Commentary: Building the developmental foundations of developmental computational psychiatry: reflections on Hauser et al. (2019)

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Hauser and colleagues’ (2019) target article illustrates the promise of applying a computational level of analysis to reveal the precise mechanisms that can explain the precipitous rise in psychopathology during the adolescent years. There is a broadly emerging view that computational approaches will accelerate progress in understanding psychiatric illness. To truly consider psychiatric illness as neurodevelopmental in nature, it is crucial to view the study of psychopathology’s symptoms, mechanisms, and course through a developmental lens. Hauser and colleagues’ optimism is welcome. By providing a road map with concise, concrete examples of the processes illuminated by computational psychiatry, Hauser, Will, Dubois, and Dolan’s (2019) review creates a framework to broaden research toward the crucial, unanswered questions about how psychopathology emerges.

We too are eager for the developmental research to reap the benefits of computational approaches. Although some progress has been made in recent years, the use of computational approaches to reveal the algorithmic changes in cognition at the algorithmic level by benchmarking on well-described adult models. For example, van den Bos, Cohen, Kahnt, and Crone (2012) show that child, adolescent, and adult learning behavior can be described equivalently well to a standard reinforcement learning model with separate parameters for positive and negative learning rates. They identify a single parameter that explains age-related differences in learning: the learning rate for negative feedback, which is elevated in children.

However, developmental research has highlighted that it is not always valid to assume the same algorithms in adult models will characterize a developmental population, just with different weights. Palminteri and colleagues, for example, found that adolescent reinforcement learning was better explained by the simplest model tested, whereas adult reinforcement learning more closely matched a more complex model that incorporated additional sources of information, including counterfactual outcomes and other contextual factors that did not influence younger individuals’ choices (Palminteri, Kilford, Coricelli, & Blakemore, 2016). In another study, whereas inaccurate instructions biased adult estimates of stimulus value in a reward learning task, children avoided this bias by relying more heavily on their own experiences (Decker, Lourenco, Doll, & Hartley, 2015) and this difference was reflected in distinct best-fitting models for these age groups. Finally, there is evidence that decision-making strategies vary qualitatively between younger and older adults as well; older adults appear to rely more heavily on simpler, heuristic-based decision strategies, whereas younger adults rely more heavily on more complex model-based decision-making (Worthy, Cooper, Byrne, Gorlick, & Maddox, 2014). Developmental computational psychiatry research will therefore need to avoid the pitfall of making assumptions that child and adolescent computational processes vary from adults only in degree and not in kind.

Development is both quantitative and qualitative

First, as described in the target article, developmental computational psychiatry will need to be grounded in developmentally informed tasks and computational models. These tools will need to be sensitive to the underlying cognitive operations used by individuals of all ages of interest. Many studies attempt to describe the developmental changes in cognition at the algorithmic level by benchmarking on well-described adult models. For example, van den Bos, Cohen, Kahnt, and Crone (2012) show that child, adolescent, and adult learning behavior can be described equivalently well to a standard reinforcement learning model with separate parameters for positive and negative learning rates. They identify a single parameter that explains age-related differences in learning: the learning rate for negative feedback, which is elevated in children.

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Testing developmental differences
Research designed to identify developmental differences in a behavior, model, symptom, or neurodevelopmental pattern requires careful consideration of the group-analytical framework used to test for these changes. Much computational-based research identifies best-fitting models of a group, or conducts between-group comparisons by directly comparing different subsets of an overall sample (i.e. case-control approaches). One common convention in early computational work has been to compare groupings based on the developmental phase (i.e. children vs. adolescents; adolescents vs. adults) and setting the boundaries between these groups in a relatively arbitrary fashion. We caution against this overly simple perspective, which tends to lump large swaths of individuals who, in actuality, are not comparable (i.e. a 13-year old and a 17-year old occupy quite distinct developmental stages, yet are frequently combined into an “adolescent” group). In our own work, we have used statistical approaches that treat age as a continuous variable to identify behavioral inflection points within the adolescent years that would not have been observable using the semi-arbitrary groupings just described (Rodman, Powers, & Somerville, 2017).

The need to consider development continuously is further reinforced in large-scale research that seeks to define the “growth curves” for different neurodevelopmental processes. A variety of metrics such as whole brain resting state connectivity, cortical volume, and behavioral markers of brain function such as self-conscious emotion (Rodman et al., 2017) have underscored that age-related change is often not a linear process (see Casey, 2015). Brain development is marked by inflection points, plateaus, and various other nonlinearities that render arbitrary groupings or simple linear fits insufficient to capture its complexity. A key challenge for the developmental computational psychiatry research community will be to represent these complex patterns of change within their analytic frameworks.

Development beyond age
Finally, Hauser and colleagues describe the need to target neurodevelopmentally grounded mechanisms of dysfunction for key psychiatric symptoms. As such, a foundational understanding of human brain development is crucial for pointing to the key processes and targets for developmental computational psychiatry. While the field of developmental cognitive neuroscience has exploded in recent years, the field is still working toward a comprehensive account of brain development. This is, in part, because development itself is a complex, multidetermined process that reflects the influences of mechanisms including (but not limited to) age, key experiences, and pubertal hormone changes.

When examining neurodevelopmental change across development, the state of brain maturation – both behaviorally and biologically – cannot necessarily be determined by age alone. Prior studies have shown that pubertal stages and hormone levels relate to differences in cortical and subcortical volumes, as well as both structural and functional connectivity and white matter tract maturation. For example, Goddings et al. (2014) investigated changes in subcortical volume in a longitudinal sample and found that changes in amygdala, hippocampal, and putamen volumes were better explained by a model that included both age and pubertal development indices than age alone. Klapwijk et al. (2013) showed that during social information processing in girls, increased functional connectivity between the dorsomedial prefrontal cortex and the right temporo-parietal junction was independent of age but instead explained by the girls’ pubertal stage. Asato, Terwilliger, Woo, and Luna (2010) similarly showed that structural white matter tracts do not asymptote in their structural change until the postpubertal stage, regardless of participant age. It is therefore important that research moves beyond considering simple age-related changes to incorporate the influence of other developmental processes, such as puberty, on cognitive development. The relevance of this issue to computational cognitive development was underscored recently by Boehme et al. (2017), who showed that contextual model-based learning was associated with more advanced pubertal development, above and beyond the influence of age. Thus, if the field hopes to utilize computational models to identify deviations in the development of cognitive processes, further work to elucidate how basic factors such as hormones and puberty interact with brain development is crucial.

In this commentary, we have underscored several ways in which incorporating a developmental framework into existing research areas is more complex than simply comparing different age groups. How can researchers traverse these challenges in their research? There is an increasing recognition that very large samples are needed to simultaneously characterize the complex mechanisms of neurodevelopment. This is reflected in large-scale investments including the Adolescent Brain and Cognitive Development study and Human Connectome Project in Development, which due to their size and scope, hold the potential to represent neurodevelopment at an appropriate level of complexity. Future computationally rooted work may benefit from following the lead from these studies by testing large samples, acquiring multiple indices of developmental stage beyond age, and structuring analytic plans to identify nonlinear patterns of change. Even studies on a smaller scale can be “developmentally informed” at the study design phase, by carefully targeting an age range of interest based on prior research and

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developmental theory, considering collateral measures of development beyond mere age, and planning to evaluate data with continuous statistics.

Here we have highlighted some of the considerations aimed to provoke thinking of how developmental computational psychiatry can progress in a way that is maximally developmentally informed. With these factors in mind, we share optimism that computational modeling will propel discoveries revealing why psychiatric illness emerges during adolescence.

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References


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