Biology of Personality and Individual Differences

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As a social species, we are in the habit of continuously predicting each other's behavior. How do we do that? How do we know what our family members, colleagues, friends (or adversaries) are probably going to do next? In 'guesstimating' behavior we use three crucial pieces of information. First, we take into account the environmental context in which we observe a person. We expect a different response to insulting behavior than to a friendly smile, but also expect that distinction to be less sharp at a party compared to a work setting or a traffic jam. Second, age and the sex of the person importantly shape our expectations regarding her or his behavior. We don't expect bungee-jumping in a 100-year-old, nor being shot by an 11-year-old (although both events have actually been recorded), and we rightfully consider recidivist female pedophiles a rare breed (although they do apparently exist). Finally, if we know the person reasonably well, we take into account her or his general tendency to respond with certain classes of behavior across a wide range of situations. This tendency is known as *personality*. Whereas the environment is in constant flux and causes changes in an individual's behavior over time, personality is considered the constant factor that causes stability of behavior over time. This constancy is likely to be hardwired in our biology, and the brain seems the obvious place for such hardwiring. The key question, of course, is the exact shape of all this hardwiring of behavior and how it exactly comes about.

The book *Biology of Personality and Individual Differences* addresses the biological processes underlying the major dimensions of personality as well as individual differences in behavioral tendencies coupled to age and sex (Canli, 2006). Integrating compelling lines of inquiry that until now have largely remained disparate, it brings together leading investigators from molecular and behavior genetics, neurobiology, neuroimaging, comparative psychology, personality psychology, clinical psychology and psychiatry. Coverage includes a wide range of topics, ranging from the structure of personality, its roots in genetics, its mapping onto brain processes, and the importance of personality in the vulnerability for psychopathology. This is a comprehensive book on the biology of individual differences that will be of interest to graduate students and researchers alike.

To set the scene, Fowles (chapter 2) opens with a comprehensive review of Jeffrey Gray's contribution to the theory of personality. In Gray's view there are two basic mechanisms that control behavior. One mechanism, the behavioral inhibition system (BIS), is activated by novelty and stimuli associated with negative reinforcement, that is, punishment or omission of reward. The other mechanism, the behavioral approach system (BAS) is activated by stimuli associated with positive reinforcement, that is, reward or termination of punishment. Gray located these systems to the septohippocampal system (to which the amygdale was later added) and the ventral striatum (including the nucleus accumbens). Engagement of both systems is associated with arousal, as reflected in changes in autonomic nervous activity and hormonal secretion. Applied to personality, Gray proposed that the most salient individual differences reflect the variation in sensitivity to stimuli associated with negative or positive reinforcement and the coupled behavioral tendencies of avoidance and approach. Specifically, individual differences in the functioning of the reward system in response to appetitive stimuli are implicated in the personality traits extraversion and sensation seeking/impulsivity. Individual differences in activity of the punishment system in response to aversive stimuli are implicated in the personality traits of neuroticism and harm avoidance. Extraversion and neuroticism, in Gray's view, relate to harm avoidance and sensation seeking/impulsivity by a 45° rotation such that sensation seeking is in the neurotic/extraverted quadrant and harm avoidance is in the neurotic/introverted quadrant.

With these main dimensions of reward and punishment sensitivity as a guideline, the book proceeds with a number of chapters on individual differences in the reward system. Zuckerman (chapter 3) reviews the biological and social factors shaping sensation seeking. As an example of their complex interplay, a twin study is cited that showed high heritability for twins raised in nonreligious families (61% for females and 49% for males) but no genetic effects for

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males raised in a religious environment and a weakening of the effects in females (h^2 = 37%). The chapter also shows how psychophysiological indicators like skin conductance responses or evoked cortical potentials can be used to show a critical aspect of sensation seeking: a larger responsiveness of the brain to novel stimuli, coupled to much faster habituation of brain responses on repeated stimulation.

Depue (chapter 4) tackles the neurobiology of extraversion by distinguishing agency (assertiveness, activity, dominance) and affiliation (warmth, positive emotions, agreeableness) as two separate aspects of extraversion with distinct neural substrates in the ventral striatal reward system. Agentic extraversion is linked to the dopaminergic incentive motivational system. Engagement of this system by natural reward or psychostimulants is associated with both positive emotional feelings such as elation and euphoria, and motivational feelings of desire, wanting, craving, vigor, and self-efficacy. The second aspect of extraversion is linked to the affiliative consummatory reward system which is engaged by nonsexual touch, comforting speech, caring faces, familiar scents, and so forth. A broad range of evidence suggests a key role for endogenous opiates in affiliative behavior. individual differences in dopaminergic incentive reward processes, which are subject to strong genetic influence, are suggested as a major source of variance in agentic extraversion. Genetic variation within the opioid system, particularly in the μ-receptors, is suggested to account for individual differences in affilitative extraversion. In keeping with the latter, strong group differences in film-induced feelings of warmth and affection between individuals high versus low in trait affiliation on Tellegen's Social closeness scale almost completely dissipated under opiate receptor blockade.

Canli (chapter 5) shows how brain reactivity to emotional stimuli correlates to the positive affective characteristics of extraversion, as well as to variation within specific genes. This is one of three chapters in the book dealing with 'genomic imaging', a strategy that may well dominate biological research on personality in the near future. In genomic imaging, individuals who are genotyped on a candidate gene for a personality trait are exposed to emotional stimuli while their brain responses are recorded using functional magnetic resonance imaging (fMRI). Canli tested individuals for extraversion and genotyped them on the dopamine D4 receptor gene (DRD4). The 7-repeat allele of this gene has been associated with higher extraversion. Individuals were exposed to a series of neutral, happy, sad, and fearful face stimuli in the MRI scanner. First, regions were identified where extraverts differed from introverts. Next it was tested whether DRD4 was associated with differential responses of these regions. Individuals with at least one copy of the 7-repeat allele were found to have higher activation of the right medial frontal gyrus and the left precuneus. This may directly implicate these areas in the genetic effects on extraversion, at least for the part that acts through the DRD4 part of the dopaminergic system. The beauty of the genomic imaging approach is that by directly linking genes, personality and brain activity it may eventually lead to the development of true causal models of personality.

Adding clever experimental design to fMRI technology, Knutson and Bhanji (chapter 6) show that different areas are activated by gain anticipation (nucleus accumbens) than by actual reward (medial frontal cortex). The degree of activation of the nucleus accumbens is seen to accurately predict individual differences in self-experienced positive arousal during gain anticipation. Moreover, fMRI data showed that extraverts have a larger sensitivity of the nucleus accumbens to gain anticipation, implying that a common neural substrate may contribute to the state of positive arousal and to trait extraversion. The obvious next step is to expand this fruitful experimental fMRI paradigm with molecular genetics, and a plea for just such a multilevel approach is made by the authors.

In chapter 7 (Herrington, Koven, Miller and Heller), substantial evidence is reviewed that points to larger activity of the left frontal cortex during positive approach emotion/approach motivation and larger activity of the right frontal cortex during negative emotion/avoidance motivation. This lateralization appears to have both state and trait components, responding to experimentally induced changes in mood, and characterizing the emotional experience of individuals with high levels of anxiety and depression. Data in support of frontal asymmetry in emotion regulation comes from brain injury, Wada testing and EEG studies, but is remarkably absent in studies using hemodynamic imaging (i.e., positron emission tomography and fMRI). Herrington and colleagues review 52 of these brain imaging studies and conclude that analytical strategies have been greatly suboptimal to detect hemispheric asymmetries in hemodynamic data. In an improved fMRI design they show an asymmetrical activation of the dorsal lateral frontal cortex during pleasant relative to unpleasant stimuli, such that left frontal activity is much larger during positive emotion. This finding is in much better keeping with EEG studies than the previous hemodynamic approaches.

In chapters 8, 9 and 10, age and sex are discussed as determinants of individual differences. As with personality, the emphasis is on the neurobiological changes with age that account for parallel changes in behavior and the neurobiological differences in the male and female brain that may underlie their differential behavior repertoire. Knight and Mather review the growing body of evidence that, in contrast to the well-documented

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decline of cognitive functioning across the adult life span, there is a relative stability and even improvement in emotion regulation and experience with age. The advantage of emotion over cognition seems related to the relative sparing of the neural mechanisms of emotion with age over those involved in higher cognition in the frontal cortex. In addition, older adults more often use executive processes to enhance positive information and diminish negative information in attention and memory, which may be related to the increased emphasis on emotional wellbeing in older individuals motivated by the perception of the diminishing time left in life.

Hamann, in the next chapter, reviews the few neuroimaging studies to date that have compared the neural responses to visual sexual stimuli across the sexes. Based on expectations from evolutionary psychology, men, who require a lesser investment of time and resources to reproduce, should have a greater ability to become quickly sexually aroused and motivated to engage in sexual activity. Visual sexual cues should be more salient to males, because they allow for rapid identification and appraisal of mating opportunities with females in the visual field, and thus facilitate their mating with a maximal number of females. Conversely, because women have a much greater parental investment in time and resources in reproduction, it would be disadvantageous for them to be as quickly and broadly responsive to mating opportunities as men are. In keeping, prominent differences in responsiveness to visual sexual stimuli are found in the same subcortical regions (amygdale and hypothalamus) that have been linked to sex-differentiated sexual behaviors in animals. The amygdale and hypothalamus are also larger in men than in women and contain high levels of sex hormone receptors. Taken together, a clear developmental difference in brain structure and function guided by differential sex hormone exposure seems a major source of stable sex differences in responsiveness to sexual cues. To what extent these differences also account for sex differences in the response to stress, sensitivity to trauma and susceptibility to mood disorders (as reviewed by Sinha in chapter 10) remains to be established.

After the section on age and sex differences the book continues with four chapters on the biology of neuroticism, anxiety and depression. In chapter 11, Gillespie and Martin review the evidence from large-scale twin studies showing the importance of genetic factors in neuroticism and harm avoidance as well as major depression and anxiety disorders. They also provide preliminary evidence for the existence of substantial genetic correlations between harm avoidance and neuroticism and harm avoidance and extraversion, supporting Gray's original idea that harm avoidance is in the high neuroticism/low extraversion quadrant of Eysenck's Big Two. In chapter 12, Middeldorp and colleagues provide convincing evidence that personality traits like neuroticism and extraversion (but not sensation seeking) are important predictors of mood disorders and as such might be valuable traits to study the molecular genetic and neurobiological basis of these disorders. Importantly, it is shown that neuroticism predicts continuous scores on trait anxiety and depression measured by the Spielberger and Beck inventories as well as discrete clinical DSM-IV diagnoses of major depression, dysthymia, generalized anxiety disorder, panic disorder and social phobia. The predictive power of neuroticism is seen to increase, furthermore, with the number of comorbid conditions.

In chapter 13 and 14, the genomic imaging strategy is applied to anxiety-related traits. Lesch and Canli first review a large body of evidence that links variation in a SNP in the serotonergic 1A receptor gene (HTR1A-1019) to neuroticism and anxiety disorders. Based on that, they submitted individuals genotyped for this variant in HTR1A to the emotional word Stroop task in the fMRI scanner. In this task word stimuli are printed in different ink colours, and participants are asked to identify, as quickly and accurately as possible, the colour of the ink for each word. Although meaning is irrelevant in this task, emotional words may be more distracting than neutral words and thus result in slower reaction times. Relative to neutral words, negative words were significantly slower for HTR1A-1019 heterozygous individuals than for both homozygous groups. This was paired to a reduced activity of the right inferior parietal lobule in the HTR1A-1019 heterozygotes. Interestingly, the effect size of the HTR1A genotype was lowest for questionnaire-based neuroticism measure, higher for to reaction speed on the emotional Stroop, and highest for localized brain activation during this task. The increase in effect size when defining traits more closer to the putative gene action in the brain is quite consistent with the theoretical rationale for the endophenotype-approach.

In chapter 14, Hariri recounts what is probably the most successful story in genomic imaging so far: the link between a variable repeat sequence variant in the promoter of the serotonine transporter gene (5-HTTLPR) and the fMRI bold response of the amygdale in response to aversive stimuli. The 5-HTTLPR genotype is associated with functional differences in synaptic serotonine clearance. A number of studies have shown that individuals carrying one or more copies of the short allele have higher anxiety and neuroticism levels and more often suffer from mood disorders (although this is not a unanimous finding). Because the amygdale plays a crucial part in the processing of aversive stimuli *and* is densely innervated by serotonergic neurons, Hariri and colleagues had individuals genotyped for 5-HTTLPR perform

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a task that is well-known to induce amygdale responses: exposure to fearful or angry human facial expressions. A significantly larger amygdale response was found in carriers of the short allele. This finding has since been replicated in three independent functional imaging studies. Interestingly, 5-HTTLPR was not associated to harm avoidance, a personality type that may be expected to emerge from amygdale hyperreactivity. Hariri interprets this as further evidence for the superior sensitivity of brain activity to gene effects in comparison to paper-and-pencil personality measures. For instance, genetic amygdale sensitivity to environmental threat may need to conspire with an actual adverse environment to create individual differences in harm avoidance.

In chapters 15 to 17 the topic of mood and personality disorders is narrowed to children. Viding and Plomin (chapter 15) present data on children with callous emotional traits (lack of empathy, lack of guilt, shallow emotions) and conduct problems that are known to greatly increase the risk for overt antisocial behavior and criminality in adulthood. Using multivariate genetic analysis on 7-year-old twins from the Twins Early Development Study (TEDS) they show high heritability of extreme callous emotional traits (h^2 = 67%) particularly if they are comorbid with conduct problems (h^2 = 81%). Callous emotional traits and conduct problems were modestly correlated (.49), and this correlation was largely due to shared genetic factors. Finding the actual genes that make up this latent genetic factor will allow future genomic imaging studies of the type described in previous chapters. An obvious hypothesis would be that antisocial behavior is linked to deviant activity in the reward processing system.

Lau and Eley address the interplay between genetic and environmental factors in the risk for anxiety and depression. Genetic factors may directly influence emotional processing by the brain, but they may also influence environmental risk exposure (gene–environment correlation) or determine the impact of environmental risk factors on depression (gene–environment interaction). As an example of the latter, adolescents whose parents reported no educational qualifications *and* who were in the highest third of scorers on a genetic risk index, were three times more likely to be grouped among the high depression scorers. As Lau and Eley point out, failing to take into account the moderating effects of environmental factors like parental education may explain why there are so many negative genetic associations with plausible candidate genes in psychiatric genetics. In such analyses many individuals who possess the risk allele may not express the associated behavioral symptoms, because they have not been exposed to an adverse environment. The genetic factors upping the risk for anxiety and depression may also lead to increased exposure to adverse events, for instance when attributional style, a heritable trait, makes some persons subjectively experience 'defeat' much more than others would in the same circumstances. This may cause avoidance behavior with social isolation and, when picked up by the environment, lead to an actual lower social ranking. This gene-environment correlation can be addressed in recent upgrades of the multivariate twin models, but Lau and Eley formulate the original idea that, in case of strong gene–environment correlation, the adverse environment itself can be used as an endophenotype for anxiety and depression.

In chapter 17, Gotlib, Joormann, Minor and Cooney review evidence that depressed and at-risk children show many of the psychobiological characteristics of depressed adults, that is, negative biases in attention and memory functioning, deregulated HPA axis functioning, and abnormalities in structure and functioning of the hippocampus, ACC and amygdale. Their own study of young girls (11–14 years) of mothers with recurrent depression reminds us of the importance of cognitive schemas in depression. Cognitive theories of depression posit that vulnerable and depressed individuals selectively attend to negative stimuli, filter out positive stimuli, and perceive negative or neutral information as being more negative than is actually the case. Because of these negative schemas, children at risk for depression are possibly characterized by a negative bias in recall, demonstrating better recall for negative than for neutral or positive material. In favour of this hypothesis, girls at risk for depression endorsed more negative adjectives as self-referent and recalled more negative adjectives during incidental recall. They also responded faster to a dot probe, when this was displayed in the spatial location of sad faces rather than happy or neutral faces. Paired to these findings, higher cortisol reactivity was found and deviant responses of the amygdale and the anterior cingulate cortex to mood repair and reward/punishment anticipation Because these girls were at risk but not yet affected these results suggest that negative cognitive schemas and deviant neural and hormonal responsivity precede rather than follow the onset of depression.

The final three chapters are intended to serve as a bridge between research on human and animal personality. Chiavegatto (chapter 18) reviews mouse models for aggressive behavior and illustrates some of the advantages of animal genetic engineering when moving from a statistical genetic association to the actual functional relevance of the gene for the behavior. For instance, the highly aggressive behavior of mice who had both alleles of the serotonine 1B receptor knocked out (5-HT_{1B} null mice) can still be reduced by administration of a nonselective $5HT_{1B}$ agonist in much the same way as in wild type mice. This suggests that 5-HT_{1B} is important for

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aggression but that other serotonine receptors (e.g., 1A) must also play a role. Moving from rodents to primates, Weiss and King (chapter 19) show how subjective wellbeing and personality traits can be reliably assessed in chimpanzees. The availability of detailed pedigrees across zoos allows estimation of variance in these traits due to genetic and environmental effects, the latter including the effects of specific zoos. Using a similar bivariate strategy as often used in human studies, they show a high genetic correlation between dominance and subjective wellbeing. Taken the relationships among chimpanzee dominance, reproductive success, and survival, shared genes between happiness and dominance suggest that happiness in chimpanzees may be a sexually selected fitness indicator. As Weiss and King put it — 'a chimpanzee's smile is like a peacock's tail'.

In the final chapter, Mehta and Gosling summarize the ways in which animal studies can contribute to an understanding of the biology of individual differences in humans. Among the benefits of animal research are (1) *greater experimental control* in terms of hormonal and pharmacological intervention, active genetic manipulation and controlled environments, (2) the *larger range of physiological parameters* that can be measured as a function of genotype or environment, for example, hormone receptor densities, gene expression and electrophysiological recordings in brain tissues (including *in vivo* recordings), (3) *greater opportunity for naturalistic observations* for greater periods of time, in more detail and in more contexts, and (4) the *shorter life span of animals*, which reduce the heavy financial costs that would required by longitudinal studies in humans targeting developmental process. This latter benefit of animal research is particularly clear when health outcomes, including mortality, need to be linked to individual differences in behavioral repertoire and/or stress exposure. Here, animal models can yield faster (and cheaper) answers than similar research in humans.

To summarize, *Biology of Personality and Individual Differences* presents a mix of original findings and excellent reviews of recent developments in psychiatric genetics, neuroimaging genetics, animal personality research and multivariate twin research on personality and correlated psychopathology. Until now, the reader who wanted to follow these exciting developments had to seek out scholarly journals from a vast array of scientific disciplines. The intention of the editor (Canli) was to have the book 'offer a current and cross-sectional overview of the field, intended for a wide audience of practicing clinicians; social, personality, and cognitive psycho-logists; neuroscientists working in neuroimaging; behavioral neuro-scientists; and behavioral and molecular geneticists'. The book succeeds remarkably well in achieving this aim, not in the least because it is written by leading authors from exactly those disciplines.

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