

Neurocognitive Mechanisms of the Active Buffer of Working Memory

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Introduction

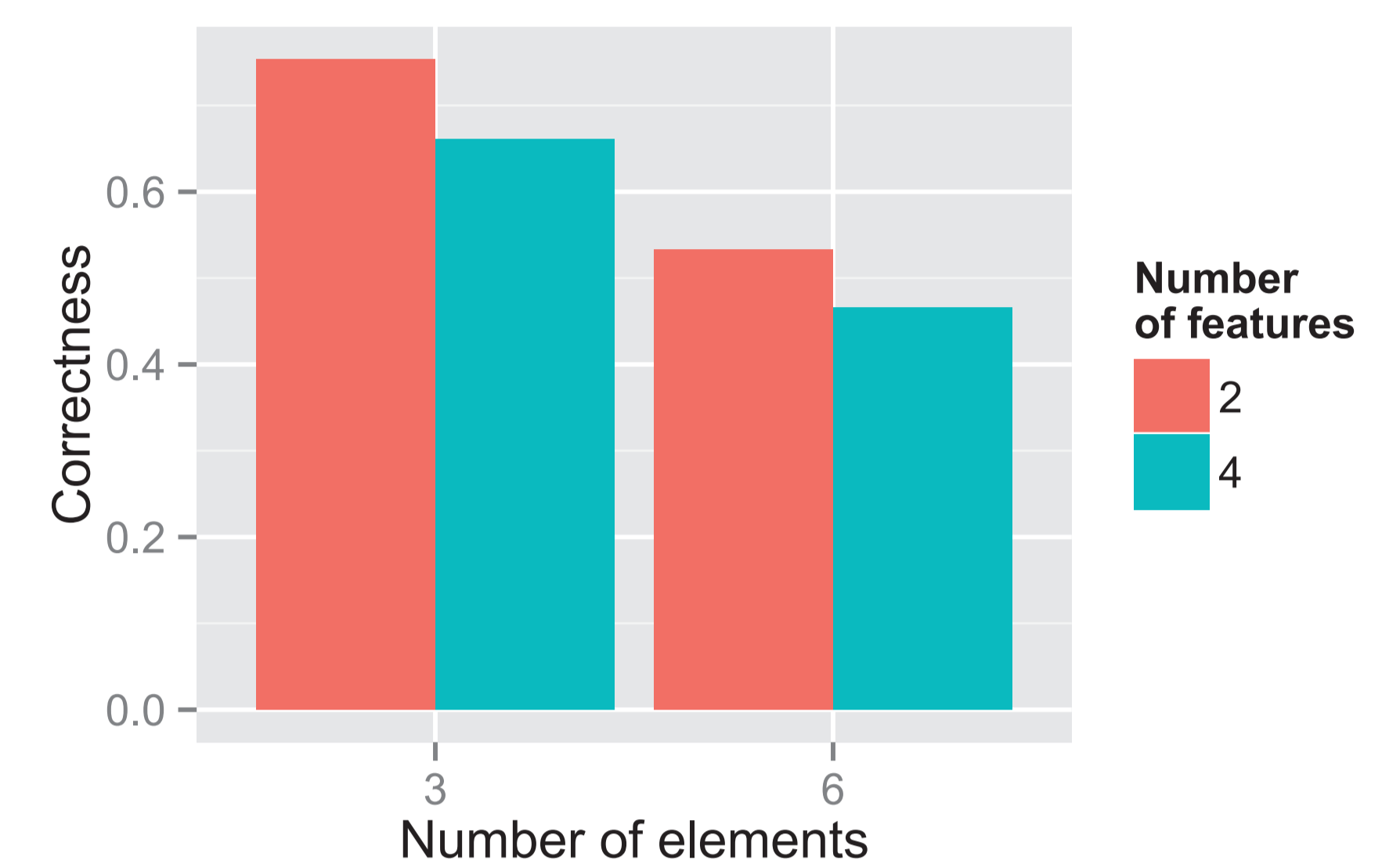
In last years a few computational models, which attempt to replicate and explain several specific phenomena observed in working memory were proposed. Many of the models are aimed in the most basic level of working of the memory which usually connects physical and algorithmic/representational levels. Some of the models focus on characteristics of neural cycles (prefrontal cortex).

We present a model of WM which is based on assumption that many of the effects observed in WM stem from features of theta and gamma cycles in prefrontal cortex. Our model simulates maintaining elements in WM by computational modelling of neural oscillations.

Effect of number of elements & features

WM is limited as regards the number of elements stored. Recent studies (Oberauer & Eichenberger 2013) reveal that WM is also limited as regards the number of features of objects maintained.

Our model is able to recreate both parts of the effect. Relation between wavelengths in θ -cycle and γ -cycle explains limitation of number of elements and growing probability of error explains limitation of number of features.



The Model

The main part of the model is a buffer which can contain a set of processed elements. Every element has three features: (a) reference, (b) output, and (c) level of activation. A reference is a pointer to some content of long term memory. An output of the element represents its level of its availability to other processes. Finally the level of activation is used for activating or inhibiting other elements in PM.

The output y of the element i in time t has been defined according to the following formula:

$$y_i(t) = \frac{1}{1 + \exp(-\delta(x_i(t) - \frac{1}{2}))}$$

Parameter δ controls the level of nonlinearity of the relation between y and x . Moderate δ values give a graded threshold function.

The level of activation x (element i) is computed as follows

$$x_i(t) = x_i(t-1) + \frac{\lambda}{1 + y_i(t-1)} + \alpha \sum_k \exp(x_k(t-1) - x_i(t-1)) - \beta \sum_j \exp(x_j(t-1) - x_i(t-1)) + \varepsilon(n).$$

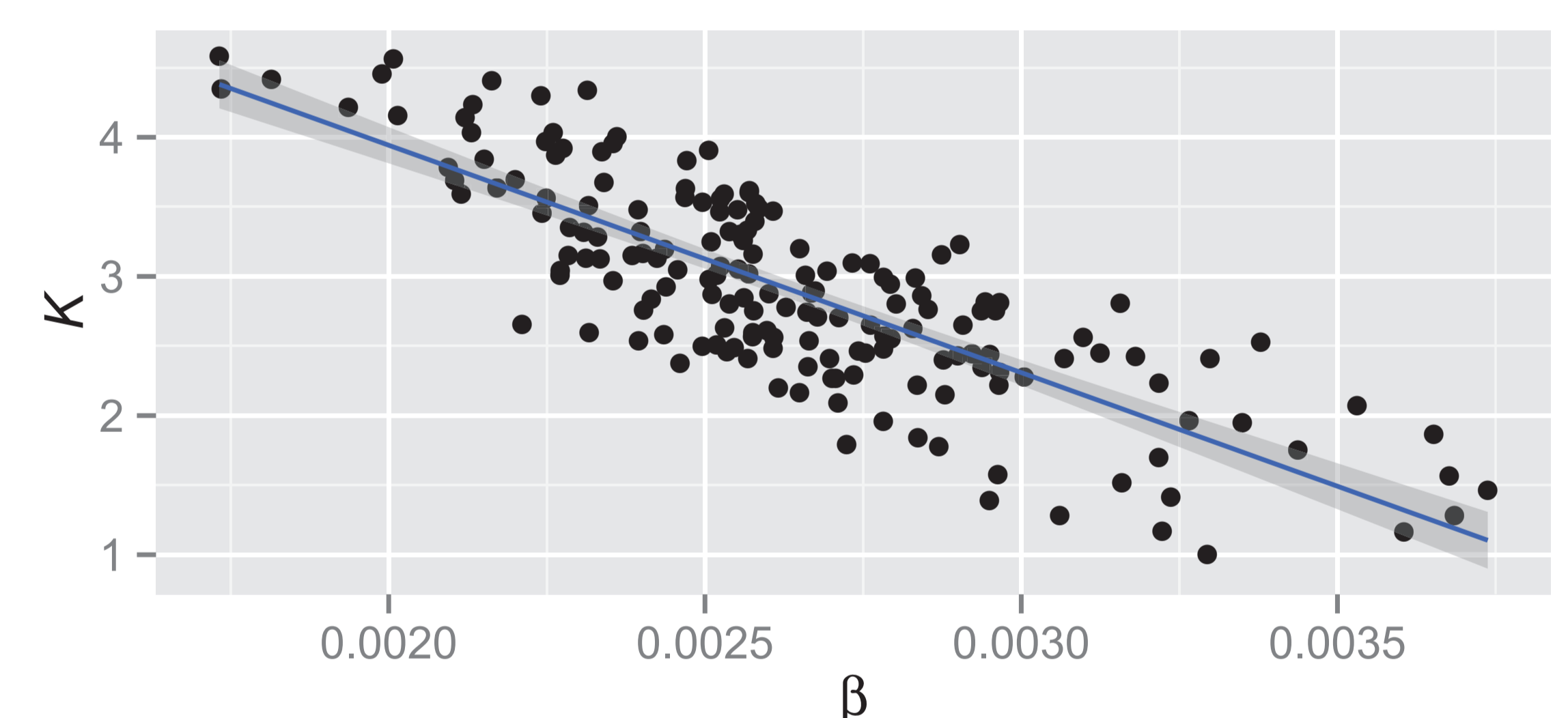
Parameter λ controls how much an element i will be auto activated by the recurrent connections feeding its output back into it, what primarily regulates the frequency of oscillations.

Index k denotes elements which output just before element i does. Parameter α determines how much the output of elements, which oscillate close to element i , will increase its activation. Parameter κ defines also the temporal resolution of bindings.

Index j denotes elements which are not k nor i elements. Parameter β controls the strength of inhibition exerted by elements j , namely how much they will decrease the activation of element i . This strength depends on a difference in the activity of elements i and j activity. The last term of equation consists of noise (ε).

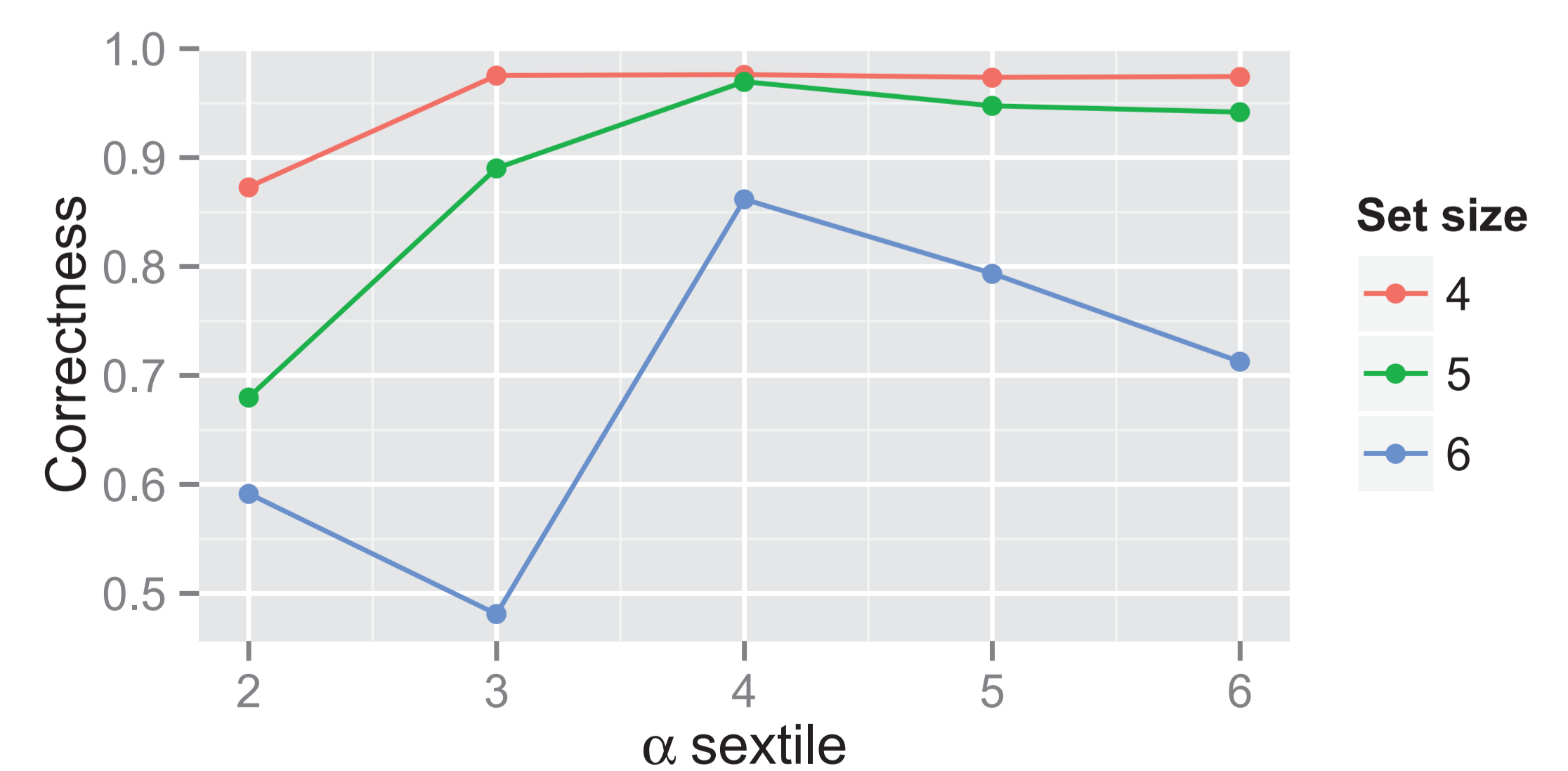
Inhibition - Capacity Relation

A parameter which mainly determines WM capacity is β . The β parameter determines strength of inhibition between all active elements. The stronger the inhibition is the less elements active in WM is required to block adding new elements. The neural substrate of the β parameter can be localised in posterior parietal cortex (Todd & Marois 2004).



Activation - Accuracy Relation

In addition to inhibition elements can activate other ones. Elements which co-oscillate (form a whole object) pass activation to each other. Strength of the activation is determined by the α parameter. The mechanism reflects top-down boosting applied by prefrontal cortex (Edin et al. 2009).



Edin, F., Klingner, T., Johansson, P., McNab, F., Tegner, J., & Compte, A. (2009). Mechanism for top-down control of working memory capacity. *PNAS*, 106, 6802–6807.
Oberauer, K., & Eichenberger, S. (2013). Visual working memory declines when more features must be remembered for each object. *Memory & cognition*. doi:10.3758/s13421-013-0333-6
Todd, J. J., & Marois, R. (2004). Capacity limit of visual short-term memory in human posterior parietal cortex. *Nature*, 428, 751–754.

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