Personality Neuroscience: An Emerging Field with Bright Prospects

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Abstract

Personality neuroscience is the study of persistent psychological individual differences, typically in the general population, using neuroscientific methods. It has the potential to shed light on the neurobiological mechanisms underlying individual differences and their manifestation in ongoing behavior and experience. The field was inaugurated many decades ago, yet has only really gained momentum in the last two, as suitable technologies have become widely available. Personality neuroscience employs a broad range of methods, including molecular genetics, pharmacological assays or manipulations, electroencephalography, and various neuroimaging modalities, such as magnetic resonance imaging and positron emission tomography. Although exciting progress is being made in this young field, much remains unknown. In this brief review, we discuss discoveries that have been made, methodological challenges and advances, and important questions that remain to be answered. We also discuss best practices for personality neuroscience research and promising future directions for the field.

Keywords

Personality neuroscience; neurobiology of traits

Personality neuroscience aims to understand the neurobiological systems—from the molecular to the macrocircuit levels—that underlie persistent individual differences in emotion, motivation, cognition, and behavior. It uses a wide range of methods, such as magnetic resonance imagining (MRI), electroencephalography (EEG), and pharmacological assays and manipulations, to measure individual differences in the structure and function of the brain. Then it tests for associations of these measurements with assessments of a wide range of psychological constructs, such as the Big Five traits, persistent symptoms of psychopathology, intelligence, working memory capacity, risk aversion, reward sensitivity, and empathy.

We adopt a broad view of the meaning of personality to include all reasonably stable psychological attributes, not merely those most prominently labeled as "personality." Further, we understand personality traits as predispositions or average tendencies to be in particular states, typically in response to common classes of stimuli, such as rewards, threats, distractions, conspecifics, or complex problems (DeYoung, 2015). Decades of research show that personality is shaped distally by both genetic and environmental forces (and by interactions between them), all of which must have an impact on the brain in order to affect personality. Thus, personality neuroscience seeks the proximal, neurobiological causes and correlates of individual differences. It is still a young field, due to the relatively recent invention of much of its measurement technology, but it is growing rapidly.

Here we discuss the current state of the field and the challenges and opportunities that lie ahead. As this is merely a brief overview, we do not attempt to be comprehensive but rather to provide a sense of the value of the field, its most solid accomplishments, and its prospects (for a longer review, see, e.g., DeYoung et al., 2021). All of the authors are in agreement regarding the assertions we make here, but naturally there are other issues on which we do not all agree, which is to be expected in a young field with much uncertainty. Given limited space, we have chosen to focus on areas of reasonable consensus rather than to explicate our various disagreements.

The Value of Personality Neuroscience

We see three broad reasons to study personality neuroscience: explanation, intervention, and incremental prediction beyond behavioral measurement modalities. The processes underlying human behavior and experience are often explained in purely psychological terms, but we believe that being able to explain psychological processes in terms of the neurobiological mechanisms that produce them is worthwhile as part of the general project of achieving consilience among different approaches to knowledge (Wilson, 1999). Integrating knowledge across different fields is valuable because it is likely to improve the knowledge of each of the fields involved. Psychological theory is more likely to be accurate if it is consistent with knowledge about how the brain works, and neuroscience is likely to make faster progress if it pursues hypotheses informed by psychological knowledge (in

part because any neuroscience attempting to understand how the brain controls behavior necessarily refers to psychological constructs on the behavioral side).

So far, this argument speaks to the value of neuroscience in psychology generally. What makes *personality* neuroscience valuable, specifically, is the fact that people's brains function differently from each other. Much research in human neuroscience attempts to make generalizations about the functioning of the average human brain. However, the knowledge of brain function thus produced is incomplete and potentially seriously misleading if we do not understand the wide variation that exists around this average, as well as the consequences of that variation for human experiences, behaviors, and outcomes. In any given study, some observed deviations from average are merely short-term, state-like deviations due to various random or situational factors and can safely be treated as noise in neuroscience research, but other deviations from average reflect long-term, trait-like patterns in brain function that correspond to each person's persistent individuality. In personality psychology, researchers commonly distinguish occasion-specific states from the longer-term patterns of states that constitute traits, and this perspective needs to be more strongly emphasized in neuroscience as well, if we are to develop accurate neurobiological explanations of personality (cf. Barch et al., 2013).

As well as facilitating explanations that cut across psychological and biological descriptions, personality neuroscience may eventually facilitate the development of interventions for the prevention and treatment of psychopathology. There is now ample evidence that most features of psychopathology are on a continuum with normal personality variation, and this is true not just of the so-called "personality disorders" but also for most other mental disorders as well (Widiger et al., 2019). Interventions for psychopathology can be roughly divided into those that directly target biological systems, such as pharmaceuticals and various brain stimulation techniques, and those that target psychological processes, such as cognitive-behavioral therapy and other forms of counseling. Personality neuroscience is especially relevant to the development of effective biological interventions, first through discovering potential neural targets of intervention and then through investigating the interventions' effects. Such efforts are in their infancy, but one recent example comes from translational research on anhedonia, where rodent models of deficits in reward processing have led to a promising clinical trial for a drug targeting opiate receptors, in people selected for elevated levels of anhedonia (Krystal et al., 2020; Pizzagalli et al., 2020). Additionally, better understanding of the underlying mechanisms involved in a given mental illness may also inform psychological interventions, by clarifying what their targets should be.

Clinical neuroscience has recently been moving away from case-control research designs based on traditional diagnostic categories and toward dimensional assessment of transdiagnostic symptoms and related mechanisms in community or treatment-seeking samples. This change has rendered clinical neuroscience contiguous with personality neuroscience and blurred the boundaries between the two fields. We see this as a positive development for both fields, as understanding general variation in brain function can help us to understand its maladaptive extremes or deviations, and vice versa.

In the clinic, personality neuroscience may usefully contribute to the rapidly growing area of neuroeducation, which is brain-based psychoeducation that aims to reduce distress and improve outcomes by helping patients understand the neurobiological processes underlying mental functioning (Miller, 2016; Servaas et al., 2021). It teaches people about brain function and how it is sculpted and regulated over time by variations in genetic and environmental forces, as well as how these processes may be related to their psychiatric symptoms. Reported benefits of integrating neuroeducation in clinical practice include increased empathy and compassion for the self, greater sense of empowerment, and better understanding of the ups and downs of the therapeutic change process (Miller, 2016).

Finally, it is possible that measurements of brain structure and function may predict important outcomes above and beyond psychological measurements that are easier and cheaper to acquire. For the purposes of basic science, effective prediction of psychological processes or outcomes from neurobiological variables might allow more accurate measurement of constructs where rater biases associated with self-reports and observer ratings, or the unreliability of task-based behavioral assessments, contribute to inaccuracy. Moving beyond basic science to clinical applications, incremental prediction may eventually contribute to our ability to identify risk for psychopathology and to diagnose it (though, currently, identifying risk is much closer to feasibility than is specific diagnosis).

At this point in time, considerably more effort has been made in research on explanation than in research on either intervention or incremental prediction. In the realm of explanation, the neural correlates of various traits can be used to develop theories designed to explain the mechanisms underlying those traits or to test hypotheses regarding existing theories. These are valuable avenues for research, but we encourage researchers to consider additionally applying personality neuroscience to projects of intervention and incremental prediction (cf. Mõttus et al., 2020).

Discoveries of Personality Neuroscience

Despite the promise of personality neuroscience, at this point few well-established findings exist in the field. In the next section, we will discuss some likely reasons for this fact, but first we want to start on a more optimistic note by describing findings that are supported by multiple replications, often in large samples. We describe research that has revealed information about the neural foundations of neuroticism, intelligence, and extraversion. This research has made important contributions to knowledge and also shows proof of concept for the ability of personality neuroscience to produce robust discoveries.

Neuroticism

Neuroticism (also known as negative affect, negative emotionality, or dispositional negativity) refers to the general tendency to experience all kinds of negative emotions and related cognitions and behaviors, such as anxiety, sadness, irritability, vulnerability, hopelessness, rumination, and self-consciousness. Neuroticism is the largest risk factor for nearly all forms of psychopathology, and it is difficult to distinguish statistically from the general factor of mood and anxiety disorders known as "internalizing" (Griffith et al., 2009; Kotov et al., 2021). In part because of its link to psychopathology, and in

part because of its conceptual connection to sensitivity to threat and punishment, there is probably more existing neuroscience research relevant to neuroticism than to any other trait, and there is also more relevant research in other species. Here we describe only some particularly well-established findings, but note that considerably more research has been done on this topic, suggesting that neuroticism is associated with a variety of brain regions (and interconnections between them), involved in the generation and regulation of negative emotion.

In both humans and non-human primates, numerous studies have shown that variation in the function of the amygdala and the adjacent bed nucleus of the stria terminalis (together often called the "extended amygdala") is related to neuroticism or internalizing (for reviews see DeYoung et al., 2021; Hur et al., 2019). One well-replicated finding is the positive association between neuroticism and the tonic or resting activity of the extended amygdala. This finding has also been supported by large studies of non-human primates, which provide evidence for a causal role of the extended amygdala in individual differences in fear and anxiety (Fox & Shackman, 2019). In human EEG research, meta-analysis shows that neuroticism is positively associated with the magnitude of an event-related waveform that occurs in the dorsal anterior cingulate cortex in response to making an error, although this association may be specific to anxiety-related subdimensions of neuroticism (Saunders & Inzlicht, 2020). Finally, a negative correlation between neuroticism and cortical surface area has been found in meta-analysis and in multiple very large human samples in a region of the dorsomedial prefrontal cortex (PFC) that appears likely to be involved in self-reflection, emotion regulation, and the subjective experience of emotion (Grasby et al., 2020; Liu et al., 2021; Valk et al., 2020). All of these findings appear consistent with the phenomenology of neuroticism and with theories of its basis in sensitivity to threat and punishment (DeYoung, 2015; Gray & McNaughton, 2000; Hur et al., 2019).

Intelligence

Intelligence, as measured by IQ and related performance tests, is a second trait that has well-replicated neural correlates (Deary et al., 2021). Before delving into these findings, we note that intelligence tests should be seen as reflecting reasoning ability, or more broadly, the ability to solve cognitive problems quickly and accurately, and that some may contain cultural biases that impede cross-cultural applications (Neisser et al., 1996). The vast majority of personality neuroscience research on intelligence (and on all other constructs, for that matter) has been conducted in populations of primarily European ancestry. Nonetheless, within the populations studied so far, some findings are quite robust.

One well-established finding is that intelligence is positively correlated with total brain volume, which may reflect the fact that individuals with larger brains are likely to have more neurons and thus more computational power to engage in problem solving and logical reasoning. Multiple meta-analyses have shown that the overall correlation is around r = .25 to .3, but also that the strength of the effect is moderated by the quality of the assessment of intelligence, such that the true correlation may be as high as .4 (Gignac & Bates, 2017; Pietschnig, 2015). Additionally, some evidence exists to support the claim that brain size contributes *causally* to intelligence (Lee et al., 2019). Nonetheless, given the size of the

correlation, it is clear that brain size could not be the only biological variable determining intelligence.

Intelligence, especially what is known as "fluid" intelligence or the ability to reason about novel problems, is also associated with structural and functional variation in large scale brain networks often labeled the "frontoparietal control" and "dorsal attention" networks (Basten et al., 2015; Santarnecchi et al., 2017; Yeo et al., 2011). This meta-analytic finding is consistent with one prominent theory of the neural basis of intelligence, the parieto-frontal integration theory (P-FIT; Jung & Haier, 2007). It is also consistent with what is known about the neural basis of working memory, which is heavily reliant on the frontoparietal control network and so strongly correlated with fluid intelligence that it is likely to be one of the main cognitive functions contributing to intelligence (Conway et al., 2003). However, regions outside of the frontoparietal control and dorsal attention networks have also been robustly associated with intelligence, including some regions in the temporal lobe and also subcortical structures such as the hippocampus and thalamus (Deary et al., 2021). Finally, robust evidence exists from diffusion MRI (which measures white matter structure) to suggest that intelligence is correlated with greater coherence of many white matter tracts (indicating greater myelination and/or more parallel trajectories of axons), especially those that connect various brain regions implicated in intelligence (Deary et al., 2021).

Extraversion

A third trait with reasonably robust findings is extraversion, the broad tendency toward assertiveness, sociability, activity, and high-energy positive emotions. One influential theory posited that extraversion is at least partly driven by sensitivity to incentive reward governed by dopaminergic function (Depue & Collins, 1999), and many studies have provided evidence supportive of this theory. At least nine studies have reported moderation of the effects of dopaminergic drugs by extraversion (for reviews of these studies, see DeYoung et al., 2021; Wacker & Smillie, 2015). Although few of these are direct replications, the consistency of the pattern provides evidence that extraversion is related to variation in dopaminergic function. Additional evidence consistent with both dopaminergic involvement and reward sensitivity has come from six studies reporting that extraversion is positively correlated with the magnitude of an event-related waveform in EEG that appears to reflect dopaminergic signaling of reward (Hauser et al., 2014; for reviews of EEG studies, see DeYoung et al., 2021 and Smillie et al., 2019). This waveform (confusingly known both as the "feedback-related negativity" and the "reward positivity") is assessed by comparing dorsomedial EEG activity following receipt of reward to the same activity following failure to receive an expected reward. Thus, we have reasonably good evidence to support the dopaminergic reward sensitivity theory of extraversion. In fact, the amount of neurobiological evidence that extraversion reflects reward sensitivity is currently greater than the amount of evidence from direct behavioral tests of reward sensitivity (judging by the number of reasonably powered studies reporting significant results of each type), a striking example of the value of neuroscience for personality theory.

Challenges for Personality Neuroscience

We could probably identify one or two more examples of well-established neural correlates of personality variables, but not many more. Despite impressive progress in the last two decades, personality neuroscience is a young field, and much remains uncertain. Many previously published findings are likely to be false positives due to the sampling variability inherent in under-powered studies using small samples. Human neuroimaging research has tended to be underpowered in general (Poldrack et al., 2017), and this problem is compounded when attempting to study individual differences, where larger samples are necessary to achieve adequate power, relative to studies of within-subject differences between experimental conditions. A sample of almost 200 is necessary to achieve 80% power to detect a correlation of .2, which is roughly the average effect size in personality psychology and in psychology as a whole (Gignac & Szodorai, 2016; Hemphill, 2003). Even larger samples are necessary to detect smaller effects, which are likely to be common in personality neuroscience, given the probable complexity of the biological systems that contribute to personality. When many causal sources contribute to a given variable, the correlation of any one of those causes with the variable in question cannot be strong, no matter how well they are measured (Ahadi & Diener, 1989; Strube, 1990). Hence, a major requirement for progress in this field is to aim for adequate sample sizes.

One boon for the field is that a number of very large MRI datasets collected by consortia, such as the Human Connectome Project and the Adolescent Brain and Cognitive Development Study, have recently become available in which to study individual differences. This is especially important in personality neuroscience because neuroimaging research is very expensive, and so collecting an adequately sized sample requires a great deal of funding. One practical strategy for acquiring larger samples, outside the context of a research consortium, is to include a standard set of personality and neural measures in smaller within-subjects studies, allowing them eventually to be aggregated into a larger sample for between-subjects research (Mar et al., 2013). More diverse samples would also be valuable, given that human neuroscience has relied for the most part on subjects of European ancestry, thereby limiting the generalizability of results.

Beyond the challenges of collecting reasonably large and representative samples are a number of other challenges that require more complicated solutions. First, is simply the complexity of the brain itself. It is clear that there is no one-to-one mapping of traits to specific neurobiological variables. Instead, each trait of interest is likely to reflect variation in many neurobiological parameters, and any particular neurobiological parameter is likely to affect multiple traits (DeYoung et al., 2021). Nor is the neural basis of any trait likely to be localized to one region of the brain, but rather it is likely to be related to large scale networks of regions, or even to complex patterns of interactions among such networks (Barbey, 2018; Markett et al., 2018; Tompson et al., 2018). Genetic research has highlighted this complexity by showing that there is no simple genetic basis for most traits. Instead, variation in most traits is influenced by thousands of different genetic variations (Lo et al., 2017).

Another problem related to the complexity of the brain is the problem of the scale of measurement. Although MRI is often touted for its spatial resolution, each voxel in a typical analysis nonetheless encompasses hundreds of thousands of neurons. The specific events within and between individual brain cells that form the mechanistic foundation of human information processing cannot be measured effectively in human beings, but nonetheless we can investigate the behavior of groups of neurons in specific parts of the brain. Unlike functional MRI (fMRI), which relies on relatively slow changes in blood oxygenation to estimate neural activity, EEG can assess neural activity on a millisecond basis, but with a great loss of spatial resolution. With EEG, one can understand the brain's broad patterns of activity in high resolution, but without being able to link that activity to very specific regions. One promising approach to these limitations is to combine fMRI with EEG. Indeed, we can say generally that combining multiple neuroscientific techniques is likely to afford new insights.

The functioning of different neurotransmitters and neuromodulators is often posited as important to the basis of various traits, but it can be assessed only indirectly. Positron emission tomography (PET) allows some assessment of variables related to specific neurotransmitters, such as receptor density, but its temporal resolution is poor, it is invasive (requiring injection of radioactive substances), and it is even more expensive than MRI. Pharmacological manipulations are sometimes able to target specific neurotransmitter systems directly (though many act on multiple neurotransmitter systems), but their effects can be measured only indirectly. In short, despite exciting technological advances, the tools of human neuroscience have a number of limitations related to their scale of measurement relative to the scale of events in the brain. Because different methods have different limitations, progress can often be made by employing multiple methods to study a particular question about personality.

The uniqueness of each human brain poses another challenge for personality neuroscience. Typical neuroimaging methods warp the image of every subject's brain to match a common template. This allows comparison of putatively anatomically equivalent points across individuals, which is certainly useful but may obscure differences in anatomical structure that can be as dramatic as the fact that some people have two gyri where others have only one (Valizadeh et al., 2018). Such differences are functionally meaningful. Each brain is somewhat unique not only in its physical structure, but also in its functional organization relative to its physical structure, meaning that not every function is localized in the same brain regions relative to anatomical landmarks, especially in the cortex (Gordon et al., 2017). Thus, even if anatomical alignment were perfectly accurate, functional neuroanatomy would nonetheless vary across individuals. We will discuss potential solutions to this problem in the next section.

So far we have been describing challenges specific to personality *neuroscience*, but of course the field also faces many of the same challenges as any other subfield within personality psychology. Because the major mode of assessment of personality is via questionnaire, measurement error is introduced by the errors that people make in describing themselves (or describing others, in the case of peer ratings). One of the likely reasons that intelligence has seen more progress than most other traits in terms of identifying

robust neural correlates is the high quality of its measurement. IQ tests are less error-prone than typical personality questionnaires because they rely on a performance metric that does not involve the distortions and biases inherent to self- or peer reports. However, this does not mean that researchers should always avoid questionnaire measures in favor of performance or task-based assessments. IQ tests have been carefully designed through extensive psychometric research and are highly reliable, but many other performance- or task-based assessments have not been well-validated and are not reliable as measures of individual differences (Enkavi & Poldrack, 2020). Further, many tasks commonly used in fMRI were not originally intended to study individual differences, rather being intended to investigate species universals. Thus, psychometrics should be brought to bear on the various tasks used in personality neuroscience. Even seemingly minor alterations to task instructions and parameters may affect the measurement properties of a tasks behavioral variables. Additionally, measurement-related concerns should not be ignored in relation to neurobiological variables; for example, we can assess their reliability (Elliott et al., 2021).

Finally, there is the difficulty of producing causal evidence in personality science, which arises primarily because we cannot easily manipulate the traits that are our primary objects of study. By definition, personality traits are persistent over time. They can and do change across the lifespan but typically do not change rapidly or dramatically. In the short term, we can manipulate behavior (e.g., by instructing people to act extraverted for some limited time; Jacques-Hamilton et al., 2019), but this does not qualify as manipulating a person's longstanding tendency toward (or away from) the trait in question. Manipulating behavior that relates to a trait is distinct from manipulating the trait itself—we can think of it as informative about *what might be*, as opposed to *what is*—because the neural parameters that govern the evoked behavior may be distinct from those that cause long-term tendencies toward such behavior. Thus, the neural effects of short-term manipulations of behavior provide only potential clues, rather than direct evidence, for the neural causes of personality. Traits themselves can potentially be manipulated in more long-term ways, for which the clearest evidence so far comes from clinical intervention studies (Roberts et al., 2017), but nonclinical interventions are beginning to be studied as well (Stieger et al., 2021). In future, longitudinal studies of neural change accompanying experimental manipulations of traits could be important for the field.

Manipulating the brain also poses challenges. Researchers studying humans rarely manipulate the brain directly, although there are important exceptions such as pharmacological manipulations and transcranial magnetic stimulation. However, researchers studying other species have more leeway in manipulating the brain and identifying accompanying changes in personality. This highlights the important role that research in other species has played in the development of theory in personality neuroscience, as with research on neuroticism (Hur et al., 2019).

In sum, much of the evidence in personality neuroscience is correlational, and it can be difficult to determine whether neural correlates are causes or effects of personality traits, or whether they are due to some third variable associated with both the neural parameter and the trait in question. Genetic data can be used to provide stronger causal evidence (e.g., Lee et al., 2019), but often we must settle for providing correlational evidence that is

consistent with some causal theory but does not indicate cause itself. The use of biologically realistic computational models fit to observed data might help us gain more confidence in our understanding of causal mechanisms (e.g., Pickering & Pesola, 2014).

Opportunities and Best Practices Going Forward

Before concluding we offer some thoughts on opportunities for progress in personality neuroscience, as well as some advice on how best to take advantage of such opportunities. The latter may also be useful for evaluating published research. Our discussion of issues and best practices is not intended to be comprehensive or definitive, but rather simply to highlight some prominent current issues in the field that we believe are important. There are other important issues, such as the value of open science and reproducibility, that we do not explicate in general terms because they are less specific to personality neuroscience, but this does not mean we consider them unimportant.

Theory- vs. data-driven research

Despite the challenges faced by personality neuroscience, we are optimistic about the future of the field. More and more large neuroimaging and genetic datasets are becoming available that include assessments of psychological individual differences, and, in the clinical world, emphasis is increasingly being placed on studying dimensional constructs in the general population, rather than focusing only on binary diagnoses and case-control designs. In fact, we may soon be in a position where our main challenge is how to deal with so much data, rather than having too little. The rise of computational power has provided one approach to dealing with such an embarrassment of riches, namely to abandon theory and to rely on data-driven exploratory approaches like machine learning (Mõttus et al., 2020; Yarkoni & Westfall, 2017). Although data-driven approaches can be powerful tools for discovery, we would like to make a plea for theory-driven research as well (DeYoung & Krueger, 2020; Oberauer & Lewandowsky, 2019).

The examples of reasonably robust findings that we highlighted in a previous section illustrate the value of both theory- and data-driven research. Sometimes data comes first and leads to theory, as with the P-FIT theory of intelligence, which was developed, and is being refined, based on patterns evident in existing findings. Sometimes theory comes first and is supported by later findings, as with the dopaminergic incentive-reward theory of extraversion. At the time the latter was formulated, only limited direct evidence was available to support it, but many studies have since been conducted to test it. The case of neuroticism provides examples both of theory-driven results (associations with the amygdala) and of data-driven results (associations with dorsomedial PFC). Both approaches are clearly valuable, and the value of both approaches will be constrained by the quality of their measures, which emphasizes the importance of selecting measures carefully.

What traits to study?

To pursue theory effectively in personality neuroscience, one can either begin with known neurobiological functions and attempt to identify dimensions of behavior to which they are likely to contribute (McNaughton & Smillie, 2018), or one can begin with psychological

traits and attempt to develop a theory of their neurobiological basis. In the latter case, the next step is to identify candidate psychological functions that seem likely to cause the various features of the trait to covary (for example, incentive reward sensitivity in the case of extraversion). To the degree that knowledge exists regarding the neural basis of these psychological functions, it can be used to develop testable hypotheses regarding associations with the trait in question.

When developing research questions in personality neuroscience, it is important to consider the hierarchical structure of traits (DeYoung et al., 2021; Mõttus et al., 2020). Most trait constructs can be split into correlated subcomponents, which may have distinct as well as shared neural correlates. Genetically informed research indicates that there is unique genetic variance at every level of the personality hierarchy, which suggests that traits and their subtraits or facets are very likely to have partially distinct neural correlates (DeYoung et al., 2021; Nagel et al., 2018). Some neural parameters may contribute to causing all of the subtraits to covary, whereas others may affect only one subtrait. Although it is important to understand why narrow traits covary in ways represented by broad traits like intelligence, the Big Five, or the dimensions of psychopathology represented in the hierarchical taxonomy of psychopathology (HiTOP; Kotov et al., 2021), it may sometimes be more tractable for personality neuroscience to study narrower traits and to develop hypotheses about their specific neural mechanisms. Ultimately, the field should try to understand the neural basis of both lower- and higher-level traits in the hierarchy. If one is taking an approach focused on a specific trait, one should nonetheless consider its potential location within trait hierarchies because any effects one identifies involving that trait may be attributable instead to related traits. This possibility typically makes it important to assess a range of traits, even if one's research question is focused only narrowly on a single trait.

The importance of neurobiological atlases

On the neurobiological side, human neuroscience in general is moving from a focus on the functional properties of specific brain regions to an approach that recognizes that most psychological functions are carried out by larger networks of regions or by interactions among such networks. Increasingly, attempts are being made to map these networks and to develop atlases that take the brain's functional organization into account and can be used to standardize research and improve comparison of results across studies (Glasser et al., 2016; Schaeffer et al., 2018; Yeo et al., 2011). These atlases are to some extent hierarchical, in the sense that networks can be identified at higher and lower levels of resolution, such that functional parcellations of the brain contain anywhere from seven to hundreds of regions, and many broader networks fractionate into narrower subnetworks. Consideration of maps based on larger networks is especially helpful for the development of cumulative knowledge and communication of results, whereas maps based on larger numbers of smaller networks or parcels may be useful for identifying more specific patterns of individual differences in neural function. Various methods can be used to study the network properties of collections of parcels, and the use of a standard atlas in such studies improves the ability to evaluate replication (Market et al., 2018; Tompson et al., 2018).

When using standard atlases in personality neuroscience, we strongly recommend the use of a new family of methods known as "individualized parcellation," which respond to the challenge posed by the fact that the functional organization of the brain varies across people in relation to anatomical landmarks (Chong et al., 2017; Gordon et al., 2017; Kong et al., 2021). Specific functional brain areas vary in their spatial location across individuals. Similarly, functional brain networks composed of numerous smaller areas or parcels also vary in their spatial layout. Reliance on standard atlases without individualized parcellation identifies inaccurate regional boundaries for each individual. This introduces noise, reduces sensitivity for characterizing functional regions and broader network topographies, and hinders the study of individual differences in neural variables. Individualized parcellation methods typically employ Bayesian algorithms to take a standard atlas and shift the parcel boundaries for each subject until each parcel is maximally functionally homogeneous for that individual. This allows accurate functional registration while preserving the ability to compare any given parcel across subjects and samples.[1]

Another viable method to achieve individualized localization of functional brain regions involves the use of functional localizers. A functional localizer is a fMRI task that reliably activates a particular brain system and thus can be used to identify a specific region or regions activated by that task in each subject before using those regions for other research purposes.[2] The obvious limitation of this method is that it can identify only those regions that are relevant to the particular task used, rather than being able to parcellate the whole brain. Individualized parcellation methods can match the functional localization of neural activity by tasks, even when derived from resting state data (Chong et al., 2017; Kong et al., 2021).

Beyond cross-sectional research in adult populations

Much important research in personality neuroscience remains to be done using large cross-sectional samples with high quality assessments of both personality and neurobiological variables. Nonetheless, we also want to encourage researchers to think big and to pursue developmental and longitudinal research. If personality neuroscience is a young field, then a subfield of *developmental* personality neuroscience is still in its infancy (DeYoung & Allen, 2019). We know very little about how the development of the brain governs the development of personality. Even in adults, we have very little data on how change in the brain over time

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[1] Individualized parcellation is preferable to dual regression, an earlier strategy to deal with the problem of individual variation in the brain's functional organization. In dual regression, a particular region or set of regions presumed to be functionally connected (often a component derived from independent components analysis) is first used as a spatial regressor to identify an aggregate time series of neural activation for that region of the brain. Then, that time series is used as a temporal regressor to identify the brain region(s) that most closely express that temporal activation pattern in that subject. Dual regression involves an obviously problematic assumption, namely that the aggregate time series within what are known to be an inaccurately specified region (or set of regions) can be used to identify the correct regional boundaries.

[2] It is important to distinguish the use of functional localizers from a superficially similar method for identifying regions of interest (ROI) that is much more common but also much more problematic for personality neuroscience. Group-level fMRI contrasts are typically used to identify ROI where a task significantly activates the brain relative to a control condition, and researchers have often subsequently examined correlations of personality traits with neural parameters only within these ROI. This is misguided because the group-level contrast ensures identification of ROI where there is sufficiently little variability across individuals that one can infer that the task tends to activate the brain similarly across individuals. In personality neuroscience, however, one is interested in studying variability, not eliminating it, and the most important regions for a trait may be ones that are not significant at the group level precisely because different brains respond differently to the task in question. Functional localizers do not suffer from this problem because they identify ROI based on contrasts at the individual level rather than the group level.

is linked to change in personality. The fact that personality traits change over time means that the neural systems that produce them must change within persons over time as well. Longitudinal research can help to shed light on these processes, and it can also provide another avenue for making stronger causal inferences.

Genetic research can also help us to understand the processes by which the neurobiology of personality develops, and with distinguishing genetic from environmental causes of personality. Although identifying genetic variations that are reliably associated with a given trait typically requires enormous samples, on the order of hundreds of thousands of people, once such genome-wide association studies (GWAS) have been conducted, polygenic scores derived from them can be calculated and used effectively in smaller samples. This makes it worthwhile to collect genetic data in studies focused primarily on other measurement modalities, such as neuroimaging. Further, even without conducting neuroscientific investigations in new samples, bioinformatics can be applied to the results of GWAS to produce evidence about which neurobiological systems are involved in a given trait (Nagel et al., 2018; Savage et al., 2018).

Finally, we recommend translational research that makes use of data and knowledge from species other than humans (e.g., Gray & McNaughton, 2000; Krystal et al., 2021; Latzman et al., 2017; Panksepp, 1998; Pizzagalli et al., 2021). This may be difficult for trait dimensions that appear in few species other than humans, such as conscientiousness (Weiss et al., 2015), but it has already proved to be a valuable approach to others, such as neuroticism (as described in previous sections). To the extent that personality traits depend on brain systems and functions that appeared earlier in phylogeny than Homo sapiens, homologous dimensions of behavioral variation are likely to be identifiable in other species. Studying those dimensions can provide a powerful approach to understanding the neural causes of personality, through the ability to manipulate the brains of those species. Although human behavior is more complex than that of other animals, many of its core dimensions of variation are likely to be rooted deep in evolutionary history. Personality neuroscience can help us to understand not only the biological basis of human personality, but also the manner in which our personalities fit into the larger history and diversity of life on earth.

Conclusion

Although personality neuroscience is a young field that faces many challenges (some unique and some inherited from personality psychology or neuroscience more generally), we are optimistic about its future, both in the immediate and in the long term. A solid base of evidence is beginning to accumulate regarding the neural basis of some important traits, and we expect that much more will follow regarding those traits and others. As psychological science as a whole moves toward larger and larger samples, more and more data are becoming available that are well-suited to studying individual differences. If you are a personality scientist curious about the neurobiological basis of psychological traits, or with a psychological question that might be informed by neurobiological evidence, we encourage you to become involved in neuroscience research. If you are a neuroscientist considering the importance of variability in brain function across individuals, we encourage you to draw on the expertise of personality neuroscientists. The field of personality neuroscience has a

crucial role to play in understanding why people do what they do and how they differ from each other.

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References

1. Ahadi S, & Diener E (1989). Multiple determinants and effect size. Journal of Personality and Social Psychology, 56(3), 398–406.

2. Barbey AK (2018). Network neuroscience theory of human intelligence. Trends in cognitive sciences, 22(1), 8–20. [PubMed: 29167088]

3. Barch DM, Burgess GC, Harms MP, Petersen SE, Schlaggar BL, Corbetta M, … & Van Essen DC (2013). Function in the human connectome: task-fMRI and individual differences in behavior. Neuroimage, 80, 169–189. [PubMed: 23684877]

4. Basten U, Hilger K, & Fiebach CJ (2015). Where smart brains are different: A quantitative meta-analysis of functional and structural brain imaging studies on intelligence. Intelligence, 51, 10–27.

5. Chong M, Bhushan C, Joshi AA, Choi S, Haldar JP, Shattuck DW, … & Leahy RM (2017). Individual parcellation of resting fMRI with a group functional connectivity prior. NeuroImage, 156, 87–100. [PubMed: 28478226]

6. Conway AR, Kane MJ, & Engle RW (2003). Working memory capacity and its relation to general intelligence. Trends in cognitive sciences, 7(12), 547–552. [PubMed: 14643371]

7. Deary IJ, Cox SR, & Hill WD (2021). Genetic variation, brain, and intelligence differences. Molecular Psychiatry, 1–19. [PubMed: 33972691]

8. Depue RA, & Collins PF (1999). Neurobiology of the structure of personality: Dopamine, facilitation of incentive motivation, and extraversion. Behavioral and Brain Sciences, 22(3), 491–569. [PubMed: 11301519]

9. DeYoung CG (2015). Cybernetic Big Five Theory. Journal of Research in Personality, 56, 33–58.

10. DeYoung CG, & Allen TA (2019). Personality neuroscience: A developmental perspective. In McAdams DP, Shiner RL, & Tackett JL (Eds.). The Handbook of Personality Development (pp. 79–105). New York: Guilford Press.

11. DeYoung CG, Grazioplene RG, & Allen TA (2021). The neurobiology of personality. In John OP & Robbins RW (Eds.), Handbook of personality: Theory and research, Fourth Edition. New York: Guilford Press.

12. DeYoung CG, & Krueger RF (2020). To wish impossible things: On the ontological status of latent variables and the prospects for theory in psychology. Psychological Inquiry, 31(4), 289–296.

13. Elliott ML, Knodt AR, & Hariri AR (2021). Striving toward translation: strategies for reliable fMRI measurement. Trends in cognitive sciences, 25, 776–787. [PubMed: 34134933]

14. Enkavi AZ, & Poldrack RA (2020). Implications of the lacking relationship between cognitive task and self report measures for psychiatry. Biological Psychiatry: Cognitive Neuroscience and Neuroimaging.

15. Fox AS, & Shackman AJ (2019). The central extended amygdala in fear and anxiety: Closing the gap between mechanistic and neuroimaging research. Neuroscience letters, 693, 58–67. [PubMed: 29195911]

16. Gignac GE, & Bates TC (2017). Brain volume and intelligence: The moderating role of intelligence measurement quality. Intelligence, 64, 18–29.

17. Gignac GE, & Szodorai ET (2016). Effect size guidelines for individual differences researchers. Personality and Individual Differences, 102, 74–78.

18. Glasser MF, Coalson TS, Robinson EC, Hacker CD, Harwell J, Yacoub E, … & Van Essen DC (2016). A multi-modal parcellation of human cerebral cortex. Nature, 536(7615), 171–178. [PubMed: 27437579]

19. Gordon EM, Laumann TO, Adeyemo B, & Petersen SE (2017). Individual variability of the system-level organization of the human brain. Cerebral cortex, 27(1), 386–399. [PubMed: 26464473]

20. Grasby KL, Jahanshad N, Painter JN, Colodro-Conde L, Bralten J, Hibar DP, … & Van Rooij D (2020). The genetic architecture of the human cerebral cortex. Science, 367 (6484).

21. Gray JA, & McNaughton N (2000). The neuropsychology of anxiety: An enquiry into the function of the septo-hippocampal system (2nd ed.). New York: Oxford University Press.

22. Griffith JW, Zinbarg RE, Craske MG, Mineka S, Rose RD, Waters AM, & Sutton JM (2010). Neuroticism as a common dimension in the internalizing disorders. Psychological Medicine, 40, 1125–1136. [PubMed: 19903363]

23. Hauser TU, Iannaccone R, Stämpfli P, Drechsler R, Brandeis D, Walitza S, et al. (2014). The feedback-related negativity (FRN) revisited: New insights into the localization, meaning and network organization. NeuroImage, 84, 159–168. [PubMed: 23973408]

24. Hemphill JF (2003). Interpreting the magnitudes of correlation coefficients. American Psychologist, 58(1), 78–79. [PubMed: 12674822]

25. Hur J, Stockbridge MD, Fox AS, & Shackman AJ (2019). Dispositional negativity, cognition, and anxiety disorders: An integrative translational neuroscience framework. Progress in brain research, 247, 375–436. [PubMed: 31196442]

26. Jacques-Hamilton R, Sun J, & Smillie LD (2019). Costs and benefits of acting extraverted: A randomized controlled trial. Journal of Experimental Psychology: General, 148(9), 1538–1556. [PubMed: 30489119]

27. Jung RE, & Haier RJ (2007). The parieto-frontal integration theory (P-FIT) of intelligence: Converging neuroimaging evidence. Behavioral and Brain Sciences, 30(02), 135–154. [PubMed: 17655784]

28. Kong R, Yang Q, Gordon E, Xue A, Yan X, Orban C, … & Yeo BTT (2021). Individual-specific areal-level parcellations improve functional connectivity prediction of behavior. Cerebral Cortex, online ahead of print. doi: 10.1093/cercor/bhab101.

29. Kotov R, Krueger RF, Watson D, Cicero DC, Conway CC, DeYoung CG, … & Wright AG (2021). The Hierarchical Taxonomy of Psychopathology (HiTOP): A quantitative nosology based on consensus of evidence. Annual Review of Clinical Psychology, 17, 19.1–19.26.

30. Krystal AD, Pizzagalli DA, Smoski M, Mathew SJ, Numberger J, Lisanby SH, et al. (2020). A randomized proof-of-mechanism trial applying the 'fast-fail' approach to evaluating κ-opioid antagonism as a treatment for anhedonia. Nature medicine, 26(5), 760–768.

31. Latzman RD, Green LM, & Fernandes MA (2017). The importance of chimpanzee personality research to understanding processes associated with human mental health. International Journal of Comparative Psychology.

32. Lee JJ, McGue M, Iacono WG, Michael AM, & Chabris CF (2019). The causal influence of brain size on human intelligence: Evidence from within-family phenotypic associations and GWAS modeling. Intelligence, 75, 48–58. [PubMed: 32831433]

33. Liu X, Lai H, Li J, Becker B, Zhao Y, Cheng B, & Wang S (2021). Gray matter structures associated with neuroticism: A meta-analysis of whole-brain voxel-based morphometry studies. Human brain mapping, 42(9), 2706–2721. [PubMed: 33704850]

34. Lo MT, Hinds DA, Tung JY, Franz C, Fan CC, … & Chen CH, 2017. Genome-wide analyses for personality traits identify six genomic loci and show correlations with psychiatric disorders. Nature Genetics, 49, 152–156. [PubMed: 27918536]

35. Mar RA, Spreng RN, & DeYoung CG (2013). How to produce personality neuroscience research with high statistical power and low additional cost. Cognitive, Affective, and Behavioral Neuroscience, 13(3), 674–685.

36. Markett S, Montag C, & Reuter M (2018). Network neuroscience and personality. Personality Neuroscience, 1, e14. [PubMed: 32435733]

37. McNaughton N, & Smillie LD (2018). Some metatheoretical principles for personality neuroscience. Personality Neuroscience, 1.

38. Miller R (2016). Neuroeducation: Integrating brain-based psychoeducation into clinical practice. Journal of Mental Health Counseling, 38(2): 103–115.

39. Mõttus R, Wood D, Condon DM, Back MD, Baumert A, Costantini G, … & Zimmermann J (2020). Descriptive, predictive and explanatory personality research: Different goals, different approaches, but a shared need to move beyond the Big Few traits. European Journal of Personality, 34(6), 1175–1201.

40. Nagel M, Jansen PR, Stringer S, Watanabe K, De Leeuw CA, Bryois J, … & Posthuma D (2018). Meta-analysis of genome-wide association studies for neuroticism in 449,484 individuals identifies novel genetic loci and pathways. Nature genetics, 50(7), 920–927. [PubMed: 29942085]

41. Neisser U, Boodoo G, Bouchard TJ Jr, Boykin AW, Brody N, Ceci SJ, … & Urbina S (1996). Intelligence: knowns and unknowns. American Psychologist, 51(2), 77.

42. Oberauer K, & Lewandowsky S (2019). Addressing the theory crisis in psychology. Psychonomic Bulletin and Review, 26(5), 1596–1618. [PubMed: 31515732]

43. Panksepp J (1998). Affective neuroscience: The foundations of human and animal emotion. New York: Oxford University Press.

44. Pickering AD, & Pesola F (2014). Modeling dopaminergic and other processes involved in learning from reward prediction error: contributions from an individual differences perspective. Frontiers in human neuroscience, 8, 740. [PubMed: 25324752]

45. Pietschnig J, Penke L, Wicherts JM, Zeiler M, & Voracek M (2015). Metaanalysis of associations between human brain volume and intelligence differences: How strong are they and what do they mean? Neuroscience and Biobehavioral Reviews, 57, 411–432. [PubMed: 26449760]

46. Pizzagalli DA, Smoski M, Ang YS, Whitton AE, Sanacora G, Mathew SJ, et al. (2020). Selective kappa-opioid antagonism ameliorates anhedonic behavior: evidence from the fast-fail trial in mood and anxiety Spectrum disorders (FAST-MAS). Neuropsychopharmacology, 45(10), 1656–1663. [PubMed: 32544925]

47. Poldrack RA, Baker CI, Durnez J, Gorgolewski KJ, Matthews PM, Munafò MR, … & Yarkoni T (2017). Scanning the horizon: towards transparent and reproducible neuroimaging research. Nature Reviews Neuroscience, 18(2), 115–126. [PubMed: 28053326]

48. Roberts BW, Luo J, Briley DA, Chow PI, Su R, & Hill PL (2017). A systematic review of personality trait change through intervention. Psychological Bulletin, 143, 117–141. [PubMed: 28054797]

49. Santarnecchi E, Emmendorfer A, & Pascual-Leone A (2017). Dissecting the parieto-frontal correlates of fluid intelligence: A comprehensive ALE meta-analysis study. Intelligence, 63, 9–28.

50. Saunders B, & Inzlicht M (2020). Assessing and adjusting for publication bias in the relationship between anxiety and the error-related negativity. International Journal of Psychophysiology, 155, 87–98. [PubMed: 32479773]

51. Savage JE, Jansen PR, Stringer S, Watanabe K, Bryois J, De Leeuw CA, … & Posthuma D (2018). Genome-wide association meta-analysis in 269,867 individuals identifies new genetic and functional links to intelligence. Nature genetics, 50(7), 912–919. [PubMed: 29942086]

52. Schaefer A, Kong R, Gordon EM, Laumann TO, Zuo XN, Holmes AJ, … & Yeo BT (2018). Local-global parcellation of the human cerebral cortex from intrinsic functional connectivity MRI. Cerebral cortex, 28(9), 3095–3114. [PubMed: 28981612]

53. Servaas MN, Schoevers RA, Bringmann LF, van Tol MJ, Riese H (2021) Trapped: Rigidity in psychiatric disorders. Lancet Psychiatry, 8(12):1022–1024. [PubMed: 34801115]

54. Smillie LD, Jach HK, Hughes DM, Wacker J, Cooper AJ, & Pickering AD (2019). Extraversion and reward-processing: Consolidating evidence from an electroencephalographic index of reward-prediction-error. Biological Psychology, 146, 107735. [PubMed: 31352030]

55. Stieger M, Flückiger C, Rüegger D, Kowatsch T, Roberts BW, & Allemand M (2021). Changing personality traits with the help of a digital personality change intervention. Proceedings of the National Academy of Sciences, 118(8), e2017548118.

56. Strube MJ (1991). Multiple determinants and effect size: A more general method of discourse. Journal of Personality and Social Psychology, 61(6), 1024–1027.

57. Tompson SH, Falk EB, Vettel JM, & Bassett DS (2018). Network approaches to understand individual differences in brain connectivity: opportunities for personality neuroscience. Personality neuroscience, 1, e5. [PubMed: 30221246]

58. Valizadeh SA, Liem F, Mérillat S, Hänggi J, & Jäncke L (2018). Identification of individual subjects on the basis of their brain anatomical features. Scientific reports, 8(1), 1–9. [PubMed: 29311619]

59. Valk SL, Hoffstaedter F, Camilleri JA, Kochunov P, Yeo BT, & Eickhoff SB (2020). Personality and local brain structure: Their shared genetic basis and reproducibility. NeuroImage, 220, 117067. [PubMed: 32574809]

60. Wacker J, & Smillie LD (2015). Trait extraversion and dopamine function. Social and Personality Psychology Compass, 9(6), 225–238.

61. Weiss A, Staes N, Pereboom JJM, Inoue-Murayama M, Stevens JMG, & Eens M (2015). Personality in bonobos. Psychological Science, 26(9), 1430–1439. [PubMed: 26209530]

62. Widiger TA, Sellbom M, Chmielewski M, Clark LA, DeYoung CG … & Wright AGC (2019). Personality in a hierarchical model of psychopathology. Clinical Psychological Science, 7, 77–92.

63. Wilson EO (1999). Consilience: The unity of knowledge. New York: Vintage Books.

64. Yarkoni T, & Westfall J (2017). Choosing prediction over explanation in psychology: Lessons from machine learning. Perspectives on Psychological Science, 12(6),1100–1122. [PubMed: 28841086]

65. Yeo BT, Krienen FM, Sepulcre J, Sabuncu MR, Lashkari D, Hollinshead M, et al. (2011). The organization of the human cerebral cortex estimated by intrinsic functional connectivity. Journal of Neurophysiology, 106(3), 1125–1165. [PubMed: 21653723]