Current perspectives and future directions in autism, dyslexia and conduct disorder: A celebration for Uta Frith

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Abstract

Earlier this year, a Festschrift was held at the Royal Society in London to celebrate the outstanding contributions that Professor Uta Frith has made to Psychology over the past 40 years. A series of exceptional talks were given by many of Uta's collaborators – themselves prominent in developmental psychology and cognitive neuroscience. The Festschrift showcased new data that provided refined insights into Uta's three main research areas – dyslexia, autism and conduct disorder. The Festschrift was broad in scope, touching on the nature and relevance of the phonological deficit in dyslexia, refinements to extant theories of autism cast against typical development, and new perspectives on the conceptualisation of conduct disorder and psychopathy. A fitting testimony to Uta's lasting influence, the Festschrift made clear the exciting and tangible possibilities for future research, which will impact upon our understanding of the origins and trajectories of developmental disorders.

At the beginning of this year, many gathered at the Royal Society in London for a Festschrift held in order to celebrate the outstanding contributions that Professor Uta Frith has made to Psychology over the past 40 years. Her manifold achievements were chronicled in an affectionate introduction by **Dorothy Bishop** (University of Oxford). Over the course of her long and prolific career, Uta supervised and collaborated with numerous people, several of whom are now influential in the field of developmental psychology and neuroscience. Many of these people joined together in honour of Uta to present and discuss three main areas of her work. The nature and causes of autism, dyslexia, and psychopathy were discussed from the perspectives of developmental psychology and cognitive neuroscience, cast against findings from typical development. The present article provides an overview of this celebratory feast for a 'modest, generous, extremely enthusiastic and decisive scientist' (Professor Simon Baron-Cohen).

Developmental Dyslexia

The widespread utility and influence of Morton and Frith's (1995) causal modelling for the conceptualisation of developmental disorders (see Figure 1) was reinforced at this conference. This framework can be used to explore the research questions that were asked about dyslexia.

What is the cognitive deficit that characterizes dyslexia?

The most widely held understanding of dyslexia is that the difficulties with reading observed at the behavioural level are caused by an underlying core phonological deficit at the cognitive level (Stanovich, 1988). The precise nature of this phonological deficit is debated. Some researchers propose that the impairment lies in the ability to process phonological information, whereas others argue that the difficulty is rooted at a lower level – namely in the quality of the phonological

representations themselves. **Franck Ramus** (Laboratoire de Sciences Cognitives et Pyscholinguistique, Paris) presented data from experiments that have addressed whether the phonological deficit in dyslexia is at the level of phonological representations or in the productive processes that operate on those representations. Phonological grammar is a set of language-specific rules governing how phonetic features are to be used within words. For proficiency in one's native language, these rules must be learnt during child language acquisition. An interesting question is whether those with dyslexia learn these rules as competently as typically developing people.

In a neat series of experiments, Ramus demonstrated that French adults with dyslexia performed as controls on a variety of tasks tapping both production and perception of phonological grammar: They demonstrated production and perception of regressive voicing assimilation (a rule of phonological grammar specific to French) in legal contexts, and did not show evidence of place assimilation (an English-specific rule) (see Ramus & Szenokvits, in press). Ramus interpreted these data as demonstrating that, contrary to prevailing opinion, adults with dyslexia have normal phonological representations; however, access to these is impaired in the presence of additional task requirements (e.g. high memory load and time constraints).

Heinz Wimmer (University of Salzburg), the colleague with whom Uta pioneered cross-linguistic studies of dyslexia, raised the provocative idea that the core phonological deficit itself should be questioned. Wimmer proposes that reading dysfluency (rather than reading accuracy) is the universal marker of dyslexia, since it is characteristic of dyslexia in both opaque languages like English and transparent orthographies like German. On this basis, he suggested some modifications to the prevalent interpretation of Morton and Frith's causal model of dyslexia. Thus, at the

behavioural level, slow reading would be the cardinal symptom. At the cognitive level, the deficit might be conceptualized as a visual-verbal connection deficit. For Wimmer's theory to hold, future research must discount the possibility that a deficit in reading fluency is a product of degraded orthographic representations, which in turn are caused by degraded phonological representations.

Maggie Snowling (University of York) set out to address the issue of whether or not developmental dyslexia is a selective reading disorder. The answer, as with all developmental disorders, is not straightforward. Historically, studies have pointed to the specificity of dyslexia: Dyslexia can be observed in the absence of more general processing impairments. However, a limitation of this view is that the majority of evidence comes from laboratory-based studies involving highly selected samples. More compelling evidence comes from longitudinal prospective studies of children at family risk for manifesting the disorder.

One such study by Snowling, Gallagher, and Frith (2003) made a useful comparison between typically developing children (TDC), and children with family risk of dyslexia who both did and did not go on to fully develop the disorder. The findings of this study also represent a challenge to the phonological deficit theory of dyslexia. Thus, children with familial risk of dyslexia who later developed the disorder displayed oral language delay (in vocabulary and narrative skills) at 3 years and 9 months. In contrast, the at-risk group who did not later gain a dyslexic diagnosis had comparable language skills to the TDC. Therefore, early language delay appears to be a risk factor for future dyslexia. Arguably, the at-risk children who do not develop dyslexia are safeguarded by their normal nonphonological oral language skills, which act as compensatory factors.

New data presented (Snowling, Muter, & Carroll, 2007) considered whether the at-risk children who were protected from developing dyslexia continued to be free of literacy problems. At 12 years of age, despite average single word reading accuracy, this group was significantly impaired relative to controls on exception word reading and spelling, and they showed a slow rate of writing. This evidence is in line with the view that the risk of dyslexia is continuous (Snowling et al., 2003). Furthermore, those at-risk children with diagnosed dyslexia exhibited oral language impairments (in expressive vocabulary and sentence recall) even at this later stage in development. To the extent that individuals with dyslexia are marked by pre-school oral language deficits, and that these remain 10 years later, the true specificity of dyslexia as a reading disorder may be questioned. Snowling concluded that the modular view of dyslexia as a specific impairment seems increasingly untenable. Rather, while the phonological deficit is the primary risk factor for dyslexia, this may be modulated by other factors such as oral language skills.

The role of intelligence in dyslexia and autism

Concerning the specificity of dyslexia, it has recently been proposed that it simply represents the extreme end of a continuum of reading ability (Plomin & Kovas, 2005), reflecting general intelligence applied to a specific task – namely reading. Such a proposition is in contrast to the predictions made by **Mike Anderson**'s (University of Western Australia) model of intelligence – the theory of minimal cognitive architecture (Anderson, 1992). In this model, dyslexia is caused by a deficit to the specific information processing module concerning phonological processing. Through a series of simulations comparing these different conceptions of dyslexia, Anderson demonstrated a moderating effect of intelligence on reading. He therefore argues that the influence of general intelligence must be explicitly

incorporated into causal theories of dyslexia, and that this may, paradoxically, aid our understanding of dyslexia as a specific disorder.

In Anderson's model there are two routes to knowledge: one through thought (central processes), which fragments into verbal and nonverbal domains, and the second through dedicated information processing modules (cognitive processes). Anderson proposes that it is the speed of one's basic processing mechanism, operating within the first route, which accounts for individual differences in general intelligence: Speed of processing constrains thought and knowledge acquisition, and therefore intelligence. Developmental cognitive disorders are, in contrast, the consequence of damage to dedicated modules. In the case of dyslexia, the deficit is in phonological processes; in autism, the deficit is theory of mind. In neither case should IQ be affected.

Although the prediction of specificity holds in the case of dyslexia, autism is known to be associated with low IQ. However, a confounding factor is poor theory of mind, which may in turn lead to depressed performance on IQ tests. Indeed, people with autism perform as typically-developing individuals on inspection tasks that tap speed of processing, and hence within Anderson's model, general intelligence. So what does intelligence theory add to our knowledge of autism? That while defined as having low IQ behaviourally, individuals with autism have normal speed of processing, signifying average general intelligence. This leaves intact the idea that autism arises from a specific deficit in the theory of mind module, which then has downstream effects (Frith & Happé, 1998) on aspects of general cognitive functioning.

Autism

Theory of Mind and Autism

Huge advances have been made in research on 'theory of mind' (ToM) during the last 25 years. Unquestionably, this has been fuelled by the discovery that children with autism have impairments in understanding false beliefs (Baron-Cohen, Leslie, & Frith, 1985).

It is widely accepted that the ability to represent the mental states of others – their thoughts, desires, beliefs, intentions, and knowledge (ToM) develops at around three and a half years. Desires and intentions are thought to be generally understood by about two years, while knowledge and belief formation are not understood until approximately three and a half years (Sodian, 2005). However, recent findings indicate that an implicit understanding of mental states precedes a full or explicit one in infants as young as 15.5 months (e.g. Onishi & Baillargeon, 2005). **Beate Sodian** (University of Munich) discussed the key question of when it is that infants begin to represent what another person sees independently of what they themselves can see.

Sodian and colleagues used preferential looking techniques to study infants' expectations about a person's search for a hidden object as a function of whether or not the person saw the object hidden (Poulin-Dubois, Sodian, Metz, Tilden & Schopper, submitted). There was no differentiation between 'visual access' and 'no visual access' conditions in 14 month olds, even when the person's gaze direction and body orientation were available as additional cues. By 18 months, a differentiation between 'visual access' and 'no visual access' conditions had emerged, when head turn and body orientation were available as cues: Infants looked longer at the screen when the person pointed to the incorrect location in the 'visual access' condition, and longer at her pointing to the correct location in the 'no visual access' condition. At 24 months, infants appeared to understand the importance of seeing for future action: they expected correct or incorrect search as a function of prior visual access when no

additional cues were available. This suggests that the link between visual perception and predicting future action develops gradually between 14 and 24 months and depends on an increasingly larger set of behavioural cues.

An optimistic interpretation of these results would bestow 18 month-olds with an implicit, nonverbal seeing-knowing understanding. A more prudent interpretation would grant infants with the ability to predict that a person will act towards an object after orienting her gaze and head towards it. Sodian argued that the ability to predict search behaviour from a previous looker-object connection indicates *some* understanding of psychological states.

However, the relationship between early aspects of social information processing and later development of ToM is in need of clarification. Sodian made a plea for research into the development of the use of behaviour cues and action-role encoding in infants with autism.

Sarah-Jayne Blakemore (Institute of Cognitive Neuroscience, University College London) presented a discussion of research on brain development during adolescence and changes in social cognition, in particular ToM, during this significant period.

Two main changes have now been noted in the brain prior to and following puberty. First, whereas sensory and motor brain regions become fully myelinated in the first few years, axons in the frontal cortex continue to be myelinated well into adolescence, implicating increases in the efficiency of neural transmission in these regions during this period. Second, there are changes in synaptic density in the prefrontal cortex (PFC) prior to and during adolescence. Blakemore's interest lies in cognitive implications of changes in the PFC at the onset of puberty and the synaptic pruning that follows it (see Blakemore & Choudhury, 2006).

The brain regions that undergo protracted development throughout adolescence include the PFC, parietal cortex and superior temporal cortex (including the superior temporal sulcus), all of which have been implicated in the circumscribed set of brain regions involved in ToM (see Frith, 2001). Thus, whilst five-year-old children begin to pass ToM tasks, the 'mentalising' brain structures undergo substantial development well beyond early childhood. The exact role each region plays and how this interacts with structural development during adolescence is yet to be determined. Blakemore calls for more research into the contributions of biological and environmental factors to the development of the social brain throughout adolescence.

Are ToM and empathy separate things?

Alexithymia is a difficulty in representing emotional states in order to identify and describe emotions (empathy), and a tendency to focus on external events. Whereas it is widely accepted that ToM is impaired in autism, it is less well known that alexithymia is characteristic of 80-85% of individuals with autism compared to only 10% of the typical population. It could be that the ToM deficit in autism may result in an impairment to consciously reflect on their emotional states. Conversely, **Geoff Bird** (University College London, Institute of Cognitive Neuroscience) argues that alexithymia may be associated with a specific emotional deficit in introspecting upon inner bodily states, subsequently implicating a distinct neural network most likely subserved by the 'interoceptive cortex' (e.g. the anterior insula).

Using fMRI Bird and colleagues have revealed that empathy, involving the representation of emotional states and inner feelings, may be neurologically distinct from the representation of mental states. Only those participants with alexithymia, regardless of autistic status, showed hypoactivity of the anterior insular and amygdala

when reflecting upon emotion and identifying subjective feelings: Participants with autism and low alexithymia showed similar brain activity to controls in the anterior insular and amygdala, and hypoactivity of the "mentalizing" system. Self-reports of alexithymia and empathy correlated with activity in the anterior insula when introspecting upon unpleasant stimuli, but did not correlate with brain activity in the ToM network.

In a further experiment, individuals with low alexithymia and autism showed similar levels of activation to controls in the cortical pain network in response to pain felt by a friend or relative. The degree of empathy on this task correlated with self reports of alexithymia. Bird emphasised that although it appears that alexithymia in autism is not related to the ToM deficit, it remains possible that ToM and alexithymia are associated with different neural networks that employ common components.

Autism and current theories

The ToM account of autism has been of massive theoretical and practical benefit in understanding, recognizing and addressing the social and communicative difficulties in this disorder. However, all deficit accounts of autism fail to explain why people with autism show not only preserved but also superior skills in certain areas.

Frith, prompted by a belief that assets and deficits in autism might have one and the same origin, proposed that autism is characterised by weak 'central coherence' (WCC) (Frith, 1989). Central coherence is the term coined for the everyday tendency to process incoming information in its context – that is, pulling information together for higher-level meaning – often at the expense of memory for detail. **Francesca Happé** (Kings College London, Institute of Psychiatry) re-examined Frith's WCC theory of autism; its origins, modifications, and alternative conceptions.

Since Frith's original conception of WCC, the suggestion of a core deficit in reduced global integration as underlying assets in local processing has moved from a primary problem to a more secondary outcome – with greater emphasis on a bias or cognitive style for local or detail-focused processing. However, questions remain as to whether autism is characterised by WCC, superior local processing, or an interaction of both. Happé and Frith (2006) note that many tasks assume or even create an inherent trade-off between processing at the local and global levels, making it impossible to tell whether patterns of performance in autism reflect reduced global processing, increased local processing, or both. For instance, in the Embedded Figures task successful performance is taken as both a measure of how salient the part is, and how (relatively) weak the camouflaging gestalt is. Hence, it remains plausible that both local superiority and global impairment might be (discrete) dimensions of WCC.

Studies conducted by Happé and colleagues suggest that individuals with autism show problems in integrating information even when attention to detail is not relevant to task performance – arguing that Uta's original conception of WCC has borne out. Their studies have revealed a trade-off between segmentation and integration ability in groups with autism. Conversely, in groups of controls the two abilities correlate when age and IQ are controlled.

Simon Baron-Cohen (University of Cambridge) proposes that autistic traits are continuously distributed in the population. Using self-report, he has shown that scientists have a higher 'Autism Quotient' (AQ) than non-scientists, and that males have a higher AQ than females. Baron-Cohen suggests that empathy (the drive to identify other people's mental states and respond to these with appropriate emotion) and systemizing (the drive to identify rules governing a system) are at either end of a sex-poled dimension: Males show significantly lower self report of empathy quotients

(EQ) than females, but significantly higher self reported systemizing quotients (SQ). In turn, Baron-Cohen's 'extreme male brain' theory argues that individuals with autism show significantly higher SQ and significantly lower EQ than typically developing males (Baron-Cohen, 2002).

In support of this theory, females perform better than males and males perform better than individuals with autism on tasks requiring the judgment of emotional expressions from the eyes. Conversely, males perform better than females and females perform better than individuals with autism on intuitive physics tests. Furthermore, Baron-Cohen has shown that fathers and mothers of children with autism are faster than adult male and female controls on the Embedded Figures task, show poorer ability to judge emotion from the eyes on the Eyes test, and show higher AQ scores. Fathers of children with autism were also more likely to be in 'systematic' careers rather than 'empathetic' careers, compared to fathers of children with Down syndrome.

These findings have been supported at the brain level. Baron-Cohen et al. (2006) used fMRI during the Embedded Figures Test and the Eyes Test with parents of children with Asperger Syndrome (AS) and sex-matched controls. For the Embedded Figures task, female controls showed more activity in extrastriate cortex than male controls, and both mothers and fathers of children with AS showed less activity in this area than controls. For the Eyes test, males showed more activity in the left inferior frontal gyrus, the left medial temporal gyrus and left dorsolateral PFC than females, and both mothers and fathers showed more activity in these areas compared to controls. Although this study needs replicating with larger samples and age-matched controls, it suggests that the genetic characteristics of parents who are carriers of the genes for autism may lead to hyper-masculinization of the brain.

Refining theories of autism

Antonia Hamilton (Department of Psychological and Brain Sciences, Dartmouth College) presented work examining the link between autism and the ability to infer and imitate goals using the mirror neuron system (MNS). The MNS has recently been discovered in the inferior parietal and inferior frontal cortex. The MNS responds when one performs an action and when one observes another's action, allowing matching between self and others, supporting the inference of goals, imitation, and intentions. The 'broken mirror' hypothesis proposes that deficits in imitation, ToM, and social cognition in autism are caused by an abnormal MNS (Williams, Whiten, Suddendorf, & Perrett, 2001).

Hamilton presented four main findings. First, there were no differences in performance between groups with autism and control groups matched for verbal mental age on goal-directed imitation, counter to the prediction of the broken mirror hypothesis. Second, both typically developing control children and children with autism showed a preference for mirror imitation of hand actions over anatomical imitation. Third, children with autism should show similar levels of motor planning to typically developing controls and took advantage of the experimenter's demonstration of an action in imitation tasks. Fourth, children with autism performed better than matched controls on a gesture recognition task. Together, data refute a strong version of the broken mirror hypothesis of autism.

However, in all of the experiments reported the children were instructed to copy the experimenter; these results may not generalise to situations of automatic mimicry. Hamilton argues for two routes for imitation: The emulation pathway (purposeful imitation or goal directed action), and the mimicry pathway (automatic, implicit imitation). Hamilton posits that while emulation is intact in autism, automatic

social mimicry may be abnormal, as suggested by findings of abnormal brain activity in autism during emotional facial imitation. Thus, different brain regions may serve emulation and mimicry and Hamilton postulates that the ToM network might exert a top-down influence on the MNS such that the use of mimicry to enhance social interaction may be impaired in autism.

Methodological and philosophical issues in theory of mind

The false belief task has been used as evidence for the view that ToM is impaired in autism. Zaitchik's (1990) false photo task has been used as non-mental analogue of the false belief task. Both feature Sally who changes her red dress for a green one while her friend Anne goes to find a matching belt (in the false belief task), or while the photo is developing (in the false photo task). Participants are asked, "What colour dress does Anne think Sally is wearing?" or, "What colour dress is Sally wearing in the photo?" In 1991 **Josef Perner** (University of Saltzburg) and **Sue Leekam** (University of Durham) found that 3 year-olds failed both tasks while older children with autism only failed the false belief task, indicating that 3-year-olds fail to understand representations while children with autism have a specific problem with mental representations.

However, Perner and Leekam argue that Anne's belief about the colour of Sally's dress is false, but the status of the photo of the dress is not. The photo is true at the time it is taken, even though it does not depict the current situation. Thus understanding assessed in the belief and photo tasks differs not only with respect to the visibility of the representational entity but also in their representational relation to their target (belief misrepresents its target; photo shows its target correctly). Hence, the belief task requires a deeper understanding of representation than the photo task

and hence has higher level cognitive demands as well as tapping domain-specific knowledge (ToM).

Parkin (1994) designed a 'false sign' task in which a signpost indicates an object in location 1, which is then moved to location 2, but the signpost is not changed and becomes false. Children have to infer where the sign shows the object is. A false sign is a non-mental, observable object, but like the false belief task, in order to understand what the false sign shows, one needs to understand that the signpost represents a situation that is different from how the signpost shows it to be.

Robust correlations have been found between the belief tasks and the sign tasks in typical children and groups with autism (Bowler et al., 2005) but not between the belief and photo tasks, suggesting only the former tasks tap a common developmental factor. This factor underlies typical children's problems understanding false beliefs and false signs, and the difficulty that children with autism have with the false belief task. Thus, autism cannot simply be characterized by a domain specific problem with understanding mental states.

Perner and colleagues investigated whether the brain areas active for processing false belief information are also employed for false signs (Perner, Aichhorn, Kronbicler, Staffen, & Ladurner, in press). They found that the right temporal parietal junction is a module involved in belief-desire reasoning (i.e. in understanding false beliefs but not false signs or photos), whereas the left temporal parietal junction was a module involved in perspective thinking (i.e. in processing false beliefs and false signs). This suggests that ToM is not processed by a narrowly circumscribed local brain region – some regions may be specialised for internal, unobservable mental states involved in belief-desire reasoning, and others may deal

with mental and non-mental problems involving a contrast of perspectives. Perner called for a shift towards a more fractionated view of ToM.

In a similar vein, **Frederique de Vignemont** (Institut de Jean-Nicod, France) also encouraged a refinement of the role ToM plays in autism. The crux of her argument is that rather than ToM having a single function, different kinds of mind-reading are required for social observation (knowledge-oriented) and for social interaction (interaction-oriented). Explicitly, it matters whether the other person is viewed from an 'egocentric' (you in relation to me) or an allocentric (he, she, they) frame of reference. An allocentric view of the world allows an understanding of social rules and social structure whereas an egocentric view (in which the self is a referent) is essential for social interaction.

The mind-reading literature shows that various empathic and inferential capacities are necessary to sway our interpretations of current situations away from natural egocentric bias. A recurrent finding in social psychology is the privilege of one's own perspective in the representation of the world. For instance, we know that most people tend to overestimate the degree to which their actions are noticed by others and that they attribute to others their own perspective more than they do the opposite. Representations of the social world tend to be egocentric by default, and to adopt the perspectives of others is a more effortful operation.

To some extent, autism may be seen as a pathological form of egocentric bias (de Vignemont & Frith, 2007). Individuals with Asperger syndrome often prefer people around them to behave as they desire and get frustrated if they are not obeyed. More specifically, the interaction between the egocentric state and the allocentric state may be disrupted in Asperger's syndrome. While the typical population is aware

when to use an egocentric or an allocentric frame of reference, people with Asperger syndrome are unable to switch appropriately from one to the other.

Poor Conduct & Psychopathology

The final contributions to the Festschrift consider a set of developmental disorders, that have only relatively recently been under cognitive investigation – conduct disorders.

Psychopathy is characterised by extreme anti-social behaviour and the presence of an affective-interpersonal impairment. Individuals with psychopathy represent approximately 25% (Hart & Hare, 1996) of individuals who meet criteria for the psychiatric classifications of conduct disorder and anti-social personality disorder. Psychopaths differ from other criminals in their lack of remorse and their difficulty in achieving normal control of behaviour. **James Blair** (Unit of Effective Emotion, Maryland) posed whether the callous unemotional component of psychopathy has similarities with the socio-emotional difficulties in autism.

Autism and psychopathy are both associated with empathic and amygdala dysfunction. However, prudent inspection suggests differences in the nature of the impairments shown by the two groups. Blair (2005) argues that empathy encompasses a variety of different functions that are mediated by partially dissociable neurocognitive systems: cognitive empathy (or ToM), motor empathy (the mirroring of motor responses of another person), and emotional empathy (the physical response to an emotional display) (Blair, 2005). When considering empathic dysfunction, one must determine the exact form of the functional impairment.

Evidence thus far suggests that autism presents difficulties with cognitive and motor empathy, related to the impaired functioning of the superior temporal cortex, but intact emotional empathy. In contrast, psychopathy is characterised by deficits in

emotional empathy but not in cognitive or motor empathy. The impairment in psychopathy is in the processing of sad, fearful, and disgusted expression, in stimulus-reward associations and the amygdala's response to rewarding and punishing stimuli, and in moral judgment and decision making associated with orbitofrontal function. Conversely, autism is free of such impairments. Blair and colleagues have revealed that individuals with autism show normal responses to a fear potential startle paradigm whereas psychopathic individuals show reduced conditioning and reduced amygdala activation.

Blair has demonstrated that psychopaths have standard mind-reading abilities but their capacities for cognitive empathy are impaired. Neuro-imaging supports this interpretation and differentiates psychopathy from autism (Blair, 2003).

Essi Viding (University College London) and **Alice Jones** (Insitute of Psychiatry, Kings College London) aimed to refine the concept of conduct disorder. Fifteen to 25% of individuals with conduct disorder display anti-social behaviour in combination with callous-unemotional traits (CU+). CU+ traits are risk factors for persistent misconduct and also for psychopathy. In better understanding the CU+ traits in individuals with anti-social behaviour problems as an antecedent to criminal activity, one may be able to intervene more effectively and prevent future seasoned criminal and psychopathic behaviour.

At the behavioural level, the CU+ profile is characterised by a lack of empathy and remorse, manipulative behaviour, and early onset of offending. In contrast, the CU- profile (in absence of callous-unemotional traits) is characterised by impulsive behaviour, reactive violence to environmental triggers, and remorse. At the cognitive level, the CU+ profile can be described as impaired activation of violence representations and stimulus-reinforcement associations, poor fear perception, and a

failure to learn from punishment. The cognitive system of the CU- profile is described as overactive and hypersensitive to anger cues. At the biological level, hyporeactivity of the amygdala has been implicated in the CU+ profile. The orbitofrontal cortex (OFC) has also been implicated in CU+ traits such as lack of emotion in decision making and poor emotional judgment. Viding and colleagues revealed that CU+ boys activated the left amydala less than control children, whereas CU- boys showed the opposite pattern. Thus, the emotional circuitry may be hypoactive in the CU+ profile but hyperactive in the CU- profile.

Viding's interest in the biological level of explanation for these profiles led to a behaviour-genetic analysis, involving over 7500 twins from the Twins Early Development study (Viding, Blair, Moffitt, & Plomin, 2005). A large genetic influence on anti-social behaviour was found for those with CU+ traits suggesting a genetic mediation of conduct disorder in such cases, whereas the genetic influence on anti-social behaviour in relation to CU- was moderate with a stronger environmental influence. Thus, CU- behaviour may be triggered by environmental cues and in line with this, it is more responsive to intervention than the CU+ profile.

Conclusion

Research both conducted and inspired by Uta has enlightened our understanding of the deficient processes that characterize developmental disorders, and provided great insight into the normal development of these processes. As highlighted by the contributors to the Festschrift, there is continuing need for greater complexity in the models of the relationships between brain and cognition that address variations in the behaviours observed within the same broad class of disorder, and that will also elucidate shared risk factors between different disorders. A further notable achievement that has not been highlighted in this Festschrift is the influence

that Uta's research has had not only on theory, but also practice – particularly in the educational and clinical professions. Uta has been and will continue to be inspirational to past, present and future researchers in the field of developmental psychology and cognitive neuroscience.

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Figure Captions

Figure 1: Morton and Frith's (1995) causal modeling framework for developmental disorders. Disorders ought to be explained at all three levels – biological, cognitive and behavioural – with a causal chain connecting each level

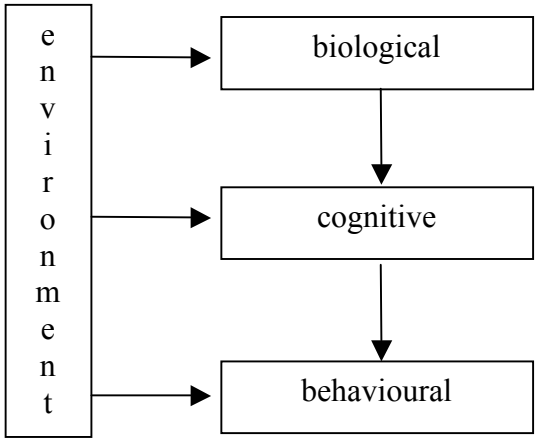

Figure 1