How We Process Information: Neurocognitive and Behavioural Traits (Keynote)

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Neuropsychology is the understanding of the relationship between what happens in our brain and the effect that this has on our emotions and behaviour. Psychologists may use computer tests, or pen and paper tests to help us understand how we process information and brain function. These tests measure thinking styles, attention, memory, recognition, planning, social behaviour, mood and feelings. Performance on these tests can be associated with function in different areas of the brain. The current presentation will focus on four different neuropsychological and behavioural traits. These include, set shifting, central coherence, emotional processing and reward sensitivity. These four features will be defined in terms of how they present in human behaviour and their neural correlates will be explained. These features are in part genetically based and also influenced by environmental factors and therefore can change over time.

The first feature to be explored is 'set shifting' which can also be defined as cognitive flexibility. When applied to real life encounters it has two aspects which are spontaneous and reactive flexibility. Spontaneous flexibility refers to the ability to generate diverse and creative responses and bypass habitual strategies in response to a single task. Reactive flexibility refers to the ability to readily shift cognitions and behaviour according to the changing demands of a situation (Eslinger and Grattan 1993).

The neural correlates associated with set shifting have been investigated by Miller and Cohen (2001) who argue that set shifting functions are dependent on the prefrontal cortices' (PFC) ability to co-ordinate activity in other parts of the brain. Studies using FMRI (functional magnetic resonance imaging) in control samples have shown that the dorso-lateral prefrontal cortex (DLPFC) is actively engaged when switching between rules (Ravizza and Carter, 2008). Moreover damage to the dorsolateral frontal lobe is associated with an impaired ability to adapt to new rules on the WCST task [Wisconsin Card Sorting Test; Heaton et al., 1993, (Milner, 1963)]. A meta-analysis of 16 studies (Barnett, Scoriels and Munafo 2008) found a small association between the Val158Met COMT genotype and the WCST perseverative errors in control individuals.

The second feature to be explored is 'central coherence'. According to the global precedence theory, perception involves a balance between global and local processes. A temporal sequence of perception usually begins with seeing the global structure followed by perceiving the finer details (Navon 1977). The overall balance between global over detail has been termed central coherence and the opposite of this, weak central coherence is considered to be one marker of autism (Happe and Booth 2008). When applied to real life encounters, weak central coherence may refer to an inability to see the bigger picture or consequences and an excessive focus on the finer details to the detriment of the overall main goal.

Investigation into neural correlates of weak coherence in autsim suggests that there may be reduced synchronisation across the cortical network (Frith 2004; Brock et al 2002). Detailed (or local) processing (measured by the Embedded Figures Test; Witkin, Dyk, Faterson, Goodenough and Karp, 1962) in autistic spectrum disorder is associated with greater activation in the ventral occipitotemporal regions (visual systems utilised for object feature analysis) (Ring et al 1999). Parents of those with autism also show superior detail processing and increased activity in the visual cortex (middle occipital and lingual gyri) (Baron-Cohen et al 2006). This contrasts with the comparison group who showed greater activation in the prefrontal cortices (Ring et al 1999). Genetic factors account for 36% of the variance in performance in control individuals on the Embedded Figures Task (EFT) version that also involves memory abilities (Smalley et al 1989).

The third feature to be presented is emotional processing. Emotional intelligence is a broad concept that refers to the ability to perceive, express, assimilate and regulate emotions (Mayer, Salovey and Caruso, 1999).

Emotion regulation is a mechanism by which we control the experience of emotion by prioritising thinking and monitoring emotions (Mayer, Salovey and Caruso, 1999). There are two main emotion regulation strategies, which use 1) conscious, controlled, or 2) unconscious, automatic processes: 'cognitive reappraisal' and 'emotion suppression'. Cognitive reappraisal, re-frames and accepts the emotion and is associated with positive self-esteem, better emotional experiences, adaptive social interactions (Gross, 2002; Saarni, 1990) and effective emotional coping strategies in response to stress (Tugade and Frederickson, 2002). On the other hand 'emotion suppression' results in increased sympathetic activation, negative emotion and worse interpersonal functioning (Gross 1998; Gross and John 2003; Gross 2002; Campbell-Sills, Barlow, Brown and Hofmann 2006). Other distinct strategies include internalising and externalising. Internalising includes avoidance, rumination and suppression of emotion (Aldao, Nolen-Hoeksema and Schweizer, 2010).

The second construct of particular relevance is emotion recognition. This refers to the ability to label complex emotions (Mayer, Salovey and Caruso, 1999). It involves both the perception of the geometric configuration of facial features in addition to the interpretation of its direct and indirect emotional (Adolphs, 2002).

The neural correlates associated with emotion processing is two interacting neural systems, the ventral and dorsal system are thought to be involved in emotional regulation. The emotional significance of a stimulus is integrated predominantly from the ventral system (amygdala, insula, thalamus and ventral striatum), whereas the dorsal system (dorsal, medial and prefrontal cortex) is involved in regulating these affective states (Phillips et al 2003; Quirk and Beer 2006; Urry et al 2006; Harriri et al 2000, Pessoa et al 2002).

Emotion recognition relies on multiple strategies utilising an array of different brain structures that work together; amygdala, orbitofrontal cortex, anterior cingulate cortex, and ventral striatum (Adolphs, 2002). The amygdala is especially important in the recognition of facial expression and social signalling (Calder, Lawrence and Young, 2001). Genetic factors account for 36% of the variance in performance in control individuals on the Embedded Figures Task (EFT) version that also involves memory abilities (Smalley et al 1989).

The fourth feature to be presented is reward sensitivity which defines a responsiveness to reward cues and subsequent positive emotion which arises from engaging in reinforcing behaviours. The Behavioural Inhibition and Behavioural Activation Systems (BIS/BAS) is a theoretical paradigm that describes the physiological mechanisms which underpin reward sensitivity (Gray, 1970, described in section 1.6.3.3). The balance between the approach (or behavioural activation) and avoidance systems (BIS) is modulated by effortful control processes (Claes et al 2010). This determines whether the person exhibits reflective or impulsive behaviours. Variations in psychological traits cause the motivation to approach reward, to differ between individuals. When applied to real life encounters, individuals with poor sensitivity to reward may display engage in more impulsive or risky behaviors such as addictive behaviors. This occurs since the person requires excessive stimulus to experience an effect. Whereas those with high sensitivity to reward may engage in more thoughtful and long-term strategies, requiring less stimulus to experience an effect.

The neural correlates of reward sensitivity involve the frontostriatal brain network which plays a key role in the balance between effortful, approach and avoidant behaviours. This balance is thought to be modulated by the serotonin and dopamine neurotransmitter systems (Congdon and Canli, 2008; Evenden, 1999; Robbins, 2005; Berridge 2007). People with the 10 allele of the DAT and the 7 repeat allele of the DRD4 in ADHD are more hyperactive (Carrasco et al 2006; Roman et al 2001; Congdon and Canli, 2008). A meta-analysis has found an association between the 7-repeat allele of the DRD4 and increased risk of ADHD, (Congdon and Canli, 2008; Faraone, Doyle, Mick & Biederman 2001; Li, Sham, Owen and He, 2001). Brain activation studies have found that healthy individuals with higher behavioural activation (measured by the BAS; Carver and White, 1994) have increased amygdala activation in response to aggressive facial expressions (Beaver et al 2008). Those with higher levels of behavioural inhibition [measured by the behavioural inhibition and activation scales (BIS/BAS scales) (Carver and White, 1994)] have increased activation in the dorsal anterior cingulate, a region known to be involved in fear conditioning (Beaver et al 2008; Phelps et al 2004; Garavan et al 2002).

Biography

Dr. Natalie Kanakam currently works as a Trainee Clinical Psychologist in the NHS. She graduated with a first class honors in Psychology BSc from Brunel University. Subsequently she completed her PhD in Behavioral Genetics – Neuropsychology of Eating Disorders at Kings' College London. Following this she worked as Postdoctoral Researcher at UCL in the Faculty of Brain Sciences in collaboration with the Computer Science Department. Subsequently Natalie work in BBC News as a researcher investigating audience opinions of TV, Radio and Online content. She then worked in management consulting in the pharmaceutical industry before training to be a Clinical Psychologist. During her training she was worked with early intervention for psychosis, health psychology, 'Improving Access for Psychological Therapies' and most recently with children and young people who have eating disorders.

References

Adolphs, R. (2002). Recognizing emotion from facial expressions: psychological and neurological mechanisms. Behavioral and Cognitive Neuroscience Reviews, 1, 21–61.

Aldao, A., Nolen-Hoeksema, S. and Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. Clinical Psychology Review, 30(2), 217–237.

Barnett, J. H., Scoriels, L. and Munafo, M. R. (2008). Meta-analysis of the cognitive effects of the catechol-O-methyltransferase gene Val158/108Met polymorphism. Biological Psychiatry, 64, 137–144.

Baron-Cohen, S., Ring, H., Chitnis, Z., Wheelwright, S., Gregory, L., Williams, S., Brammer, M. and Bellmore, E. (2006). fMRI of parents of children with Asperger Syndrome: a pilot study. Brain and Cognition, 61, 122–130.

Beaver, J. D., Lawrence, A. D., Passamonti, L. and Calder, A. J. (2008). Appetitive motivation predicts the neural response to facial signals of aggression. The Journal of Neuroscience. 28(11) 2719–2725.

Berridge, K. (2007). The debate over dopamine's role in reward: the case for incentive salience. Psychopharmacology, 191, 391–431.

Calder, A. J., Lawrence, A. D., and Young, A. W. (2001). Neuropsychology of Fear and Loathing. Nature Reviews Neuroscience, 2(5), 352–363.

Campbell-Sills, L., Barlow, D. H., Brown, T. A. and Hofmann, S. G. (2006). Effects of suppression and acceptance on emotional responses of individuals with anxiety and mood disorders. Behaviour Research and Therapy. 44(9), 1251–1263.

Carver, C. S. & White, T. L. (1994). Behavioral Inhibition, Behavioral Activation, and Affective Responses to Impending Reward and Punishment: The BIS/BAS Scales. Journal of Personality and Social Psychology, 67(2), 319–333.

Carrasco, X. Rothhammer, R. Moraga, M. Henriquez, H. Chakraborty, R. Aboitiz, F. And Rothammer, F. (2006) Genotypic interaction between DRD4 and DAT1 loci is a high risk factor for attention deficit/hyperactivity disorder in Chilean families. American Journal of Medical Genetics B: Neuropsychiatric Genetics, 141(1), 51–54.

Claes, L., Robinson, M.D., Muehlenkamp, J. J., Vandereycken, W. and Bijttebier, P. (2009). Differentiating bingeing/purging and restrictive eating disorder subtypes: The roles of temperament, effortful control and cognitive control. Personality and Individual Differences. 48. 166–170.

Congdon, E. and Canli, T. (2008). A neurogenetic approach to impulsivity. Journal of Personality, 76(6), 1447–1484.

Eslinger, P. J., & Grattan, L. M. (1993). Frontal lobe and frontal-striatal substrates for different forms of human cognitive flexibility. Neuropsychologia, 31(1), 17–28.

Evenden, J. L. (1999) Varieties of impulsivity. Psychopharmacology (Berl), 146(4), 348–361.

Faraone, S. V., Doyle, A. E., Mick, E., Biederman, J.. (2001). Meta-analysis of the association between the 7-repeat allele of the dopamine D(4) receptor gene and attention deficit hyperactivity disorder. American Journal of Psychiatry. 158(7), 1052–1057.

Frith, C. D. (2004). Is autism a disconnection disorder? Lancet Neurology. 3, 577.

Garavan, H., Ross, T. J., Murphy, K., Roche, R. A.P. and Stein, E. A., (2002). Dissociable executive functions in the dynamic control of behaviour: inhibition, error detection and correction. NeuroImage. 17. 1820-1829. doi:10.1006/nimg

Gray, J. A. (1970). The psychophysiological basis of introversion-extraversion. Behaviour Research and Therapy. 8, 249–266

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. Journal of Personality and Social Psychology, 85, 348–362.

Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. Psychophysiology, 39, 281–291.

Gross, J. J. (1998). Antecedent- and response-focused emotion regulation: divergent consequences for experience, expression, and physiology. Journal of Personality and Social Psychology, 74, 224 –237.

Happe, F. and Booth, R. (2008). The power of the positive: revisiting weak coherence in autism spectrum disorders. Quarterly Journal of Experimental Psychology, 61, 50–63.

Li, D., Sham, P. C., Owen, M.J. and He, L. (2006). Meta-analysis shows significant association between Dopamine system genes and attention deficit hyperactivity disorder (ADHD). Human Molecular Genetics, 15(14), 2276–2284.

Mayer, J. D., Salovey, P., & Caruso, D. (2000). Models of emotional intelligence. In Sternberg, R. J. ed. The handbook of intelligence, New York: Cambridge University Press. 396–420.

Miller, E. K. & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. Annual Review of Neuroscience, 24, 167–202.

Milner B. (1963). Effects of different brain lesions on card sorting: The role of the frontal lobes. Archives of neurology, 9, 90.

Navon, D. (1977). Forest before trees - precedence of global features in visual perception. Cognitive Psychology, 9, 353–83.

Phelps, E. A., Delgado, M. R., Nearing, K. I. and LeDoux, J. E. (2004). Extinction learning in humans: role of the Amygdala and vmPFC. Neuron, 43, 897–905.

Phillips, M., Young, A., Senior, C., Brammer, M., Andrew, C., & Calder, A. (1997). A specific neural substrate for perceiving facial expressions of disgust. Nature, 389, 495–498.

Ravizza, S. M. & Carter, C. S. (2008). Shifting set about task switching: Behavioral and neural evidence for distinct forms of cognitive flexibility. Neuropsychologia, 46, 2924–2935.

Ring, H. A., Baron-Cohen, S., Wheelwright, S., Williams, S. C., Brammer, M., Andrew, C., & Bullmore, E. T. (1999). Cerebral correlates of preserved cognitive skills in autism: a functional MRI study of embedded figures task performance. Brain, 122(7), 1305–1315.

Robbins, T. W. (2005). Chemistry of the mind: neurochemical modulation of prefrontal cortical function. Journal of Comparative Neurology. 493(1), 140–146.

Roman, T., Schmitz, M., Polanczyk, G., Eizirik, M., Rohde, L. A., Hutz, M. H. (2001). Attention-deficit hyperactivity disorder: a study of association with both the dopamine transporter gene and the dopamine D4 receptor gene. American Journal of Medical Genetics. 105(5), 471–478.

Quirk, G. J. & Beer, J. S. (2006). Prefrontal involvement in the regulation of emotion: Convergence of rat and human studies. Current Opinions in Neurobiology, 16, 723–727.

Saarni, C. (1990). Emotional competence: How emotions and relationships become integrated. In: Thompson, R. A. ed. Nebraska Symposium on Motivation: Socioemotional development. Current theory and research in motivation, Lincoln, NE, US: University of Nebraska Press. 36, 115–182.

Smalley, S. L., Thompson, A. L., Spence, M. A., Judd, W. J. and Sparkes, R. S. (1989). Genetic influences on spatial ability: transmission in an extended kindred. Behaviour Genetics. 19(2).

Tugade, M. M., & Fredrickson, B. L. (2002). Positive emotions and emotional intelligence. In: Barrett, L. F. & Salovey, P. eds. The wisdom of feelings: Psychological processes in emotional intelligence, New York: Guilford Press. 319–340.

Urry, H. L., van Reekum, C. M., Johnstone, T., Kalin, N. H., Thurow, M. E. & Schaefer, H. S. (2006) Amygdala and ventromedial prefrontal cortex are inversely coupled during regulation of negative affect and predict the diurnal pattern of cortisol secretion among older adults. Journal of Neuroscience, 26, 4415–4425.

Witkin, H. A., Dyk, R. B., Faterson, H. F., Goodenough, D. R. & Karp, S. A. (1962). Psychological differentiation: studies of development. Potomac, MD: Lawrence Erlbaum Associates.