# Understanding divergence: Placing developmental neuroscience in its dynamic context

**Duncan E. Astle[1,2], Dani S. Bassett[3,4] & Essi Viding[5]**

1. *Department of Psychiatry, University of Cambridge*
2. *MRC Cognition and Brain Sciences Unit, University of Cambridge*
3. *Departments of Bioengineering, Electrical & Systems Engineering, Physics & Astronomy, Neurology, and Psychiatry, University of Pennsylvania*
4. *The Santa Fe Institute*
5. *Psychology and Language Sciences, University College London*

**Corresponding author:**

Professor Duncan Astle,
15 Chaucer Road,
Cambridge,
CB2 7EF
Duncan.astle@mrc-cbu.cam.ac.uk

**Abstract**

Neurodevelopment is not merely a process of brain maturation, but an adaptation to constraints unique to each individual and to the environments we co-create. However, our theoretical and methodological toolkits often ignore this reality. There is growing awareness that a shift is needed that allows us to study divergence of brain and behaviour across conventional categorical boundaries. However, we argue that in future our study of divergence must also incorporate the developmental dynamics that capture the emergence of those neurodevelopmental differences. This crucial step will require adjustments in study design and methodology. If our ultimate aim is to incorporate the developmental dynamics that capture how, and ultimately when, divergence takes place then we will need an analytic toolkit equal to these ambitions. We argue that the over reliance on group averages has been a conceptual dead-end with regard to the neurodevelopmental differences. This is in part because any individual differences and developmental dynamics are inevitably lost within the group average. Instead, analytic approaches which are themselves new, or simply newly applied within this context, may allow us to shift our theoretical and methodological frameworks from groups to individuals. Likewise, methods capable of modelling complex dynamic systems may allow us to understand the emergent dynamics only possible at the level of an interacting neural system.

**Keywords:** development, systems neuroscience, neurodevelopmental condition, mental health, computational neuroscience

## Main Text

The mental processes by which we represent the world shape our behaviour, our interactions with each other, and our engagement with our wider environments. Crucially, these cognitive processes, and their underpinning neurobiology, differ widely across individuals. Multiple perspectives, often drawing heavily on evolutionary theory, posit that this variety is not a *bug*, but rather a *feature* of human development (Honegger & de Bivort, 2018; Hiesinger & Hassan, 2018). At a societal level some features of this variability are more salient than others because they impact our social interactions, learning, health or wellbeing. On certain measures, and at certain thresholds, divergence from what is considered normative will result in formal clinical recognition, and in some cases intervention. *When does this divergence emerge? How would we detect it? What factors drive it? And can (or should) we do anything to address it?* As we outline in the following sections, these foundational questions are incredibly hard to answer because the very differences we are seeking to understand cannot be divorced from their developmental, and indeed dynamic, context.

From the outset it should be stressed that we use the term 'divergence' in its most basic statistical sense. Put simply, we use this term as a shorthand for *when trajectories of change become different across individuals*. It is important to distinguish our meaning from 'neurodivergent', a term now commonly used within the 'neurodiversity paradigm'. Neurodiversity is an important area of scholarship *in its own right*, drawing widely on disciplines including psychology, philosophy, and biology, with far reaching implications for clinical support, education, politics and social justice.

### 1. *The importance of developmental timing*

During neurodevelopment the brain undergoes a continual process of adaptation, across multiple levels of analysis and many time scales. Each individual's brain adapts in response to the environment in which it develops, which they themselves have a hand in creating, and

according to unique characteristics conferred by their genetic background. This complex process of adaptation starts early, even prenatally, and over time results in diverse trajectories of brain development, cognition, and behaviour. In other words, a continual adaptive ontogeny creates unique individuals, but also makes pinpointing moments of divergence between individuals extremely challenging. Establishing the specific brain mechanisms that drive that divergence is more challenging still.

An emergent property of these overlapping adaptive processes is the existence of 'sensitive periods' — epochs of more dramatic positive change during which capacities emerge rapidly. These periods can also be windows of potential vulnerability. For example the period between 6 months and 3 years of age has been identified as a sensitive period for the acquisition of oral language (Norrman & Bylund, 2016), and adolescence has been identified as a sensitive period for the development of new social cognitive skills (Foulkes & Blakemore, 2018). The emergence of behavioural, learning and mental health difficulties also has a developmental context. Inattention and hyperactivity tend to first be recognised in early to middle childhood, with the majority of children who experience these externalising difficulties first being identified between the ages of 6 and 10 years (Sayal et al., 2018; Scahill & Schwab-Stone, 2000). Difficulties with literacy and numeracy are often identified when children enter school, typically between ages 5 and 7 years (Bull et al., 2008). In contrast, difficulties with mood and anxiety most commonly first appear during adolescence (Kessler et al., 2010). These rough windows reflect population-level data. In reality, just as individuals are adapting to a unique combination of constraints, so too developmental trajectories vary considerably between individuals. The key point, regardless, is that divergence only really makes sense within its developmental context.

Divergence of trajectories in cognitive and brain development is a natural part of human existence and diversity. But our understanding of when *divergence* becomes *difficulty* is currently constrained by diagnostic frameworks that chart a list of symptoms and assign individuals to one or more discrete categories at a particular point in time. In short, we deploy

a framework largely inherited from adult psychiatry in which we categorise divergence, which can itself be problematic. Existing diagnostic classification systems, such as the DSM-5 (American Psychiatric Association, 2013) or ICD-11 (World Health Organization, 2019) have been widely critiqued (Astle et al., 2021; Astle & Fletcher-Watson, 2020; Happé et al., 2006; Pennington, 2006), with many scholars and practitioners questioning whether they are fit for purpose, in both research and practice. Our aim here is not to provide a detailed overview of the conceptual and methodological shortcomings of the diagnostic orthodoxy. This has already been done (e.g. Astle et al., 2021): these frameworks are thought to be ill equipped to accommodate the high degree of symptom overlap *across* diagnoses, incorporate variability *within* diagnoses, or capture the needs of those with difficulties that do not conform to a diagnostic standard. Furthermore, it is clear from longitudinal population representative data that many individuals migrate between diagnostic criteria across development (Caspi et al., 2020). Our efforts to understand underpinning mechanisms or deliver effective support will inevitably be impeded by ill-fitting criteria.

The concerns about the existing diagnostic frameworks have led many to call for a shift towards a 'transdiagnostic' framework in which categorical boundaries are relaxed, redrawn, or removed altogether (Coghill & Sonuga-Barke, 2012; Levy & Ebstein, 2009; Sonuga-Barke et al., 2016; Sonuga-Barke & Coghill, 2014). Broadly speaking this alternative framework focuses instead on the variability of particular characteristics within the population rather than on predefined categories and could be helpful when considering preventative, clinical staging models of care (McGorry & Mei, 2021). In research settings, deployment of a transdiagnostic framework means that recruitment strategies are broadened and data-driven methodologies are applied, capturing *dimensions* (Astle et al., 2019) or *clusters* (Astle et al., 2019; Bathelt et al., 2021; Kushki et al., 2019; Siugzdaite et al., 2020) that provide parsimonious accounts of the variability within these more representative cohorts. In some cases these new data-driven accounts can be used to query the utility of existing diagnoses (Bathelt et al., 2018; Kushki et al., 2019), but more commonly they are used to bring fresh insight to the scope and nature of

the neurodevelopmental difficulties themselves (Bathelt et al., 2021). A transdiagnostic framework also opens the door for a deeper conceptual shift. Abandoning the supremacy of diagnostic status shifts the emphasis to capturing the profile of an individual, regardless of their formal label. This shift allows us to focus on understanding characteristics that are impactful in daily life, whether they match a diagnostic prototype or not. In some respects, the shift also mirrors similar changes within the neurodiversity movement, which focuses on 'differences' rather than 'disorders', and resists the default pathologisation of young people, whilst recognising that some may experience differences that are disabling (Chapman, 2020).

The absence of developmental context from the existing diagnostic criteria is particularly troubling. The symptoms are treated as static. In reality the likelihood of any particular set of characteristics being either an enduring or sole defining feature of a person's condition across their entire development is slim (Caspi et al., 2020). We will struggle to identify the drivers of divergence if we group individuals according to their cross-sectional diagnostic status, ignoring their vastly different profiles of strength and difficulty, their differing developmental trajectories, or the developmental context in which their difficulties occur. Likewise, an intervention selected on the basis of a diagnostic label at a single time-point is unlikely to be optimally effective. Put simply, viewing developmental differences through a stationary diagnostic lens may not have served us well. It may not be enough to simply revisit diagnostic boundaries; we also need to map the timing and sequencing of difficulties (Figure1).

This paper takes as its backdrop the aspiration of a transdiagnostic approach. This reframing of neurodevelopmental divergence is an incredibly useful lens through which to understand and study the interrelationships between brain and behaviour. However, we argue that efforts to characterise the broad, mixed population of young people encountering neurodevelopmental difficulties using a transdiagnostic approach will fail if that approach does not also incorporate the *developmental dynamics* that capture how difficulties emerge.

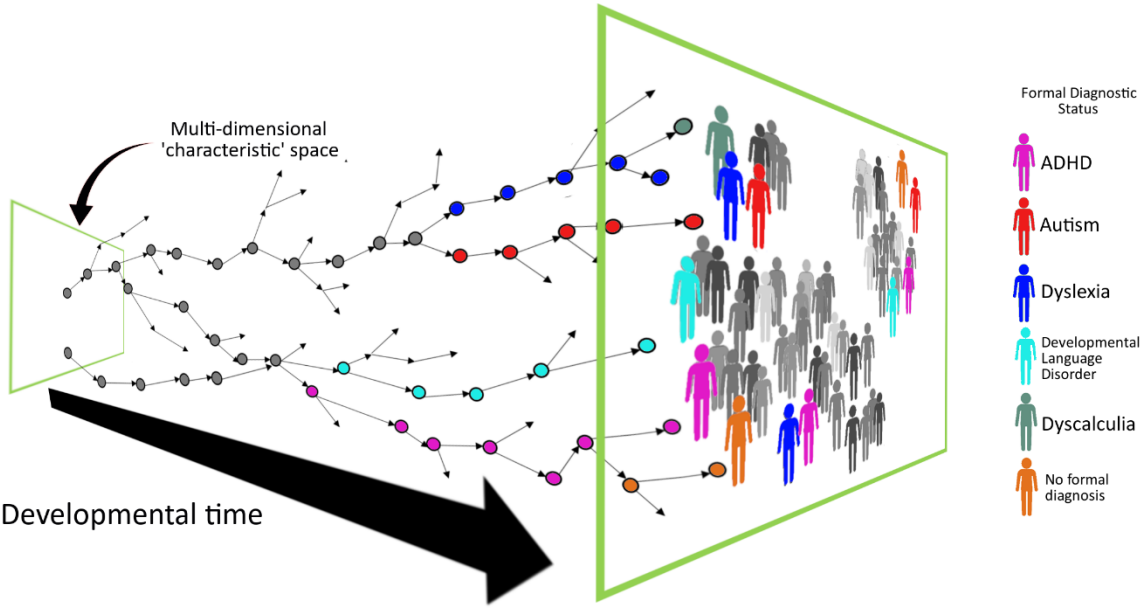


***Figure 1:*** *A schematic depicting some notional multi-dimensional 'characteristic' space (perhaps constructed from symptom data, cognitive tasks, or brain data), projected on a 2D plane, across which individuals differ regardless of diagnostic status. The image also captures the progression of developmental time, illustrating that different profiles within that 'characteristic' space do not simply need to be mapped at a single time, but to be understood within their developmental context.*

### *2. Capturing divergence*

Better understanding divergence and its origins requires that we first briefly consider how we would measure it. A crucial first step is recognising that different designs can provide different ways of capturing that divergence.

*2a. Recruitment:* Cross-group comparisons, in which multiple diagnostic groups are recruited, can provide a simple way of testing whether developmental characteristics span existing boundaries (Kushki et al., 2019). However, these designs tend to recapitulate the problems of existing diagnostic frameworks, since they still use the established rubric for the recruitment and exclusion of participants. Alternative sampling techniques, such as functional recruitment on the basis of clinical or educational need (Holmes et al., 2019) are likely to yield samples more representative of neurodivergent young people, because they side-step the various biases, and exclusion criteria, inherent in the diagnostic system (though may still include biases that reflect inequalities in the recognition of those needs). In short, the first

consideration for capturing divergence is to establish who is studied. Tethering recruitment more closely to the existing categorical criteria will make the findings more directly relevant to the existing diagnoses; looser sample frames will likely achieve samples that are more representative of the real underlying population of young people experiencing difficulties. In reality there is no perfect sampling solution. Truly population representative datasets, such as the Millennium Cohort Study (Connelly & Platt, 2014), clearly offer the best chance of capturing the underlying diverse reality of children experiencing difficulties. However, studies of this scale are often so vast and multi-purpose that it is practically difficult to cram in time-consuming detailed assessments. Moreover, the sub-populations of interest might still be relatively small. The obvious alternative, of targeted recruitment within the clinic, will inevitably introduce multiple biases. The nature and impact of those biases will depend upon the national or local context that dictates who gets a clinic referral. Whatever sampling frame is deployed, it is crucial to acknowledge the potential biases when drawing theoretical conclusions. One particularly promising recruitment framework is a collaborative multi-pronged approach in which participants can be recruited through multiple routes (e.g., in clinics, schools, and community settings; Holmes et al. 2019). This method may allow us to not only minimise biases but also explicitly test for them by contrasting data collected with different recruitment approaches.

*2b: Measurement:* Once the sample of interest is identified, the next ingredient for capturing divergence is a set of measures to characterise that sample. Diagnostic studies tend to focus measurement heavily towards the characteristics thought to be most relevant for a particular group: theory of mind (ToM) assessments for studies of autism, phonological awareness assessments for studies of dyslexia, inhibitory control assessments for studies of ADHD, and so on. This selective measurement can itself skew our understanding of the relevant characteristics for defining developmental difficulties. For this reason transdiagnostic studies often cast a wider net, revealing that developmental difficulties are rarely as selective as previously thought.

Particularly relevant here are issues of measurement sensitivity and quality. Many well-established tasks from the cognitive neuroscience and experimental psychology traditions have poor psychometric properties, including poor test-retest reliability (Elliott et al., 2020; Enkavi et al., 2019; Parsons et al., 2019). This poverty arises because tasks optimised to capture sometimes subtle between-trial effects at a group level do not automatically lend themselves to reliably and sensitively measuring individual differences. Tasks we like as experimentalists, designed to carefully tease apart different component processes, are unlikely to be optimally informative for understanding the differences between individuals. Indeed, experimental tasks are often chosen explicitly to minimise those differences. A related obstacle is the 'single-shot' nature of most cognitive performance metrics. In contrast to questionnaire measures, which call upon the respondent to estimate particular characteristics based on multiple exposures over a period of time (e.g., "in the last year, how often…"), task-based neuroimaging or experimental psychology paradigms typically sample behaviour on a single occasion (albeit with multiple trials). This sort of sampling will limit the ability of the metric to capture enduring characteristics of an individual, and increase the effect of stochastic performance-influencing factors. Pezzoli et al. (2023) provide a good overview of the challenges of measurement sensitivity and quality within developmental samples, and outline opportunities to overcome those challenges.

*2c. Analysis.* Once we have recruited and measured our samples, the final key ingredient for studying divergence is the toolkit we use to characterise them. If group membership is loosened, or abandoned altogether, then we need methods that go beyond conventional univariate comparisons. To date, transdiagnostic studies have tended to adopt one of two alternative approaches. The first route is to use data reduction methods that identify broad dimensions of difference that characterise the sample. The second route is to use data-driven clustering algorithms to identify subgroups of individuals. This is also essentially a data reduction technique, but it results in a novel data-driven grouping (Archibald et al., 2013; Bathelt et al., 2018; Kernbach et al., 2018).

In summary, transdiagnostic frameworks, whilst in their infancy, provide useful ways of capturing the underlying differences between individuals. They require us to rethink recruitment, measurement and analytic tools, relative to the traditional diagnostic forebearer. However, even this transdiagnostic reframing of divergence has rarely placed those differences within a developmental context, in which mechanisms and characteristics *change with time*. In turn, this neglect of developmental context makes it difficult to understand how complex profiles of difficulty can emerge over time, as difficulties cascade.

### *3. Hitting a moving target–why does development make it so hard to capture divergence?*

Measurement sensitivity of a particular task is likely to change dramatically across infancy, childhood and adolescence. Furthermore, the same measure can draw on different processes at different developmental stages. This is a particular example of the 'task impurity problem' (Hartung et al., 2018). Poor choice of measures can thus give the false appearance of developmental change, or conversely can mask genuine change. To provide an example, Simpson-Kent and colleagues (Simpson-Kent et al., 2020) used a wide battery that could be broadly characterised as capturing both 'fluid' and 'crystallised' cognitive abilities, in a sample from age 5 to 18 years. Vocabulary assessments are usually labelled as a measure of crystallised performance, because the assessment is designed to tax the participant's existing word knowledge. However, the authors demonstrated that the inter-individual variability captured by the assessment varies with age. Using SEM-Trees, a method that can partition structural equation models according to the measurement invariance (see Brandmaier et al., 2013), they established that in children under 9 years of age vocabulary predominantly captures variance associated with fluid measures (e.g., spatial working memory, matrix reasoning), and only from 9 years onwards does it predominantly capture variance associated with crystallised measures (e.g., literacy). In short, despite being a standardised validated measure, it appears to tax different aspects of cognition at different points in development.

The above example both illustrates the challenge and its potential solution. Capturing multiple measures, and then modelling their interrelationships, can provide insight as to *what* is being measured and how this measurement sensitivity may change across individuals and over time. In the example above, the aspects of cognition being taxed by the task change with age–for younger children the vocabulary measure draws upon fluid reasoning skills.

In short, there is a dearth of work validating paradigms to assess individual differences across the lifespan, or across the ability spectrum. There are some solutions, like collecting broader sets of measurements and modelling interrelationships, but even these solutions have their limits—partly due to the construction of current experimental tasks and to the lack of data on the most appropriate ways of measuring particular constructs over development.

As highlighted above, age can act as a strong moderating variable, shifting the target of an assessment. Another challenge of capturing divergence in developing populations is that the moderator could be a different aspect of cognition itself. This possibility becomes increasingly important as we try to accurately capture a range of neurodevelopmental profiles. Take as an example verbal working memory—the ability to hold in mind and manipulate phonological information for brief periods of time (Gathercole et al., 2004). Tasks probing this cognitive function could measure something different in children with a phonological awareness difficulty (Holmes et al., 2015). In this example, one cognitive function (phonological awareness) may 'gatekeep' another (working memory). If a participant cannot form good phonological representations, then the task may cease to be a good measure of their working memory.

This 'gatekeeping' phenomena can itself reflect a cognitive system that is self-organising over time. Consider again verbal working memory as an example. Because tasks like this recruit multiple component processes that depend upon one another for task performance, this task interdependence can itself come to shape neurocognitive development. Difficulties in verbal working memory tasks have been implicated across a wide-variety of neurodevelopmental disorders, including ADHD (Holmes, Guy, et al., 2021), autism (Williams et al., 2005) and dyslexia (Majerus & Cowan, 2016). However, because there are multiple cognitive

components that contribute to performance, there are different reasons why a child might find this task difficult and we cannot, on the basis of current assessments, be confident that all children present with 'working memory difficulties' because of comparable cognitive challenges. Studies with more inclusive recruitment criteria and a broad battery of cognitive tasks reveal that verbal working memory difficulties are common both in children with a wider executive function difficulty (alongside difficulties in spatial working memory, fluid reasoning etc.), and in children with relatively good executive function skills but poor phonological processing (alongside difficulties in phonological awareness and verbal short-term memory) (Astle et al., 2019). In short, because one cognitive domain might 'gatekeep' another, when this is allowed to play out over development, difficulties can cascade. If enough gatekeeping happens over time, then it constrains the development of other processes, and in turn shapes how the cognitive system organises itself (Karmiloff-Smith, 2009). To use the language of adaptation, difficulties within one aspect of functioning will shape how others develop.

This constructivist perspective, in which cognitive processes are interdependent, and this interdependence in turn shapes cognition across development (Karmiloff-Smith, 1994), highlights why development itself poses a substantive challenge to capturing and characterising divergence. Cognitive or behavioural difficulties are unlikely to remain selective across the course of development. For example, early difficulties in hyperactivity may shape emerging pragmatic language skills (Hawkins et al., 2016), which in turn may shape wider social skills and elevate the risk of peer rejection (de Boo & Prins, 2007). Population-level cohorts, provided they have sufficiently rich data, may provide a way of exploring these cascades between different aspects of functioning. These studies could provide invaluable prospective longitudinal data, sometimes since birth, that demonstrate how complex profiles of relative strength and difficulty in late childhood and adolescence could reflect cascading differences over many years. The challenge is to improve our measures and to create longitudinal cohorts that indeed have that breadth of assessment over time.

The inevitable consequence of this interconnectedness is that difficulties at one particular level can arise from different causal routes at lower levels, a notion commonly termed equifinality (Bishop, 1997; Cicchetti & Rogosch, 1996). One of the key demonstrations that makes the case for the transdiagnostic approach—that some common behavioural or cognitive characteristics appear to arise in supposedly distinct diagnostic groups—may itself reflect equifinal developmental mechanisms. Put simply, certain symptoms are common because they can be arrived at via multiple causal routes (Astle et al., 2019). Elsewhere in medicine this mechanistic convergence is commonplace—surface level features, like fever, can have multiple causal antecedents (e.g., viral infection, bacterial infection, heat exhaustion, inflammatory conditions, etc.). In summary, capturing divergence in developing populations is challenging because assessments likely measure different things at different points in time and because one cognitive difficulty might 'gatekeep' and shape others.

So what is the solution to these challenges posed by developmental biology and cognition to our understanding of individual differences? *It may be that we should consider softening our adherence to group-averages.* The majority of our knowledge of development, and indeed divergence, stems from a group-average approach. We typically identify children with a particular diagnostic label, group them and compare groups on a host of measures. A significant difference on a measure is then thought to reflect the underlying 'core deficit' that gives rise to the wider diagnostic phenotype. This logic has given rise to the majority of our theoretical accounts of neurodevelopmental divergence, from the theory of mind 'deficit' account of autism (Baron-Cohen, 1995) to the executive function deficit theory of ADHD (Willcutt et al., 2005). However, this logic has been widely critiqued (Bishop, 1997; Happé et al., 2006; Pennington, 2006). Moreover, these theories have been notoriously difficult to validate empirically. Deficits apparently 'core' to a particular condition are often present among other groups, so they are not nearly as distinctive after all; not all individuals with a particular diagnosis have the supposed deficit, so they are not nearly so shared as previously thought; and they typically fail to account for the wide range of differences seen across individuals with

the same diagnostic label, so, as explanations go, not so powerful in the end (see Astle & Fletcher-Watson, 2020 for a review). This way of framing divergence–a difference between group means–is at the heart of why these theories have so often proven to be a conceptual dead end. The critique is highly overlapping with that of the diagnostic framework in which those theories are situated. Neither the labels, nor their associated theories, can capture the variability within a category, explain the characteristics that are commonly shared across supposedly distinct categories, or illuminate the co-occurrence of difficulties (see Astle et al., 2021 for a review). Especially crucial for our purposes here, the adherence to group-average approach makes it very difficult to place difficulties within a developmental context, simply because developmental routes into a particular profile are lost within a group average.

If our current understanding of divergence is largely informed by group averaging then *what are the alternatives*? There are many recent computational advances that could provide an alternative methodological, and in turn conceptual, approach, relative to conventional group comparisons. The first example is Hierarchical Bayesian Modelling (HBM; McGlothlin & Viele, 2018). This family of models allows the researcher to separate and estimate parameters at multiple levels, thereby characterising the learning of an individual (Figure 2). For instance, it is possible to model how individuals learn about the likelihood of different stimuli being presented under different conditions. Because the model is fit to the individual, the researcher can in principle disentangle the potential influence of different sources of information in that learning process. The combination of this type of modelling with carefully designed behavioural paradigms, in which those sources of information are varied systematically, provides a formal way of characterising the underlying processes by which individuals integrate information as they perform the task (e.g., Lawson et al., 2017). A full review of the application of HBM is beyond the scope of the current review; moreover, there are likely multiple other methods that would do just as well. The crucial ingredient for our illustrative purposes is that this method provides mechanistic insight into behaviour without recourse to group average performance. A second example methodology is ‘idiographic symptom

networks' (Figure 2). These are an extension of the more commonly used tools of group-level network science, in which nodes correspond to particular measures and the edges between them correspond to the partial relationships between them (e.g., Dalmaijer et al., 2021). Network analysis, when applied to the group, provides a way of mapping the interactions between multiple symptoms simultaneously. The application of graph theory can provide a way of characterising the organisational properties of that network, such as whether particular nodes act as hubs, connecting other more disparate symptoms. Where there is sufficiently rich data available for a given individual, these networks can be computed at the level of an individual (Fisher et al., 2017; Howe et al., 2020; Lydon-Staley, Leventhal, et al., 2021; Piccirillo & Rodebaugh, 2019). Put simply, understanding *how* and *when* divergence in cognition or behaviour emerges may require us to move beyond the group average, and instead to use methods that allow us to model causes at the level of an individual.

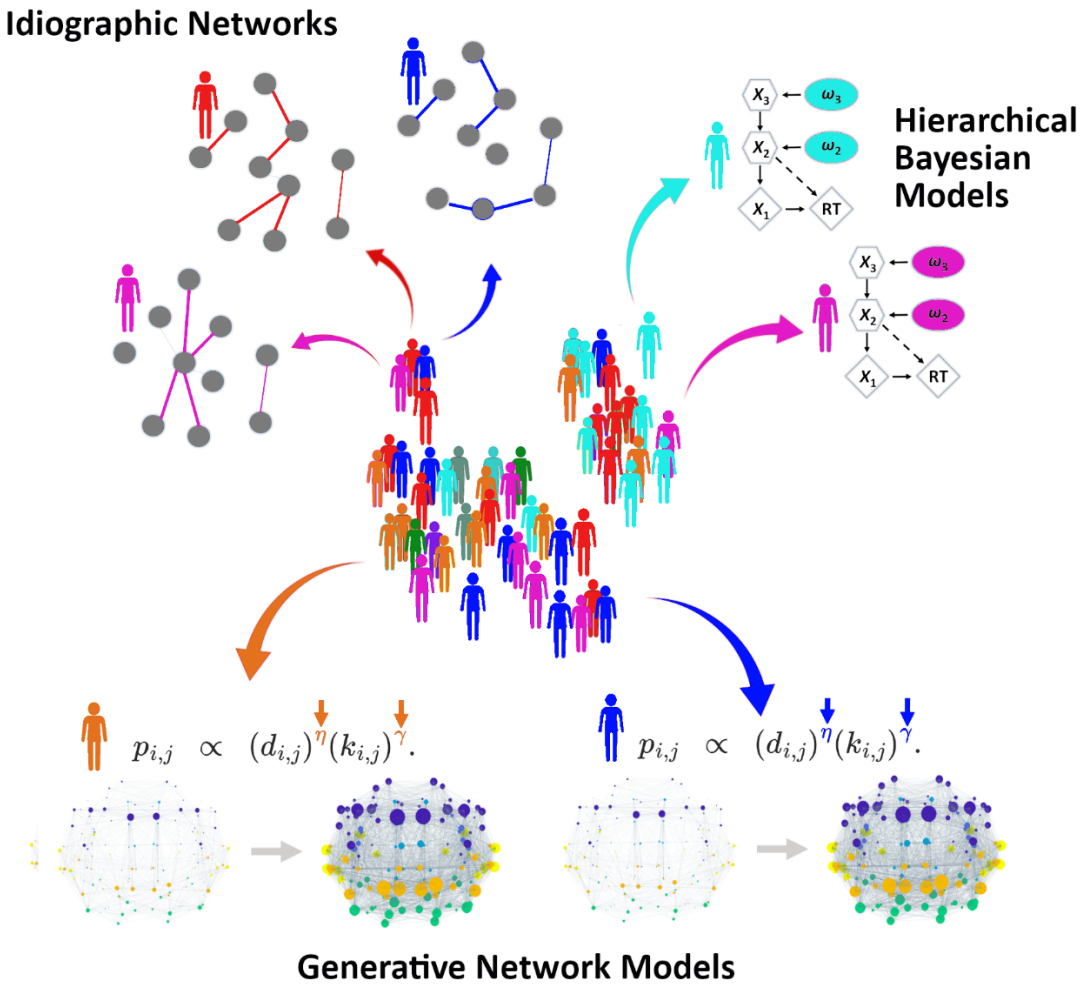

***Figure 2:*** *Methods for moving beyond group averages. This figure depicts three methods that allow us to characterise individuals in different ways. First, Idiographic Networks use repeated measures of relevant characteristics, such as clinical symptoms, to build network models that capture the organisation of, and relationships between, those characteristics at the level of an individual. Second, Hierarchical Bayesian models use repeated measures, for instance performance across trials, to build a multi-component model that fits the performance of an individual. Careful paradigm design allows the researcher to separate different hierarchically nested processes. Third, Generative Network models use a simple wiring equation to simulate the formation of a network that is fit to an individual's observed connectome. The probability (p) of nodes i and j connecting reflects the trade-off between the costs of those connections forming (d) and the topological value that a connection would confer (k). This process simulates the formation of complex networks via the iterative addition of individual connections. The resulting parameters from the best fit models provide a compressed representation of the economic constraints that shape the formation of an individual's connectome.*

## *4. Divergent neurobiology: how and when do we detect differences in the brain?*

Multiple theoretical perspectives agree that the functional specialisation of particular brain regions or circuits is not deterministic, but instead emerges probabilistically (Fair et al., 2007; Johnson, 2000, 2011; Jones et al., 2021). That is, the functional role of any neural assembly is shaped gradually over developmental time, and in part this shaping depends upon the connectivity of that assembly, in particular its inputs (Gottlieb, 2005, 2007). Likewise, cognitive processes do not simply come 'online' when a neural circuit matures. Instead nascent functional circuits support emerging cognitive processes, and as they are engaged in the service of those processes this repeated co-activity will itself shape future functional specialisation (Johnson, 2011). Put simply, relationships between brain and behaviour are not static. Their adaptive nature means that they will shape one another over development. This theoretical standpoint seems at odds with discoveries of particularly strong relationships between individual brain regions or circuits and particular behaviours of task performance (Kowalczyk et al., 2021). We are not the first to outline the challenge of dynamic brain-behaviour relationships over time (e.g., Karmiloff-Smith, 2010; Lessov-Schlaggar et al., 2016). The field is broadly aware that we tend to rely on individual measures to establish univariate brain-behaviour relationships, when in reality we are assessing a highly interconnected system that is changing dynamically with time, in individuals whose brain-behaviour relationships are also likely changing. Despite this challenge being articulated previously,

progress towards redressing it has been slow. Part of the problem is undoubtedly statistical power, to which the solution is shared datasets that afford analyses at scale. However, new larger datasets that have sampled individuals across the population (e.g., ACBD, Karcher & Barch, 2021) or selectively sampled children at neurodevelopmental risk (e.g., CALM, PDN) afford more than just additional power *per se*. A key ingredient to the success of many of these large-scale studies is that they are, in essence, multi-lab endeavours. This collaborative approach allows us to gather samples with greater generalizability, capture different contextual or cultural contexts that might be relevant for dynamic developmental processes, and facilitate sharing of best practice and enhanced reproducibility. This collaborative approach also spreads the recruitment load, something valuable in any project but especially so if the sample is enriched for neurodivergent young people. A larger sample size means that we can run analyses on a training sample while holding out a testing or replication sample. This capacity is important because many contemporary multivariate analytic tools, such as CCA or partial least squares (PLS) risk over-fitting. The ability to test whether relationships between neural measures and behaviour measures generalise to unseen data is crucial for establishing more reliable brain-behaviour relationships.

However, the barrier to identifying robust brain-behaviour relationships in developmental samples is not just one of statistical power afforded by a large sample size; our challenge is also conceptual. Just as group-averaging at a behavioural level will inevitably mask developmental trajectories into a profile, the same logical problem applies to the hunt for neural correlates of divergence. For instance, grouping individuals according to diagnostic status and comparing neural characteristics voxel by voxel, in search of the 'neural seat of [*insert diagnosis*]', has yielded remarkably inconsistent results. For example, ADHD has been associated with differences in grey matter within the anterior cingulate cortex (Amico et al., 2011), caudate nucleus (Onnink et al., 2014), pallidum (Frodl & Skokauskas, 2012), striatum (Greven et al., 2015), cerebellum (Mackie et al., 2007), prefrontal cortex (Dirlikov et al., 2015), premotor cortex (Mahone et al., 2011), and most parts of the parietal lobe (Shaw et al., 2006).

The variety of brain regions identified is itself not problematic; we might expect group differences to be distributed. More troubling is the difficulty in replicating group average differences across studies. Indeed, a recent meta-analysis of almost 2000 participants found *no consistent* significant functional connectivity differences between ADHD participants and controls (Figure 3; Cortese et al., 2021). This inconsistency does not just reflect a lack of power. As noted earlier, relationships between brain and behaviour measures are non-stationary. Their adaptive nature means that brain and behaviour will shape one another over development, and there are likely many neural routes to the same behavioural profile.

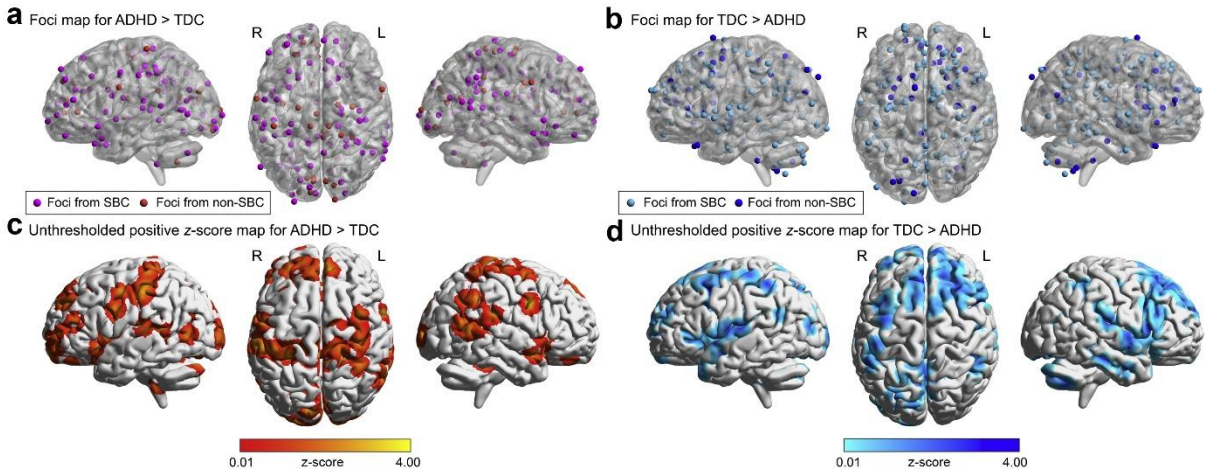


***Figure 3:*** *Taken with permission from Cortese et al. (2021). Foci with hyperconnectivity in attention-deficit/hyperactivity disorder (ADHD) reported in seed-based connectivity (SBC) studies and non-SBC studies are shown in purple and red, respectively (a). Foci with hypoconnectivity in ADHD reported in SBC and non-SBC studies are shown in light blue and dark blue, respectively (b). Unthresholded positive z-score maps are shown for hyperconnectivity (c) and hypoconnectivity (d). L = left; R = right; TDC = typically developing controls.*

Possible solutions could be found in the nascent neuro-AI movement. Growing numbers of researchers are building artificial models inspired by (e.g., Achterberg, Akarca et al. 2023), or directly implementing (e.g., Suarez et al. 2022), neurobiology. For example, generative network modelling (GNM) (Betzel et al., 2016; Kaiser & Hilgetag, 2004; Vértes et al., 2012) is a biophysically grounded computational framework in which the formation of a functional (Vértes et al., 2012) or structural connectome (Akarca et al., 2021) is simulated for a given individual (Betzel & Bassett, 2017). Within the generative model connections form

probabilistically to reflect the trade-off between the energetic cost of forming that connection and the topological value the connection would yield. This framework allows the researcher to formalise different theoretical accounts of the basis upon which networks form over developmental time, and then to implement, test, and refine those models. Crucially, this simulation can occur at the level of an individual's connectome, meaning that each simulation is unique to that person. This outcome is achieved by individually calibrating the relative trade-off between the components of the generative model (Figure 3). The typical approach is to trade off the relative distance a new connection must span as a measure of 'cost', against the proportion of shared neighbours as the measure of topological 'value'. Whilst the costs are fixed across time, a node's neighbourhood is updated dynamically as connections are added, thereby creating a stochastic computational process (Carozza, Akarca & Astle, 2023). Recent work has used this approach to show that each brain forms a network according to a subtly different economic trade-off (Akarca et al., 2021). These subtle differences cascade and amplify over development to produce the large amount of variability in connectome organisation that we see across children during development. This approach is currently constrained by its relative biological plausibility–whilst it is biophysically embedded, the networks only form one connection at a time. Future changes will need to shift this framework to capture more realistic weighted changes within networks (see Akarca et al., 2023). Nonetheless, the approach provides another example of how we can model and understand developmental processes without recourse to group averaging. Whilst promising, a key barrier to the application and utility of AI-informed methodologies to developmental contexts is simply the difficulties in their operation and interpretability, particularly in those without backgrounds in engineering or computer sciences. The best solution is surely multi-disciplinary teams who speak a common language that allows for the integration of computational expertise with theoretical insight.

### *5. Divergent systems*

While the group average hampers our ability to understand the individual person, the focus on individual brain areas hampers our ability to understand the emergent dynamics only possible from a group of brain areas. Hence, while drilling down from the sample to the single human, we must simultaneously expand upward, from the individual brain region to the circuit and connectome. This expansion mirrors more closely the nature of cognition and behaviour, which together arise from the complex system of the brain-body union. That tighter connection, between method and the nature of the object of study, fosters greater potential for fundamental understanding. But this tighter connection also requires increasingly careful development of practical tools and notional structures, from algorithms to theory. As noted in a previous section, our methodological approach and conceptual framework must go hand-in-hand.

The recognition that complex systems like the brain cannot be explained by their separate components has gained momentum over recent years (Bassett & Gazzaniga, 2011; Bullmore et al., 2009). The associated progress in approaching neuroimaging data from the complex systems' perspective has benefited greatly from the near simultaneous development and expansion of network science (Newman, 2010). A relatively young scholarly field of study, network science saw its first formal institutes around the year 2000, and its first undergraduate and graduate degrees about ten years later. Spanning from models and data analytics to conceptual frameworks, network science studies (and formalises the study of) complex systems composed of many interacting parts. Of particular interest are such systems in which the pattern of part-to-part interactions is critically important for the observed function. A counterpoint to reductionist thinking, network science stems from the acknowledgement that the ability to isolate and understand simple units does not imply the ability to start from those units and reconstruct the system. Rather, one must understand how the parts are related, and how those relationships both constrain and support system processes.

The approach is particularly relevant to our focus here, as the study of neurodevelopmental differences has historically relied on unit-level models, which have precluded holistic understanding. Specifically, neural correlates of such differences have typically been

evaluated through the lens of a long-standing model from adult neuropsychology, in which damage to an established brain unit can result in a specific profile of difficulties (Bishop, 1997). Neurodevelopmental differences do not fit well with this model for two key reasons. First, such differences do not reflect differences in individual brain regions. No single brain area underpins inattention or hyperactivity, or poor working memory (Siugzdaite et al., 2020). In fact, more broadly, the map from unit to difficulty profile (or vice versa) is not one-to-one: several units are implicated in a single difficulty, and a single unit is altered across many difficulties. This many-to-many map between region and profile calls for a connectivity-based approach. Such an approach is well-placed to provide a greater understanding of developmental phenomena like equi- and multifinality. Second, neurodevelopmental differences emerge across time as the system engineers itself (Gottlieb, 2007). They arise on the backdrop of a time-evolving set of interactions between brain regions, between brain and behaviour, and between behaviours. Understanding the provenance of these difficulties, and targeting effective intervention, hence requires an understanding of network dynamics across brain and behaviour.

In what follows, we will briefly describe approaches that expand the study of neurodevelopmental differences beyond the model of adult neuropsychology, and in a manner that foregrounds emergence across brain networks and across time.

*Emergence across brain networks*

Approaches that expand beyond single brain regions to better understand the neural dynamics that accompany neurodevelopmental differences span from descriptive to predictive network approaches. Descriptive network approaches typically use graph metrics to quantitatively map variation in the architecture of connectivity patterns (Rubinov & Sporns, 2010). Such approaches have elucidated links between variation in network topology and variation in cognition and symptomatology (Chu-Shore et al., 2011; Parkes et al., 2020; Zhang et al., 2021). More recently, predictive network approaches have focused on how activity in one or a few brain regions can predictably drive changes in other brain regions. Two key examples include the finite impulse response approach (Blaauw et al., 2017; de Zwart et al., 2005) and

network control theory (Lynn & Bassett, 2019). Impulse response analysis uses the pattern of connections between regions to predict the degree to which a change in the activity of region *i* (i.e., an *impulse*) will drive a change in the activity of other regions *j* (i.e., a *response*) (Lydon-Staley, Cornblath, et al., 2021). The approach has proven useful in distinguishing regional influence on task-based fMRI activity in conditions ranging from schizophrenia to 22q11.2ds (Cornblath et al., 2021; Woodward et al., 2015), and in cognitive processes ranging from working memory to emotion discrimination (Goghari et al., 2017; Sanford et al., 2020). Moreover, the approach has also shown extensive utility in predicting activity flow in a second kind of network—state networks, where single states, emotions, behaviours, or symptoms are represented by network nodes and where inferred causal relations between nodes are represented by network edges. In this context, impulse response analysis has been used to pinpoint influential emotions and symptoms, and their temporal dynamics, in depression, anxiety, and tobacco withdrawal (Bos et al., 2018; Lydon-Staley, Leventhal, et al., 2021; Yang et al., 2019).

A step beyond impulse response models, network control theory expands the question of interest from “How does a single unit drive change in the system?” to “How can time-varying changes in multiple units predictably drive the system to a desired state?”. Network control theory provides a model, analytic solutions, and associated algorithms to answer this question, and has been quite broadly developed and applied across mechanical, robotic, technological, physical, and biological systems (Cornelius et al., 2013; Liu et al., 2011; Pasqualetti et al., 2014; Ruths & Ruths, 2014). In the context of the brain, network control theory has been used to determine how the development of brain connectivity constrains (and supports) the attainment of an increasingly diverse dynamical repertoire (Tang et al., 2017); how individual differences in that development explain individual differences in executive function (Cui et al., 2020) and positive psychosis spectrum symptoms (Parkes, Moore, Calkins, Cieslak, et al., 2021); and how variations in dopamine and serotonin receptor function (either by natural biology or by drug) alter the ease versus difficulty of reaching particular brain and cognitive

559 states (Braun et al., 2021; Singleton et al., 2021). The approach is absolutely central to the
560 understanding of neurodevelopmental differences, as it provides an explicit formal link
561 between brain network organisation and system function as evinced by dynamical transitions
562 in state with relevance to behaviour.

563 *Emergence across time*

564 To complement the expansion from region to network, we now turn to questions posed by the
565 fact that brain, behaviour, environment, and their interrelations, change over a range of
566 timescales from moments to years. Precisely how do neurodevelopmental differences reflect
567 emergent properties of the system organising itself across developmental time according to
568 different underlying constraints? What tools are available to study these dynamics? One
569 particularly powerful approach is known as *normative modelling*, which maps the typical (or
570 "normative") developmental trajectory, function, or curve of a brain feature (Marquand et al.,
571 2016), and then measures how single individuals diverge from that curve (Wolfers et al., 2018,
572 2020; Zabihi et al., 2019). The approach has been used to map the heterogeneous
573 phenotypes of schizophrenia, bipolar disorder, ADHD, and autism (Wolfers et al., 2018, 2020;
574 Zabihi et al., 2019). In applying this approach to transdiagnostic dimensions of
575 psychopathology, recent work has further shown that modelling features of cortical brain
576 structure as deviations from normative neurodevelopment improves the prediction of
577 psychiatric symptoms in out-of-sample testing (Parkes, Moore, Calkins, Cook, et al., 2021).
578 Importantly, more general deviations can also take the form of acceleration or delay of 'typical'
579 development, which is itself associated with a variety of factors including socioeconomic status
580 (Tooley et al., 2021).

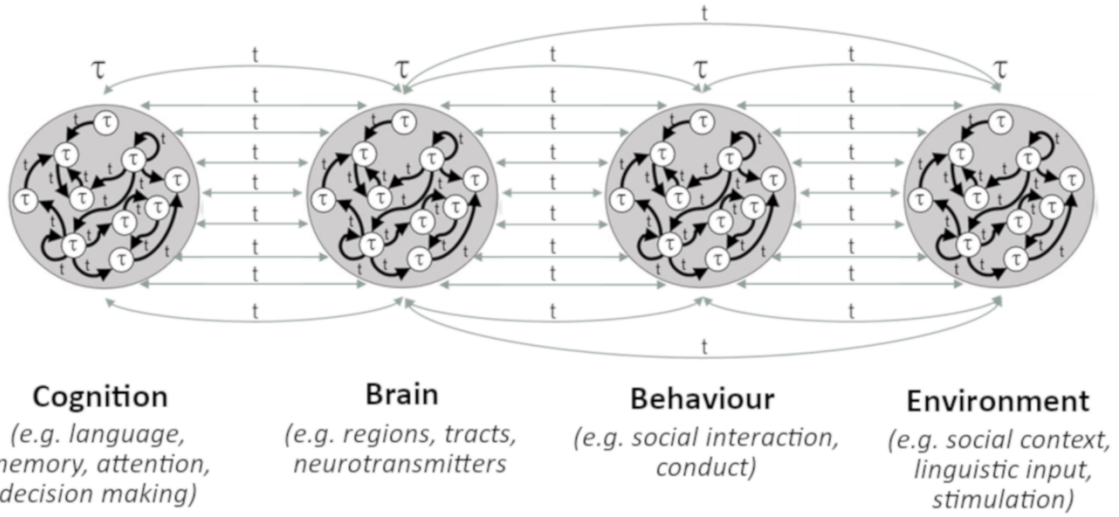


***Figure 4:*** *The network of networks. A depiction of the interconnectedness both within and across levels of analysis. The principle challenge of studying neurodevelopment, and the differences therein, is to understand how the system organises itself at one particular level, and how deviations within that level can influence, and be influenced by, emerging organisation at different levels.*

Now, how do we expand beyond a single factor whose normative trajectory can be modelled, and instead understand how advance, delay, or deviation in one factor might change the possible trajectories of another factor? Gaining such an understanding is further complicated by the fact that inter-factor dependencies arise bidirectionally in time (Figure 4). For example, we know that changes in brain structure can drive changes in brain function, just as changes in brain function can drive changes in brain structure. Similarly, changes in function can drive changes in gene expression, just as changes in gene expression can drive changes in function. Changes in the environment can alter one's experiences—and by extension—neural representations and adaptations, just as changes in neurodevelopment can alter the way in which one engages with the environment—and by extension—one's experiences. These complex bidirectional relationships call for approaches that embrace the time-dependent nature of interconnections both within and between brain, behaviour, and environment. In the parlance of network science, the question is one of understanding a dynamic *network of networks*, and expanding impulse response and network control approaches to this fuller picture.

## Conclusion

Understanding divergence and its drivers requires that we place differences within a developmental context. More representative sampling frames and valid assessments are merely the first step towards this goal. The ultimate aim is to incorporate the developmental dynamics that capture how, and ultimately when, divergence takes place. We need an analytic toolkit that is equal to these ambitions. There is greater potential for fundamental advances in our understanding of neurodevelopmental differences with a tighter coupling between our conceptual and methodological frameworks; if our methods are only able to capture simple relationships, at isolated moments in time, then our theories will be similarly constrained.

**References**


Achterberg, J., Akarca, D., Strouse, D. J., Duncan, J., & Astle, D. E. (2023). Spatially embedded recurrent neural networks reveal widespread links between structural and functional neuroscience findings. Nature Machine Intelligence, 1-13.

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). https://doi.org/10.1176/appi.books.9780890425596

Akarca, D., Schiavi, S., Achterberg, J., Genc, S., Jones, D., & Astle, D. (2023). A weighted generative model of the human connectome. bioRxiv, 2023-06.

Akarca, D., Vértes, P. E., Bullmore, E. T., & Astle, D. E. (2021). A generative network model of neurodevelopmental diversity in structural brain organization. *Nature Communications*, *12*(1), 4216. https://doi.org/10.1038/s41467-021-24430-z

Amico, F., Stauber, J., Koutsouleris, N., & Frodl, T. (2011). Anterior cingulate cortex gray matter abnormalities in adults with attention deficit hyperactivity disorder: A voxel-based morphometry study. *Psychiatry Research*, *191*(1), 31–35. https://doi.org/10.1016/j.pscychresns.2010.08.011

Archibald, L. M. D., Cardy, J. O., Joanisse, M. F., & Ansari, D. (2013). Language, Reading, and Math Learning Profiles in an Epidemiological Sample of School Age Children. *PLOS ONE*, *8*(10), e77463. https://doi.org/10.1371/journal.pone.0077463

Astle, D. E., Bathelt, J., & Holmes, J. (2019). Remapping the cognitive and neural profiles of children who struggle at school. *Developmental Science*, *22*(1), e12747. https://doi.org/10.1111/desc.12747

Astle, D. E., & Fletcher-Watson, S. (2020). Beyond the Core-Deficit Hypothesis in Developmental Disorders. *Current Directions in Psychological Science*, *29*(5), 431–437. https://doi.org/10.1177/0963721420925518

Astle, D. E., Holmes, J., Kievit, R., & Gathercole, S. E. (2021). Annual Research Review: The transdiagnostic revolution in neurodevelopmental disorders. *Journal of Child Psychology and Psychiatry*. https://doi.org/10.1111/jcpp.13481

Baron-Cohen, S. (1995). *Mindblindness: An essay on autism and theory of mind.* (pp. xxii, 171). The MIT Press.

Bassett, D. S., & Gazzaniga, M. S. (2011). Understanding complexity in the human brain. *Trends in Cognitive Sciences*, *15*(5), 200–209. https://doi.org/10.1016/j.tics.2011.03.006

Bathelt, J., Holmes, J., & Astle, D. E. (2018). Data-Driven Subtyping of Executive Function–Related Behavioral Problems in Children. *Journal of the American Academy of Child and Adolescent Psychiatry*, *57*(4), 252-262.e4. https://doi.org/10.1016/j.jaac.2018.01.014

Bathelt, J., Vignoles, A., & Astle, D. E. (2021). Just a phase? Mapping the transition of behavioural problems from childhood to adolescence. *Social Psychiatry and Psychiatric Epidemiology*, *56*(5), 821–836. https://doi.org/10.1007/s00127-020-02014-4

Betzel, R. F., Avena-Koenigsberger, A., Goñi, J., He, Y., de Reus, M. A., Griffa, A., Vértes, P. E., Mišic, B., Thiran, J.-P., Hagmann, P., van den Heuvel, M., Zuo, X.-N., Bullmore, E. T., & Sporns, O. (2016). Generative models of the human connectome. *NeuroImage*, *124*(Pt A), 1054–1064. https://doi.org/10.1016/j.neuroimage.2015.09.041

Betzel, R. F., & Bassett, D. S. (2017). Generative models for network neuroscience: Prospects and promise. *Journal of The Royal Society Interface*, *14*(136), 20170623. https://doi.org/10.1098/rsif.2017.0623

Bishop, D. V. (1997). Cognitive neuropsychology and developmental disorders: Uncomfortable bedfellows. *The Quarterly Journal of Experimental Psychology. A, Human Experimental Psychology*, *50*(4), 899–923. https://doi.org/10.1080/713755740

Blaauw, F. J., van der Krieke, L., Emerencia, A. C., Aiello, M., & de Jonge, P. (2017). Personalized advice for enhancing well-being using automated impulse response analysis—AIRA. *ArXiv:1706.09268 [Cs]*. http://arxiv.org/abs/1706.09268

Bos, F. M., Blaauw, F. J., Snippe, E., van der Krieke, L., de Jonge, P., & Wichers, M. (2018). Exploring the emotional dynamics of subclinically depressed individuals with and without anhedonia: An experience sampling study. *Journal of Affective Disorders*, *228*, 186–193. https://doi.org/10.1016/j.jad.2017.12.017

Brandmaier, A. M., von Oertzen, T., McArdle, J. J., & Lindenberger, U. (2013). Structural equation model trees. *Psychological Methods*, *18*(1), 71–86. https://doi.org/10.1037/a0030001

Braun, U., Harneit, A., Pergola, G., Menara, T., Schäfer, A., Betzel, R. F., Zang, Z., Schweiger, J. I., Zhang, X., Schwarz, K., Chen, J., Blasi, G., Bertolino, A., Durstewitz, D., Pasqualetti, F., Schwarz, E., Meyer-Lindenberg, A., Bassett, D. S., & Tost, H. (2021). Brain network dynamics during working memory are modulated by dopamine and diminished in schizophrenia. *Nature Communications*, *12*(1), 3478. https://doi.org/10.1038/s41467-021-23694-9

Bull, R., Espy, K. A., & Wiebe, S. A. (2008). Short-Term Memory, Working Memory, and Executive Functioning in Preschoolers: Longitudinal Predictors of Mathematical Achievement at Age 7 Years. *Developmental Neuropsychology*, *33*(3), 205–228. https://doi.org/10.1080/87565640801982312

683 Bullmore, E., Barnes, A., Bassett, D. S., Fornito, A., Kitzbichler, M., Meunier, D., & Suckling,
684 J. (2009). Generic aspects of complexity in brain imaging data and other biological
685 systems. *NeuroImage*, *47*(3), 1125–1134.
686 https://doi.org/10.1016/j.neuroimage.2009.05.032
687 Carozza, S., Akarca, D., & Astle, D. (2023). The adaptive stochasticity hypothesis: modelling
688 equifinality, multifinality and adaptation to adversity. *Proceedings of the National*
689 *Academy of Sciences*, 120 (42) e2307508120
690 Caspi, A., Houts, R. M., Ambler, A., Danese, A., Elliott, M. L., Hariri, A., Harrington, H.,
691 Hogan, S., Poulton, R., Ramrakha, S., Rasmussen, L. J. H., Reuben, A., Richmond-
692 Rakerd, L., Sugden, K., Wertz, J., Williams, B. S., & Moffitt, T. E. (2020). Longitudinal
693 Assessment of Mental Health Disorders and Comorbidities Across 4 Decades Among
694 Participants in the Dunedin Birth Cohort Study. *JAMA Network Open*, *3*(4), e203221.
695 https://doi.org/10.1001/jamanetworkopen.2020.3221
696 Chapman, R. (2020). Neurodiversity, disability, wellbeing. In *Neurodiversity Studies*.
697 Routledge.
698 Chu-Shore, C. J., Kramer, M. A., Bianchi, M. T., Caviness, V. S., & Cash, S. S. (2011).
699 Network analysis: Applications for the developing brain. *Journal of Child Neurology*,
700 *26*(4), 488–500. https://doi.org/10.1177/0883073810385345
701 Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental
702 psychopathology. *Development and Psychopathology*, *8*(4), 597–600.
703 https://doi.org/10.1017/S0954579400007318
704 Coghill, D., & Sonuga-Barke, E. J. S. (2012). Annual research review: Categories versus
705 dimensions in the classification and conceptualisation of child and adolescent mental
706 disorders--implications of recent empirical study. *Journal of Child Psychology and*
707 *Psychiatry, and Allied Disciplines*, *53*(5), 469–489. https://doi.org/10.1111/j.1469-
708 7610.2011.02511.x
709 Connelly, R., & Platt, L. (2014). Cohort profile: UK millennium Cohort study (MCS).
710 International journal of epidemiology, 43(6), 1719-1725.

Cornblath, E. J., Mahadevan, A., He, X., Ruparel, K., Lydon-Staley, D. M., Moore, T. M., Gur, R. C., Zackai, E. H., Emanuel, B., McDonald-McGinn, D. M., Wolf, D. H., Satterthwaite, T. D., Roalf, D. R., Gur, R. E., & Bassett, D. S. (2021). Altered functional brain dynamics in chromosome 22q11.2 deletion syndrome during facial affect processing. *Molecular Psychiatry*, 1–9. https://doi.org/10.1038/s41380-021-01302-y

Cornelius, S. P., Kath, W. L., & Motter, A. E. (2013). Realistic control of network dynamics. *Nature Communications*, *4*(1), 1942. https://doi.org/10.1038/ncomms2939

Cortese, S., Aoki, Y. Y., Itahashi, T., Castellanos, F. X., & Eickhoff, S. B. (2021). Systematic Review and Meta-analysis: Resting-State Functional Magnetic Resonance Imaging Studies of Attention-Deficit/Hyperactivity Disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, *60*(1), 61–75. https://doi.org/10.1016/j.jaac.2020.08.014

Cui, Z., Stiso, J., Baum, G. L., Kim, J. Z., Roalf, D. R., Betzel, R. F., Gu, S., Lu, Z., Xia, C. H., He, X., Ciric, R., Oathes, D. J., Moore, T. M., Shinohara, R. T., Ruparel, K., Davatzikos, C., Pasqualetti, F., Gur, R. E., Gur, R. C., … Satterthwaite, T. D. (2020). Optimization of energy state transition trajectory supports the development of executive function during youth. *ELife*, *9*, e53060. https://doi.org/10.7554/eLife.53060

Dalmaijer, E. S., Gibbons, S. G., Bignardi, G., Anwyl-Irvine, A. L., Siugzdaite, R., Smith, T. A., Uh, S., Johnson, A., & Astle, D. E. (2021). Direct and indirect links between children's socio-economic status and education: Pathways via mental health, attitude, and cognition. *Current Psychology*. https://doi.org/10.1007/s12144-021-02232-2

de Boo, G. M., & Prins, P. J. M. (2007). Social incompetence in children with ADHD: Possible moderators and mediators in social-skills training. *Clinical Psychology Review*, *27*(1), 78–97. https://doi.org/10.1016/j.cpr.2006.03.006

de Zwart, J. A., Silva, A. C., van Gelderen, P., Kellman, P., Fukunaga, M., Chu, R., Koretsky, A. P., Frank, J. A., & Duyn, J. H. (2005). Temporal dynamics of the BOLD fMRI

impulse response. *NeuroImage*, *24*(3), 667–677. https://doi.org/10.1016/j.neuroimage.2004.09.013

Dirlikov, B., Shiels Rosch, K., Crocetti, D., Denckla, M. B., Mahone, E. M., & Mostofsky, S. H. (2015). Distinct frontal lobe morphology in girls and boys with ADHD. *NeuroImage. Clinical*, *7*, 222–229. https://doi.org/10.1016/j.nicl.2014.12.010

Elliott, M. L., Knodt, A. R., Ireland, D., Morris, M. L., Poulton, R., Ramrakha, S., Sison, M. L., Moffitt, T. E., Caspi, A., & Hariri, A. R. (2020). What Is the Test-Retest Reliability of Common Task-Functional MRI Measures? New Empirical Evidence and a Meta-Analysis. *Psychological Science*, *31*(7), 792–806. https://doi.org/10.1177/0956797620916786

Enkavi, A. Z., Eisenberg, I. W., Bissett, P. G., Mazza, G. L., MacKinnon, D. P., Marsch, L. A., & Poldrack, R. A. (2019). Large-scale analysis of test–retest reliabilities of self-regulation measures. *Proceedings of the National Academy of Sciences*, *116*(12), 5472–5477. https://doi.org/10.1073/pnas.1818430116

Fair, D. A., Dosenbach, N. U. F., Church, J. A., Cohen, A. L., Brahmbhatt, S., Miezin, F. M., Barch, D. M., Raichle, M. E., Petersen, S. E., & Schlaggar, B. L. (2007). Development of distinct control networks through segregation and integration. *Proceedings of the National Academy of Sciences of the United States of America*, *104*(33), 13507–13512. https://doi.org/10.1073/pnas.0705843104

Fisher, A. J., Reeves, J. W., Lawyer, G., Medaglia, J. D., & Rubel, J. A. (2017). Exploring the idiographic dynamics of mood and anxiety via network analysis. *Journal of Abnormal Psychology*, *126*(8), 1044–1056. https://doi.org/10.1037/abn0000311

Foulkes, L., & Blakemore, S. J. (2018). Studying individual differences in human adolescent brain development. Nature neuroscience, 21(3), 315-323.

Frodl, T., & Skokauskas, N. (2012). Meta-analysis of structural MRI studies in children and adults with attention deficit hyperactivity disorder indicates treatment effects. *Acta Psychiatrica Scandinavica*, *125*(2), 114–126. https://doi.org/10.1111/j.1600-0447.2011.01786.x

767 Gathercole, S. E., Pickering, S. J., Ambridge, B., & Wearing, H. (2004). The structure of
768 working memory from 4 to 15 years of age. *Developmental Psychology*, *40*(2), 177–
769 190. https://doi.org/10.1037/0012-1649.40.2.177
770 Goghari, V. M., Sanford, N., Spilka, M. J., & Woodward, T. S. (2017). Task-Related
771 Functional Connectivity Analysis of Emotion Discrimination in a Family Study of
772 Schizophrenia. *Schizophrenia Bulletin*, *43*(6), 1348–1362.
773 https://doi.org/10.1093/schbul/sbx004
774 Gottlieb, G. (2005). *Handbook of Developmental Psychology* (By pages 3-17). SAGE
775 Publications Ltd. https://doi.org/10.4135/9781848608306
776 Gottlieb, G. (2007). Probabilistic epigenesis. *Developmental Science*, *10*(1), 1–11.
777 https://doi.org/10.1111/j.1467-7687.2007.00556.x
778 Greven, C. U., Bralten, J., Mennes, M., O'Dwyer, L., van Hulzen, K. J. E., Rommelse, N.,
779 Schweren, L. J. S., Hoekstra, P. J., Hartman, C. A., Heslenfeld, D., Oosterlaan, J.,
780 Faraone, S. V., Franke, B., Zwiers, M. P., Arias-Vasquez, A., & Buitelaar, J. K.
781 (2015). Developmentally stable whole-brain volume reductions and developmentally
782 sensitive caudate and putamen volume alterations in those with attention-
783 deficit/hyperactivity disorder and their unaffected siblings. *JAMA Psychiatry*, *72*(5),
784 490–499. https://doi.org/10.1001/jamapsychiatry.2014.3162
785 Happé, F., Ronald, A., & Plomin, R. (2006). Time to give up on a single explanation for
786 autism. *Nature Neuroscience*, *9*(10), 1218–1220. https://doi.org/10.1038/nn1770
787 Hartung, J., Doebler, P., Schroeders, U., & Wilhelm, O. (2018). Dedifferentiation and
788 differentiation of intelligence in adults across age and years of education.
789 *Intelligence*, *69*, 37–49. https://doi.org/10.1016/j.intell.2018.04.003
790 Hawkins, E., Gathercole, S., Astle, D., The CALM Team, & Holmes, J. (2016). Language
791 Problems and ADHD Symptoms: How Specific Are the Links? *Brain Sciences*, *6*(4),
792 50. https://doi.org/10.3390/brainsci6040050
793 Hiesinger, P. R. & Hassan, B. A. The Evolution of Variability and Robustness in Neural
794 Development. Trends in Neuroscience. 41, 577–586 (2018).

795 Holmes, J., Bryant, A., Gathercole, S. E., & the CALM Team. (2019). Protocol for a
796 transdiagnostic study of children with problems of attention, learning and memory
797 (CALM). *BMC Pediatrics*, *19*(1), 10. https://doi.org/10.1186/s12887-018-1385-3
798 Holmes, J., Butterfield, S., Cormack, F., van Loenhoud, A., Ruggero, L., Kashikar, L., &
799 Gathercole, S. (2015). Improving working memory in children with low language
800 abilities. *Frontiers in Psychology*, *6*, 519. https://doi.org/10.3389/fpsyg.2015.00519
801 Holmes, J., Guy, J., Kievit, R. A., Bryant, A., Mareva, S., CALM Team, & Gathercole, S. E.
802 (2021). Cognitive dimensions of learning in children with problems in attention,
803 learning, and memory. *Journal of Educational Psychology*, *113*(7), 1454–1480.
804 https://doi.org/10.1037/edu0000644
805 Honegger, K. & de Bivort, B. Stochasticity, individuality and behavior. Current Biology 28,
806 R8–R12 (2018).
807 Howe, E., Bosley, H. G., & Fisher, A. J. (2020). Idiographic network analysis of discrete
808 mood states prior to treatment. *Counselling and Psychotherapy Research*, *20*(3),
809 470–478. https://doi.org/10.1002/capr.12295
810 Johnson, M. H. (2000). Functional brain development in infants: Elements of an interactive
811 specialization framework. *Child Development*, *71*(1), 75–81.
812 https://doi.org/10.1111/1467-8624.00120
813 Johnson, M. H. (2011). Interactive Specialization: A domain-general framework for human
814 functional brain development? *Developmental Cognitive Neuroscience*, *1*(1), 7–21.
815 https://doi.org/10.1016/j.dcn.2010.07.003
816 Jones, J. S., CALM Team, & Astle, D. E. (2022). Segregation and integration of the
817 functional connectome in neurodevelopmentally ‘at risk’children. *Developmental*
818 *science*, *25*(3), e13209.
819 Kaiser, M., & Hilgetag, C. C. (2004). Modelling the development of cortical systems
820 networks. *Neurocomputing*, *58–60*, 297–302.
821 https://doi.org/10.1016/j.neucom.2004.01.059

822 Karcher, N. R., & Barch, D. M. (2021). The ABCD study: Understanding the development of
823 risk for mental and physical health outcomes. *Neuropsychopharmacology*, *46*(1),
824 131–142. https://doi.org/10.1038/s41386-020-0736-6
825 Karmiloff-Smith, A. (2009). Nativism versus neuroconstructivism: Rethinking the study of
826 developmental disorders. *Developmental Psychology*, *45*(1), 56–63.
827 https://doi.org/10.1037/a0014506
828 Karmiloff-Smith, B. A. (1994). Beyond Modularity: A Developmental Perspective on
829 Cognitive Science. *European Journal of Disorders of Communication*, *29*(1), 95–105.
830 https://doi.org/10.3109/13682829409041485
831 Kernbach, J. M., Satterthwaite, T. D., Bassett, D. S., Smallwood, J., Margulies, D., Krall, S.,
832 Shaw, P., Varoquaux, G., Thirion, B., Konrad, K., & Bzdok, D. (2018). Shared endo-
833 phenotypes of default mode dysfunction in attention deficit/hyperactivity disorder and
834 autism spectrum disorder. *Translational Psychiatry*, *8*(1), 1–11.
835 https://doi.org/10.1038/s41398-018-0179-6
836 Kessler, R. C., McLaughlin, K. A., Green, J. G., Gruber, M. J., Sampson, N. A., Zaslavsky, A.
837 M., Aguilar-Gaxiola, S., Alhamzawi, A. O., Alonso, J., Angermeyer, M., Benjet, C.,
838 Bromet, E., Chatterji, S., de Girolamo, G., Demyttenaere, K., Fayyad, J., Florescu,
839 S., Gal, G., Gureje, O., … Williams, D. R. (2010). Childhood adversities and adult
840 psychopathology in the WHO World Mental Health Surveys. *The British Journal of*
841 *Psychiatry*, *197*(5), 378–385. https://doi.org/10.1192/bjp.bp.110.080499
842 Kowalczyk, O. S., Mehta, M. A., O'Daly, O. G., & Criaud, M. (2021). Task-Based Functional
843 Connectivity in Attention-Deficit/Hyperactivity Disorder: A Systematic Review.
844 *Biological Psychiatry Global Open Science*.
845 https://doi.org/10.1016/j.bpsgos.2021.10.006
846 Kushki, A., Anagnostou, E., Hammill, C., Duez, P., Brian, J., Iaboni, A., Schachar, R.,
847 Crosbie, J., Arnold, P., & Lerch, J. P. (2019). Examining overlap and homogeneity in
848 ASD, ADHD, and OCD: A data-driven, diagnosis-agnostic approach. *Translational*
849 *Psychiatry*, *9*(1), 1–11. https://doi.org/10.1038/s41398-019-0631-2

Lawson, R. P., Mathys, C., & Rees, G. (2017). Adults with autism overestimate the volatility of the sensory environment. *Nature Neuroscience*, *20*(9), 1293–1299. https://doi.org/10.1038/nn.4615

Lessov-Schlaggar, C. N., Rubin, J. B., & Schlaggar, B. L. (2016). The Fallacy of Univariate Solutions to Complex Systems Problems. *Frontiers in Neuroscience*, *10*. https://www.frontiersin.org/article/10.3389/fnins.2016.00267

Levy, Y., & Ebstein, R. P. (2009). Research review: Crossing syndrome boundaries in the search for brain endophenotypes. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *50*(6), 657–668. https://doi.org/10.1111/j.1469-7610.2008.01986.x

Liu, Y.-Y., Slotine, J.-J., & Barabási, A.-L. (2011). Controllability of complex networks. *Nature*, *473*(7346), 167–173. https://doi.org/10.1038/nature10011

nLydon-Staley, D. M., Cornblath, E. J., Blevins, A. S., & Bassett, D. S. (2021). Modeling brain, symptom, and behavior in the winds of change. *Neuropsychopharmacology*, *46*(1), 20–32. https://doi.org/10.1038/s41386-020-00805-6

Lydon-Staley, D. M., Leventhal, A. M., Piper, M. E., Schnoll, R. A., & Bassett, D. S. (2021). Temporal networks of tobacco withdrawal symptoms during smoking cessation treatment. *Journal of Abnormal Psychology*, *130*(1), 89–101. https://doi.org/10.1037/abn0000650

Lynn, C. W., & Bassett, D. S. (2019). The physics of brain network structure, function and control. *Nature Reviews Physics*, *1*(5), 318–332. https://doi.org/10.1038/s42254-019-0040-8

Mackie, S., Shaw, P., Lenroot, R., Pierson, R., Greenstein, D. K., Nugent, T. F., Sharp, W. S., Giedd, J. N., & Rapoport, J. L. (2007). Cerebellar development and clinical outcome in attention deficit hyperactivity disorder. *The American Journal of Psychiatry*, *164*(4), 647–655. https://doi.org/10.1176/ajp.2007.164.4.647

Mahone, E. M., Ranta, M. E., Crocetti, D., O'Brien, J., Kaufmann, W. E., Denckla, M. B., & Mostofsky, S. H. (2011). Comprehensive Examination of Frontal Regions in Boys and Girls with Attention-Deficit/Hyperactivity Disorder. *Journal of the International*

878 *Neuropsychological Society : JINS*, *17*(6), 1047–1057.
879 https://doi.org/10.1017/S1355617711001056
880 Majerus, S., & Cowan, N. (2016). The Nature of Verbal Short-Term Impairment in Dyslexia:
881 The Importance of Serial Order. *Frontiers in Psychology*, *7*, 1522.
882 https://doi.org/10.3389/fpsyg.2016.01522
883 Marquand, A. F., Rezek, I., Buitelaar, J., & Beckmann, C. F. (2016). Understanding
884 Heterogeneity in Clinical Cohorts Using Normative Models: Beyond Case-Control
885 Studies. *Biological Psychiatry*, *80*(7), 552–561.
886 https://doi.org/10.1016/j.biopsych.2015.12.023
887 McGlothlin, A. E., & Viele, K. (2018). Bayesian Hierarchical Models. *JAMA*, *320*(22), 2365–
888 2366. https://doi.org/10.1001/jama.2018.17977
889 McGorry, P. D., & Mei, C. (2021). Clinical Staging for Youth Mental Disorders: Progress in
890 Reforming Diagnosis and Clinical Care. *Annual Review of Developmental*
891 *Psychology*, *3*(1), 15–39. https://doi.org/10.1146/annurev-devpsych-050620-030405
892 Newman, M. E. J. (2010). *Networks: An introduction*. Oxford University Press; /z-wcorg/.
893 Norrman, G., & Bylund, E. (2016). The irreversibility of sensitive period effects in language
894 development: Evidence from second language acquisition in international adoptees.
895 *Developmental Science*, *19*(3), 513–520. https://doi.org/10.1111/desc.12332
896 Onnink, A. M. H., Zwiers, M. P., Hoogman, M., Mostert, J. C., Kan, C. C., Buitelaar, J., &
897 Franke, B. (2014). Brain alterations in adult ADHD: Effects of gender, treatment and
898 comorbid depression. *European Neuropsychopharmacology: The Journal of the*
899 *European College of Neuropsychopharmacology*, *24*(3), 397–409.
900 https://doi.org/10.1016/j.euroneuro.2013.11.011
901 Parkes, L., Moore, T. M., Calkins, M. E., Cieslak, M., Roalf, D. R., Wolf, D. H., Gur, R. C.,
902 Gur, R. E., Satterthwaite, T. D., & Bassett, D. S. (2021). Network Controllability in
903 Transmodal Cortex Predicts Positive Psychosis Spectrum Symptoms. *Biological*
904 *Psychiatry*, *90*(6), 409–418. https://doi.org/10.1016/j.biopsych.2021.03.016

905 Parkes, L., Moore, T. M., Calkins, M. E., Cook, P. A., Cieslak, M., Roalf, D. R., Wolf, D. H.,
906 Gur, R. C., Gur, R. E., Satterthwaite, T. D., & Bassett, D. S. (2021). Transdiagnostic
907 dimensions of psychopathology explain individuals' unique deviations from normative
908 neurodevelopment in brain structure. *Translational Psychiatry*, *11*(1), 232.
909 https://doi.org/10.1038/s41398-021-01342-6
910 Parkes, L., Satterthwaite, T. D., & Bassett, D. S. (2020). Towards precise resting-state fMRI
911 biomarkers in psychiatry: Synthesizing developments in transdiagnostic research,
912 dimensional models of psychopathology, and normative neurodevelopment. *Current*
913 *Opinion in Neurobiology*, *65*, 120–128. https://doi.org/10.1016/j.conb.2020.10.016
914 Parsons, S., Kruijt, A.-W., & Fox, E. (2019). Psychological Science Needs a Standard
915 Practice of Reporting the Reliability of Cognitive-Behavioral Measurements.
916 *Advances in Methods and Practices in Psychological Science*, *2*(4), 378–395.
917 https://doi.org/10.1177/2515245919879695
918 Pasqualetti, F., Zampieri, S., & Bullo, F. (2014). Controllability Metrics, Limitations and
919 Algorithms for Complex Networks. *ArXiv:1308.1201 [Physics]*.
920 http://arxiv.org/abs/1308.1201
921 Pennington, B. F. (2006). From single to multiple deficit models of developmental disorders.
922 *Cognition*, *101*(2), 385–413. https://doi.org/10.1016/j.cognition.2006.04.008
923 Pezzoli, P., Parsons, S., Kievit, R. A., Astle, D. E., Huys, Q. J., Steinbeis, N., & Viding, E.
924 (2023). Challenges and solutions to measurement of neurocognitive mechanisms in
925 developmental settings. *Biological Psychiatry: Cognitive Neuroscience and*
926 *Neuroimaging*.
927 Piccirillo, M. L., & Rodebaugh, T. L. (2019). Foundations of idiographic methods in
928 psychology and applications for psychotherapy. *Clinical Psychology Review*, *71*, 90–
929 100. https://doi.org/10.1016/j.cpr.2019.01.002
930 Rubinov, M., & Sporns, O. (2010). Complex network measures of brain connectivity: Uses
931 and interpretations. *NeuroImage*, *52*(3), 1059–1069.
932 https://doi.org/10.1016/j.neuroimage.2009.10.003

Ruths, J., & Ruths, D. (2014). Control Profiles of Complex Networks. *Science*, *343*(6177), 1373–1376. https://doi.org/10.1126/science.1242063

Sanford, N., Whitman, J. C., & Woodward, T. S. (2020). Task-merging for finer separation of functional brain networks in working memory. *Cortex; a Journal Devoted to the Study of the Nervous System and Behavior*, *125*, 246–271. https://doi.org/10.1016/j.cortex.2019.12.014

Sayal, K., Prasad, V., Daley, D., Ford, T., & Coghill, D. (2018). ADHD in children and young people: Prevalence, care pathways, and service provision. *The Lancet. Psychiatry*, *5*(2), 175–186. https://doi.org/10.1016/S2215-0366(17)30167-0

Scahill, L., & Schwab-Stone, M. (2000). Epidemiology of Adhd in School-Age Children. *Child and Adolescent Psychiatric Clinics of North America*, *9*(3), 541–555. https://doi.org/10.1016/S1056-4993(18)30106-8

Shaw, P., Lerch, J., Greenstein, D., Sharp, W., Clasen, L., Evans, A., Giedd, J., Castellanos, F. X., & Rapoport, J. (2006). Longitudinal mapping of cortical thickness and clinical outcome in children and adolescents with attention-deficit/hyperactivity disorder. *Archives of General Psychiatry*, *63*(5), 540–549. https://doi.org/10.1001/archpsyc.63.5.540

Simpson-Kent, I. L., Fuhrmann, D., Bathelt, J., Achterberg, J., Borgeest, G. S., & Kievit, R. A. (2020). Neurocognitive reorganization between crystallized intelligence, fluid intelligence and white matter microstructure in two age-heterogeneous developmental cohorts. *Developmental Cognitive Neuroscience*, *41*, 100743. https://doi.org/10.1016/j.dcn.2019.100743

Singleton, S. P., Luppi, A. I., Carhart-Harris, R. L., Cruzat, J., Roseman, L., Deco, G., Kringelbach, M. L., Stamatakis, E. A., & Kuceyeski, A. (2021). *Psychedelics Flatten the brain's energy landscape: Evidence from receptor-informed network control theory* (p. 2021.05.14.444193). bioRxiv. https://doi.org/10.1101/2021.05.14.444193

959 Siugzdaite, R., Bathelt, J., Holmes, J., & Astle, D. E. (2020). Transdiagnostic Brain Mapping
960 in Developmental Disorders. *Current Biology*, *30*(7), 1245-1257.e4.
961 https://doi.org/10.1016/j.cub.2020.01.078
962 Sonuga-Barke, E. J. S., & Coghill, D. (2014). Editorial perspective: Laying the foundations
963 for next generation models of ADHD neuropsychology. *Journal of Child Psychology*
964 *and Psychiatry, and Allied Disciplines*, *55*(11), 1215–1217.
965 https://doi.org/10.1111/jcpp.12341
966 Sonuga-Barke, E. J. S., Cortese, S., Fairchild, G., & Stringaris, A. (2016). Annual Research
967 Review: Transdiagnostic neuroscience of child and adolescent mental disorders –
968 differentiating decision making in attention-deficit/hyperactivity disorder, conduct
969 disorder, depression, and anxiety. *Journal of Child Psychology and Psychiatry, and*
970 *Allied Disciplines*, *57*(3), 321–349. https://doi.org/10.1111/jcpp.12496
971 Suárez, L. E., Richards, B. A., Lajoie, G., & Misic, B. (2021). Learning function from structure
972 in neuromorphic networks. Nature Machine Intelligence, 3(9), 771-786.
973 Tang, E., Giusti, C., Baum, G. L., Gu, S., Pollock, E., Kahn, A. E., Roalf, D. R., Moore, T. M.,
974 Ruparel, K., Gur, R. C., Gur, R. E., Satterthwaite, T. D., & Bassett, D. S. (2017).
975 Developmental increases in white matter network controllability support a growing
976 diversity of brain dynamics. *Nature Communications*, *8*(1), 1252.
977 https://doi.org/10.1038/s41467-017-01254-4
978 Tooley, U. A., Bassett, D. S., & Mackey, A. P. (2021). Environmental influences on the pace
979 of brain development. *Nature Reviews Neuroscience*, *22*(6), 372–384.
980 https://doi.org/10.1038/s41583-021-00457-5
981 Vértes, P. E., Alexander-Bloch, A. F., Gogtay, N., Giedd, J. N., Rapoport, J. L., & Bullmore,
982 E. T. (2012). Simple models of human brain functional networks. *Proceedings of the*
983 *National Academy of Sciences*, *109*(15), 5868–5873.
984 https://doi.org/10.1073/pnas.1111738109
985 Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity
986 of the executive function theory of attention-deficit/hyperactivity disorder: A meta-

analytic review. *Biological Psychiatry*, *57*(11), 1336–1346. https://doi.org/10.1016/j.biopsych.2005.02.006

Williams, D. L., Goldstein, G., Carpenter, P. A., & Minshew, N. J. (2005). Verbal and spatial working memory in autism. *Journal of Autism and Developmental Disorders*, *35*(6), 747–756. https://doi.org/10.1007/s10803-005-0021-x

Wolfers, T., Beckmann, C. F., Hoogman, M., Buitelaar, J. K., Franke, B., & Marquand, A. F. (2020). Individual differences v. the average patient: Mapping the heterogeneity in ADHD using normative models. *Psychological Medicine*, *50*(2), 314–323. https://doi.org/10.1017/S0033291719000084

Wolfers, T., Doan, N. T., Kaufmann, T., Alnæs, D., Moberget, T., Agartz, I., Buitelaar, J. K., Ueland, T., Melle, I., Franke, B., Andreassen, O. A., Beckmann, C. F., Westlye, L. T., & Marquand, A. F. (2018). Mapping the Heterogeneous Phenotype of Schizophrenia and Bipolar Disorder Using Normative Models. *JAMA Psychiatry*, *75*(11), 1146–1155. https://doi.org/10.1001/jamapsychiatry.2018.2467

Woodward, T. S., Tipper, C. M., Leung, A. L., Lavigne, K. M., Sanford, N., & Metzak, P. D. (2015). Reduced functional connectivity during controlled semantic integration in schizophrenia: A multivariate approach. *Human Brain Mapping*, *36*(8), 2948–2964. https://doi.org/10.1002/hbm.22820

Yang, X., Ram, N., Lougheed, J. P., Molenaar, P. C. M., & Hollenstein, T. (2019). Adolescents' emotion system dynamics: Network-based analysis of physiological and emotional experience. *Developmental Psychology*, *55*(9), 1982–1993. https://doi.org/10.1037/dev0000690

Zabihi, M., Oldehinkel, M., Wolfers, T., Frouin, V., Goyard, D., Loth, E., Charman, T., Tillmann, J., Banaschewski, T., Dumas, G., Holt, R., Baron-Cohen, S., Durston, S., Bölte, S., Murphy, D., Ecker, C., Buitelaar, J. K., Beckmann, C. F., & Marquand, A. F. (2019). Dissecting the Heterogeneous Cortical Anatomy of Autism Spectrum Disorder Using Normative Models. *Biological Psychiatry. Cognitive Neuroscience and Neuroimaging*, *4*(6), 567–578. https://doi.org/10.1016/j.bpsc.2018.11.013

Zhang, J., Kucyi, A., Raya, J., Nielsen, A. N., Nomi, J. S., Damoiseaux, J. S., Greene, D. J., Horovitz, S. G., Uddin, L. Q., & Whitfield-Gabrieli, S. (2021). What have we really learned from functional connectivity in clinical populations? *NeuroImage*, *242*, 118466. https://doi.org/10.1016/j.neuroimage.2021.118466

World Health Organization. (2019). ICD-11: International classification of diseases (11th revision). Retrieved from https://icd.who.int/