Editorial

The mismatch hypothesis of psychiatric disease

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The mismatch hypothesis is an evolutionary relevant hypothesis on the origins of health and disease. According to the mismatch hypothesis individuals who have been able to correctly 'predict' their later environment from early life circumstances are better off compared to individuals who face a mismatch between early and later life circumstances. The focus of this special section in Physiology and Behavior is on the question if and how mismatches between the psychosocial environment in early and later life could be used to explain the occurrence of psychiatric disease.

The idea for testing the mismatch hypothesis of psychiatric disease first came up during a Summer School about Neurodevelopmental Programming and Phenotypic Plasticity in 2009. Paul Brakefield introduced the concept of phenotypic programming in Bicyclus butterflies [1]. In these butterflies, a mismatch between predicted and actual later environments compromised fitness. A butterfly with the warm and wet season phenotype living in the cold and dry season would either be preadapted because of its colorful wings or starve because of its fast metabolism. On the other hand, a butterfly with the cold and dry season phenotype living in the wet and dry season would not be able to find a mate because it is not as good looking as the other phenotype [2]. Immediately, the question arose whether this mechanism applies to psychosocial aspects of the environment as well.

On the second day of the summer school Danielle Champagne presented her results on context dependence of the adaptiveness of phenotypic programming. She showed that rats who received little maternal licking and grooming developed phenotypes that enhanced learning under stressful conditions [3]. By now, quite some studies support the idea that psychosocial aspects of the environment can cause adaptive phenotypic programming [4–6]. For example, van Hasselt et al. [7] present evidence in this special section that maternal licking and grooming of male rats are associated with later social play. This suggests that indeed the early psychosocial environment has the capacity for developmental programming.

The question also kept coming back at consecutive meetings of researchers involved in EuroSTRESS. Although in the central theme of the European Science Foundation (ESF) coordinated EuroSTRESS consortium the classical cumulative stress model is central, many researchers in this consortium have a special interest in evolutionary relevant explanations of the effects of stress (for example, [8]). That’s why this theme was chosen for a symposium accompanying the final EuroSTRESS meeting from which this special section in Physiology and Behavior is a result.

Both the symposium and this special issue commenced with theoretical and methodological issues [9]. Nederhof and Schmidt summarize empirical evidence regarding the long-term effects of phenotypic programming by the psychosocial environment. Evidence in support of both the mismatch hypothesis and the classical cumulative stress hypothesis can be found in the literature [9].

In this special section, Daškalakis et al. [10] present evidence that supports the mismatch hypothesis in a rat model for psychosis susceptibility. They showed that a mismatch between the early and later environment increases psychosis susceptibility. However, rats who experienced both an adverse early and an adverse later environment showed increased psychosis susceptibility when exposed to high levels of the stress hormone cortisone, supporting the cumulative stress hypothesis [10].

Also in this special section, Pesonen and Raikkonen [11] review results from studies into the effects of child evacuation during World War II in the Helsinki Birth Cohort. This review provides evidence for the cumulative stress hypothesis; Longer evacuation showed largest effects.

Nederhof and Schmidt [9] integrated the mismatch hypothesis with the cumulative stress hypothesis by placing them on opposite ends of a sensitivity to programming axis. Results of now numerous papers point into the direction that individuals with a high (heritable) sensitivity to programming are indeed disproportionally influenced by positive as well as negative aspects of the environment (for recent examples see [12–15], for reviews see [16–18]).

Glover and Mill [19] extended Nederhof and Schmidt’s integrated model further by suggesting that the effects of programming and therefore the adaptiveness of phenotypes might be sex specific. In this special section, Glover and Mill reviewed the effects of prenatal stress on responsiveness of the major stress systems and on psychopathology concluding that more research on the sex specificity of the effects is needed. I agree with their conclusion that, indeed, a lot of work needs to be done to test Nederhof and Schmidt’s integrated model [9].

References


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