

Simon Baron-Cohen, Daniel Bor,
Jac Billington, Julian Asher,
Sally Wheelwright and Chris Ashwin

Savant Memory in a Man with Colour Form-Number Synaesthesia and Asperger Syndrome

Abstract: *Extreme conditions like savantism, autism or synaesthesia, which have a neurological basis, challenge the idea that other minds are similar to our own. In this paper we report a single case study of a man in whom all three of these conditions co-occur. We suggest, on the basis of this single case, that when savantism and synaesthesia co-occur, it is worthwhile testing for an undiagnosed Autism Spectrum Condition (ASC). This is because savantism has an established association with ASC, and the combination of ASC with synaesthesia may increase the likelihood of savantism. The implications of these conditions for philosophy of mind are introduced.*

The Assumption that all Human Minds are Wired the Same

The problem of other minds has been of interest to researchers in fields as diverse as philosophy of mind (Goldman, 2006; Nagel, 1974; Wittgenstein, 1958), developmental and clinical psychology (Baron-Cohen & Cross, 1992; Baron-Cohen, Golan *et al.*, 2004; Baron-Cohen, Wheelwright & Jolliffe, 1997; Perner, 1991) and neuroimaging (Baron-

Correspondence:

Simon Baron-Cohen *et al.*, Autism Research Centre, Department of Psychiatry, University of Cambridge, Douglas House, Cambridge CB2 2AH, UK.
Daniel Bor, MRC Cognition and Brain Sciences Unit, 15 Chaucer Rd, Cambridge CB2 2EF, UK.

Cohen & Ring, 1994; Baron-Cohen, Ring *et al.*, 2000; Frith & Frith, 1999; Iacoboni *et al.*, 2005).

This article focuses on two clinical conditions, autism and synaesthesia, that challenge the assumption that all human minds are wired the same. Even though only one percent of the population at most may have each of these conditions (Baird *et al.*, 2006; Baron-Cohen, Burt *et al.*, 1996), this illustrates a basic problem that there may be other minds that are wired differently at a neuronal level. Neurodevelopmental conditions like autism and synaesthesia invalidate the assumption that all humans share similar conscious experiences because they share similar neural architecture.

In the next section, we summarize previous studies of these two neurodevelopmental conditions, before reporting a single case study of a young man who has both. One reason for reporting this case is that currently it is not known how rare or common it is to have both conditions. We wish to encourage other clinicians or researchers to document such cases in order for us to understand if they are related in any way. If each condition separately has a prevalence of 1%, and if these were truly independent, then the probability (p) of them co-occurring would be calculated using the multiplication law: $p(\text{synaesthesia}) \times p(\text{autism}) = 0.01 \times 0.01 = 1$ in 10,000 (i.e. quite rare). However, if they share some common causal mechanism, such as neural over-connectivity (Baron-Cohen, Harrison *et al.*, 1993; Belmonte *et al.*, 2004; Rouw and Scholte, 2007), then they may co-occur more often than chance. Their possible independence or association remains to be tested and epidemiological methods are required to find out if they are related.

The single case study we report below in whom both conditions co-occur is all the more interesting because he has a third 'condition': savantism. We not only wish to document his unusual profile but also wish to speculate on the relationship between these three conditions. In particular, we put forward the proposal that whenever autism and synaesthesia co-occur, the likelihood of savantism is increased. Such a proposal requires empirical validation through the documentation of further cases.

Synaesthesia

Synaesthesia is defined as occurring when stimulation of one sensory modality automatically triggers a perception in a second modality, in the absence of any direct stimulation to this second modality (Cytowic, 1989, 1993; Marks, 1975; Motluk, 1994; Vernon, 1930). For example, a sound automatically and instantly triggers the perception of vivid colour: a person describes the sound of the word MOSCOW as

'Darkish grey, with spinach green and pale blue' (Baron-Cohen, Wyke & Binnie, 1987). Many combinations of synaesthesia are reported to occur naturally, including sound giving rise to visual percepts ('coloured-hearing') and smell giving rise to tactile sensation (Cytowic, 1993).

The standard test for the presence of synaesthesia involves assessing a subject's *consistency* in reporting sensory descriptions for words across two or more occasions, when the subject has no prior warning of the retest, and irrespective of the length of interval between testing sessions (Baron-Cohen, Harrison *et al.*, 1993; Baron-Cohen, Wyke & Binnie, 1987). Using this method, consistency is typically as high as 90%, even when retested over years and even when stringent criteria are set for retest descriptions. Recently the 'Test of Genuineness' (TOG) has been revised for precise quantitative scoring (Asher *et al.*, 2006), and psychophysical studies of synaesthesia (Ramachandran & Hubbard, 2001) also confirm that synaesthesia is highly consistent within an individual.

'Developmental synaesthesia' is distinguished from acquired or drug-induced synaesthesia and has several characteristics:

1. childhood onset before 4 years of age;
2. differentially diagnosed to hallucination, delusion or other psychotic phenomena;
3. distinct from imagery arising from imagination;
4. not induced by drug use;
5. vivid;
6. automatic/involuntary; and
7. unlearned.

Regarding the latter claim, that synaesthesia is unlearned, the key arguments against a learning account are as follows:

1. The sex ratio in synaesthesia is 6:1 (f:m);
2. consecutive letters may be closely related colours (e.g. 'M' = olive green, 'N' = emerald green, 'O' = washed out pale green). Coloured alphabet books logically go to great lengths to ensure that consecutive letters are printed in very different colours;
3. the coloured alphabets of family members often show substantial variation, ruling out imitation;
4. typically subjects lack of recollection of any learning.

Two biological theories of synaesthesia have been put forward. The *neural connectivity* theory is based on the evidence of connective pathways between auditory and visual areas of the brain in other

species (Dehay *et al.*, 1984; Kennedy *et al.*, 1996; Kennedy *et al.*, 1989). These projections are transient, typically disappearing approximately 3 months *post partum*. These transitory pathways may get 'pruned' as part of the biological maturation of the brain. Synaesthesia might be due to the persistence of neural information passing from auditory to visual brain areas, beyond the neonatal stage. At the cognitive level, the neural connectivity theory would be compatible with the *modularity breakdown* theory. This states that whereas in non-synaesthetes audition and vision are functionally discrete, in individuals with synaesthesia a breakdown in this modularity has occurred, such that there is cross-talk (Baron-Cohen, Harrison *et al.*, 1993).

Secondly, the *genetic* theory argues that synaesthesia is heritable (Galton, 1883). This has some support from a family study in which the pedigrees of seven families of probands suggested that the condition is transmitted as an autosomal dominant X chromosome linked condition (Baron-Cohen, Burt *et al.*, 1996). This is currently being tested (Asher *et al.*, submitted). Candidate mechanisms include genes that regulate the migration and maturation of neurons within the developing brain, or those that regulate 'neuronal pruning' (apoptosis). The first total genome linkage study of synaesthesia has recently been completed (Asher *et al.*, submitted).

As mentioned above, a neuroimaging study of synaesthesia using PET (Paulesu *et al.*, 1995) compared brain activity in synaesthetes and control whilst listening to either words or pure tones. Activity was seen in the synaesthetic group alone during auditory stimulation by words, in the posterior infer-temporal cortex and the parietal-occipital junction, both of which have known involvement in colour perception. An fMRI study (Nunn *et al.*, 2002) also found activity in the colour-selective regions V4/V8, as would be predicted. Both of these neuroimaging studies confirm atypical brain function in synaesthesia, though do not allow us to test the modularity breakdown theory directly. It may be that diffusion tensor imaging (DTI) would enable such a test (cf. Rouw and Scholte, 2007). But from the functional neuroimaging studies it has been argued that this condition is a good illustration of how atypical neural wiring can produce radically different conscious experience (Gray *et al.*, 2002).

The case of synaesthesia we report below is a man who came to our attention because of his savant memory: he is the European champion for memorizing the number Pi. His name is Daniel Tammet (DT). His identity can be disclosed because he has written an autobiography (Tammet, 2006) and he agreed to take part in a television documentary

investigating the nature of his savantism (*The Boy with the Incredible Brain*, May 2005, Channel 5). In 2004 he recited Pi to 22,514 decimal places from memory. His synaesthesia takes the form of numbers being experienced as colours with texture and shape. In many ways, DT is the modern-day Shereshevsky (S), otherwise known as Luria's 'Mnemonist' (Luria, 1988). Like DT, S could recall long lists of words or letters in order, could recall the list in reverse, his memory was described as 'seemingly limitless', and recall was possible decades later. Like DT, for S each letter had a shape and a colour, and he described his memory as going along 'the mental walk', where he could 'see' each item to be remembered as landmarks in a mental landscape. Today, like DT, S would be regarded as a 'savant'.

Such cases raise 4 possible theories:

1. *Savant memory is caused by synaesthesia.* It is easy to rule this theory out as there are many documented cases of savant memory in whom there is no apparent synaesthesia. (The names of winners of international memory championships — so-called mnemonists — are available on the internet). Whilst it might be objected that people who have trained their memories are not really savants, even in cases of savants (e.g. Kim Peek), it could be argued that synaesthesia may not have been formally tested and there would have been no reason to ask about synaesthesia prior to the present case study. It remains the case that even one case of a memory savant without synaesthesia would disprove the theory that savant memory is the result of synaesthesia.

2. *Synaesthesia is caused by savant memory.* Like the previous theory, this is also easy to rule out because there are many synaesthetes who have average (but not superior) memory (Baron-Cohen, Wyke & Binnie, 1987).

3. *Synaesthesia has a facilitation effect on memory:* This theory has some plausibility. Since each item to be recalled has a visual (colour, shape, texture) dimension, this might enable a superior mnemonic strategy to be used. For example when S listened to a pure 250 Hz tone with 64 decibels, he saw 'a velvet cord with fibres jutting out on all sides. The cord was tinged with a delicate pink-orange hue'. This was quite distinct from what he saw when he heard a 250 Hz tone that was 86 decibels. DT also reported vivid 3D descriptions for numbers, with many facets to them, including colour, shape, height, size and texture. One might therefore imagine that their unusually rich form of synaesthesia facilitated the development of their prodigious memory abilities.

To test this theory would require a comparison of two groups: those with synaesthesia and those without (matched for age, education and

IQ) and assessed using standardized memory tests. As far as we know, such a relatively straight-forward experiment has not yet been conducted. Note that if the null hypothesis was supported (no difference in memory between the two groups) this would disprove the theory. If the synaesthetic group was found to have memory abilities superior to controls, this would be consistent with this third theory, though it would not be water-tight proof for it, since correlation does not prove cause.¹

4. *In cases of savantism, there is also an autism spectrum condition (ASC).* In this article we can begin to test this fourth theory. This theory is at least plausible because savant skills are most often found in ASC (Hermelin, 2002). That is, it is already established that ASC increases the likelihood of savantism, so at a minimum it is important to test cases of savants for whether they have an ASC. Even well-documented cases of savants who have apparently intact social skills cannot be taken as clear counter-evidence for this theory, if they have never been formally tested for an ASC. ASC exist on a spectrum and a reliable way to measure this spectrum within high functioning individuals is to use the Autism-Spectrum Quotient (AQ) (Baron-Cohen, Wheelwright *et al.*, 2001). So a way of testing this fourth theory is to ask if savants have an elevated AQ. Whilst DT had no prior ASC diagnosis (and no prior measure of his AQ), in the study reported below, we conducted a full diagnostic assessment for Asperger Syndrome (AS) (a sub-group on the autistic spectrum), to test the hypothesis that in DT's case the AS might be present but undiagnosed. Before we describe our study, we first summarize what is meant by ASC.

Autism Spectrum Conditions

Autism is defined in terms of abnormalities in social and communication development, in the presence of marked repetitive behaviour and limited imagination (A.P.A, 1994). Asperger Syndrome (AS) is defined in terms of the individual meeting the same criteria for autism but with no history of cognitive or language delay, and not meeting the criteria for Pervasive Development Disorder (PDD) (I.C.D-10, 1994). Language delay itself is defined as not using single words by two years of age and/or phrase speech by three years of age. There is growing evidence that autism and AS are of genetic origin. The evidence is strongest for autism and comes from twin and behavioural genetic family studies (Bailey *et al.*, 1995). Family pedigrees of AS also implicate heritability (Gillberg, 1991).

[1] While this paper has been in press, a study has been published showing that synaesthesia does facilitate memory (see Yaro and Ward, 2007).

Children and adults with AS show empathizing deficits on age-appropriate tests (Baron-Cohen, Jolliffe *et al.*, 1997). This deficit in their empathizing is thought to underlie the difficulties in social and communicative development (Tager-Flusberg, 1993) and in the imagination of others' minds (Baron-Cohen, 1987). Children and adults with AS also show intact or even superior systemizing, defined as the drive to analyse systems, in order to understand and predict the behaviour of inanimate events (Baron-Cohen, 2002). Studies suggest systemizing in autism is at least in line with mental age, or superior (Baron-Cohen, Richler *et al.*, 2003). The hyper-systemizing that may be the core characteristic of ASC (Baron-Cohen, 2006) may be the key reason for the strong association between ASC and savantism. Put differently, savantism may be nothing more than the end-product of good systemizing. If one systemizes calendars, one could show the signs of 'calendrical calculation' (Hermelin & O'Connor, 1986). If one systemizes drawing, one could develop remarkable accuracy as an artist (Myers *et al.*, 2004). If one systemizes number patterns, one could develop a facility for identifying prime numbers (Baron-Cohen & Bolton, 1993). And if one systemizes syntax, one could develop a talent for acquiring languages (Hermelin, 2002).

Anatomical abnormalities have been identified in many brain areas in autism. These include the cerebellum (Courchesne *et al.*, 1994), the brain stem (Hashimoto *et al.*, 1995), frontal lobes (Carper & Courchesne, 2000), parietal lobes (Courchesne *et al.*, 1993), hippocampus (Aylward *et al.*, 1999) and the amygdala (Aylward *et al.*, 1999). In terms of neuropathology, the number of Purkinje cells in the cerebellar cortex is abnormally low (Williams *et al.*, 1980). Abnormalities in the density of packing of neurons in the hippocampus, amygdala and other parts of the limbic system have also been reported (Bauman & Kempner, 1985). Using either MRI volumetric analysis or measures of head circumference, the autistic brain appears to involve transient postnatal macrocephaly (Courchesne, 2002). The *overgrowth* may reflect a failure of synaptic pruning or an excess of synaptogenesis. We now turn to describe DT, to confirm his synaesthesia and to test him for AS.

Daniel Tammet (DT)

Biographical information

At the time of testing DT was 26 years old (born 31 January 1979). He had epilepsy at age 3 years. He did well at school in terms of academic progress, though reports having been unhappy as a child and teenager, in feeling isolated from others. He has 3 A levels, in History, French,

German (grade Bs). He has worked as a mathematics tutor. Currently he lives with his male partner whom he met through the internet, and he runs his own website providing language-learning tutorials.

Family structure

He is the oldest of nine siblings, one of whom has AS. All nine siblings have an area of strong narrow interest, ranging from music through to literature and politics. His father has a diagnosis of schizophrenia.

His savantism

DT speaks 10 languages, including Estonian and Finnish, has invented his own language (Manti) and learnt Spanish in one weekend. He performs mathematical calculations at lightning speed, including multiplying six-digit numbers together. He commented that 31, 19, 79 and 1979 are all prime numbers, an indication of how he sees patterns in numbers very rapidly. As mentioned earlier, as part of a formal competition he recited Pi to 22,514 decimal places, earning the title of European champion. He did not do this as part of a competition but to raise money for an epilepsy charity.

His synaesthesia

DT reports that he has always seen numbers as shapes, colours and textures. He also experiences some words as having colour. When calculating, the shape of two numbers combines to produce a new shape (the solution). This has been investigated at the neural level using fMRI, the results of which are reported elsewhere (Bor *et al.*, submitted).

Tests and Results

1. *The Test of Genuineness-Revised* (Asher *et al.*, 2006). On this test to validate synaesthesia, DT was over 90% consistent. This confirms his synaesthesia.

2. *The Autism Spectrum Quotient (AQ)* (Baron-Cohen, Wheelwright *et al.*, 2001). On the AQ he scored 39. People with AS score a mean of 35.8 (sd = 6.5), whilst controls score a mean of 16.3 (sd = 6.2). He is therefore in the clinical range on the AQ. The recommended clinical cut-off on the AQ for AS is 32 or more out of 50.

3. *The Empathy Quotient (EQ)* (Baron-Cohen & Wheelwright, 2004). On the EQ he scored 8 out of a maximum of 80. People with AS score a mean of 20.4 (sd = 11.6) and controls score a mean of 45.3 (sd = 10.5). This is clearly in the below average range. The recommended clinical cut-off for AS on the EQ is 30 or less. Whilst the AQ and EQ

are not diagnostic of AS, they are screens for clinical use to check for possible AS. DT's scores are below the cut-off on the EQ and above the cut-off on the AQ, indicating likely AS on both measures. In order to test for AS, a full diagnostic assessment was undertaken, described next.

4. *The Adult Asperger Assessment (AAA)* (Baron-Cohen, Wheelwright *et al.*, 2005). The AAA was used as part of a formal diagnostic assessment for AS. DT's mother served as informant for his developmental history. He scored 13 out of a maximum of 18 symptoms, and a diagnosis is made if the individual scores at least 10. His *social difficulties* were evident across his development. For example, he had no friends at school and instead counted leaves in the playground. He reported that numbers were his friends. He taught himself eye-contact at 13 years old. He tends to take things literally and is reported to commit frequent *faux pas*. He avoids social situations and finds parties confusing. He is aware that he talks too much and has taught himself to stop. He has also been told that he doesn't notice if someone is upset. Examples of his *obsessions* are that he has to have strict order in his routines and he showed severe tantrums at change of routine as a child. He constructed a library in his house, alphabeticizing the books and giving out tickets. He collected hundreds of ladybirds as a child and read books about numbers for hours as a child. He was obsessed with play-doh shapes for numbers, and with Rubik cubes. He showed head-banging in his cot. As a child, he sat with fingers in his ears in primary school and with his eyes tight shut.

The combination of his social difficulties and his obsessional interests were the basis for giving him a diagnosis of AS. He fulfilled the criteria because these symptoms had interfered with his development, causing him unhappiness whilst at school when he did not understand why he couldn't fit in. The fact that he has made an excellent adjustment in his adulthood raised the question as to whether he still needed the diagnosis of AS, even though there was no question that it would have been helpful to him as a child. This was discussed with him and on balance he decided he would like the diagnosis because it helped him understand his own development.

5. *The Systemizing Quotient (SQ)* (Baron-Cohen, Richler *et al.*, 2003). On the SQ he scored 50 out of a maximum of 80. Controls score a mean of 27.2 (sd = 7.6) and people with AS score a mean of 35.9 (sd = 15.2). He is therefore above average on the SQ.

6. *Standard memory tests*. On the visual digit span test he scored 11.5 (where controls score 6.5). On the spatial span task he scored 6.5 (where controls score 5.3). His memory for faces was tested by

showing him 82 photographs of faces expressing one of the basic emotions or neutral. He was then given a surprise face memory task one hour later, with 42 of the photos taken from the face task he had just done and 42 foil face photos that were novel, and asked to say whether he had seen each face in the previous task or not. DT had an accuracy rate of 57.1% for the faces he had seen only an hour before, which is at chance level or guessing. His performance on the foil faces was somewhat better, with an accuracy score of 69%. These facial memory accuracy scores by DT are comparable to data in a current study in our lab involving 6–8 year old children. Thus, his short-term memory appears normal, his face memory appears impaired, whilst his number memory is superior.

Discussion

In this article we report a case of a savant memory. He had self-reported synaesthesia which we validated using the Test of Genuineness-Revised (Asher *et al.*, 2006). Because of the well-established association between ASC and savantism, we predicted he might also have one form of ASC, namely Asperger Syndrome (AS). This was confirmed. Whilst the existence of his AS may be sufficient to explain his savantism, we speculate that his unusual combination of conditions (synaesthesia *and* AS) may have increased the likelihood of his savant memory. This idea requires testing in a group study.

Future work needs to address the following questions. First, what percentage of people with an ASC also have synaesthesia? Currently this is unknown, perhaps because in classic autism it would be difficult to distinguish. Synaesthesia depends on verbal self-report, and in classic autism the language skills and self-reflection may not be sufficient. But in people with AS, this question should be answerable. Certainly, there are autobiographical accounts of people with AS or High Functioning Autism who have strong visual processing or sensory hyper-sensitivity (Grandin, 1996). We are currently conducting such a survey in our lab.

Second, what percentage of people with synaesthesia have an ASC? This has never been investigated as far as we are aware. If these two conditions are significantly associated, is this for genetic reasons? We mentioned earlier that neurological theories of both autism and synaesthesia refer to an excess of neural connectivity, perhaps due to a failure of pruning or apoptosis. It is interesting to speculate as to whether these two apparently very different conditions may share a common neural abnormality.

Third, does having both an ASC (which involves strong systemizing (Baron-Cohen, 2006) and synaesthesia (which arguably allows for mnemonic enhancement) increase the likelihood of savantism over and above the rate that would be expected if the person only had an ASC? To test this would require comparison of separate populations (no diagnosis, vs. ASC alone, vs. ASC with synaesthesia) in terms of their prevalence rates of savantism.

In this article we are studying DT as a savant with autism and synaesthesia. We regard the first of these (savantism) as a possible effect of the other two (autism and synaesthesia). Since savantism is the main focus of the study, we have not spent much time discussing the nature of autism. However, given the focus of this Special Issue is on consciousness, it is worth mentioning that autism has attracted considerable attention from philosophers of mind because of the idea that — hardwired into the typical brain — there is an innate module for ‘mind-reading’ which for genetic reasons is impaired in autism. Such ‘mindblindness’ would be expected to have major implications for consciousness. It would mean that whilst the person could think with ease about objects in the world, or about facts and patterns in the world, their idea of what another person might be thinking, and especially of what another person might be thinking about them, might be quite limited. A person with mindblindness might spend hours thinking about a favourite topic, becoming lost in the details and going deeply into it, all the while remaining relatively oblivious of how they appear to others or what others think of their behaviour.

Such a state was certainly true of DT when he was a child in school, since it appears he had little insight into how odd his behaviour seemed to the other children in the class. When he sat on the carpet during story-time, with his eyes tight shut and his fingers in his ears, picturing numbers in his mind and their shapes and colours, whilst the other children looked at each other or at the teacher and listened to the story, DT was in some sense in a world of his own. With age, DT has developed more of an idea of how to behave and how he seems to others, raising the possibility that mindreading skills are not completely absent but are simply delayed. It helped when, at the age of 13, his mother was able to give him some feedback and tell him to look at others’ eyes and not at his own feet. This suggests that in individuals on the autistic spectrum, for whom such social insight and consciousness of others’ minds does not develop naturally at the right point in development, learning to consciously attend to key parts of the environment (faces, eyes, expressions) may help. A recent study evaluating whether people on the autistic spectrum could learn to recognize facial

expressions with conscious effort suggests that this is possible (Golan *et al.*, 2006). Quite what the relationship is between such atypical development of mindreading and the presence of savantism remains to be established.

In conclusion, following the validation of both synaesthesia and ASC in one case of savantism, we recommend that in future savants should be tested for both of these possible co-occurring conditions. It is of course possible that savant memory could be the result of the application of mnemonic training strategies, such as the ‘loci’ method, and without any effects of factors such as ASC or synaesthesia. Whether it is possible to distinguish such ‘acquired’ savantism from the kind shown by cases such as DT or by others with ASC will be important to establish. Finally, concerning the relevance of atypical minds for theories of consciousness, we contend that there is much to learn from the study of such rare cases. They illustrate the general principle that other minds might think or feel differently, if they are wired differently.

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