B., Strayer D.L. Effect of stimulus repetition on positive and negative identity priming. *Attention, Percept. & Psychophys.* 1995. V. 57. P. 657–667.
266. de Fockert J.W., Mizon G.A., D'Ubaldo M. No negative priming without cognitive control. *J. Exp. Psychol. Hum. Percept. Perform.* 2010. V. 36. P. 1333–1341.
267. Mayr S., Buchner A., Dentale S. Prime retrieval of motor responses in negative priming. *J. Exp. Psychol. Hum. Percept. Perform.* 2009. V. 35. P. 408–423.
268. Henson R.N., Eckstein D., Waszak F., Frings C., Horner A.J. Stimulus-response bindings in priming. *Trends Cogn. Sci.* 2014. V. 18. P. 376–384.
269. Klein R.M. Inhibition of return. *Trends Cogn. Sci.* 2000. V. 4. P. 138–147.
270. Samuel A.G., Kat D. Inhibition of return: A graphical meta-analysis of its time course and an empirical test of its temporal and spatial properties. *Psych. Bull. and Review.* 2003. V. 10. P. 897–906.
271. Posner M., Cohen Y. Components of visual orienting. In: *Attention and performance X: Control of language processes*, V. 32. Eds. Bouma H., Bouwhuis D.G. 1984. P. 531–556.
272. Francis L., Milliken B. Inhibition of return for the length of a line? *Percept. Psychophys.* 2003. V. 65. P. 1208–1221.
273. Hu F.K., He S., Fan Z., Lupiáñez J. Beyond the inhibition of return of attention: reduced habituation to threatening faces in schizophrenia. *Front. Psychiatry.* 2014. V. 5.
274. Zhou X., Chen Q. Neural correlates of spatial and non-spatial inhibition of return (IOR) in attentional orienting. *Neuropsychologia.* 2008. V. 46. P. 2766–2775.
275. Hu F.K., Samuel A.G., Chan A.S. Eliminating inhibition of return by changing salient nonspatial attributes in a complex environment. *J. Exp. Psychol. Gen.* 2011. V. 140. P. 35–50.
276. Martin-Arevalo E., Chica A.B., Lupianez J. No single electrophysiological marker for facilitation and inhibition of return: A review. *Behav. Brain Res.* 2016. V. 300. P. 1–10.
277. Pérez-Dueñas C., Acosta A., Lupiáñez J. Reduced habituation to angry faces: increased attentional capture as to override inhibition of return. *Psychol. Res.* 2014. V. 78. P. 196–208.
278. Satel J., Hilchey M.D., Wang Z., Story R., Klein R.M. The effects of ignored versus foveated cues upon inhibition of return: an event-related potential study. *Atten. Percept. Psychophys.* 2013. V. 75. P. 29–40.
279. Tian Y., Klein R.M., Satel J., Xu P., Yao D. Electrophysiological explorations of the cause and effect of inhibition of return in a cue-target paradigm. *Brain Topogr.* 2011. V. 24. P. 164–182.
280. Zhao Y., Heinke D. What causes IOR? Attention or perception? - Manipulating cue and target luminance in either blocked or mixed condition. *Vision Res.* 2014. V. 105. P. 37–46.

281. Chan M.Y., Park D.C., Savalia N.K., Petersen S.E., Wig G.S. Decreased segregation of brain systems across the healthy adult lifespan. *Proc. Natl. Acad. Sci. USA*. V. 2014. V. 111. P. 4997–5006.
282. Deco G., Kringelbach M.L., Jirsa V.K., Ritter P. The dynamics of resting fluctuations in the brain: metastability and its dynamical cortical core. *Sci. Rep.* 2017. V. 7. Article No. 3095.
283. Massobrio P., de Arcangelis L., Pasquale V., Jensen H.J., Plenz D. Criticality as a signature of healthy neural systems. *Front. Syst. Neurosci.* 2015. V. 9. Article No. 22.
284. Marques P., Moreira P., Magalhães R., Costa P., Santos N., Zihl J., Soares J., Sousa N. The functional connectome of cognitive reserve. *Hum. Brain Mapp.* 2016. V. 37. P. 3310–3322.
285. Deco G., Kringelbach M.L. Great expectations: using whole-brain computational connectomics for understanding neuropsychiatric disorders. *Neuron*. 2014. V. 84. P. 892–905.
286. Sporns O. Structure and function of complex brain networks. *Dialogues Clin Neurosci.* 2013. V. 15. P. 247–262.
287. Backus A.R., Schoffelen J.M., Szebényi S., Hanslmayr S., Doeller C.F. Hippocampal-prefrontal theta oscillations support memory integration. *Curr. Biol.* 2016. V. 22. P. 450–457.
288. Shine J.M., Aburn M.J., Breakspear M., Poldrack R.A. The modulation of neural gain facilitates a transition between functional segregation and integration in the brain. *Elife*. 2018. V. 7. Article No. e31130.
289. Deco G., Cabral J., Saenger V.M., Boly M., Tagliazucchi E., Laufs H., Van Someren E., Jobst B., Stevner A., Kringelbach M.L. Perturbation of whole-brain dynamics *in silico* reveals mechanistic differences between brain states. *Neuroimage*. 2018. V. 169. P. 46–56.
290. Deco G., Tononi G., Boly M., Kringelbach M.L. Rethinking segregation and integration: contributions of whole-brain modeling. *Nat. Rev. Neurosci.* 2015. V. 16. P. 430–439.
291. Kenett Y.N., Medaglia J.D., Beaty R.E., Chen Q., Betzel R.F., Thompson-Schill S.L., Qiu J. Driving the brain towards creativity and intelligence: A network control theory analysis. *Neuropsychologia*. 2018. V. 118. P. 79–90.
292. Koch C., Massimini M., Boly M., Tononi G. Neural correlates of consciousness: progress and problems. *Nat. Rev. Neurosci.* 2016. V. 17. P. 307–321.
293. Raichle M.E., MacLeod A.M., Snyder A.Z., Powers W.J., Gusnard D.A., Shulman G.L. A default mode of brain function. *Proc. Natl. Acad. Sci. USA*. 2001. V. 98. P. 676–682.
294. Raichle M.E. The brain's default mode network. *Ann. Rev. Neurosci.* 2015. V. 38. P. 433–447.
295. Fox M.D., Snyder A.Z., Vincent J.L., Corbetta M., Van Essen D.C., Raichle M. The human brain is intrinsically organized into dynamic, anticorrelated functional networks. *Proc. Natl. Acad. Sci. USA*. 2005. V. 102. P. 9673–9678.
296. Greicius M.D., Krasnow B., Boyett-Anderson J.M., Eliez S., Schatzberg A.F., Reiss A.L., Menon V. Regional analysis of hippocampal activation during memory encoding and retrieval: fMRI study. *Hippocampus*. 2003. V. 13. P. 164–174.
297. Fransson P. Spontaneous low-frequency BOLD signal fluctuations: an fMRI investigation of the resting-state default mode of brain function hypothesis. *Hum. Brain Mapp.* 2005. V. 26. P. 15–29.
298. Golland Y., Bentin S., Gelbard H., Benjamini Y., Heller R., Nir Y., Hasson U., Malach R. Extrinsic and intrinsic systems in the posterior cortex of the human brain revealed during natural sensory stimulation. *Cerebr. Cortex*. 2007. V. 17. P. 766–777.
299. Buckner R.L., Andrews-Hanna J.R., Schacter D.L. The brain's default network anatomy, function, and relevance to disease. *Ann. NY Acad. Sci.* 2008. V. 1124. P. 1–38.

300. Jack A.I., Dawson A., Begany K., Leckie R.L., Barry K., Ciccio A., Snyder A.Z. fMRI reveals reciprocal inhibition between social and physical cognitive domains. *Neuroimage*. 2012. V. 66C. P. 385–401.
301. Rochford K.C., Jack A.I., Boyatzis R.E., French S.E. Ethical leadership as a balance between opposing neural networks. *J. Business Ethics*. 2017. V. 144. P. 755–770.
302. Boyatzis R.E., Rochford K., Jack A.I. Antagonistic neural networks underlying differentiated leadership roles. *Front. Hum. Neurosci*. 2014. V. 8. P. 114.
303. Dixon M.L., Andrews-Hanna J.R., Spreng R.N., Irving Z.C., Mills C., Girn M., Christoff K. Interactions between the default network and dorsal attention network vary across default subsystems, time, and cognitive states. *Neuroimage*. 2016. V. 147. P. 632–649.
304. Fornito A., Zalesky A., Pantelis C., Bullmore E.T. Schizophrenia, neuroimaging and connectomics. *Neuroimage*. 2012. V. 62. P. 2296–2314.
305. Foster B.L., Rangarajan V., Shirer W.R., Parvizi J. Intrinsic and task-dependent coupling of neuronal population activity in human parietal cortex. *Neuron*. 2015. V. 86. P. 578–590.
306. Betzel R.F., Fukushima M., He Y., Zuo X.N., Sporns O. Dynamic fluctuations coincide with periods of high and low modularity in resting-state functional brain networks. *Neuroimage*. 2016. V. 127. P. 287–297.
307. Vatansever D., Menon D.K., Manktelow A.E., Sahakian B.J., Stamatakis E.A. Default mode dynamics for global functional integration. *J. Neurosci*. 2015. V. 35. P. 15254–15262.
308. Douw L., Wakeman D.G., Tanaka N., Liu H., Stufflebeam S.M. State-dependent variability of dynamic functional connectivity between frontoparietal and default networks relates to cognitive flexibility. *Neuroscience*. 2016. V. 339. P. 12–21.
309. Ossandón T., Jerbi K., Vidal J.R., Bayle D.J., Henaff M.A., Jung J., Minotti L., Bertrand O., Kahane P., Lachaux J.P. Transient suppression of broadband gamma power in the default-mode network is correlated with task complexity and subject performance. *J. Neurosci*. 2011. V. 31. P. 14521–14530.
310. Baker A.P., Brookes M.J., Rezek I.A., Smith S.M., Behrens T., Smith P.J., Woolrich M. Fast transient networks in spontaneous human brain activity. *Elife*. 2014. V. 3. Article No. e01867.
311. Hari R., Parkkonen L. The brain timewise: how timing shapes and supports brain function. *Philos. Trans. R Soc. London, B Biol. Sci*. 2015. V. 370. Article No. 20140170.
312. Runyan C.A., Piasini E., Panzeri S., Harvey C.D. Distinct timescales of population coding across cortex. *Nature*. 2017. V. 548. P. 92–96.
313. Linkenkaer-Hansen K., Nikouline V.V., Palva J.M., Ilmoniemi R.J. Long-range temporal correlations and scaling behavior in human brain oscillations. *J. Neurosci*. 2001. V. 21. P. 1370–1377.
314. Chang C., Glover G.H. Time-frequency dynamics of resting-state brain connectivity measured with fMRI. *NeuroImage*. 2010. V. 50. P. 81–98.
315. Hasson U., Chen J., Honey C.J. Hierarchical process memory: memory as an integral component of information processing. *Trends Cogn. Sci*. 2015. V. 19. P. 304–313.
316. Bassett D.S., Wymbs N.F., Porter M.A., Mucha P.J., Carlson J.M., Grafton S.T. Dynamic reconfiguration of human brain networks during learning. *Proc. Natl. Acad. Sci. USA*. 2011. V. 108. P. 7641–7646.
317. Bassett D.S., Yang M., Wymbs N.F., Grafton S.T. Learning-induced autonomy of sensorimotor systems. *Nat. Neurosci*. 2015. V. 18. P. 744–751.
318. Poldrack R.A., Laumann T.O., Koyejo O., Gregory B., Hover A., Chen M.Y., Gorgolewski K.J., Luci J., Joo S.J., Boyd R.L., et al. Long-term neural and

- physiological phenotyping of a single human. *Nat. Commun.* 2015. V. 6. Article No. 8885.
319. Betzel R.F., Byrge L., He Y., Goñi J., Zuo X.N., Sporns O. Changes in structural and functional connectivity among resting-state networks across the human lifespan. *Neuroimage*. 2014. V. 102. P. 345–357.
320. Gu B.M., van Rijn H., Meck W.H. Oscillatory multiplexing of neural population codes for interval timing and working memory. *Neurosci. Biobehav. Rev.* 2015. V. 48. P. 160–185.
321. Kiebel S.J., Daunizeau J., Friston K.J. Driving the brain towards creativity and intelligence: A network control theory analysis. *Neuropsychologia*. 2008. V. 118. P. 79–90.
322. Stephens G.J., Honey C.J., Hasson U. A place for time: the spatiotemporal structure of neural dynamics during natural audition. *J. Neurophysiol.* 2013. V. 110. P. 2019–2026.
323. Cocchi L., Sale M.V., Gollo L., Bell P.T., Nguyen V.T., Zalesky A., Breakspear M., Mattingley J.B. A hierarchy of timescales explains distinct effects of local inhibition of primary visual cortex and frontal eye fields. *Elife*. 2016. V. 6. Article No. e15252.
324. Chaudhuri R., Knoblauch K., Gariel M.A., Kennedy H., Wang X.J. A large-scale circuit mechanism for hierarchical dynamical processing in the primate cortex. *Neuron*. 2015. V. 88. P. 419–431.
325. Mattar M.G., Kahn D.A., Thompson-Schill S.L., Aguirre G.K. Varying timescales of stimulus integration unite neural adaptation and prototype formation. *Curr. Biol.* 2016. V. 26. P. 1669–1676.
326. Ponce-Alvarez A., He B.J., Hagmann P., Deco G. Task-driven activity reduces the cortical activity space of the brain: experiment and whole-brain modeling. *PLoS Comput. Biol.* 2015. V. 11. Article No. e1004445.
327. Bola M., Sabel B.A. Dynamic reorganization of brain functional networks during cognition. *Neuroimage*. 2015. V. 114. P. 398–413.
328. Canolty R.T., Knight R.T. The functional role of cross-frequency coupling. *Trends Cogn. Sci.* 2010. V. 14. P. 506–515.
329. Brier M.R., Thomas J.B., Ances B.M. Network dysfunction in Alzheimer's disease: refining the disconnection hypothesis. *Brain Connect.* 2014. V. 4. P. 299–311.
330. Geerligs L., Renken R.J., Saliassi E., Maurits N.M., Lorist M.M. A brain-wide study of age-related changes in functional connectivity. *Cerebr. Cortex*. 2015. V. 25. P. 1987–1999.
331. Esposito R., Cieri F., Chiacchiarretta P., Cera N., Lauriola M., Di Giannantonio M., Tartaro A., Ferretti A. Modifications in resting state functional anticorrelation between default mode network and dorsal attention network: comparison among young adults, healthy elders and mild cognitive impairment patients. *Brain Imaging Behav.* 2017. V. 12. P. 127–141.
332. Spreng R.N., Stevens W.D., Viviano J.D., Schacter D.L. Attenuated anticorrelation between the default and dorsal attention networks with aging: evidence from task and rest. *Neurobiol. Aging*. 2016. V. 45. P. 149–160.
333. Yeo B.T., Tandi J., Chee M.W. Functional connectivity during rested wakefulness predicts vulnerability to sleep deprivation. *Neuroimage*. 2015. V. 111. P. 147–158.
334. Tagliazucchi E., Behrens M., Laufs H. Sleep neuroimaging and models of consciousness. *Front. Psychol.* 2013. V. 4. P. 256.
335. Kinnison J., Padmala S., Choi J.M., Pessoa L. Network analysis reveals increased integration during emotional and motivational processing. *J. Neurosci.* 2012. V. 32. P. 8361–8372.
336. Godwin D., Barry R.L., Marois R. Breakdown of the brain's functional network modularity with awareness. *Proc. Natl. Acad. Sci. USA*. 2015. V. 112. P. 3799–3804.

337. Westphal A.J., Wang W., Rissman J. Episodic memory retrieval benefits from a less modular brain network organization. *J. Neurosci.* 2017 V. 7. P. 3523–3531.
338. Sadaghiani S., Poline J.B., Kleinschmidt A., D'Esposito M. Ongoing dynamics in large-scale functional connectivity predict perception. *Proc. Natl. Acad. Sci. USA.* 2015. V. 112. P. 8463–8468.
339. Yang G.J., Murray J.D., Wang X.J., Glahn D.C., Pearlson G.D., Repovs G., Krystal J.H., Anticevic A. Functional hierarchy underlies preferential connectivity disturbances in schizophrenia. *Proc. Natl. Acad. Sci. USA.* 2016. V. 13. P. 219–228.
340. Yu Q., Sui J., Kiehl K.A., Pearlson G., Calhoun V.D. State-related functional integration and functional segregation brain networks in schizophrenia. *Schizophr. Res.* 2013. V. 150. P. 450–458.
341. Li J.M., Bentley W.J., Snyder L.H. Functional connectivity arises from a slow rhythmic mechanism. *Proc. Natl. Acad. Sci. USA.* 2015. V. 112. P. 2527–2535.
342. Hearne L.J., Cocchi L., Zalesky A., Mattingley J.B. Reconfiguration of brain network architectures between resting-state and complexity-dependent cognitive reasoning. *J. Neurosci.* 2017. V. 37. P. 8399–8411.
343. Baum G.L., Ciric R., Roalf D.R., Betzel R.F., Moore T.M., Shinohara R.T., Kahn A.E., Vandekar S.N., Rupert P.E., Quarmley M. et al. Modular segregation of structural brain networks supports the development of executive function in youth. *Curr. Biol.* 2017. V. 27. P. 1561–1572.
344. Di X., Biswal B.B. Dynamic brain functional connectivity modulated by resting-state networks. *Brain Struct. Funct.* 2015. V. 220. P. 37–46.
345. Shine J.M., Bissett P.G., Bell P.T., Koyejo O., Balsters J.H., Gorgolewski K.J., Moodie C.A., Poldrack R.A. The dynamics of functional brain networks: integrated network states during cognitive task performance. *Neuron.* 2016. V. 92. P. 544–554.
346. Cohen J.R., D'Esposito M. The segregation and integration of distinct brain networks and their relationship to cognition. *J. Neurosci.* 2016. V. 36. P. 12083–12094.
347. Mohr H., Wolfensteller U., Betzel R.F., Mišić B., Sporns O., Richiardi J., Ruge H. Integration and segregation of large-scale brain networks during short-term task automatization. *Nat. Commun.* 2016. V. 3. Article No. 13217.
348. Gallen C.L., Baniqued P.L., Chapman S.B., Aslan S., Keebler M., Didehbani N., D'Esposito M. Modular brain network organization predicts response to cognitive training in older adults. *PLoS One.* 2016. V. 11. Article No. e0169015.
349. Bullmore E., Sporns O. The economy of brain network organization. *Nat. Rev. Neurosci.* 2012. V. 13. P. 336–349.
350. Zalesky A., Fornito A., Cocchi L., Gollo L.L., Breakspear M. Time-resolved resting-state brain networks. *Proc. Natl. Acad. Sci. USA.* 2014. V. 111. P. 10341–10346.
351. Barttfeld P., Uhrig L., Sitt J.D., Sigman M., Jarraya B., Dehaene S. Signature of consciousness in the dynamics of resting-state brain activity. *Proc. Natl. Acad. Sci. USA.* 2015. V. 112. P. 887–892.
352. Mudrik L., Faivre N., Koch C. Information integration without awareness. *Trends Cogn. Sci.* 2014. V. 18. P. 488–496.
353. Mudrik L., Breska A., Lamy D., Deouell L.Y. Integration without awareness: expanding the limits of unconscious processing. *Psychol. Sci.* 2011. V. 22. P. 764–770.
354. Faivre N., Mudrik L., Schwartz N., Koch C. Multisensory integration in complete unawareness: evidence from audiovisual congruency priming. *Psychol. Sci.* 2014. V. 25. P. 2006–2016.
355. Cole M.W., Reynolds J.R., Power J.D., Repovs G., Anticevic A., Braver T.S. Multi-task connectivity reveals flexible hubs for adaptive task control. *Nat. Neurosci.* 2013. V. 16. P. 1348–1355.

356. Geib B.R., Stanley M.L., Dennis N.A., Woldorff M.G., Cabeza R. From hippocampus to whole-brain: The role of integrative processing in episodic memory retrieval. *Hum. Brain Mapp.* 2017. V. 38. P. 2242–2259.
357. Cole M.W., Yarkoni T., Repovs G., Anticevic A., Braver T.S. Global connectivity of prefrontal cortex predicts cognitive control and intelligence. *J. Neurosci.* 2012. V. 32. P. 8988–8999.
358. Sugihara T., Diltz M.D., Averbach B.B., Romanski L.M. Integration of auditory and visual communication information in the primate ventrolateral prefrontal cortex. *J. Neurosci.* 2006. V. 26. P. 11138–11147.
359. Liao Y., Tang J., Liu J., Xie A., Yang M., Johnson M., Wang X., Deng Q., Chen H., Xiang X., Liu T., Chen X., Song M., Hao W. Decreased Thalamocortical Connectivity in Chronic Ketamine Users. *PLoS One.* 2016. V. 11. Article No. e0167381.
360. Liao X., Cao M., Xia M., He Y. Individual differences and time-varying features of modular brain architecture. *Neuroimage.* 2017. V. 152. P. 94–107.
361. Gopinath K., Krishnamurthy V., Cabanban R., Crosson B.A. Hubs of anticorrelation in high-resolution resting-state functional connectivity network architecture. *Brain Connect.* 2015. V. 5. P. 267–275.
362. Stratton P., Wiles J. Global segregation of cortical activity and metastable dynamics. *Front. Syst. Neurosci.* 2015. V. 9. Article No. 119.
363. Shen Ren, Junhua Li, Taya F., deSouza J., Thakor N.V., Bezerianos A. Dynamic functional segregation and integration in human brain network during complex tasks. *IEEE Trans. Neur. Syst. Rehabil. Eng.* 2017. V. 25. P. 547–556.
364. Wig G.S. Segregated systems of human brain networks. *Trends Cogn. Sci.* 2017. V. 21. P. 981–996.
365. Kelso J.A. Multistability and metastability: understanding dynamic coordination in the brain. *Philos. Trans. R Soc. London, B Biol. Sci.* 2012. V. 367. P. 906–918.
366. Cruzat J., Deco G., Tauste-Campo A., Principe A., Costa A., Kringelbach M.L., Rocamora R. The dynamics of human cognition: increasing global integration coupled with decreasing segregation found using iEEG. *Neuroimage.* 2018. V. 172. P. 492–505.
367. Cocchi L., Zalesky A., Fornito A., Mattingley J.B. Dynamic cooperation and competition between brain systems during cognitive control. *Trends Cogn. Sci.* 2013. V. 17. P. 493–501.
368. Dixon M.L., Thiruchselvam R., Todd R., Christoff K. Emotion and the prefrontal cortex: An integrative review. *Psychol. Bull.* 2017. V. 143. P. 1033–1081.
369. Zippo A.G., Della Rosa P.A., Castiglioni I., Biella G.E.M. Alternating dynamics of segregation and integration in human eeg functional networks during working-memory task. *Neuroscience.* 2018. V. 371. P. 191–206.
370. Parlatini V., Radua J., Dell'Acqua F., Leslie A., Simmons A., Murphy D.G., Catani M., Thiebaut de Schotten M. Functional segregation and integration within fronto-parietal networks. *Neuroimage.* 2017. V. 146. P. 367–375.
371. Casali A.G., Gosseries O., Rosanova M., Boly M., Sarasso S., Casali K.R., Casarotto S., Bruno M.A., Laureys S., Tononi G., Massimini M. A theoretically based index of consciousness independent of sensory processing and behavior. *Sci. Transl. Med.* 2013. V. 5. Article No. 198ra105.
372. Konishi M., McLaren D.G., Engen H., Smallwood J. Shaped by the past: The default mode network supports cognition that is independent of immediate perceptual input. *PLoS One.* 2015. V. 10. Article No. e0132209.
373. Fransson P. How default is the default mode of brain function? Further evidence from intrinsic BOLD signal fluctuations. *Neuropsychologia.* 2006. V. 44. P. 2836–2845.
374. Shine J.M., Keogh R., O'Callaghan C., Muller A.J., Lewis S.J., Pearson J. Imagine that: elevated sensory strength of mental imagery in individuals with Parkinson's disease and visual hallucinations. *Proc. Biol. Sci.* 2015. V. 282. Article No. 20142047.

375. Hellyer P.J., Scott G., Shanahan M., Sharp D.J., Leech R. Cognitive flexibility through metastable neural dynamics is disrupted by damage to the structural connectome. *J. Neurosci.* 2015. V. 35. P. 9050–9063.
376. Kucyi A., Hove M.J., Esterman M., Hutchison R.M., Valera E.M. Dynamic brain network correlates of spontaneous fluctuations in attention. *Cerebr. Cortex.* 2017. V. 27. P. 1831–1840.
377. Yue Q., Martin R.C., Fischer-Baum S., Ramos-Nuñez A.I., Ye F., Deem M.W. Brain modularity mediates the relation between task complexity and performance. *J. Cogn. Neurosci.* 2017. V. 29. P. 1532–1546.
378. Thompson W.H., Fransson P. The frequency dimension of fMRI dynamic connectivity: Network connectivity, functional hubs and integration in the resting brain. *Neuroimage.* 2015. V. 121. P. 227–242.
379. Florin E., Baillet S. The brain's resting-state activity is shaped by synchronized cross-frequency coupling of neural oscillations. *Neuroimage.* 2015. V. 111. P. 26–35.
380. Popa D., Popescu A.T., Paré D. Contrasting activity profile of two distributed cortical networks as a function of attentional demands. *J. Neurosci.* 2009. V. 29. P. 1191–1201.
381. Sara S.J. Sleep to Remember. *J. Neurosci.* 2017. V. 37. P. 457–463.
382. Finc K., Bonna K., Lewandowska M., Wolak T., Nikadon J., Dreszer J., Duch W., Kühn S. Transition of the functional brain network related to increasing cognitive demands. *Hum. Brain Mapp.* 2017. V. 38. P. 3659–3674.
383. Kelly A.M., Uddin L.Q., Biswal B.B., Castellanos F.X., Milham M.P. Competition between functional brain networks mediates behavioral variability. *Neuroimage.* 2008. V. 39. P. 527–537.
384. Amico E., Marinazzo D., Di Perri C., Heine L., Annen J., Martial C., Dzemidzic M., Kirsch M., Bonhomme V., Laureys S., Goñi J. Mapping the functional connectome traits of levels of consciousness. *Neuroimage.* 2017. V. 148. P. 201–211.
385. Hudson A.E., Calderon D.P., Pfaff D.W., Proekt A. Recovery of consciousness is mediated by a network of discrete metastable activity states. *Proc. Natl. Acad. Sci. USA.* 2014. V. 111. P. 9283–9288.
386. Sharp P., Melcher D., Hickey C. Endogenous attention modulates the temporal window of integration. *Atten. Percept. Psychophys.* 2018. V. 80. P. 1214–1228.
387. Alnæs D., Kaufmann T., Richard G., Duff E.P., Sneve M.H., Endestad T., Nordvik J.E., Andreassen O.A., Smith S.M., Westlye L.T. Attentional load modulates large-scale functional brain connectivity beyond the core attention networks. *Neuroimage.* 2015. V. 109. P. 260–272.
388. Wiesman A.I., Heinrichs-Graham E., Proskovec A.L., McDermott T.J., Wilson T. Oscillations during observations: Dynamic oscillatory networks .serving visuospatial attention. *Hum. Brain Mapp.* 2017. V. 38. P. 5128–5140.
389. Nakatani C., Raffone A., van Leeuwen C. Efficiency of conscious access improves with coupling of slow and fast neural oscillations. *J. Cogn. Neurosci.* 2014. V. 26. P. 1168–1179.
390. Doesburg S.M., Green J.J., McDonald J.J., Ward L.M. Rhythms of consciousness: binocular rivalry reveals large-scale oscillatory network dynamics mediating visual perception. *PLoS One.* 2009. V. 4. Article No. e6142.
391. De Ridder D., van der Loo E., Vanneste S., Gais S., Plazier M., Kovacs S., Sunaert S., Menovsky T., van de Heyning P. Theta-gamma dysrhythmia and auditory phantom perception. *J. Neurosurg.* 2011. 114. P. 912–921.
392. Swann N.C., de Hemptinne C., Maher R.B., Stapleton C.A., Meng L., Gelb A.W., Starr P.A. Motor system interactions in the beta band decrease during loss of consciousness. *J. Cogn. Neurosci.* 2016. V. 28. P. 84–95.

393. Wiley J., Jarosz A. F. How working memory capacity affects problem solving. In: *The psychology of learning and motivation*. Ed. Ross B.H. Elsevier Academic Press, 2012. P. 185–227.
394. Vytal K., Hamann S. Neuroimaging support for discrete neural correlates of basic emotions: a voxel-based meta-analysis. *J. Cogn. Neurosci.* 2010. 22. P. 2864–2885.
395. Saarimäki H., Gotsopoulos A., Jääskeläinen I.P., Lampinen J., Vuilleumier P., Hari R., Sams M., Nummenmaa L. Discrete neural signatures of basic emotions. *Cerebr. Cortex.* 2016. V. 26. P. 2563–2573.
396. Kringelbach M.L., Berridge K.C. The affective core of emotion: Linking pleasure, subjective well-being, and optimal metastability in the brain. *Emot. Rev.* 2017. V. 9. P. 191–199.
397. Storbeck J., Maswood R. Happiness increases verbal and spatial working memory capacity where sadness does not: Emotion, working memory and executive control. *Cogn. Emot.* 2016. V. 30. P. 925–938.
398. Mattar M.G., Cole M.W., Thompson-Schill S.L., Bassett D.S. A functional cartography of cognitive systems. *PLoS Comput. Biol.* 2015. V. 11. Article No. e1004533.

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