A theoretical muddle of the conditional strategy: a comment on Neff and Svensson

Bruno A. Buzatto¹, Wade N. Hazel² and Joseph L. Tomkins¹

¹Centre for Evolutionary Biology, School of Animal Biology (M092), The University of Western Australia, 35 Stirling Highway, Crawley, Western Australia 6009, Australia
²Department of Biology, DePauw University, Greencastle, IN 46135, USA

Neff & Svensson [1] recently reviewed the literature relating to the theoretical modelling of alternative mating tactics. Their review presents insightful future perspectives on how genomics could unveil the molecular genetics and proximate mechanisms of tactic expression, and we fully agree with their statements that a better understanding of the field can be achieved through the integration between quantitative and molecular genetics. However, we feel that it is important to draw attention to two significant oversights: their review (i) neglects previous studies that provided theoretical and empirical contributions to the understanding of the genetics underlying the conditional expression of alternative mating tactics (i.e. conditional strategies) and (ii) presents ideas that have been previously published (by other authors) as a new ‘unified theory for the evolution and phenotypic expression of alternative mating tactics’. Below we elaborate why we believe that Neff and Svensson’s verbal model (‘the conditional alternative strategy’) is not a new model, but is instead based on a misinformed view that current models do not account for genetic variation underlying conditional strategies. As a result, Neff and Svensson’s ‘conditional alternative strategy’ unnecessarily muddles our understanding of conditional strategies.

Phenotypic plasticity is a ubiquitous evolutionary phenomenon [2] and nowhere perhaps, is plasticity more apparent than when single genotypes can produce different alternative phenotypes depending upon environmental conditions [3]. Such polyphenisms are synonymous with conditional strategies and have been described in a wide range of traits, including the presence, shape and colour of morphological traits, alternative mating tactics, diet, sex and caste determination, as well as paedogenesis and diapause [4]. Understanding their genetic architecture and evolution is therefore vital for our understanding of these important adaptations. Dawkins’ conditional evolutionary stable strategy model [5] provided an important theoretical framework for understanding polyphenisms, but also conveyed the message that genetic polymorphism is not necessary for a conditional strategy to be evolutionary stable. This message was an attempt to distinguish conditional strategies from ‘alternative strategies’, which represent a genetic polymorphism with Mendelian inheritance. However, it also led numerous authors to erroneously associate conditional strategies with complete genetic monomorphism [1,6,7]. It is well established, however, that underlying genetic variation is prevalent in plastic traits [8,9], and its role in polyphenisms has been modelled [10,11] and supported empirically numerous times [12–15]. The conditional strategy was recently criticized based on this false assumption of genetic monomorphism [7]—a misconception that was subsequently laid to rest [16]. Neff and Svensson do cite this [16] correction of the record, but in our view failed to convey an understanding of its content, because the assumption of monomorphism re-appears as the driver of Neff & Svensson’s [1] ‘new model’.

Sewall Wright [17,18] pioneered the idea that discontinuous traits could result from continuous polygenic variation, coupled with a threshold mechanism that generates discontinuity in trait expression. Such traits were named
threshold traits [19], and the concept of continuous polygenic variation underlying their expression became the central idea of the ‘liability model’ of quantitative genetics [20,21]. This model assumes liability is a continuously variable quantitative trait, influenced by genetic and environmental factors. Individuals produce one or the other of two alternative phenotypes depending on whether their liability exceeds some fixed threshold [20]. Wright’s model can be modified to account for the alternative tactics of conditional strategies by allowing the position of the threshold relative to the continuous distribution of liabilities to be a function of the environmental cues that influence the expression of the alternative tactics. Thus, according to this ‘environmental threshold model’, additive genetic variation, not genetic monomorphism, underlies the expression of the alternative tactics of the conditional strategy [4,10,16].

Unarguably, the view that genetic variation can influence the conditional expression of alternative tactics is neither new nor commonly disregarded—the three main references on the quantitative genetics of conditional strategies, namely Hazel et al. [10], Roff [4] and Hazel et al. [11] sum more than 360 citations in Web of Science to date. Despite briefly citing some of these studies, Neff & Svensson [1] disregard the ideas and models in them, and then present the conceptualization of genetic variation for switch points as if it were a new idea (see their section ‘Genetic and plastic alternative mating tactics’). Not only has this idea been described previously, it has also been recently reviewed [14,16] and empirically supported [12,13,22–25]. Interestingly, Neff & Svensson [1] acknowledge and cite previous work that demonstrates that threshold reaction norms have underlying genetic variation and can therefore evolve [14,26,27], but by and large discuss the subject as if this awareness had not penetrated the field as a whole. They also neglect the actual quantitative genetics models for threshold traits, and go as far as proposing a verbal model that fuses alternative strategies and conditional strategies [1]. This fusion, however, does not provide any new theoretical framework for our understanding of the evolution of alternative tactics, and hence the term ‘conditional alternative strategies’ is an unnecessary confusion.

We feel that the manner in which Neff & Svensson [1] express how phenotypic plasticity can evolve, as a ‘novel’ point in their model, wrongly leads the reader to believe that the previous models for conditional strategies do not account for evolving phenomena. We believe that verbal models are very important when they present new ideas. However, given that previous (mathematically formalized) models for conditional strategies do account for genetic variation and the evolvability of conditional strategies, we question the value of a verbal model that does the same, but without mathematical formality. Neff & Svensson advocate their model by proposing that alternative and conditional strategies “…represent the extremes on a continuum and that most AMTs fall somewhere in between the two, with both genes and environment contributing to phenotypic expression of the tactics” [1, p. 8]. As we argued above, current genetic models incorporate genetic variation in liability underlying conditional strategies [16] as well as environmental influences on where the distribution of liabilities is divided by a threshold. Hence, this point does not generate a need for fusing conditional and alternative strategies. The idea of a continuum between alternative and conditional strategies could still be applied to cases where one or a few genes have a large effect on the expression of the alternative tactics in response to environmental variation. Again, this possibility has been modelled [11,13,28,29] and empirically supported [12]. Unfortunately, most of these studies were also overlooked by Neff & Svensson [1].

In conclusion, the game-theoretical models of conditional and alternative strategies do not need to be fused in order to accommodate genetic variation for conditional polymorphisms, because this has already been comprehensively done under the umbrella of the environmental threshold model for conditional strategies. Presenting this fusion and labelling it as a putative new unified theory [1] is unnecessary and mathematically unsupported. Much progress has been done in the last few decades on the genetic basis of conditional strategies, and it is clear that genetic variation in conditional strategies is prevalent in natural populations. The genetic monomorphism-oriented criticism of conditional strategies present in Neff & Svensson [1] (see also [7]) adds nothing to the progress already made [10,11,14,16]. Finally, since a new verbal model is proposed to do what previous mathematically supported models have already been doing for years, we believe that the ‘conditional alternative strategies’ model is unjustified, and even muddies the water when it unnecessarily fuses alternative strategies with conditional strategies.

References


