

WJEC Psychology A-level

Autistic Spectrum Behaviours

Notes

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Part 1: The Role of Amygdala Dysfunction

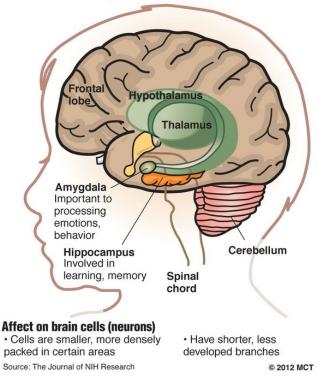
 ¹Research has suggested that amygdala dysfunction may be responsible for several characteristics of autism, such as abnormal eye contact, difficulties associated with face processing and also a lack of mental understanding. Therefore, consideration of autism in terms of a self-relevance detection system may be more appropriate. This theory is based on Baron-Cohen et al's research into the Eyes Task, where adults and adolescents with both autism and Apperant's Syndrome, were unable to

Asperger's Syndrome, were unable to identify the emotions displayed by an individual whose eyes were only visible, which also corresponded to reduced amygdala activity, as measured through an fMRI scan.

- More recent research has refuted the idea that the amygdala is the main area of the brain regulating emotional behaviours within the limbic system, as suggested by Prather et al (2011), who found that abnormal fear behaviours were displayed by primates who'd undergone surgical lesioning of the amygdala. Disruption of the laterobasal nuclei group of the amygdala is particularly linked with autism, due to such an area processing sense-related information (i.e. visual, auditory and olfactory), as suggested by Bzdok et al (2012). Despite such areas being key in primates, the connection between the amygdala and the anterior cingulate cortex in humans is particularly associated with autistic/atypical behavioural patterns.
- Duerden et al, concluded that ²"clear maturational differences exist in social cognition and limbic processing regions only in children/adolescents and not in adults with ASD, and may underlie the emotional regulation that improves with age

Autism and the brain

The areas of the brain affected by autism, which stems from abnormal brain development:



in this population", on the basis that ASD sufferers had a smaller grey matter volume in their medial prefrontal cortices.

- The anygdala does not work in isolation, but rather forms a priority map of self-relevant events through continuous interactions with the ventromedial prefrontal cortex. Therefore, this suggests that it is inappropriate and reductionist to assume that amygdala dysfunction alone can be responsible for autism, but rather that this disorder is better explained through a holistic theory of brain function (and not localised!).

- There are issues with the early basis of the amygdala dysfunction theory, where recent was primarily conducted on animals who, arguably, have a smaller range of emotions compared to humans (and a lower intensity), as well as having different amygdala systems and emotional regulation mechanisms. This means that such a theory may have low ecological validity.

+ However, there has been some evidence supporting the role of amygdala dysfunction in humans, and particularly the research conducted by Duerden et al, which demonstrated that individuals with ASD have neurological differences compared to neurotypical individuals, which may coincide with differences in amygdala functioning also. This supporting evidence increases the validity of the theory as an explanation for autism.

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¹ Zalla, T. And Sperduti, M., The amygdala and the relevance detection theory of autism: an evolutionary perspective. *Frontiers of Human Neuroscience*, 2013, 7: 894

² Duerden, E.G., Mark-Fan, K.M., Taylor, M.J. and Roberts, S.W., Regional differences in grey and white matter in children and adults with autism spectrum disorders: an activation likelihood estimate (ALE) meta-analysis, *Autism Research*, 2012, 5(1):49-66.



Part 2: Genetic Predisposition for Autism

- In a ³recent review of research into the risk factors for autism, conducted by Chaste et al (2012), several genetic risk factors had been identified.
- 1. The appears to be a biological basis for autism, where there are concordance rates of 12-20% for siblings where one member has autism. This is particularly important when considering that such siblings share 50% of their genetic information with each other.
- Autism has been associated with mutations of the following genes: NLGN3, NLGN4X, SHANK3 and the 12q11-q13 maternal allele. The incidence of these mutations in the autistic population has been confirmed through the use of genome-wide studies. These candidate genes (where each genetic variation slightly increases the likelihood of developing autism) are specifically implicated in the maintenance of the synapse, and there are up to ⁴234 candidate genes contributing to the development of autism.
- Autism may also considered a ⁵sex-linked disorder, due to the fact that it affects four times as many males as females. This strongly suggests a link between autism and sex, meaning that mutations on the sex chromosome may be particularly important in the development of autism.
- Despite there being the possibility of candidate genes existing for autism, it is important to consider the diathesis-stress model where if an individual has specific candidate genes, they are not destined to develop autism. For ⁶example, there is a greater incidence of autism in first-born children in Nordic countries, and autism is associated with pre-natal factors such as an abnormally low birth weight and neonatal anemia. Therefore, an interactionist stance may be more beneficial when explaining the genetic/biological basis of autism and the implications that this has.

- There has been a lack of replication of the results produced by several family studies investigating the risk factors associated with autism. This makes it more difficult to draw a clear and reliable 'cause and effect' relationship between candidate genes and autism, thus reducing the validity of genetic explanations for autism.

- There is a very large number of candidate genes coding for the development of autism. This poses a practical issue because it is unlikely that in the future, potentially, genetic tests will be able to screen for so many candidate genes with a high degree of accuracy. This number also poses a theoretical issue because it is increasingly difficult for researchers to assess the relative significance of each of these genes towards the development of autism, and so it is tricky to decide which genes to target in potential therapies.

- Genetic/biological determinism = The focus on the biological basis of autism means that it is easy to simply view autism as a biologically-determined disorder. However, through adopting an interactionist stance, this is not the case, due to the disorder being the result of an interaction between both genetic and environmental risk factors. Therefore, this 'middle-ground' could be considered as a more viable and valid explanation for autism.

Part 3: Theory of Mind

- Theory of Mind (ToM) can be described as the ability to understand/identify what other people are thinking and feeling, through a 'mind-reading'-like process.
- Those with autism may have a deficit of ToM, meaning that they cannot understand the emotions of others, or even comprehend that individuals can have emotions different to their own. Such misunderstandings may explain why those with autism have impairments in empathy, social communication and social imagination.

³ Chaste, P. And Leboyer, M. Autism risk factors: genes, environment, and gene-environment interactions, *Dialogues of Clinical Neuroscience*, 2012, 14(3): 281-292.

⁴ Sanders SJ., Ercan-Sencicek AG., Hus V., et al. Multiple recurrent de novo CNVs, including duplications of the 7q11.23 Williams syndrome region, are strongly associated with autism. *Neuron.* 2011;70:863–685

⁵ Fombonne E. Epidemiology of autistic disorder and other pervasive developmental disorders. *J Clin Psychiatry.* 2005;66 (suppl 10):3–8

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⁶ Gardener H., Spiegelman D., Buka SL. Perinatal and neonatal risk factors for autism: a comprehensive meta-analysis. *Pediatrics.* 2011;128:344–355

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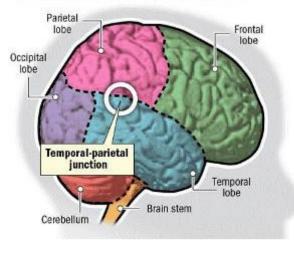


- Other social deficits caused by ToM impairments include a lack of understanding that behaviour impacts how others think and/or feel, alongside problems differentiating fact from fiction, as characterised by poor performance on 'false-belief' tasks, such as the Sally-Anne task.
- In this case, where the participants were asked to identify where Sally would look for her marble
 after it had been moved without her knowledge, 85% of the control group (14 with Down's
 Syndrome and 27 neurotypical children) correctly answered, compared to 20% of the autistic
 group. This supports the idea that a ToM deficit is responsible for autistic children being unable
 to understand that people can believe something that is not true. This lack of understanding of
 others' viewpoints and emotions may also explain another characteristic trait of autism:
 difficulties predicting the behaviour or emotional states of others.
- ToM can also be assessed specifically in children below the age of 2 years old, as suggested by Meltzoff (1988), using intentional reasoning tasks. In such tasks, Meltzoff found that 18 month olds, after observing an adult struggling to place beads into a jar, dropped no beads and so imitated the intention of the adult, as opposed to the actual action (which is what would have been predicted by social learning theory). Therefore, this we can assume that children as young as one and a half years old can understand and imitate intention, on the basis of observable behaviour, and so appear to have at least some understanding of ToM.
- Since adults with Asperger's Syndrome can easily perform on false belief tasks, they appear to perform less successfully on 'The Eyes Task', which involves identifying the emotion displayed by a character whose eyes can only be seen. Baron-Cohen et al concluded that since adults with AS continued to perform poorly on such tasks, that they still suffered from ToM deficits, but these deficits simply had to be assessed in another way. This is in line with the original ToM theory and its link with autism!

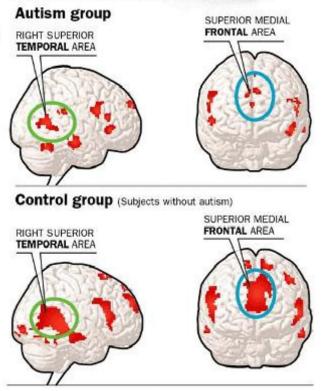
Making the connection

Studies conducted by Carnegle Mellon University suggest that one of the key problems in autistic people's brains is that the brain areas that do "theory-ofmind" processing, figuring out what is in someone else's mind, are badly connected.

Researchers tested this by asking a group of people with high-functioning autism and another group that did not have the disorder to watch **line-drawing animations** while lying in a magnetic resonance scanner, which can measure blood flow in the brain. The **people with autism** had a much harder time figuring out what the animated characters were doing and why.



Below, brain images taken while the autistic people were struggling with this task showed some activity in one key area of their theory-of-mind network, the **temporal-parietal junction**. The scans also showed that other important spots in the **frontal lobe** of the brain were less active than in the control group and **were not synchronized well** with areas farther back in the brain.



Previous research, such as that carried out by Baron-Cohen et al (1985) using the Sally-Anne tasks, has focused on the link between ToM and the cognitive impairments suffered by those with AS. Although this does serve as a comprehensive explanation, ToM deficits cannot explain the desirable characteristics which belong to AS sufferers, such as advanced numerical and logical reasoning. This means that ToM is a limited, and not universal, explanation for autism.
 ToM has close links with perspective-taking, as both involve understanding another person's thoughts and emotions, and thus allowing the observer to take on the perspective of another.

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However, this also makes drawing the distinction between the two increasingly difficult. For example, Meltzoff's intentional reasoning tasks can be explained in terms of the child taking on the perspective and thus intention of the adult (perspective-taking) as well as understanding the struggles and aims experienced by the adult (ToM). Therefore, this means that it is difficult to differentiate between the mechanisms of ToM and perspective-taking, limiting the theoretical value of both explanations.

- The Eyes Task can be said to have low mundane realism, because the procedure does not represent everyday life where we are usually able to look at the entire person's face and facial expressions, in order to assess their emotions. Verbal cues, such as the tone of their voice, as well as language provide us with essential information about their feelings. Therefore, such studies may produce findings with little ecological validity, because they cannot be generalised beyond the original, specific research settings.

Part 4: Gender Differences

- The idea that autism is four times more likely to occur in males compared to females, has sparked further research into the individual differences and basis of sex differences in the incidence of autism.
- ⁷It may be that despite males suffering from fewer risk factors for autism than females, a
 phenomenon which has been described as the 'female protective effect', females are less likely
 to develop the symptoms associated with ASD, meaning that these symptoms may be too mild
 or even masked to merit a diagnosis.
- A key issue associated with the diagnosis of ASD is ⁸that the symptoms appear to develop in later life or adolescence. This may be an example of gender bias because the appearance of symptoms may be due to different communication skills and stages of maturational development experienced by each gender, which are also affected by individual differences. This means that males may not necessarily always have higher incidence rates of autism, but merely appear to do so.
- Researchers have given different explanations for these differences in incidence rates between
 males and females. For example, Wing (1981) concluded that ⁹"the excess of males was much
 more marked in language and socially impaired children who were of higher ability. The findings
 were linked to hypotheses of genetically greater variability in males, and to male-female
 differences in visuospatial and language skills". Therefore, this suggests that the differences in
 autism between males and females is also in line with differences in other cognitive abilities,
 which may increase the likelihood of diagnosis for autism in males.
- ¹⁰The National Autistic Society, using evidence provided by Gould and Ashton-Smith (2011), suggested that females are less frequently diagnosed with autism because they are more capable of masking their symptoms, they interact socially with others more frequently, they experience greater social expectations and have more active imaginations and so role-play more frequently.

— There is co-morbidity between ASD and other social developmental/communicative disorders, such as ADHD, as demonstrated by ¹¹Willcutt (2012). This comorbidity is stronger in males compared to females, which further supports the idea that males may simply be at a greater risk of developing autism because of their greater likelihood of developing other disorders, rather than candidate genes or mutations on the sex chromosomes (particularly the Y chromosome).

⁷ Halladay, A.K. et al, Sex and gender differences in autism spectrum disorder: summarising evidence gaps and identifying emerging areas of priority, *Molecular Autism*, 2015, 6:36.

⁸ Ozonoff S, Young GS, Carter A, Messinger D, Yirmiya N, Zwaigenbaum L, et al. Recurrence risk for autism spectrum disorders: a Baby Siblings Research Consortium study. Pediatrics. 2011;128:e488–95.

⁹ Wing, L., Sex ratios in early childhood autism and related conditions, *Psychiatry Research*, Volume 5, Issue 2, 129 - 137

¹⁰ Gender and Autism, The National Autistic Society, Accessed on 24.08.17, Accessed through <u>http://www.autism.org.uk/about/what-is/gender.aspx</u>

¹¹ Willcutt EG. The prevalence of DSM-IV attention-deficit/hyperactivity disorder: a meta-analytic review. Neurotherapeutics. 2012;9:490–9

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Therefore, this suggests that biological differences between males and females are not enough to explain differences in the incidence rates of autism.

- The idea of gender differences in autism has little practical value, because it has been suggested that the symptoms of ASD only develop in later life i.e. in adolescence. This means that it is difficult to diagnose young children with ASD and so prevent treatments or therapies for their respective symptoms.

+ There is a practical application associated with an increased understanding of the gender differences underlying autism. This means that diagnoses techniques should be catered more specifically towards both males and females. For example, with the Sally-Anne studies conducted by Baron-Cohen et al., there may have been a smaller autism incidence rate for females because they are more accustomed to role play and playing with dolls compared to boys. Such gender differences should be considered a key element in diagnoses.

Part 5: Refrigerator Mother

• ¹²The Refrigerator Mother theory was developed in the 1940s by Kanner and saw autism as the result of a lack of maternal warmth during childhood, and so a consequence of overly-harsh



parenting and a lack of a secure bond/ attachment.

•The theory argues that a lack of maternal warmth means that the child's personality is not fully developed, and compared the family climate created by a refrigerator mother as being akin to the conditions within a World War II concentration camp, which sparked particular controversy and debate.

•The aftermath of the Refrigerator Mother theory could still be seen in later theories, such as Mahler and Furer (1959) viewing autism as the product of the child attempting to dedifferentiate themselves in response to a lack of maternal warmth and an inability to form social bonds with their mother (initially).

•The focus of previous child physical and emotional abuse is still cited to this day as the

cause of autism. For example, Frances Tustin, a prominent autism researcher, concluded that 'psychogenic' autism may arise as ¹³"a reaction to brain damage or to sensory defect, as well as to a traumatic situation which seems to threaten life and limb".

— This theory has the particular problem of leading to parent-blaming. Parents, who are also likely to be responsible for the care of their autistic child, are already upset and traumatised by the idea that their child is ill, and would be further hurt by the idea that their parenting may be the cause for autism. This may explain the ever-decreasing support for this theory - parents simply no longer tolerate it.

- This theory focuses almost entirely on situational factors, such as the presence of an overly harsh mother. Therefore, no consideration is given towards biological factors, such as candidate genes, which may increase the child's likelihood of developing autism, the idea that autism is four times more frequently diagnosed in males compared to females, the broken mirror neutron hypothesis or the role of perspective-taking and theory of mind in the development of autistic behaviours. Such a reductionist approach is unlikely to provide an accurate and reliable explanation for autism!

— There may be other confounding variables associated with a harsh family climate and a 'refrigerator mother', which may have been more significant in the development of autism rather than Kanner's original theory. For example, social deprivation, a lack of educational opportunities, personality differences, genetic predispositions and family histories of autism can all be

¹² Refrigerator Mother - A Discredited Cause of Autism Fact Sheet, Synapse-Reconnecting Lives, Accessed on 24.08.17, Accessed through <u>http://www.autism-help.org/points-refrigerator-mothers.htm</u>

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¹³ Frances, T. Revised understandings of psychogenic autism, *The International Journal of Psycho-Analysis*, 72.4 (Jan 1, 1991): 585.





considered as risk factors for autism, where no single risk factor immediately means that autism will develop!

Part 6: The Empathising - Systemising Theory

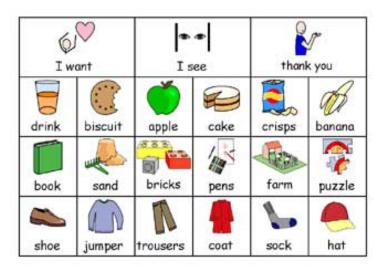
- ¹⁴Autism can be explained as a result of deficits in empathy and abnormal development of empathy, which may act as compensation for high levels of systematising shown by ASD patients. This was based on the idea that ASD patients show abnormally low levels of affective empathy (involved in emotional self-regulation and identifying emotions in others) as measured by the Empathy Quotient.
- The larger the difference between an individual's empathy and systematising levels, the more likely they are to develop autism. High levels of systematising means that ASD patients can identify patterns within the following systems: collectable, mechanical, numerical, abstract, natural, social and motoric. This may explain why individuals with ASD have a greater need or desire to systematise or identify patterns within these systems, such as showing an exceptional understanding of the complex mechanisms of a camera, as demonstrated by Baron-Cohen et al (1985).
- + The ES theory can explain both the social and behavioural symptoms of autism in a simple and precise manner. For example, a high need for systematising can explain repetitive behaviour and strong analytical skills, characterised by superior temporal and reasoning skills.
- + Both the ES theory and the idea of Weak Central Coherence share the same advantage of providing a detailed explanation for autism, where individuals pay particular attention to small details which all have a role within each system, and so are considered important by the individual. Therefore, the ES theory can easily explain behaviours which are specific to autism.

— However, there are differences between the ES theory and the idea of Weak Central Coherence. For example, the first theory sees an almost obsession over detail as undesirable and negative, whereas the ES theory sees such an obsession as desirable and demonstrating a positive symptom of autism, which has been demonstrated with exceptional individuals with autism, such as Richard Borcherds.

+ An improved understanding of the ES theory, particularly in relation to the ideas of Weak Central Coherence and the extreme male brain theory, means that there are practical applications in education as a way of accommodating such individuals. These methods, as suggested by Baron-Cohen et al, would be suited towards the hallmarks of autism, such as narrow interests and an urge to carry out repetitive behaviours associated with an obsession of detail.

Part 7: Picture Exchange Communication System (PECS)

- ¹⁵Research has shown that PECS systems can be successfully used to increase the number of words spoken by autistic children, and specifically more complex words, as well as reducing the frequency of non-verbal vocalisations used by young children, thus building a stable platform for future effective social communication skills.
- The system works through children making associations between giving someone a card with a picture on it, and receiving an item in return for this card i.e. positive reinforcement in operant conditioning terms. This means that requests made by young



¹⁴ Baron-Cohen, S., The empathising-systemising theory of autism: implications for education, *Tizard Learning Disability Review*, 2009, 14(3).

¹⁵ Ganz, J.B. and Simpson, R.L., Effects on Communicative Requesting and Speech Development of the Picture Exchange Communication System in Children with Characteristics of Autism, Journal of Autism and Developmental Disorders, 2004, 34(4), pp. 395-409.

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autistic patients are clear to both them and the recipient. Such a system can be used at home and in educational settings.

- ¹⁶There are 6 phases used by the PECS: Learning how to communicate, appreciating the importance of distance and persistence, recognising picture discrimination, constructing appropriate sentence structures, learning how to answer questions and give suitable comments on such answers.
- + There is evidence to support the effectiveness of PECS systems. For example, Charlop-Christy et al (2002) found that ¹⁷"all 3 children met the learning criterion for PECS and showed concomitant increases in verbal speech. Ancillary gains were associated with increases in social-communicative behaviours and decreases in problem behaviours". This suggests that the use of PECS has a proven practical application!

However, evidence has also suggested that there may be problems with ¹⁸maintenance and generalisation, as suggested by Flipping et al (2010). This means that PECS may not be as effective as portrayed, with the possibility of children quickly forgetting how to use the system if not applied consistently, or generalising the cards to other items which are not featured.
 According to the ¹⁹Research Autism group, there are several issues with the research supporting PECS. These include the issue of generalisability because most of the studies are small-scale with few participants, meaning that the findings are unlikely to be applied to the general autistic population. Secondly, the frequent lack of control groups within these trials means that statistical analyses or baseline comparisons with the autistic group are not possible, so we do not know whether the use of PECS has a significant impact. Thirdly, some of the participants have disabilities other than autism, which makes it difficult to evaluate whether the apparent improvements in communication are due to the alleviation of autistic symptoms, or of the symptoms of the other learning disabilities which participants may have, and so this acts as a confounding variable.

Part 8: Relationship Development Intervention

According to ²⁰Autism Speaks, relationship development interventions (RDT) depends on developing dynamic intelligence and improving interpersonal relationships within a family. The aims of RDT are: emotional referencing, social coordination, the use of declarative language, flexible thinking, relational information processing, foresight and hindsight. Therapy sessions involve developing these skills, with a particular focus on non-verbal communication.
 A key issue associated with RDI is that there have been no independent studies carried out to assess the effectiveness of the treatment. The original study conducted by Gurstein et al (2007) found positive results for the small sample size used (only 16 children) but even reported that

¹⁷ Charlop-Christy, M. H., Carpenter, M., Le, L., LeBlanc, L. A. and Kellet, K. (2002), USING THE PICTURE EXCHANGE COMMUNICATION SYSTEM (PECS) WITH CHILDREN WITH AUTISM: ASSESSMENT OF PECS ACQUISITION, SPEECH, SOCIAL-COMMUNICATIVE BEHAVIOR, AND PROBLEM BEHAVIOR. *Journal of Applied Behavior Analysis*, 35: 213–231. doi:10.1901/jaba. 2002.35-213

¹⁸ Flippin, M., Reszka, S., and Watson, L.R. Effectiveness of the Picture Exchange Communication System on Communication and Speech for Children With Autism Spectrum Disorders: A Meta-Analysis, *American Journal of Speech-Language Pathology*, 2010, 19(1), pp.178-195.

¹⁹ Picture Exchange Communication System and Autism, Research Autism - *Improving the Quality of Life*, Accessed on 24.08.17, Accessed through <u>http://www.researchautism.net/interventions/36/picture-exchange-communication-system-and-autism/Current%20Research</u>

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²⁰ Relationship Development Intervention, Autism Speaks, Accessed on 24.08.17, Accessed through <u>https://www.autismspeaks.org/what-autism/treatment/relationship-development-intervention-rdi</u>

¹⁶ The Picture Exchange Communication System, *National Autism Resources*, Accessed on 24.08.17, Accessed through <u>https://www.nationalautismresources.com/the-picture-exchange-communication-system-pecs/</u>



²¹"generalisability of current findings is limited by the lack of a control or comparison group, constraints on age and IQ of treated children, parent self-selection, and parent education conducted through a single clinic setting". This reduces the validity of RDI as a potential treatment for autism.

- Secondly, there has been no peer review of any published studies concerning RDI. This means that the choice of statistical test, formulation of hypotheses, significance levels and discussion of results has not been independently checked by other experts in the field. This reduces the reliability and validity of the conclusions drawn about the effectiveness of RDI.

²¹ Gurstein, S.E., Burgess, A.F. and Montfort K., Evaluation of the relationship development intervention program, *Autism*, 2007, 11(5):397-411.

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