

The Role Played by Eye Contact in Yawn Contagion

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Abstract

Contagious yawning occurs when an individual yawns as a result of viewing, listening to, reading or thinking about another individual yawning. There are several mechanisms proposed to underlie yawn contagion. This study will focus on the hypothesis that yawn contagion is a response to social cues, especially eye contact. Individuals with autism spectrum disorder (ASD) have shown a reduced susceptibility towards yawn contagion. However, when told to focus on the eye area, children with ASD catch yawns as frequently as typically developing (TD) children. This suggests that a lack of eye contact and the associated abnormalities of the social brain, specifically the amygdala and superior temporal sulcus, play a role in the lack of yawn contagion previously associated with autism spectrum disorder. Similarly, a lack of yawn contagion has previously been found among TD children aged 6 years and younger, however, in the current study these children showed comparable levels of yawn contagion to children above the age of 6 years, when eye contact was controlled for. This implies that increasing focus on the eyes may overcome the poor attention skills characteristic of children aged 6 years and younger.

Keywords: yawn contagion; autism spectrum disorder; social cues; eye contact

Background

Yawning is a common, spontaneous occurrence observed in humans as well as other animal species (Baenninger, 1987). Alternatively, yawning can be a contagious event (Provine, 1986). There are several mechanisms proposed to underlie yawn contagion. Most recently contagious yawning has been interpreted as having communicative functions. For instance, yawns have been viewed as social cues that coordinate group behaviour (Daquin, Micallef, & Blin, 2001). Individuals with ASD, who display social deficits, have shown a reduced susceptibility towards yawn contagion (Senju et al., 2007). This has been attributed to abnormalities of the so called ‘social brain’, particularly the amygdala and superior temporal sulcus (STS) (Schürmann et al., 2005). In addition, the inability of individuals with ASD to catch yawns has been attributed to their aversion to eye contact (Klin, Jones, Schultz, Volkmar, & Cohen, 2002), which serves as an important tool for social interaction and communication (Senju, Yaguchi, Tojo, & Hasegawa, 2003; Senju, Kikuchi, Hasegawa, Tojo, & Osanai, 2008). Thus, certain mechanisms related to the identification and processing of social cues may underlie yawn contagion.

Yawn Contagion

Introduction to contagious and spontaneous yawning. Contagious yawning occurs when an individual yawns as a result of viewing, listening to, reading or thinking about another individual yawning (Rizzolatti & Craighero, 2004; Provine, 1986). Although yawn contagion is a well-documented occurrence (Anderson & Meno, 2003; Senju et al., 2007; Platek et al., 2003; Provine, 1986), knowledge regarding the mechanisms, functioning and development of yawn contagion remains limited (Senju et al., 2007). While spontaneous yawning is a phenomenon experienced by all vertebrates and mammals, contagious yawning has only been observed in humans (Baenninger, 1987), certain primates, such as chimpanzees (Anderson, Myowa-Yamakoshi, & Malsuzawa, 2004) and macaques (Paukner & Anderson, 2006), and perhaps dogs (Joly-Mascheroni, Senju, & Shephard, 2008).

Spontaneous and contagious yawning in children. Very little research has been conducted regarding yawning in infancy and childhood (Anderson & Meno, 2003). Spontaneous yawning occurs in the foetus from the second to third trimester of pregnancy (de Vries, Visser, & Prechtel, 1982). Contagious yawning is only observed much later, although there is disagreement as to when children start to yawn contagiously. One study reported that yawn contagion begins in the second year of life (Piaget, 1951). Another study reported that yawn contagion can only be observed reliably in children over the age of 6 years (Anderson & Meno, 2003). The absence of contagious yawning in infants is supported by studies done on chimpanzees as the infant chimpanzees were not shown to catch yawns (Anderson et al., 2004). However, there were a limited number of infant chimpanzees present in the study which limits the generability of these observations. The lack of yawn contagion among children below the age of 6 years contrasts with the high frequency of spontaneous yawning found in these children. Different times of onset of spontaneous and contagious yawning suggest that different mechanisms underlie these two phenomena (Giganti & Esposito Ziell, 2009). A greater susceptibility to yawn contagion among older children may be linked to aspects such as heightened self- and social awareness. However, there is some inconsistency in the argument that reduced self- and social- awareness is the reason for the lack of yawn contagion among children younger than 6 years as it is unlikely that primates and dogs, which are capable of yawn contagion (Anderson et al., 2004; Paukner & Anderson, 2006; Joly-Mascheroni, Senju, & Shephard, 2008), are more self-aware than young children. It is more plausible that the lack of yawn contagion previously found may be due to inattention, specifically to the eye region. Further research is required to determine when children start to yawn contagiously and what mechanisms underlie this occurrence. Specifically, no research has been done on yawn contagion in young children where attention to the eyes has been regulated. This study will focus on the hypothesis that contagious yawning happens as a response to social cues, especially those conveyed by eye contact.

Eye contact and the social brain. Eye contact forms an important element of social communication in humans. According to Senju and Johnson (2009, p. 127) “the eye contact effect is defined as the phenomenon that perceived eye contact modulates the concurrent and/or immediately following cognitive processing and/or behavioural response”. It has been suggested that eye contact modulates the development of the social brain network. The social brain is a network of brain regions that are proposed to underlie human social interaction. The

social brain is responsible for the interpretation of social cues, such as yawning, and comprises of a network of regions including ventral and medial prefrontal cortex, superior temporal gyrus, fusiform gyrus (FG), cingulate gyrus and amygdala. A neuroimaging study has shown that viewing others yawn and susceptibility to yawn contagion are associated with activation of the superior temporal sulcus (STS) and periamygdalar areas (Schürmann et al., 2009). The STS and amygdala are key structures in the social brain. Therefore, it has been suggested that the mechanisms underlying yawn contagion are related to the mechanisms underlying other aspects of social cognition.

There is still much debate about the mechanisms and developmental processes concerned with eye contact and the social brain. Eye contact stimulates the activation of parts of the social brain, but this interaction is influenced by task requirements and social context to determine which regions of the social brain network are activated (Senju & Johnson, 2009). Abnormalities in structures that are associated with the eye contact effect, including the amygdala and its connections with other structures, could be responsible for the lack of eye contact effect found in individuals with ASD.

Amygdala. Social perceptual limitations in facial expression perception may be the result of abnormality towards the beginning of the amygdala's development. The amygdala is often identified as playing a key role in theories of social perception and cognition (Adolphs, 2008). The amygdala responds to emotionally charged stimuli, notifying other areas of the brain of the salience of the situation. It is important in the early period of facial expression processing and has been linked to facial emotional processing (Adolphs, 2008; Schultz, 2005). Activation of the periamygdala has been linked to the emotional weight of social cues, especially those conveyed by human faces (including yawning) (Critchley, Daly, Phillips, Bullmore, Williams, Van Amelsvoort, Robertson, David, & Murphey, 2000). Individuals' self-reported propensity to yawn negatively covaries with activation of the left periamygdalar region (perceived contagiousness increases as amygdala activation decreases). This implies that there is a link between yawn contagion and de-activation of the amygdala. Schürmann et al. (2005) have suggested that this negative covariance alludes to a relationship between the susceptibility to contagious yawning and the face-processing-related emotional interpretation during social interaction. The amygdala is important for processing certain facial features, particularly the eye region. In cases of bilateral damage to the amygdala, there is an inability to focus on the eye region of faces spontaneously and an increased tendency to fixate on the

mouth. However, the amygdala is not responsible for sophisticated calculations of facial expressions. These are attributed to cortical areas including the STS.

Superior temporal sulcus. Activation of the STS occurs when individuals observe others yawn (Schürmann, Hesse, Stephan, Saarela, Zilles, Hari, & Fink, 2005). Schürmann et al. (2005) found a significant increase in the blood oxygen level dependant (BOLD) signal in the right posterior STS and bilaterally in the anterior STS. This signal is unique to observing yawning opposed to observing other mouth movements. This supports the great affinity of the STS to social cues. The STS is an important component during the normal activation sequence observed while viewing and imitating facial gestures, such as yawning. In contrast, activation was not found in Broca's area, a central aspect of the mirror neuron system (MNS) that links action observation with execution, as a result of exposure to yawn stimuli. This suggests that yawn contagion is the result of automatically released behavioural acts instead of truly imitated motor patterns.

Yawn Contagion and Autism

Autism. The diagnostic criteria for autistic disorder, as specified in the text revision of the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (APA, 2000), are as follows: Individuals must show impairments or atypical functioning in each of the following domains; social interaction, communication and restricted/repetitive play (See Appendix A). The onset of symptoms must occur before the age of 3 years. The DSM-IV provides a broader category, namely autism spectrum disorder (ASD) which includes autism, Asperger syndrome and Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS) and other developmental disorders such as Rett's syndrome (APA, 2000). These disorders are characterized by the same core deficits as autism, but individuals do not meet all the criteria necessary for a diagnosis of autistic disorder.

Yawn contagion as it relates to ASD. Children with ASD display normal spontaneous yawning but reduced contagious yawning (Senju et al., 2007; Giganti & Esposito Ziell, 2009). There is no difference between higher and lower functioning children on the autism spectrum in terms of frequency of yawn contagion (Giganti & Esposito Ziell, 2009). The reason for this reduced yawn contagion among individuals with autism remains unclear (Senju et al., 2009).

A possible reason for reduced yawn contagion is abnormal face fixation in ASD. As discussed above, eye contact is a particularly important aspect of social interaction and communication (Senju et al., 2003; Senju et al., 2008). Individuals with ASD, who display significant social and communicative shortcomings, display deviant patterns of eye contact behaviour (Senju et al., 2003). Autism is characterised by a reduced tendency to make eye contact (Hermans et al., 2009) as individuals with ASD are more inclined to focus on the mouth (Klin et al., 2002). The idea that yawn contagion is influenced by eye contact has been supported by a recent study which found that when told to focus on the eye area, children with ASD catch yawns as frequently as TD children when exposed to yawn stimuli (Senju et al., 2009). This contrasts with the studies conducted by Senju et al. (2007) and Esposito Ziehl (2009) which found that children with ASD catch yawns less frequently than TD children. These studies followed the same protocol as that of Senju et al. (2009) except that children were not encouraged to focus on the eyes of the faces presented. Atypical facial orienting and processing, abnormalities of the social brain and a lack of eye contact as well as the interplay among these components has an important influence on yawn contagion among individuals with ASD and will be discussed in greater detail below.

Atypical facial orientation and processing. Reciprocal social interactions and interpersonal communication rely on the capacity of individuals to interpret facial expressions and gain other socially pertinent information from faces (Hadjikhani, Joseph, Snyder, Chabris, Clark...Steele, 2002; Pelphrey Sasson, Reznick, Paul, Goldman, & Piven, 2002). Thus, it is important to determine whether the atypical perceptions of faces, and the associated social cues, may be a factor in the social deficits associated with ASD (Hadjikhani et al., 2002).

A lack of use of nonverbal communicative behaviours is a characteristic of the autism phenotype (APA, 1994). This characteristic follows a particular developmental course. Early indications can be found very soon in ontology, such as an atypical focus on inanimate objects and a disinterest in human faces (Kanner, 1943). This disinterest in human faces is present in the infant's first 6 months and is one of the strongest predictors of an ASD diagnosis later on in life (Maestro, Muratin, Cavallaro, Pei, Stern, Golse, Palacio-Espasa, 2002). Retrospective studies of home movies have shown that individuals who have been diagnosed with ASD later on in life displayed significantly less interest in individuals' faces as infants. The face-directed behaviours of infants go on to form a developmental pathway through which a limited set of basic abilities are transformed into the complex face processing system present in TD individuals (Pelphrey et al., 2002)

Various studies suggest that ASD individuals process faces differently than TD individuals (Pelphrey et al., 2002). These deficits may contribute to impairments in face perception and the recognition of facial affect among individuals with ASD. TD individuals tend to focus on the core facial features: namely, the eyes, nose and mouth, while paying less attention to the non-feature parts of the face. Individuals with ASD, in contrast, pay more attention to non-feature facial regions than core features. Studies have stressed that TD individuals, who do not suffer from social deficits, focus on the eye region to gain information about the mental states of others. In contrast individuals with ASD show a reduced ability to interpret emotional states from the eyes (Baron-Cohen et al., 1999).

While TD children apply holistic facial processing techniques, children with ASD do not (Schultz, 2005). In order to detect a certain face or facial expression, TD individuals tend to depend on the spatial orientation of the face's central features, namely the eyes, nose and mouth (Schürmann et al., 2005). TD individuals employ a different processing technique when viewing nonface objects (Pelphrey et al., 2002). Instead of the holistic approach described above, TD individuals use a fragmented approach when processing nonface objects, focusing on the detection of separate features rather than the overall composition of the object. The different techniques employed for face and nonface processing in TD individuals are not present in individuals with ASD. Individuals with ASD tend to depend on the separate aspects of the face for identification (for instance, the mouth and lower region of the face), instead of its overall composition.

The fusiform face area has been identified as playing a key role in the recognition of faces (Kanwisher, McDermott, & Chun, 1997). It consists of a part of the cortex, in the right fusiform gyri. This area has been found to show greater activation during the observation of faces than any other form of visual stimuli among TD individuals. In a study conducted by Schultz et al. (2005) investigating face and nonface perception using functional magnetic resonance imaging (fMRI), it was found that individuals with ASD showed significantly higher activation of the right inferior temporal gyri and lower activation in the right fusiform gyrus than TD individuals when viewing face or nonface objects. Conversely, TD individuals display the normal pattern of higher fusiform gyrus activation during face processing and higher right inferior temporal gyri activation during the observation of nonface objects. This indicates that individuals with ASD may interpret faces as though they were nonface objects, using the fragmented processing techniques that are similar to those used by TD individuals while viewing nonface objects.

Autism and abnormalities of the social brain. Autism researchers and individuals interested in the social brain have paid much attention to the study of the amygdala (Schultz, 2005). Problems with the amygdala of ASD individuals may result in the inability to orient to social cues such as yawning. fMRI studies of the amygdala and fusiform face area (FFA) of individuals with ASD have found a hypoactive response to face perceptual tasks (Baron-Cohen et al., 1999). These findings have been linked to inadequate emotional perception characteristic of individuals with ASD.

It has recently been recognised that individuals with ASD do not display a typical use of information from the eye region as they tend to focus on the mouth when viewing a face. The length of eye contact has been correlated with modulation of the amygdala (Adolphs, 2008; Magnée, Gelder, van Engeland, & Kemmer, 2007). This suggests that eye contact is associated with an increased emotional response in individuals (Magnée et al., 2007). Thus the lack of eye contact displayed by individual with ASD, resulting in a lack of regulation of amygdala activation could play an important role in the lack of yawn contagion among individuals with ASD. A lack of attention to the eye region can be reduced or eliminated by instructing individuals with ASD to focus on the eyes of the face (Hadjikhani et al., 2002).

Rationale for the Study

Yawn contagion among ASD children. A reduced tendency to make eye contact among individuals with ASD could be the reason for lower levels of yawn contagion among these individuals compared to TD individuals (Klin et al., 2002). As observation of the eye area is an important trigger for yawn contagion (Provine, 1989), it is possible that the lack of eye contact made by individuals with ASD inhibits them from yawning contagiously. Instructed focus on the eyes of a yawning individual brings about contagious yawning in individuals with ASD, and supports this explanation of yawn contagion (Senju et al., 2009). However, the notion that yawn contagion is dependent on eye contact alone is contradicted by the finding that children with ASD, unlike TD children, also show a lack of yawn contagion when listening to other individuals yawning (Giganti & Esposito Ziell, 2009). This may suggest that that a general inability to interpret social cues, not eye contact exclusively, is responsible for reduced yawn contagion among children with ASD. The recent finding on the importance of eye contact in contagious yawning could have significant implications (Senju et al., 2009). This finding could support the idea that yawn contagion is linked to the social capacities of individuals as eye contact is an important social cue. Further research into

the phenomenon of yawn contagion, when eye contact is controlled for, could provide new insight into yawn contagion.

Yawn contagion among TD children above and below 6 years. The reason for the lack of YC in children below the age of 6 years requires further investigation. The lack of contagious yawning among children below the age of 6 years contrasts with the high frequency of spontaneous yawning found in children of the same age group. There is a shortage of research into the social influences, including eye contact, on contagious yawning in infancy and childhood (Anderson & Meno, 2003). Inattention to the eye region has not been explored as a reason for the reduced yawn contagion in this age group. As children aged 6 years and younger have poorer attention skills than children older than 6 year, the lack of yawn contagion found within this group may be due to a lack of attention to the eye regions. Therefore, prompting young children to look at the eyes during a yawn task may induce yawn contagion, as seen in ASD.

Specific Aims/Hypotheses

Yawn Contagion among ASD Children

The primary aim of this study was to replicate the study conducted by Senju et al. (2009). Thus the study aimed to verify the finding of Senju et al., (2009) that children with ASD show comparable levels of yawn contagion to TD children when eye contact is controlled for. This study will therefore attempt to confirm the finding that eye contact is a factor in the lack of yawn contagion among ASD children.

H1: When the lack of eye contact associated with individuals with ASD is controlled for, they are likely show similar levels of yawn contagion to TD children.

TD Children Above and Below 6 Years

In addition, the study aimed at determining whether yawn contagion is possible in TD children aged 6 years and younger when eye contact is controlled for. As eye contact has been found to induce yawn contagion among children with ASD (Senju et al., 2009), it would be interesting to see if the same happens in children 6 years old and younger. Previous studies conducted on children below the age of 6 years have not controlled for eye contact (Anderson & Meno, 2003), which is an important aspect of social communication. Therefore this study

attempted to determine whether eye contact is a factor in the lack of yawn contagion among TD children 6 years and younger.

H2: When eye contact is controlled for among TD children below the age of 6 years, they are likely show similar levels of yawn contagion to TD children above the age of 6.

Methods

Research Design

The study made use of two separate cross-sectional comparisons. Firstly, an ASD group was compared to a TD control group. The study used a quasi-experimental design as the participants were grouped based on the pre-established criterion of an ASD diagnosis. Secondly, a TD group 6 years and younger was compared to a TD group above the age of 6 years old. The groups were compared on scores obtained from a yawn contagion task. This study implemented protocols based on the designs of prior research in this area (Senju et al., 2009).

Participants

The section of the study concerning children with ASD included 20 children with ASD who were matched with TD control children. The second section of the study, concerning TD children of different age groups, consisted of 28 TD children 6 years and younger and matched children above the age of 6 years old. Participants were recruited from schools for TD children as well as schools for children with ASD in the Western Cape. The schools for children with ASD have an admission requirement of a diagnosis of autism made by a qualified clinician, with no association to this study, according the specifications of the DSM-IV-TR (APA, 2000). In addition, participants were sourced from a preexisting research participation pool recruited via support groups, the internet or personal referral. These participants were required to have been diagnosed with ASD, according to the specifications of the DSM-IV-TR (American Psychiatric Association, 2000), by a qualified clinician independent from the study in order to be included in the study.

The TD children were paired with the children with ASD according to chronological age, gender and socio-economic status (SES). As the studies conducted by Senju et al. (2007) and Senju et al. (2009) have not shown any correlation between IQ and yawn contagion, participants were not matched on IQ scores. TD children in the two age groups were matched on gender and SES. The relevant demographic characteristics of the participants are displayed in Table 1 and Table 2.

Table 1.

Demographic Characteristics of the Autism Spectrum Disorder (ASD) and Typically Developing (TD) Control Groups

Demographic Information	ASD (<i>n</i> = 20)	TD (<i>n</i> = 20)
Age Range (Years: Months)	7:3-16:6	7:2-15:6
Age (Years)		
<i>Mean (SD)</i>	11.25 (2.67)	11.43 (2.51)
Gender		
<i>Male: Female</i>	4:1	4:1
Socio-economic Status		
<i>High: Medium: Low</i>	11:8:0*	11:7:1
	*1 unknown	

Table 2.

Demographic Characteristics of the Typically Developing (TD) Age Groups

Demographic Information	≤6 years (<i>n</i> = 28)	>6 years (<i>n</i> = 28)
Age Range (Years: Months)	3:0-6:11	7:7-16:3
Age (Years)		
<i>Mean (SD)</i>	4.96 (1.05)	12.18 (2.46)
Gender		
<i>Male: Female</i>	1:1	1:1
Socio-economic Status		
<i>High: Medium: Low</i>	22:5:1	23:5:0

Inclusion and exclusion criteria. A history of head injury, infantile meningitis, or any neurological condition impacting the central nervous system lead to TD participants and participants with ASD being excluded from the study. Participants with ASD were excluded if they suffered from additional neurological conditions, while TD participants with any form of neurological condition were excluded. Moreover, TD participants with a diagnosis or

history of social disorders, including conduct disorder, oppositional defiant disorder, attention disorders (for instance, Attention Deficit/Hyperactivity Disorder), a pervasive developmental disorder or affective disorders were excluded from the study.

Ethical considerations. Parents or legal guardians were asked for consent before the study commenced (Appendix B & Appendix C). Informed assent was obtained from participants before testing took place (Appendix D). In addition, the parents or guardians were asked to complete a demographic questionnaire (Appendix E). This questionnaire concerned information— such as age, gender, home language and SES— required for the selection of control group participants.

The Ethics Committee of the University of Cape Town's Department of Psychology and the University of Cape Town's Faculty of Health Sciences Research Ethics Committee awarded ethical approval to the larger research project that this study fell under. The Western Cape Education Department granted permission for participants to be sourced from public schools in the province (Appendix F).

Measures

Yawn contagion. The protocol implemented in this task was derived from that used by Senju et al. (2009) which tested yawn contagion as follows: Yawn contagion was tested using a video composed of footage of young adults either yawning or opening their mouths. Six individuals, three males and three females, unknown to the participants, were used as models. The video displayed 6 clips of yawning models and 6 clips of models opening their mouths (which served as a control condition). Control clips were shown to control for imitation behaviour. Each clip lasted for 7 -seconds. These clips were presented in a pseudorandom order. Before each clip, a 1cm x 1cm fixation stimulus (white crosses) appeared for 1 second on the position where the model's eyes were situated. Before the video started, participants were instructed to focus on the fixation points when they appeared on the screen. Each successive clip was separated by a 30-second silent cartoon. The purpose of the cartoon was to maintain the child's concentration on the video. The 30-second time period in which the cartoon was shown allowed the child time to respond to the previous clip. If the child lost concentration during the video, he/she was reminded to pay attention to the screen.

The video was screened on a LCD laptop monitor. Participant responses were recorded during the screening using a built-in web camera. These recordings were scored at a later stage by 3 independent raters to determine the number of yawns caught and imitations

displayed. In order to increase the accuracy of results, yawn and imitation responses were only coded when two or more of the raters noted the response.

In previous studies a yawn was understood as “the stereotyped motor pattern of deep inspiration, a brief pause accompanying eye occlusion, and following expiration with open mouth” (Province 1986, as cited in Senju et al., 2009, p. 1600). However, when this description was used on a subsection of the participants ($n = 15$) we found poor agreement among rater results for yawn responses with a Cronbach’s alpha level of 0.53. Consequently, new criteria were established for the purpose of this study. A yawn was identified as a response in which three of the following criteria were presented: stereotyped mouth movements (including opening the mouth, curling the lips or a noticeable distortion of the mouth), eye movement (including closing the eyes, rapid blinking and opening the eyes wide for a brief period preceding the yawn), flaring of the nostrils, chest expansion, raising of the shoulders, and covering the mouth with a hand. Imitations were defined as the opening of the mouth, with or without closing the eyes, with no visible signs of deep inhalation. Using these criteria, inter-rater reliability was high with a Cronbach’s alpha level of 0.86 for yawn scores and 0.91 for imitation scores.

Procedure

The study fell under a larger research project studying individuals with ASD. Therefore, IQ, executive functions, spatial navigation and Theory of Mind tests were conducted in addition to the yawn contagion task. This study focused exclusively on the measures of yawn contagion. In order to prevent fatigue effects, tests were not conducted in a pre-determined order. Participants were allowed to take a short break if required.

Data Analysis

Statistical analyses were conducted using PASW Statistics 18. Demographic data was analysed using descriptive statistics. Direct comparisons of yawn and imitation responses across different groups were analysed by mixed design ANOVA analyses. The difference in yawn and imitation responses elicited by yawn and control clips was examined using non-parametric Wilcoxon's signed-rank tests. This non-parametric test was used as Levene's test for homogeneity of variance was significant for the data on frequency of yawns caught and imitations displayed across the different clips.

Results

Comparison across ASD and TD Control Groups

Contrast of yawn and imitation responses across groups. A mixed design 2 (group) x 2 (clip) ANOVA was run in order to contrast yawn and imitation responses¹. A significant interaction was found ($F_{1, 38} = 5.554$; $p = .024$; partial $\eta^2 = 0.13$), using Greenhouse-Geisser values.

The interaction was then further examined using simple effects analysis. This indicated that there was a statistically significant difference between yawn and imitation responses in the ASD group, $p = .008$; $M (SD)$ 0.6 (0.94) vs. 1.4 (2.44), who displayed less yawns than imitations. In contrast, there was no statistically significant difference between yawn and imitation responses among the TD group ($p = .602$).

A simple effects analysis also showed that there was no statistically significant group difference in yawn responses. ASD and TD children caught yawns at similar rates ($p = .446$). A difference bordering on statistical significance was found in imitation responses for the two groups with children with ASD displaying more imitation responses than TD children, $p = .05$; $M (SD)$ 1.4 (2.44) vs. 0.25 (0.72).

The different responses to yawn and control clips were then examined (refer to Table 3). A non-parametric Wilcoxon's signed-rank test² was run to compare the yawn responses across the various clips. No difference in the number of yawns elicited by yawn and control clips was found in the ASD group ($z = -0.707$, $p = .363$). However, a statistically significant difference was found in the TD group ($z = -2.121$, $p = .031$) with more yawns being elicited by yawn clips.

¹The assumption of homogeneity of variance was not upheld for imitation responses ($p = .001$). However, this was not considered to be problematic as cell sizes were equal and the assumption of normality was upheld.

² Exact significance levels for 1-tailed tests were used as children were expected to yawn more after yawn clips than after control clips.

When the same test was run to compare imitation responses across the different clips, no difference was found in the number of imitations elicited by yawn and control clips in the ASD group ($z = -0.531, p = .625$) or the TD group ($z = -0.577, p = 1.00$).

Table 3.

Comparison of Various Responses across ASD and TD Groups

Total Number of Given Response within Each Group	ASD ($n = 20$)	TD ($n = 20$)
Yawns after yawn clip	7	7
Yawns after control clip	5	1
Total yawns (i.e. yawns caught)	12	8
Imitations after yawn clip	13	3
Imitations after control clip	15	2
Total imitations	28	5

Comparison across Age Groups

Contrast of yawn and imitation responses across age groups. A mixed design 2 (group) x 2 (clip) ANOVA was run in order to contrast yawn and imitation responses³. A significant interaction was found ($F_{1, 54} = 5.74; p = .02$; partial $\eta^2 = 0.096$), using Greenhouse-Geisser values. The interaction was then further examined using simple effects analysis. This indicated that there was a statistically significant difference between yawn and imitation responses among the 6-years-and-younger age group, $p = .011$; $M (SD)$ 0.75 (1.53) vs. 1.25(1.69) with more imitations being coded. In contrast, there was no statistically significant difference between yawn and imitation responses among the older than 6 years age group ($p = .455$).

Importantly, there was no statistically significant group difference in yawn responses across the 2 age groups ($p = .22$). However, a statistically significant difference was found in imitation responses with the younger group imitating more than the older age group, $p = .004$; $M (SD)$ 1.25 (1.69) vs. 0.21 (0.69).

³ The assumption of homogeneity of variance for yawn ($p = .038$) and imitation ($p < .0001$) responses was not upheld. However, this was not considered to be problematic as cell sizes were equal and the assumption of homogeneity was upheld.

The various responses to the different types of clips were then investigated using Wilcoxon's signed-rank tests⁴ (refer to Table 4). Yawn clips elicited more yawns in children above 6 years than the control clips did ($z = -2.271, p = .016$); in contrast there was no difference in the number of yawns elicited by yawn or control clips in children 6 years and younger ($z = -0.302, p = .5$).

A Wilcoxon's signed-rank tests indicated that there was no statistically significant difference in the number of imitations elicited by yawn versus control clips among children below 6 years ($z = -1.941, p = .092$). Moreover, no statistically significant difference was found in the number of imitations elicited by yawn versus control clips among children above the age of 6 years ($z = 0.00, p = 1.00$).

Table 4.

Comparison of Various Responses across Age Groups

Total Number of Given Response within Each Group	<6 years ($n = 28$)	> 6 years ($n = 28$)
Yawns after yawn clip	12	9
Yawns after control clip	9	1
Total yawns (i.e. yawns caught)	21	10
Imitations after yawn clip	14	3
Imitations after control clip	21	3
Total imitations	35	6

⁴ Exact significance levels for 1-tailed tests were used as children were expected to yawn more after yawn clips than after control clips.

Discussion

Responses across ASD and TD Groups.

Yawn responses. The findings of this study are in line with those of Senju et al. (2009). The hypothesis that children with ASD would show comparable levels of yawn contagion to TD children, when eye contact is controlled for, was confirmed. These results suggest that a lack of eye contact plays a role in the lack of yawn contagion previously associated with ASD. The importance of eye contact can be supported by a previous study by Provine (1989) which found that more yawns were elicited by videos of yawning faces when the mouth was blocked out and only the eyes were visible than clips when the eyes were blocked out and the mouth was visible. This supports the assumption of this study that it is the focus on the eyes, encouraged by the fixation stimuli, that enabled yawns to be elicited among ASD children.

The results of the present study, and that of Senju et al. (2009), compliment the explanation that irregular functioning of the ‘social brain’ network associated with ASD can be attributed, in part, to atypical orienting to social stimuli, including the eyes, which may be the result of atypical functioning of the amygdala (Grelotti et al., 2002; Hadjikhani et al., 2004; Schultz 2005; Senju & Johnson, 2009). The amygdala serves as an ‘eye contact detector’ which in turn directs social orienting and regulates neurological reactions to social stimuli (Johnson, 2005; Senju & Johnson, 2009). The results of this study, and that of Senju et al. (2009), allude to the idea that the same structures may play a role in yawn contagion by assisting spontaneous orienting towards the eyes (Schürmann et al., 2005).

The findings of this study are also compatible with the theory that the ‘social brain’ is modulated by the ‘eye contact effect’. The findings are interesting in light of the idea of the ‘eye contact effect’ which suggests that eye contact modulates the activation of the social brain network (Senju & Johnson, 2009). Deficits in structures that are associated with the ‘eye contact effect’, including the amygdala and its connections with other structures, have been proposed to underlie the lack of eye contact found in individuals with ASD. Problems with the amygdala of ASD individuals may result in the inability to orient to social cues such as yawning. This study controlled for the lack of eye contact typically found among individuals with ASD by drawing attention to the eyes of yawning faces. Interestingly, comparable levels of yawn contagion were found among ASD and TD children, which was not the case in similar studies conducted by Senju et al. (2007) and Pileggi (2009) which did not encourage focus on the eyes. It therefore seems likely that eye contact modulates the activation of the ‘social brain’ network, as proposed by the ‘eye contact effect’ (Senju &

Johnson, 2009), probably as a result of the de-activation of the amygdala which has been correlated with susceptibility to yawn contagion (Schürmann et al., 2005). Thus the lack of eye contact typically displayed by individuals with ASD appears to play an important role in the lack of yawn contagion among individuals with ASD.

The results support the idea that atypical social cognition and social behaviour displayed by individuals with ASD may be interconnected to atypical orientating to social stimuli, which may be the result of atypical development and functioning of structures such as the amygdala (Adolphs, 2008; Grelotti et al., 2002; Schultz 2005; Senju and Johnson, 2009). The finding that children with ASD show comparable levels of yawn contagion to TD children when eye contact is controlled for supports the idea that yawn contagion is linked to the social capacities of individuals, as eye contact is an important social cue. Furthermore, the results are consistent with the idea that atypical perceptions of faces, and the consequent lack of eye contact, inhibit ASD individuals' abilities to interpret social cues and may be a factor in the social deficits associated with ASD (Hadjikhani et al., 2002) as they are able to pick up on the social cue of yawning when eye contact is controlled for.

Imitation responses. Both TD and ASD children imitated yawns, a response that was not reported in the previous study by Senju et al. (2009). Although no statistical difference was found between the two groups, this result bordered on significance. Thus, there is a possibility that this finding may be due to limited power given the small sample size. The presence of imitations among the ASD group is particularly interesting as individuals with ASD have been found to suffer from deficits in imitation (Oberman & Ramachandran, 2007). These deficits in are significant as facial imitation is fundamental for the development of affective, social and communicative skills (Rogers & Pennington, 1991). A possible explanation for the imitative responses displayed in this study may be that greater attention to the facial region has been found to induce empathetic responses, including spontaneous facial expression mimicry among individuals with ASD (Magnée et al., 2007).

The occurrence of facial mimicry among ASD children is important as it has certain implications for empathy and bonding. Facial imitation has been linked to social and empathetic development (Stel & Vonk, 2010). It has been proposed that the facial feedback processes associated with mimicry are related to empathy. When an individual assumes the same facial expression as another, the involved facial muscles send signals to the brain to evoke the related emotions (Hess, Kappas, McHugo, Lanzetta, & Kleck, 1992). A recent study (Stel, van Baaren, & Vonk, 2008) demonstrated this 'emotional contagion' by showing

that individual who imitated the expressions of faces shown in a video experienced a stronger sense of empathy towards the individual in the video, than when they did not imitate facial expressions. Another function of mimicry is that it can foster a sense of bonding. When facial imitation takes place within an interaction, it has been shown to make the individuals involved in the interaction to feel closer towards one another, and towards others in general (Stel & Vonk, 2010). Facial imitation has been found to foster empathic development and promote bonding-aspects that form part of the core disability of autism. Children with ASD struggle with these vital components of social interactions. It may therefore appear that the act of imitation may enhance social and emotional responses. However, lower levels of yawn contagion than imitation among these children suggest that an inability to process yawning and other complex social cues persists among ASD children.

Comparison across Age Groups

Yawn responses. The hypothesis that children 6 years and younger would show comparable levels of yawn contagion to children above the age of 6 years when eye contact is controlled for was confirmed. This is an interesting finding as previous literature has reported that children only display reliable yawn contagion above the age of 6 years (Anderson & Meno, 2003). In fact, no children below the age of 6 years caught yawns in their study. The lack of yawn contagion previously observed in children below the age of 6 years may have been due to poor attention skills which this study overcame by drawing children's attention to the eyes. Yawn contagion is therefore possible in children 6 years and younger when eye contact is controlled for. The findings of this study suggest that eye contact plays a key role in contagious yawning among younger children.

Imitation responses. Children aged six years and younger displayed more imitation responses than those older than 6 years. The high levels of imitation among the younger age group is not surprising as infants as young as 12-21 days old have been found to imitate open mouth movements (Meltzoff & Moore, 1977). Imitation among infants is thought to serve social and communicative functions (Meltzoff & Moore, 1999). This imitation seems to decrease substantially with age as far less imitation responses were displayed by children older than 6 years.

The finding that children above the age of 6 years show comparable levels of imitation and yawn responses is interesting as it would be expected they would show lower levels of imitations than yawns. However, all of the children in this age group that displayed

imitations are 7 years old, except one who is 9 years old. This suggests that the tapering off of imitation responses that is typically found among children may only occur after the age of 7 years and may vary among children.

Similarities between the ASD and 6 years and younger TD group

The ASD groups showed similar response patterns to the TD group aged 6 years and younger. Neither of these groups show yawn contagion without prompting attention to the eyes (Anderson & Meno, 2003; Senju et al., 2007). This suggests that a general lack of eye contact may be characteristic of each of these groups and of the lack of yawn contagion previously associated with them. However, the reasons for this lack of eye contact differ across the groups. Among the ASD children an inattention to the eyes has been explained as resulting from abnormalities of the amygdala (Schürmann et al., 2005). In younger children it is likely that the lack of eye contact is due to general poor attention skills characteristic of children of that age.

In addition, both groups displayed more imitations than yawns. This suggests that children with ASD and young children are more capable of imitation than yawn responses. The act of imitation, which serves some social and communicative functions (Meltzoff & Moore), appears to occur more readily among these groups than the more complex perception of social cues associated with yawn contagion.

Limitations and Recommendations for Future Research

It is important to note the limitations of the current study and how they can be improved upon in future research. Due to the time constraints in which this study had to be conducted, the sample sizes were relatively small. It would be beneficial to conduct a study with larger sample sizes for the ASD and TD control comparison as well as for the comparison across different TD age groups. Using larger sample sizes could be advantageous as one could then divide the ASD children into separate groups according to their level of functioning. Comparing ASD children at different levels on the spectrum could produce interesting results as it could show whether there is a difference in yawn contagion across these groups. Although the study across TD age groups is useful as it found that yawn contagion is possible in TD children aged six years and younger, it could be beneficial to make use of smaller age bands in future research to determine whether a developmental trajectory can be identified.

Another clear limitation of the study is the short time span separating yawn and control clips. In the study comparing ASD and TD groups, TD children yawned more frequently after yawn clips than control clips. In contrast, ASD children showed similar levels of yawn contagion after the different clips. Similarly, the study comparing TD children across age groups found that children in the older age group yawned more frequently after yawn clips than control clips, while the younger children showed similar levels of yawn contagion after the different clips. These findings among the ASD and younger TD groups may be due to the carry-over effect (Senju et al., 2009) as yawn clips may have promoted yawn contagion and this may have lasted longer than the 30 second period separating clips in this study. The latency of yawn contagion varies and ranges from seconds to minutes after the yawn stimulus has been viewed (Provine, 1989). Therefore delayed responses to a yawn clip may have been incorrectly interpreted as a response to control clips. A tentative explanation for this finding in ASD and younger TD children and not TD control and older TD children may be that children with ASD and children below the age of 6 years could possibly have longer latencies of yawn contagion than TD children above the age of 6 years. As the latency of yawn contagion has been found to vary from seconds to minutes, it is important that the clips be separated by longer time intervals in order to reduce the influence of carry over effects on results. Despite these potential difficulties, the decision to make use of short intervals in this study is justifiable as young children and children with ASD would have had difficulties paying attention to a longer video. Alternatively, it may be decreased attention to faces (as has been proposed to explain decreased yawn contagion when eye-fixation stimuli are not present), rather than different latency periods, that lead the ASD children and young TD children to yawn as frequently on control clips as yawn clips. It may be possible that children aged 6 years and younger, and ASD children, are less sensitive to the particular stimulus. These children might have paid less attention to the face and therefore did not discriminate between open mouth and yawn movements. Consequently, once they 'caught' a yawn, they yawned after both yawn and control clips.

Lastly, as suggested by Senju et al. (2009), future research should make use of an eye tracker. This would allow researchers to determine the exact areas of fixation of the participants gaze. It would then be possible to better determine the influence of precise points of focus on yawn contagion.

Conclusion

Comparable levels of yawn contagion were found across ASD and TD control groups as well as across older and younger TD age groups when eye contact was controlled for. These findings suggest that social cues relayed through eye contact play a key role in yawn contagion. This also implies that when eye contact is made, individuals with ASD better are able to pick up on certain social cues and respond accordingly. A bi-directional relationship appears to exist between the functioning of the ‘social brain’ and the ‘eye contact effect’: atypical functioning of the ‘social brain’, the amygdala in particular, leads to reduced eye contact (Grelotti et al., 2002; Hadjikhani et al., 2004; Schultz 2005; Senju & Johnson, 2009), while a lack of eye contact results in a lack of regulation of amygdala activation (Senju & Johnson, 2009). Although one may not be able to change the pre-existing deficits of the ‘social brain’ associated with ASD, increasing attention to eye contact allows for activation of parts of the social brain similar to that found among TD individuals. In addition this study showed that yawn contagion is in fact possible among children aged 6 years and younger when focus on the eye region is controlled for. This suggests that poor attention skills, which may result in a lack of attention to social cues, may have been the reason for the lack of yawn contagion previously associated with young children. Overall, it appears that the social cues conveyed by eye contact appear to play an important role in yawn contagion.

