Autism's Direct Cause? Failure of Infant-Mother Eye-Contact in a Complex Adaptive System.

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Abstract

This paper shows why an experimental hypothesis is plausible and merits testing. In brief the hypothesis is that autism begins with a failure in early learning and that changing the environment of early learning would dramatically change its incidence. Strong statistical evidence which supports this hypothesis has already been published by Waldman et al. (2008) but that evidence has largely been ignored, perhaps because it challenges prevalent beliefs about autism.

This paper also suggests that the current epidemic of autism is serious enough, and intellectually mysterious enough, to merit attention from a wider community of cognitive scientists: new ideas are needed. A confirmation of this paper's hypothesis would have interesting implications for cognitive science.

Keywords

Autism spectrum disorder; complexity; complex adaptive system; self-organize; emergent; eye-contact;

infant-mother; intersubjectivity; oxytocin; television/computer/smartphone.

Introduction

Most clinicians and researchers in the field of autism (Klin and Jones 2007; Johnson and Morton 2009; Baron-Cohen 1994) agree that autism is a *pervasive developmental disorder*, that is, that a primary deficit in the fetus or infant begins (to a greater or lesser degree) a cascade of secondary developmental failures which constitute the autistic syndrome. After decades of research, however, the primary deficit has not been identified (Muratori & Maestro 2007). This paper departs from the work of the researchers listed above only in arguing that the primary deficit which begins the cascade of developmental failures is not a genetic defect but is instead a failure in early learning.

Autism calls for an interdisciplinary approach; such an approach is difficult because insights from unfamiliar disciplines may not be well understood. (This paper explains some theory for readers for whom it may be unfamiliar and, where possible, uses non-technical language.) Geneticists have found that autism is strongly associated with a large variety of genetic lesions. Understandably, geneticists emphasize the relevance of their own results for research in autism. Researchers who are not themselves geneticists have tended to overlook the important distinction between a genetic association and a genetic cause. This has led to the widespread assumption that autism is initiated/caused by a genetic defect.

But experts in autism genetics (Abrahams & Geschwind 2008) have shown, as I detail below, that *none* of the genetic lesions associated with autism seems to cause it directly: each genetic lesion seems rather to compromise overall brain development so that the individual is more *likely* to develop autism and other syndromes. Autism's direct cause needs to be identified, both to understand its mechanism and to prevent it. To this end all pertinent information should be considered.

Working with infants (or older children), several teams of researchers (Kaiser et al. 2010; Klin et al. 2009; Auyeung et al. 2010; Elsabbagh et al. 2009), have recently described anatomical or behavioral traits which are correlated with autism. These researchers tend to assume that such traits are genetically

determined. In no case, however, have they eliminated the possibility that the trait might be acquired by experience in the first days, months, or years after birth.

Bergman (1985) documented the healing of an autistic child through psychoanalytic treatment. Deborah Fien (Poitras 2010) recently documented that at least 10%, and perhaps 20% of children who have clearly satisfied the criteria for a diagnosis of autism or autism spectrum disorder are able, given intensive behavioral therapy before age 4 or 5, to "move off the autism spectrum". A genetic deficit is unlikely to be reversed by psychoanalytic or behavioral therapy.

I begin by describing the evidence which led to my hypothesis that *the direct cause of autism is a failure of infant-mother* [endnote 1] *eye-contact*.

Why is it plausible that a failure of eye-contact may cause autism? To answer this I discuss the new paradigm of complexity the validity of which has been confirmed in economics and computer science and by much hard evidence from biochemistry, molecular biology, entomology, and ecology. We have not fully absorbed the new paradigm's implications: we are still inclined to think of ourselves linearly as genetically-predetermined structures.

Autism Spectrum Disorders and Intelligence

The autistic syndrome varies greatly in severity. A child with Asperger's syndrome (mild or high-functioning autism) has normal intelligence and language but has a social deficit and a narrow range of interests and activities. A child with classic autism may sometimes have high intelligence, but has more severe social symptoms.

Hans Asperger (Frith 1991) noted:

> ... autistic individuals, as long as they are intellectually intact, can almost always achieve professional success, usually in highly specialized academic professions, often in very high positions, with a preference for abstract content. We found a large number of people whose mathematical ability

> determines their professions; mathematicians, technologists, industrial chemists and high-ranking civil servants.

It is common for scientists and mathematicians to show some degree of Asperger's traits (Baron-Cohen et al. 2001). According to standard criteria it seems very likely that Newton, Einstein, and Cavendish had Asperger's syndrome (James 2003). Clearly autism's direct cause does not necessarily affect intelligence and must specifically target social abilities.

The Incidence of Autism is Increasing Rapidly

The California Department of Developmental Services (2007) reported that, from 1987 to 2007, the number of people with autism spectrum disorders grew 1148%. This increase is

> significant when compared to increases of 73% for cerebral palsy, 66% for epilepsy, and 95% for mental retardation. During this same period, California's general population grew 27%.

In 1999 it was estimated that between 0.1 and 0.2% of all children are autistic (Gillberg & Wing 1999). In 2007 the Center for Disease control estimated that the incidence in the united states was 0.75 percent. In 2009 their estimate increased to 1% (Kogan et al. 2009). Ganz (2006; 2007) estimated that, due to the high costs of treating and caring for a typical autistic individual over his or her lifetime, the annual cost of autism to US society is thirty-five billion dollars.

Byrd et al. (2002) have shown that the increase in California from 1987 to 1998 cannot be accounted for by changes in diagnostic criteria, by mis-classification, or by the immigration of autistic children. Hormatertz-Picciotto & Delwiche (2009) have shown in a rigorous statistical study that the increase in California cannot be explained by differential migration, diagnosis at earlier ages, changes in diagnostic criteria, and inclusion of milder cases. The incidence of autism rose 7- to 8-fold in California

from the early 1990s to 2008. Changes in diagnostic criteria seemed to contribute a 2.2-fold increase, inclusion of milder cases a 1.56-fold increase, and earlier age at diagnosis a 1.24-fold increase. These increases total a 5-fold increase, which leaves a 2-to 3-fold (very large) increase unaccounted for. While other statistical artifacts have not yet been fully excluded (for example, wider awareness, greater motivation of parents to seek services as a result of expanding treatment options, and increased funding for treatment), the most plausible explanation for these figures is that there has been a sharp increase in the real incidence of autism.

Yazbak (2003) commented:

> Suggesting that a sudden and exponential increase in autistic disorders is not real, and results only from better diagnosis, amounts to denial ... it does not seem reasonable to insist that the present autism outbreak is solely caused by hereditary factors. Genetic disorders have never presented as epidemics, and investing the scant available resources solely in genetic research diverts them from the scientific exploration of more plausible environmental etiological factors.

Genetic factors cannot account for a rapid increase in autism in many countries because the gene pool cannot change rapidly without extremely large migrations. The increase must, therefore, be triggered by a rapid change (in many countries) in one or more as-yet-unidentified environmental factor(s).

A Recent Review of Genetic Research

In a review titled "Advances in autism genetics: on the threshold of a new neurobiology," Abrahams & Geschwind (2008: 341) summarize the data as follows:

> Thus, in contrast to the complete absence of any biological understanding of ASD (autism spectrum disorders) as recently as 30 years ago, we now know that defined mutations, genetic syndromes and

de novo copy number variations account for about 10–20% of ASD cases. However, the striking finding that none of these known causes accounts for more than 1–2% of cases is reminiscent of *mental retardation,* an overlapping but distinct neurodevelopmental syndrome for which there is no single major genetic cause, but rather many relatively rare mutations.

New data on de novo copy number variations which are linked to autism was published this year by the Autism Genome Project (Scherer et al. 2010) but the new data does not change the above percentages nor their meaning. Scherer et al. confirmed Abrahams & Geschwind's percentages by noting that, taken together, all known genetic variants could only account for 20% of cases of autism and that, of the genetic variants they had found, none were present in more than 1% of individuals with autism.

A direct cause of autism should be both necessary (without it, no autism) and sufficient (when present, so is autism). No one genetic factor accounts for more than 2% of cases, which means that no one genetic factor is a necessary cause since at least 98% of cases occur in its absence. Neither is any one genetic factor, with the possible exception of Potocki–Lupski syndrome, a sufficient cause. Elsewhere in this review the authors state that "there is a growing list of single genetic lesions, each of which seems to be largely sufficient to cause an ASD". But their data shows that no more than 20-70% of the individuals who have a single lesion also have an ASD, which means, by definition, that the genetic lesions are not sufficient.

Abrahams & Geschwind themselves state that no genetic factor seems to be a *direct* cause of autism. They note:

Overall, none of the molecules or syndromes currently linked to the ASDs have been shown to selectively cause autism. Instead, each seems to result in an array of abnormal neurobehavioural phenotypes, including autism, Asperger syndrome, non-syndromic mental retardation and other neurodevelopmental abnormalities (p. 348).

This indicates that genetic lesions "cause" autism only in the *indirect* sense that genes are a

precondition for all biological phenomena. A familiar example of indirect "causation" is genetically-based blindness which, though it may be strongly associated with illiteracy, is neither necessary nor sufficient to cause it. Blindness blocks everything which depends upon vision and thus *indirectly* "causes" illiteracy. Another example is genetically-based fair skin which, though strongly associated with skin cancer, is neither necessary nor sufficient to cause it and is not skin cancer's direct cause. When a disease does have a direct genetic cause - familiar examples are Huntington's disease and sickle-cell anemia - then the mutant allele is both necessary and sufficient: it has its effect by changing the building blocks which are available to self-organization.

In the first paragraph quoted above, the authors note that autism's genetic footprint is like that of the "overlapping but distinct neurodevelopmental syndrome," mental retardation, "for which there is no single major genetic cause, but rather many relatively rare mutations." Mental retardation reflects the overall functioning of the cerebral cortex and thus is a frequent result of anything which interferes with brain development. The many not-necessary genetic lesions associated with mental retardation are therefore likely to be "causes" only in the indirect sense. The same is true for the many neither-necessary-nor-sufficient genetic lesions associated with autism.

The authors describe autism as a "neurodevelopmental" and "neurobehavioral" syndrome. These adjectives imply that neurological abnormalities are causal but this has not been proven. Characteristic changes in neuroanatomy may be associated with autism (see, for example, Elsabbagh et al. 2009) but, since brain anatomy is plastic and extensively shaped by early learning, such changes may readily be a secondary consequence of autistic behavior.

Under the subheading "Autism has a strong genetic basis" the Abrahams & Geschwind summarize current evidence as follows:

> Several lines of evidence support genetic factors as a predominant cause of the ASDs. First is the growing body of literature demonstrating that mutations or structural variation in any of several genes can dramatically increase disease risk. Second, the relative risk of a child being diagnosed with autism is increased at least 25-fold over the population prevalence in families in which a sibling is

affected. Third, siblings and parents of an affected child are more likely than controls to show subtle cognitive or behavioral features that are qualitatively similar to those observed in probands (the broader autism phenotype); this is consistent with the segregation of quantitative sub-threshold traits within these families. Fourth, independent twin studies, although small, indicate that concordance rates for monozygotic twins (70–90%) are several-fold higher than the corresponding values for dizygotic twins (0–10%) (refs). An important question for future work will be to clarify how environmental and genetic factors interact to influence risk and presentation (2008: 342).

The statement made above in the subheading, "autism has a strong genetic basis", is justified only in the indirect sense by the evidence given. First, some mutations dramatically increase the risk: these mutations may be "causes" only in the indirect sense. Second, siblings are 25-fold more likely to develop autism: again the genetic similarities may be "causes" only in the indirect sense; moreover, sibling data does not distinguish between genetic and environmental factors since siblings share physical, chemical, biological, psychological, and social environments. Third, siblings and parents are more likely to show subtle autism-like symptoms: the same two qualifications apply. Fourth, independent twin studies of monozygotic twins compared with dizygotic twins show a several-fold increase in concordance: again these studies do not distinguish between a genetic association and a genetic cause.

Since this paper is authoritative and summarizes all current evidence, we must conclude that autism is sometimes associated with genetic defects but that such defects are likely to be "causes" only in the indirect sense that genes are a precondition for all biological phenomena. Though sophisticated tools exist for studying genetic lesions, this may not be a promising direction for research in autism. Our understanding of autism would be greatly advanced if we could identify a defect which is autism's direct, immediate cause.

Tracking the Mother's Face

The correlation between autism and congenital infant blindness (Brown et al. 1997; Hobson et al. 1999; Bower 2009) is very high. If the mother of a blind baby relates to it actively by means of touch and sound then her baby's development may be close to normal; if the mother does not do so then her baby is very likely to develop autism (Als et al. 1980). These autistic children do not have higher-than-normal rates of any other biological factor. What they have in common is blindness and a consequent failure to relate to the mother in infancy.

There is also a high incidence of autism in children who were institutionalized at birth (Fraiberg 1977, pp. 185-7). There was a high incidence, for example, in children who were adopted in the U.K. from orphanages in Romania (Rutter et al. 2001). These children are normally sighted but were neglected in cribs during infancy. The Romanian children do not have higher-than-normal rates of any biological factor. Infants who have cranial nerve palsy with consequent paralysis of eye muscles are also liable to develop autism (Stromland et al. 2000).

These three different groups, congenitally blind children, children institutionalized at birth, and children with cranial nerve palsy, share one common deficit: they are all unable to track a care-giver's face. It seems that early deprivation in visual stimulus by a care-giver's face often leads to autism.

A Deficit in Psychosocial Development with *Secondary* Brain Differences

An autistic child seems not to know that his or her mother has a subjective self. Hobson et al. (1999, p. 55) suggested that the "final common pathway" (primary deficit) is a failure to develop a *theory of mind*. A theory of mind (or the lack thereof) may be inferred from a child's visual behavior. An autistic child makes little eye-contact, pays little attention to the mother's face, and ignores the mother's facial expressions. Consequently the child lacks *social referencing*: it cannot evaluate an ambiguous situation by checking the mother's expression. The child also lacks shared attention: it cannot follow the mother's eyes see what she is seeing, nor point to share feelings about something with the mother. Such deficits have recently been demonstrated at less than six months (Maestro et al. 2001). By studying home movies of

infants later diagnosed to be autistic, (Muratori & Maestro 2007) showed that some of these infants appear never to achieve eye-contact while others achieve some eye-contact, then lose it in subsequent months. Since some degree of eye-contact normally begins at birth (Farroni et al. 2002), a deficit in eye-contact may also begin at birth.

From evidence of this kind, Trepagnier (1998, p. 158) proposed that brain differences are secondary in autism. She suggested that a failure in face-processing (due to very-early-onset anxiety/ affective disorder) is the key. Klin et al. (2002) showed that an autistic person looks preferentially at the mouth rather than the eyes in another's face. A normal person does the opposite. Klin et al. suggested that this preference may represent a core social deficit in autism.

I have proposed that the primary deficit in autism is the failure of the infant-mother pair to achieve or retain eye-contact (McDowell 2002, 2004a, b)[endnote 2]. (Corollaries are: provided a blind infant is actively mothered, the infant-mother pair may achieve an equivalent contact through the other senses; the attainment or functioning of eye-contact may be disturbed to varying degree.)

To understand how an early failure of eye-contact could lead to autism, we must first discuss complexity.

Complexity

Human Complexity

A human being, body and personality, represents an astronomically high degree of complexity which is not commensurate with the relatively small amount of information stored in genes. One estimate is that human body contains about $5x10^{28}$ bits of information in its molecular arrangement (Elman et al. 1996, p. 319) while the total number of different genes in our chromosomes is very small. We have only about five times as many genes (20,500) as a bacterium (3-5,000) (Broad Institute of MIT and Harvard 2008; Alberts et al. 2007, p. 31). A microscopic nematode, one of the simplest animals, has about 19,000 genes. But our structure is astronomically more complex than that of a bacterium or nematode.

A gene functions by predetermining the sequence of bases in a ribonucleic acid and the sequence of amino acids in a polypeptide. The three-dimensional structure of an enzyme or regulatory protein then emerges from the polypeptide's spontaneous folding (self-organization) under the chemical constraints of the intracellular environment (Alberts et al. 2007 pp. 134-7, 387-8)[footnote 3]. An enzyme then catalyzes a chemical reaction while a regulatory protein modifies gene expression. The other structures within the cell - for example the DNA double helix, chromosomes (Wood and Crowther 1983; Alberts et al. 2007, pp. 195-245), transfer RNA molecules, ribosomes (ibid pp. 378-9), membranes (ibid p. 9) and mitochondria - all self-organize spontaneously. Since genes do not predetermine structure even at the level of the folded form of enzymes and regulatory proteins, they certainly do not predetermine multicellular structure.

Moreover, we evolved from a species of ape - the common ancestor of humans and chimpanzees - within the last six million years. The rate at which genes mutate is unchanging: six million years is only long enough to permit a small number of mutations. Current estimates are that humans and chimps differ by perhaps 2,000 significant mutations (Chimpanzee Sequencing and Analysis Consortium 2005; Lovgren 2005). The genetic difference between humans and chimps is no more than that between two species of mice. In humans these genetic differences achieve their dramatic effects mainly by changing the *timing* of the processes by which a fertilized egg develops into an adult (McKinney & McNamara 1991). For example, one genetically-determined difference between a human and an ape is that, in human development, the cerebral cortex continues to grow in *volume* for a longer period of time. The result is that we have a bigger cortex. The greater *complexity* of our cortex cannot be predetermined by mutations because, by astronomically large orders of magnitude, there are not enough mutations to do so. Its greater complexity must be achieved by other means.

The brain's complexity is much informed by its environment. For example, when a child learns to speak the language centers in the human cortex expand relative to adjacent cortical areas. If the child does not learn to speak those areas do not expand. Thus the information required for the formation of language centers in the brain is mostly acquired by learning to speak (Karmiloff-Smith 2009). Experiments with ferrets have confirmed this (Sharma et al. 2000). Visual information from the eyes was surgically

redirected to the auditory region of the newborn ferrets' developing cortex; the auditory region became a visual region.

A New Paradigm

To understand complexity Holland (1998, see pp. 8-9, 229-31), Gell-Man and others have elucidated a new paradigm: complexity *emerges* (*self-organizes*) when a small number of simple components interact spontaneously according to simple rules and the environment provides simple selection criteria.

This can best be understood by means of an example. In chess (Holland 1998, p. 23) there are only six different pieces - 32 pieces in all - set up in a simple pattern on a board of 64 squares. Each of the six pieces moves according to its own simple rules. When all the pieces interact, however, there emerges an astronomically large number of possible sequences of moves. This complexity is nowhere specified in the game's rules but is an emergent consequence of those rules. Most sequences are nonsensical. By applying a few simple selection criteria, however, we can easily select serviceable games. Examples of selection criteria are: only sequences ending in checkmate or stalemate; only sequences shorter than 20 moves. The pieces and their simple rules generate possibilities while simple selection criteria ensure that only serviceable games emerge. Because each selection criterion has a dramatic, pervasive effect on the outcome, a small increase in the number of selection criteria can make the outcome highly predictable. Thus complex, functional, predictable organization can be achieved from minimal information: this is what is needed to account for human complexity.

Dynamic Systems

Live phenomena are not static, like a finished chess game, but dynamic. A dynamic system is a spontaneous organization which requires a continual flow of energy. An example is the whirlpool in your sink: it forms only while the kinetic energy of moving water flows through it. If you block the drain, the kinetic energy subsides and the whirlpool ceases to exist.

A dynamic system is:

self-organizing or emergent. It is not formed according to an external blueprint, but develops spontaneously from the inherent properties of its components. A whirlpool is formed by water, its mass and fluidity, the structure of the sink, gravity, and mathematical laws of motion.

homeostatic; when perturbed it tends to restore its original equilibrium. If you distort the whirlpool in your sink by putting a finger in it, then remove your finger, it will quickly restore its original form.

constrained by its environment. Your finger affects the form of the whirlpool. Thus a dynamic system absorbs information from, and adapts to, its environment.

regulated by the supply of its input and the demand for its output. A fire in a fireplace (a dynamic system) is regulated by the rate at which fuel and oxygen are supplied and the rate at which carbon dioxide is removed.

Dynamic systems also exist in living cells: the Krebs cycle, for example, burns fuel molecules to produce heat, carbon dioxide and new energy-storing molecules.

Complex Dynamic Systems

The whirlpool and the Krebs cycle are *simple dynamic systems*. A collection of several simple dynamic systems may spontaneously interact with each other according to simple rules, thereby forming a complex dynamic system. Rules of interaction are not imposed from an external blueprint, but arise spontaneously from the intrinsic properties of the component systems.

In the living cell the Krebs cycle is one of many cycles/pathways, each a simple dynamic system. Each "structure" within a cell also behaves like a cycle/pathway and is also a simple dynamic system. Each cycle produces molecules which are consumed by other cycles. Each cycle is homeostatic. The activity of each cycle is regulated by the supply of its input molecules and the demand for its output. Thus

"supply and demand" is one of the simple rules by which cycles interact (Alberts et al. 2007 pp. 88-102; Mitchel 2009). All these component simple systems, interacting together, form a homeostatic network which is the cell. In this (schematic [footnote 4]) description, a cell is a "first-order" complex dynamic system.

Many cells interact spontaneously to form a kidney. Each cell creates products which are used by other cells. Each cell is regulated by other cells according to simple rules. Together the cells form a homeostatic network. In this (schematic) description, an organ is a "second-order" complex dynamic system (Holland 1998, pp. 225-31). Organs interact spontaneously in a homeostatic network to form an individual plant or animal, a "third-order" complex dynamic system. For bees, ants, and termites (Bonabeau et al. 1997), individuals interact spontaneously to form a colony, a "fourth-order" complex dynamic system. Plants, animals, and micro-organisms interact spontaneously according to simple rules to form an ecosystem (Levin 1998; Scheffer 2009; Loeuille 2009), a "fourth- or fifth-order" complex dynamic system. An ecosystem integrates myriad interactions to form a flexible but intensively-ordered whole. And yet there are no genes to order an ecosystem.

Complex Adaptive Systems

Because it adapts spontaneously to its environment, a higher-order complex dynamic system is sometimes termed a complex *adaptive* system. An ecosystem, for example, adapts to climate changes to maximize biomass. A market-based economy (Arthur 1999) is a complex adaptive system whose component systems are individual businesses. It adapts spontaneously to its environment to maximize productivity.

Some complex adaptive systems have the capacity not only to adapt, but also to *learn* an adaptation, that is, the adaptation includes *persistant internal changes* which influence the system's future behavior. Such systems can then move progressively through a series of further adaptations. In the evolution of a species, learning occurs at the level of population: a population evolves over many generations. An ecosystem not only *adapts* to environmental changes but also *evolves* in the direction of greater diversity and greater biomass.

It is now known that everything which is alive, without exception, is a complex adaptive system.

Is this also true of the human personality?

The Human Personality:

Carver and Scheier (2000) defined the personality as "a dynamic organisation, inside the person, of psychophysical systems that create a person's characteristic patterns of behavior, thoughts, and feelings." For the purposes of this argument I define the personality more psychologically and include the interpersonal: the personality is a person's system of desires, affects, feelings, bonds, ambitions, ideals, expectations, sensations, images, memories, dreams, intuitions, ideas, habits, skills and activities, conscious and unconscious, interior and interpersonal [endnote 5]. (A physical substrate for these psychological aspects is assumed, but is not relevant to my argument.)

Self-Organizes from Elemental Systems. The personality is a dynamic system which can only exist while psychological energy flows through it, that is, while it receives psychological stimulation and expresses itself (Stolorow 1997, Beebe et al. 1997, Beebe & Lachmann 1998).

A system is considered "alive" if it fulfills the following criteria: reacts to its environment; adapts to its environment; reproduces; grows; obtains and uses energy; is composed of cells; is capable of evolving. The personality fulfills all these criteria: it reacts and adapts to its environment, it reproduces itself in the next generation, it grows, it is nourished by "input" psychological energy (love, companionship, play, stimulation, challenge, education, experience), it generates "output" psychological energy (aggression, hurt, grief, excitement, love, joy, creativity), and it evolves. For the personality an individual person is the equivalent of a cell.

In the realm of psychology, therefore, the personality must be considered alive. But we know that all living things are complex adaptive systems which emerge from the self-organization of simpler systems. It follows that the same must be true for the personality. Given the evolutionary and genetic constraints discussed above, the human personality must necessarily organize itself by means of the spontaneous interactions, according to simple rules, of a small number of *psychological* simple dynamic systems. It should therefore be possible to identify some of these elemental systems.

Is a System of Interactions. A crucial difference between humans and other mammals is socialization. Many human attributes have evolved in whole or in part for social signaling:

Flattened face, which often lacks facial hair, and our enlarged, mobile lips and prominent teeth.

Color contrasts in hair and skin which, especially for light-skinned people, changes color dramatically when we tire or blush. Color contrasts formed by teeth, lips, iris, pupil, whites of the eyes, eyelashes and eyebrows.

Complex innervation and musculature of the face, including its skin with its wrinkles, which enables facial expressions.

Individual variation in features, coloring, and body form.

Pronounced sexual dimorphism, monthly oestrus cycle and continual sexual appetite.

Upright stature and hairless skin which exposes to view the whole body surface.

Many changes in the appearance of face and body which signal age.

Prolonged dependent childhood shared with multiple siblings and peers.

Mobility of arms, hands and fingers which enables social gesturing.

Tears, whimpering, sobbing, smiling, giggling, laughing, guffawing, and vocal range, all of which express affects and moods.

Verbal language.

The cerebral cortex seems to have evolved to its modern size mainly to enable social interactions (Dunbar 1992). Performing arts, literature, visual art, and programs of training, education, and research all revolve around socialization, as does much of technology.

The personality emerges mostly in interactions with other people [endnote 6], first in the infant-mother interaction. The personality does not exist like the brain or the heart, as a complex dynamic system inside of a single person. It is more like an on-going conversation, a system of interaction which can only exist between two or more people (Stolorow 1997, Lachmann 2000). Beebe and Lachman (1998) reviewed how the infant's personality evolves as the infant-mother pair interact. For stability and vitality, the adult personality also needs continued interaction. The elemental psychological systems from which the personality self-organizes, therefore, are likely to be simple dynamic *systems of interaction*.

Failure of an Elemental Dynamic System of Interaction. If one of the personality's elemental systems failed to initiate, the effects upon the personality's development would be pervasive. If an elemental system operated for some months and then ceased to operate, the effects would also be pervasive. (A complex adaptive system requires the continual functioning of its component systems.) If an elemental system operated at a lower-than-normal level, the effects would be pervasive but less severe.

The above is consistent with the pattern of effects in autism: autism is a pervasive cascade of disordered development which often begins very early; some infants achieve a degree of interactive functioning in the first year but subsequently become autistic; amongst individuals the severity of symptoms varies greatly.

Eye-Contact

Eye-contact is a simple dynamic system of interaction. It has a profound affect on both participants, alerting each, making each more aware of the other's subjectivity, and forming at least a momentary bond between the two.

Among mammals, a human infant is unique in that a human infant is biologically designed to establish eye-contact. When a mother breast- or bottle-feeds her infant the distance between her infant's eyes and her own eyes corresponds exactly to the distance at which a new-born's eyes can focus (at birth the range of focus is narrow). While breast feeding, a human mother spends about 70% of her time gazing at her infant's eyes (Stern 1977, p. 35-6). At about six weeks the infant learns to make eye-contact with the mother. *Eye-contact stimulates the mother to play more with the infant.* The mother plays with facial expression, with voice, with face presentations, with head movements, and with proximity games (ibid, p. 37). Such a positive feedback loop must inevitably reinforce the subsequent progression of stimulating and comforting interactions (Beebe et al. 1997). As I discuss below, these "socially pleasant sensory experiences" have a permanent effect on the infant's oxytocin and vasopressin levels and these, in turn, enhance subsequent interactions.

The foregoing vividly illustrates self-organization in infant development. Eye-contact itself begins so early that it is likely to be one of the original simple dynamic systems of interaction from which the personality self-organizes.

The Evolution of Social Gaze

For many species of birds, an image of the eyes is a potent signal warning of the danger of predators (Janzen et al. 2010). We can deduce an evolutionary sequence by comparing modern primates because humans evolved from ancestral primates very like modern primates. Within that evolutionary sequence, social gaze became increasingly important.

In most primates the visible eye consists mainly of dark iris with only a very small proportion of sclera. The sclera is almost always brown or light brown rather than white. But in humans the visible eye

has a large expanse of white sclera which provides a sharp contrast to the dark iris (Emery 2000, pp. 583, 585). This helps an observer to determine the precise direction of a human's gaze.

When viewing faces, a monkey shows an extreme bias for looking at the eyes and the small region surrounding the eyes. The eyes play a central role in a primate's facial expression and thus in emotional communication (ibid., pp. 585–6). Staring and frequency of eye-contact are key social signals. The direction of one monkey's attention, coupled with its facial expression, conveys crucial information to a second monkey about the object of attention (joint attention). It may inform the second monkey about the social status of a third, or about food or about danger.

Apes, but not monkeys, appear to use the direction of another's gaze as a clue to the other's intended behavior. This is more complex than joint attention because it requires an awareness of the other as a repository of potential behavior. It seems that only humans, however, practice *shared attention* (ibid., pp. 588, 594), in which two individuals are each aware of the direction of the other's gaze (I know that she sees it and she knows that I see it). Shared attention is a component of theory of mind. In psychoanalytic terms its equivalent is *intersubjectivity*. These observations are direct evidence that, as the primate's eye has evolved in appearance (towards more visual contrast between iris and sclera and hence towards a better signal of gaze direction), so it has evolved in psychological function (towards enabling intersubjectivity).

This is supported by experiments with human subjects (Baron-Cohen et al. 1997, pp. 323–5). Subjects were asked to identify complex emotional states (scheme, admire, interest, thoughtful, arrogant) from photographs of the whole face, from cropped photographs showing only the mouth, and from cropped photographs showing only the eyes and eyebrows. When the whole face or the eyes alone were seen, accuracy of identification was equally high. When the mouth alone was seen, accuracy was low. Thus the other's complex subjective state is conveyed by the appearance of the eyes. This last conclusion is confirmed by the work of Klin et al. (2002, p. 809).

The Development of Intersubjectivity

Intersubjectivity is Emergent

Winnicott (1971/86, p. 112) said:

> when [the baby] looks at the mother's face . . . what the baby sees is himself or herself . . . the mother is looking at the baby and what she looks like is related to what she sees there.

Stern (1985, p. 139) described how the infant develops a sense of self within the infant-mother interaction. At what he termed the intersubjective phase, beginning around eight months, the infant senses that infant and mother each have inner mental states which they can share (I am excited and I know that she feels my feeling.) Just as the infant's body develops in relationship with the mother's body and her actions, so the infant's sense of self develops in relationship to the mother's awareness of and inner response to the infant. This is particularly the case for the intersubjective sense of self (I see that my mother sees me as lovable, so I see myself that way too). In the words of Stern et al. (1998, pp. 907–8):

> . . . the intersubjective goal . . . [is] a mutual recognition of each other's motives, desires, and implicit aims that direct actions, and the feelings that accompany this process . . . [This] also implies a signaling or ratifying to one another of this sharing . . . The work is asymmetrical, with the caregiver . . . doing the lion's share.

Achieving eye-contact

Stern's model of development shows why eye-contact is important. The mother senses the presence of the infant's inner life in part through the infant's eyes. Likewise, the infant senses the mother's inner life in part through the mother's eyes.

Infant and mother also engage each other through smell, taste, touch and sound. An infant has remarkable cross-modal perception. Infants recognize the smell of their own mother's milk (Stern 1985, pp. 39–40). Beebe, Lachman and co-workers have shown that by four months the infant-and-mother pair

have developed vocal-rhythm coordination, the degree of which predicts both attachment and cognition at twelve months (Jaffe et al. 2001; Gergely & Watson 1996; Beebe 2005; for examples of mother and five-week-old infant interactions which show the centrality of eye-contact, Beebe et al. 1997, pp. 153–4).

If the baby is born blind then it must detect the mother's inner life through the other senses. Infant-observation studies by Fraiberg (1977, pp. 3–9) and Als et al. (1980) compared groups of blind and sighted infants (all mothers were sighted):

> For the blind infant, containment is more difficult. The visual focus is missing. Closer tactile contact, nuzzling, and more continuous enveloping with the mother's voice make up for it . . . The sighted infant's interaction with the mother is similarly structured, yet not as explicit, nor are the cycles repeated as often, as containment and goal attainment are facilitated by visual feedback . . . the process of implementation is much more conscious for the mother of the blind infant (ibid., pp. 198, 201).

The studies by Fraiberg and Als et al. show that, *for sighted infants* (the vast majority), eye-contact with the mother is central to establishing an infant-mother bond.

One might object that the infant interacts visually with the mother's whole face, not just the eyes. Visually, however, the eyes are highlighted in the face by virtue of their duplication, intense color, extreme light–dark contrasts, sharpness of edge, regular shape (which is echoed by moving curved lashes and brows), and rapid movements. That the movements of the mother's eyes follow (are contingent with) the infant's movements must make the eyes the more compelling. Gergely (2001, p. 411) notes that "young infants are very sensitive to the contingent relationships between their motor responses and consequent stimulus events". The mother also follows her infant with ears and nose, but these are visually undistinguished and they give no evidence of following. Even to an adult the eyes are visually compelling; as an infant first learns to distinguish forms, the eyes are likely to be the first image the infant masters.

In order for the infant to achieve eye-contact, the infant must learn to *visually recognize* the

mother’s eyes.

How the Infant Learns to Recognize the Mother’s Eye

Visual Edges in the Image of the Eye. A reflex is not peculiar to humans but is intrinsic to the function of an organ. Blinking and coughing are examples. When the eyelid evolved in reptiles it necessarily had a movement reflex (Gans and Parsons 1973). A reflex is a simple element of behavior which involves only a few muscles. It behaves as though it were hard-wired into the nervous system. I will show that such reflexes help a newborn to recognize the mother’s eye.

In a newborn infant some behaviors seem to precede learning. For example, Spitz and Wolf (1946) found that an infant gazed preferentially at a human face or at a schematic drawing of a face. A line drawing of two eyes, a nose, and a mouth was sufficient. They suggested that the infant inherits a schematic image of the human face and seeks a match for that image in its immediate environment.

Friedman (1964) and Haaf and Bell (1967) proved, however, that the infant gazed preferentially only at some elements of the face. It made no difference if the elements were scrambled in the drawing. The infant gazed preferentially at moving edges. The most attractive moving edges within the mother's face were the sharp angles at the corner of the eyes (two edges intersecting), the light/dark contrasts between the pupil and the white sclera of the eye (an edge), and the contrast between eyebrow and skin (an edge) (Stern 1977, p. 37). There are also edges around the lips and around the outer rim of the head.

Haith (1966) and Salapatek (1975) proved that, from birth to about one month, an infant seeks out any visual stimuli which includes both movement and 'edge density', that is, the concentration of edges in a given space. By two months the infant's vision is beginning to mature: the infant begins to recognize when facial features are scrambled and shows a preference for naturally-ordered features.

Visual Edges and the Cortex. Research on the vision of adult vertebrates (fish, frogs, cats, and monkeys) helps to explain the human infant’s preference for visual edges (Michael 1969). All vertebrates analyze visual input primarily into a series of moving edges. The analysis is done by neurons in the retina of the eye and by neurons in areas of the visual cortex which are adjacent (closely connected) to the retina. This

means that the brain does not initially 'see' a whole image. Rather it initially 'sees' a series of moving edges.

A computer analyzes an image into a stream of digits, zeros and ones, which it records. The stream of digits contains the information needed to recreate the image but is not itself a spatial analog of the image. In a similar fashion the retina and the adjacent visual cortex analyze the image into multiple streams of moving edges. Elsewhere within the brain, this data must be “synthesized” in some way to form an “internal image” which permits later recognition of the same visual input. I imply nothing about the mechanism by which this is accomplished, nor about the nature of the internal image. These are not known. It is logically necessary, however, that an internal “image” must form and that it is not achieved whole, like a photograph, but by synthesis.

The experiments of Friedman and Haaf and Bell (*v.s.*) distinguished a stage through which the infant must pass before it learns to see like an adult. Since the newborn recognizes moving edges, we know that the neurons in the newborn's retina and adjacent cortex are pre-wired (or very quickly wired) to analyze visual input into edges. The newborn also has functioning muscle reflexes which enable it to focus its eyes, to fix upon an object, and to track an object (Stern 1977, p. 34; 1985, p. 40). All this very early wiring provides for the visual reflexes by which the newborn tracks the visual edges in its mother's face.

Cortical neurons have been identified which only fire when the infant recognizes a face (Elman et al. 1998, p. 116). Only after the infant has learned to synthesize an internal “image” of the face could the infant discriminate between naturally-ordered and scrambled images of the face. This explains what Salapatek (*v.s.*) observed experimentally, that at two months infants begin to distinguish between scrambled and ordered features.

During breast feeding, as noted earlier, the mother's eyes are within the newborn’s (limited) focal range and the mother spends 70% of her time gazing at her infant's face. The infant-mother pair is thus designed to ensure that the newborn will stare at the sharp, high-contrast, moving edges of its mother's eyes. Thus the infant will learn to make eye-contact and to associate it with breast (or bottle) feeding.

It is perhaps a human’s sense of self which most distinguishes him or her from other mammals. It

is remarkable, therefore, that the human sense of self is initiated by the geometry of breast feeding. A cat's eyes are equally sensitive to moving edges. Because of the way a kitten feeds, however, it does not begin life gazing at its mother's eyes.

Two Lines of Current Research which Support the Eye-Contact Hypothesis

Oxytocin, Vasopressin, and a Developmental Cascade

Children who were institutionalized at birth show *secondary brain differences* which interfere with face recognition and bonding.

In infants, the level of the neuropeptides oxytocin and vasopressin is normally increased by socially pleasant sensory experiences (Fries et al. 2005). In turn these neuropeptides promote social bonding, the display of selective infant-parent attachments and the formation of memories of these interactions. Fries et al. (ibid) showed that, in adopted children who had previously been raised in orphanages, there were very large reductions in the baseline level of vasopressin. They also showed that there were very large reductions in that elevation of oxytocin which, in normal controls, is triggered by close contact with the mother.

Rimmele et al. (2009) showed that, when administered to normal adults, oxytocin specifically improves recognition memory for faces but not for nonsocial stimuli. Andaria et al. (2010) found that when adult subjects with autism inhaled oxytocin, they interacted preferentially with more social partners, felt more trust and preference and, when looking at faces, spent more time gazing at the eyes. Guastella et al. (2010) found that inhaled oxytocin improved emotion recognition in young adults with autism spectrum disorders.

Thus a developmental cascade, which is triggered in part by early infant-mother eye-contact and which is disrupted in autism, is being deciphered at the molecular level:

(1) Infant-mother eye-contact triggers increased maternal attention (above).

(2) Early maternal attention permanently increases (a) baseline vasopressin and (b) that oxytocin release which is normally triggered by subsequent socially pleasant sensory experiences. Lack of early maternal attention permanently blocks both increases.

(3) Vasopressin and oxytocin, in turn, promote face recognition, gazing-at-the-eyes, emotion recognition, and social bonding in both normal and autistic adults.

Precipitation Levels and Autism Incidence are Strongly Linked

When I first proposed that failure of early eye-contact was the immediate cause of autism (McDowell 2002, 2004a, b), I pointed out that this hypothesis led to a testable prediction: autism would be statistically linked to early non-maternal child care which includes time spent watching television/video screens.

There are a number of reasons to suspect that television watching might trigger autism. Because the recent increase in autism is world-wide, its environmental trigger must also have recently increased worldwide. Television watching by very young children has grown dramatically over the last few decades (Kaiser Family Foundation 2003; 2006). There is evidence that early television watching hurts language, cognitive, and attentional development (Anderson & Pempek 2005). For example Christakis et al. (2004) found a statistical link between early television watching and later diagnosis of ADHD (see also Christakis 2009). The American Academy of Pediatricians Committee on Public Education (2001) recommends no television viewing for children below the age of two. Olmsted (2005a,b) found that the incidence of autism amongst the Amish, who watch no television, is extremely low.

It is self-evident that children's time spent indoors now involves much use of television/ computer/smartphone screens. Waldman et al. (2006) confirmed statistically that childhood television watching is linked to precipitation. Waldman et al. (2008) then published rigorous proof that autism and precipitation rates are linked. Precipitation rates appear to account for 40% of autism diagnoses, a very

high proportion. Waldman et al. (2006) also showed that, in Pennsylvania and California from 1972 to 1989, autism rates correlated strongly with county-by-county introduction of cable television. Cable also increases television watching.

The statistical associations proved by Waldman et al. could conceivably be due to indoor air pollution or vitamin D deficiency. Both must be tested but both are implausible as causes of autism. If either caused neurological damage, the damage would be non-specific; it is very unlikely that either would *specifically* target social interaction.

The most plausible explanation for Waldman et al.'s results is that television/computer/smartphone use interferes (by distracting either the mother or the infant or both) with an early, critical stage of infant-mother bonding. It is likely that eye-contact would be the component of early bonding most directly affected by competition with the glowing screens to which we have become addicted (Christakis 2010).

Conclusion and a Clinical Study which Addresses Prevention

I have presented theory and evidence to suggest that autism may be directly caused by a failure in early infant-mother eye-contact, a failure which becomes more frequent with the increasing use of television/computer/smartphone screens. More generally, it may be possible to identify some of the early simple dynamic systems of interaction from which the personality self-organizes: if this were confirmed it would have interesting implications for brain science.

Those invested in other theories of autism may tend to dismiss the eye-contact hypothesis. Parents of autistic children, for example, are sensitive to being "blamed" for their children's condition. Early breast feeding may offer a helpful analogy here. For a few hours after birth an infant has spontaneous mouth movements by which it seeks to suck the mother's nipple. If the newborn is promptly applied to the nipple, breast feeding tends to begin easily. If breast feeding is delayed then it is more difficult to establish; sometimes bottle feeding is then favored. Parents might be be comforted by

understanding that failure to establish a behavior during a very-early critical period does not reflect upon the overall quality of their parenting.

The forgoing suggests a clinical study which would address prevention. It would be possible to recruit a select group of parents who undertake (a) to greatly reduce, from birth for the first few years, their whole family's exposure to television, computer and smartphone screens, and (b) to keep records of such exposure which may occur. It would not be necessary or desirable to ask these parents to forgo other strategies for preventing or treating autism. Waldman's research suggests that, compared with the general population, the children in this study might show a large reduction in the incidence of autism. Such a result, in an initial study, would encourage more parents to take part in a larger study.

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Endnotes

1. The terms 'mother' and 'maternal' refer to the very early mothering function, which may sometimes be fulfilled by a person other than the mother.

2. Earlier I proposed that the failure was to acquire or retain the image of the mother's eye. Eye-contact itself is a more measurable phenomenon. Eye-contact requires recognition which requires that the infant acquire an image, in some sense, of the mother's eye.

3. Molecular biologists have described in fine detail the mechanisms by which macromolecules self organize. In their writing, however, molecular biologists do not always distinguish between the invariance of primary structure (invariant because it is directly specified by genes) and the invariance of secondary,

tertiary, and quaternary structure (invariant because, in the normal intracellular environment, the outcome of macromolecular self-organization is predictable)

4. This account, including my use of ordinal numbers, is highly schematic. My purpose is to clarify the hierarchical nature of complexity in biological systems.

5. This inclusive definition is compatible with most theories of personality, for example with trait, type, psychoanalytic, behaviorist, social cognitive, and humanistic theories.

6. Some of the personality emerges though interactions with impersonal factors.