Babies and infants

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Age of identification

'The earlier intervention can begin, the better the outcome.'[1]

Data from both the USA (e.g. California[2]) and Europe (e.g. Denmark[3]), show that the average and median ages at which childhood autism diagnoses are made are steadily dropping. However, there are some subtleties in the pattern; for example, our analysis[4] suggested that in England the average age of diagnosis for the youngest children (0–2 years) went up marginally between 1998 and 2018, perhaps because of increased demand for diagnoses and long waiting times.

Claims for identification of pre-symptomatic predictors of autism are now being made for very young babies.[5, 6] Using brain imaging, one group has noted autism-specific 'features' in six-month-old babies,[7] while another, which received world-wide media attention, used eye-tracking technology to identify subtle differences in the way affected babies responded to visual prompts:[8] 'Autism can be identified in babies as young as two months, early research suggests'.[9] Studies such as these, and others, are used to define infants 'at risk' of autism. To be 'at risk' is to be in danger of falling outside the statistical norm – a state requiring expert advice, intervention, parental regulation and surveillance.

The narrative of earlier-is-better (EIB) transcends autism to pervade child psychiatry, education and infant development and beyond: dementia, diabetes and hypertension spring to mind as examples of ways in which medicine has extended its jurisdiction.[10] Identifying potential early signs and signals of autism makes earlier diagnosis, detection and intervention possible. However, although the evidence base is regularly reviewed, the evidence that earlier intervention results in more successful outcomes for the child is poor.[11, 12] A recent UK review of evidence on screening infants for autism, conducted in 2011, concluded:

- Diagnoses of very young children may not be stable.
- Current screening tools are insufficiently sensitive and may not be accepted by a significant proportion of parents.
- The outcomes of interventions are variable.
- It is not known if short-term improvements continue in the long term.[13]

Baby-sibs

The collection of studies known as 'baby-sibs' research gives 'at-risk' status to new-born and unborn children who have siblings with autism.[14] At-risk status is given because autism is heritable and geneticised.[15–18] Sharing a genomic profile with autistic siblings, that is, being a sibling of someone with autism, therefore puts you at risk of autism. Estimates of the extent of familial heritability over 40 years ago were that around 90% of variance in autistic traits is attributable to inherited factors,[19] whereas today around 50% of variance is attributed to inherited factors.[20]

These two types of identification of the youngest children (an autism diagnosis in babyhood and the 'at-risk' status given to babies and unborn children) are related but distinct processes. Earlier autism diagnosis has consistently been associated with more severe autism and more severe impairment.[21–26] In childhood studies, the factors associated with an earlier diagnosis include greater language delay, need for a greater degree of support, more cognitive and intellectual disability, greater parental concern, an autism (as opposed to Asperger's) diagnosis and the severity of autistic beahviours.[21–26] The picture is one in which more severe autism is more obvious, therefore is picked up earlier in a child's life. Put simply, babies with more extreme neurodevelopmental difference are, and were before 1990, easier to spot.

A raft of EIB studies tells the story of how the earlier a child can be recognised, the more effective early intervention is, and so it must be brought into place.[27–30] The longer diagnosis is delayed, the greater the chances of missing a critical developmental period.[22] Once this window is missed, brain plasticity is lost and interventions may be ineffective.

At-risk babies (such as baby siblings, through their shared inheritance of a genetic predisposition) may be anywhere in the broad autism phenotype, which includes sub-clinical (milder) levels of autistic traits.[31] Baby-sibs studies look for early indicators of autism but necessarily include children who go on to develop milder, and in some cases, few or zero, autistic traits. Many but not all, baby-sibs studies follow up on later autism diagnosis.

Precursor signs of autism in infants, which have been deduced from baby-sibs and retrospective studies, can be loosely divided into behavioural signs, genetic predisposition and neurological differences. *Behaviours* include types of movements or lack of motor skills, imitation impairments, lack of physical exploration of objects in the environment with less object manipulation[32] and lack of joint attention. Many studies identify abnormal movement as a precursor, including gross motor, fine motor and postural control[28, 33–36] and babies' head lag.[37]

The larger catchment of 'at risk' of, as opposed to diagnosed with, autism presumably results in some studies widening the net of potential early signs of autism gleaned from siblings' behaviours and abilities. An aspect that is not often dwelled on is that a researcher denoting a baby sibling as 'at risk' surely makes an autism diagnosis more likely, not only through relatedness but also if parents

see their baby as being in a proto-autism group. This, one would assume, will increase the likelihood of referral to a clinic and, once in the clinic, the interpretation of behaviour as autism. At the same time, by defining new signs of precursor autism using behaviours in the 'at-risk' group, all 'at-risk' babies' behaviours start to be understood as signalling autism. It seems circular: what counts as a specific 'signifier' of autism, most often motor difficulties, becomes connected to identification of autism in the group from whom the 'signifier' was determined.

Earlier diagnosis and risk

As well as identifying early indicators, autism studies have early diagnosis of autism as a core objective.[38] EIB is most commonly operationalised in intervention research. The assumption is that there is a fixed disorder that is present from birth, can be correctly identified soon after birth and which intervention will ameliorate. Advocacy, funding and charity organisations also strongly promote earlier diagnosis; for example, Autistica's report, *One in a Hundred*, emphasises the importance of diagnosis at the youngest possible age.[39] This report is typical of policy guidelines in higher-income countries but the rhetoric of early diagnosis is also visible in narratives aimed at broader publics. In the USA, five million coffee cups were released by Starbucks in a campaign aimed at raising the profile of autism, put together by the founder of the charity *Autism Speaks* (Figure 2.1).

In an inspired analysis, Anne McGuire argued the Coffee Cup casts the non-normatively developing child as non-valuable and perhaps even non-viable in a market-driven economy (of Starbucks).[40] Certainly, this widely distributed declaration contributed to the cultural recognition of autism as a threat, something to be dreaded and something to be identified (by parents' surveillance) as early as possible so that it can be fixed. And the younger the better. It also invokes a moral obligation for parents to monitor their children, if they wish to qualify as good parents.

The Coffee Cup uses non-gender-specific language. Despite this, it is interpretable as an exhortation to good mothering. The word 'parent' is gender-blind and obliterates oppressive imbalances in the roles and experience of mothers by

'The way I see it' slogan reads:

Every 20 minutes… less time than it will take you to drink your coffee, another child is diagnosed with autism. Learn the early warning signs of autism, and if you're concerned about your child's development, talk to your doctor. Early intervention could make a big difference to your child's future.

Figure 2.1 The Coffee Cup example: Starbucks Autism Awareness campaign.

using the gender-neutral language of 'parenting'.[41] In autism discourse, 'parent' is often a synonym for 'mother', because the vast majority of primary carers of autistic children are mothers. Studies of parental attitudes, parent-rated behaviour scales and parent-mediated interventions often overwhelmingly rely on mothers to participate. Good mothering tacitly means offering as much therapy as possible to the child, at the expense of any other career; Gil Eyal and colleagues[42] refers to this as the 'vocation' of autism parenting.

The threat of autism, this framing of risk, prompts anxiety which demands action. EIB targets the family, in partnership with medical institutions, as the site of early detection and intervention. Intervention may involve one-to-one teaching or up to 40 hours of speech, occupational and Applied Behavioural Analysis (ABA) therapies a week.[42] One US survey suggested parents use as many as 111 different therapies;[43] the mean number used at any one time was seven. The more severe the autism, the more types of treatments parents experimented with. In her work on attention deficit hyperactivity disorder (ADHD), Singh situates mothers' actions in the context of the multiple pressures they feel from so many sources, such as the Coffee Cup campaign.[44] A patriarchal culture that allows mothers to be culpable of their children's behaviour, responsible for monitoring the progress of the child and for 'doing something' if autism is detected, is a driver for the adoption of highly suspect therapies. The neurodiversity movement (Chapter 4) encourages better choices by situating autism as non-problematic – a condition that cannot, and perhaps should not, be 'fixed'.

The moral obligation for mothers to treat, monitor and report to clinicians is not new;[45] through a sociological lens, it is a form of surveillance medicine.[46, 47] Surveillance medicine, the screening, monitoring and establishment of early risk factors, involves monitoring across a whole population, including healthy people.[46] Sociologists such as Ulrich Beck have pointed to a 'politics of anxiety' in the risk society.[48] David Armstrong writes about how infants were the first population to be scrutinised and surveyed for potential risks to normal childhood, such as being of a height and weight that fall outside statistical norms.[46]

Concepts of surveillance draw on Michel Foucault's work, particularly his book *Discipline and Punish*,[49] in which he describes how people are monitored, understood and regulated via institutions, which for babies include nurseries, research institutes, health visits and baby clinics. Foucault describes how people are first trained and observed in institutional settings to produce knowledge about disciplinary norms (for example, the observation of babies in maternity hospitals that produces knowledge about paediatrics, or the knowledge production of baby-sibs studies) and subsequently populations become monitored and subject to regulatory controls. Screening and surveillance therefore promote framing and recognition of differences as problems that were formerly not part of a medical remit. Hence, for good or ill, surveillance fosters medicalisation. The community is encouraged to monitor others in the community, providing normative standards of behaviour. This community policing and neighbourly surveillance were heightened during the 2020 Covid-19 lockdown to maintain social and behavioural norms.

Foucault wrote about the historical steps from a past model of external monitoring and top-down surveillance by powerful actors in the establishment, such as monarchs and lawyers, to community surveillance that provides a net-like power structure in which everyone is responsible for upholding normal behaviour, to the inculcation of internalised self-surveillance, the internalisation of bio-power, so that one comes to 'subjectivise' oneself and discipline one's own body.[49] Mothers' internalisation of vigilance and responsibility for the monitoring of their child seem to be an example of a relational form of bio-power.

In the case of the Coffee Cup, an exhortation for parents to perform surveillance of their child invokes anxiety, with autism described like a threatening disease. The Coffee Cup therefore promotes both pathologisation and vigilance and invokes autism as an object in itself, distant and removed from the individual person, meaning a person may become alienated from it.[50] For Foucault, a condition such as autism is objectified or 'spatialised' by its description as an entity that exists independently of the person (in texts and on coffee cups, etc.). Diagnosis locates autism in a second space, the brain, but autism also requires a third space, the social realm, because it is rendered in interaction. According to Foucault, 'truth' is produced through these levels of spatialisation, exercised by the professional gaze.[51] Once objectified, autism (or any condition) is subject to discipline, and through its control, subjection leads to the subjectification of people who are diagnosed. Although young children may not be able to resist this, adults can – a topic I will return to in Chapter 4. But Foucault was a master of the rhetorical device; others see power dynamics very differently, with less sinister overtones.

A similar rhetorical device to that of the Coffee Cup (risk, threat, requiring action) appears in most medical funding applications that try to identify early signs of autism. Research into either biomarkers or behavioural markers in infancy usually starts with a statement about autism's terrible impact on personal outcomes, families and the economy. Autism is often positioned as an object that is thoroughly bad news, the threat of which provokes anxiety and should be eliminated as early as possible.

Selective interpretation of data justifies the use of language to back up the EIB story, such as Green and colleagues' study of intervention for at-risk babies in which parents delivered the intervention.[52] Results were described as 'encouraging' despite there being no significant improvement in the primary outcome (attentiveness to parent); indeed, a few babies had a worse outcome. The abstract describes first how 'point estimates suggest the intervention increased the primary outcome of infant attentiveness', although qualifies this as 'including possibilities ranging from a small negative treatment effect to a strongly positive treatment effect' (actually it had a non-significant effect). The positioning and wording of reporting, in this and other literature, bolster the EIB narrative by accentuating the positive and diminishing the negative of EIB. Green and colleagues correctly reported the possibility of a negative outcome but the results were nevertheless framed as 'exciting' in the promise of intervention research.

Another example is a research paper, published in the journal *Autism*, that analysed the socio-demographic and child-based factors that predict late

diagnosis.[26] The discussion describes how children are at risk of late diagnosis (after five years old): 'our understanding of "*red flags*" for *missed* diagnosis, that is early characteristics for children *at risk* of receiving a late diagnosis' (my italics). The phrase 'red flags' indicates autism is something that should raise an alarm and being 'at risk' of receiving a late diagnosis is troubling.

The Coffee Cup, and other forms of the EIB narrative, exhorts parents (specifically mothers) to perform surveillance and early childhood monitoring, to report proto-autism behaviours and, if possible, to intervene early. This surely leads to more early referrals and ultimately more diagnoses, contributing, perhaps in a small way, to autism's rise.

Caveats to EIB

There is a lack of evidence that diagnosis is stable at younger ages.[13] At very young ages, it is difficult to distinguish an autistic from an allistic (non-autistic) child, to distinguish a toddler who is not speaking because they may continue to display traits of autism later in life from a toddler who is a slow developer and will catch up. Some children grow out of autistic traits: 30% of children who are given a diagnosis at two years old no longer meet the criteria for an autism spectrum disorder (ASD) diagnosis at four.[53]

There is more uncertainty about future trajectories when screening procedures for autism begin before the child is two.[54] Our work followed the trajectories of two groups of children from two years old to 12; both groups were measured with comparably severe autistic-type traits at age two. The children in one group received an autism diagnosis, while those in the other did not.[55, 56] At adolescence, the children without an autism diagnosis were better on a range of outcomes. In other words, some pre-school-age children who have autistic traits can improve to sub-clinical levels without having ever been diagnosed or treated. In these cases, 'wait and see' may indeed be the best strategy.

To me, our work underlined that the human child is born in an immature state and learns adaptive behaviours as they grow. Many behaviours characteristic of developmental disorders are noticeable in all younger children: hand flapping, hyperactivity, inattention and motor difficulties are all common in toddlerhood. Resolving, at a very early stage, who has a lifelong impairment (and what impairment) and who will catch up is extremely difficult. In medical parlance, the specificity of these early signs in predicting autism may be very low, with many false positives. In a prospective Danish cohort of more than 75,000 children, in infancy the signs that distinguished autism from intellectual disability were unclear and at 18 months old, the positive predictive values (the probability that subjects with a positive test truly have autism) were below 10% for both individual predictors and aggregated risk scores.[57]

In addition, as children grow up the extent of autistic behaviours tends to diminish.[58] The age effect is illustrated by the seasonal influence on ADHD diagnosis. Summer-born children are more likely to be diagnosed with ADHD; a systematic review showed ADHD is consistently diagnosed more often in children

who are young for their school year (which starts in the autumn in the UK),[59] not because they have more ADHD but simply because, relative to their peers, younger children display more behavioural characteristics of ADHD. Taking a developmental perspective therefore throws up challenges to the current recommendation for the reduction of age of diagnosis of autism to very young children.

Another counter to EIB is that diagnosis is not a neutral process of identification but shapes how others react to the baby. Given a specific childhood diagnosis, the people around the child (parents, teachers and clinicians), tend to interpret the child's behaviour in the diagnostic frame.[60] This may lead to an expectancy bias, in the classroom for example, that negatively affects outcomes.[61–63] Thus, very early labelling is problematic even if you consider a young baby either categorically has autism or does not, which is debatable. If the diagnosis is a false positive, those around the child might look at them through an autism lens; could this not negatively affect their trajectory?

Advocates of early diagnosis, on the other hand, see early identification as a crucial step to enable access to support and accommodations that benefit all children; diagnosis opens the gateways to intervention.[64] Autism can certainly act as an explanatory frame for differences in a child's biological and psychological make-up, which can radically improve the functioning of the family. As we have seen, autism researchers have emphasised the critical importance of intervening early in autistic children's lives to give them the best chance of meaningful communication.

A final caveat is that, despite the overwhelming call for early intervention, systematic reviews suggest research into early interventions is of poor quality and the effectiveness of early intervention is not proven for children with autism.[12] The rhetoric around early identification is widespread, and therefore should be underpinned by a rigorous evidence base. In fact, randomised controlled trials (RCTs) on early interventions are rare. One systematic review uncovered a replicated finding that many children who receive early intensive intervention, across methodologies, do *not* demonstrate dramatic gains in social, cognitive, adaptive and educational functioning or autism-specific behaviours.[12] A more recent review on the effects of ABA concluded there is weak or very weak evidence that ABA is a useful behavioural treatment for some children with autism and none that it alters core autism symptoms.[65]

The best that can be concluded is that some interventions improve some areas of functioning and sometimes improve cognition, in some young autistic children, some of the time. What is not often acknowledged is that early interventions for autism have high costs both for the children and in terms of parents' financial and time commitment. Programmes involving more than 40 hours of intensive therapy a week may be exhausting for parents (disproportionately mothers) and children alike.[42] The extra parenting work (usually mothering work) is implicitly expected to be done at home, even though a better outcome is not guaranteed. Nor is it currently possible for a clinician to confidently recommend a particular treatment for a particular child. There seems to be a disjunction between the level of actual evidence for the efficacy of early interventions for autism and what

I would term the rhetoric of early intervention and surveillance that designates good mothering.

Biomarkers

'At-risk' status can also be assigned from the evidence of biomarkers: objective, biological, measurable differences. For some conditions, biomarkers are physical attributes such as weight or heart rate; for autism, the biomarkers are usually neurological or genetic differences.

Some researchers use indices of risk or algorithms that calculate from a combination of biomarkers. For example, for ADHD, a genetic risk profile combines a number of genetic markers into an overall at-risk-of-ADHD score, a polygenic risk score.[66] In this way, researchers increase the predictive power of their models and, based on a risk index, can calculate a person's estimated probability of developing a condition. Considering an at-risk group in this way often gives access to larger and younger populations than would be possible if only confirmed cases were considered.

Perhaps the ultimate in baby surveillance is an electronic romper suit that monitors all aspects of the wearer's behaviour for 'warning' signs. In 2015, I interviewed a technology expert with many years' experience of designing computer algorithms to detect mouse movements in the laboratory. He described his company's on-going project to design romper suits to be used in the home to recognise the autism behavioural phenotype and help detect autism.[67] This 'smart' baby suit has sensors woven into the fabric that monitor the baby's heart rate, respiration, mobility and movement against normal parameters and automatically and securely transmit the data to the researchers' lab. The design was commissioned by at-risk-of-autism researchers but perhaps will be rolled out to the general population. Late development and missed milestones will ring 'alarm bells', raise 'red flags' and provide the required 'early warnings'.

For many years, there has been a push to detect biomarkers of autism because, as some argue, a biomarker is considered to be a more objective measure, and potentially a better mechanism of identification, than behavioural clinical assessments[68] which are subjective and dependent on the settings in which they are recorded. Plausible biomarkers for autism are measures such as brain circumference, genetic profile or a particular pattern of activity in the brain during a certain task, normally revealed by magnetic resonance imaging (MRI), but studies have identified many others.[69] Some scientists have advocated the fusing of behavioural definitions with biological, particularly neurological, indicators, for all psychiatric classes.[70] Perhaps, neither is 'better'; just different. Publicising, operationalising or adjusting either definitions or indicators will both influence our understanding of the autism category and alter who is in it. Autism is partly a product of how it is measured and identified.

In medical discourse, ethical arguments regarding 'at-risk' status are often founded on the notion of false positives. Statements about diagnosis and at-risk status use terms such as inaccuracy, misdiagnosis, false positive and validity. In this

language, being named at-risk-of-autism may not *accurately* reflect your status (you may be a *false positive*), leading to *misdiagnosis* and raising questions about the *validity* of the at-risk category. These terms confer notions of 'truth', 'fact' and an 'objectivity' to be striven for. Again, these words assume there is a true fixed autism to be measured against and that conferring at-risk status does not in itself shape how we understand and define autism, how often we refer for autism and how deeming babies at risk could alter their developmental trajectory.

To take an example, let's say a neuro-marker is discovered, for example differences in white-matter tracts,[69] that forms a biomarker to identify autism. The at-risk group of babies thus identified will be a slightly different bunch to the babies identified as at risk by their behaviours, such as head lag. In this hypothetical example, publicising the neuro-work leads to understandings of autism as a neural condition (the white-matter tract difference). Atypical white-matter tract at-risk babies are more likely to be referred and diagnosed. Thus, the net effect of finding biomarkers contributes to what being 'at risk' of autism looks like, and who qualifies as having autism may be very slightly reshaped.

Although biomarker results are frequently described as 'promising', they are not often replicated or applicable to the whole spectrum. However, the search for genetic markers for autism has revealed some very useful markers of rare syndromes, for example Williams syndrome. The genetics of autism are complex, with different genetic sub-profiles that involve multi-faceted interactions with the environment.[71–73] Because what is diagnosable as autism is a slowly moving target, the search for a fixed set of biomarkers against which to compare is like having moving goalposts; it may be better to search for sub-groups across the spectrum. The latest iteration of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) has dropped the distinction between Asperger's disorder and autistic disorder but acknowledges differences within the autism spectrum, which is now stratified by the severity both of social communication impairment and restricted and repetitive patterns of behaviour, and with and without co-occurring intellectual disability. In DSM-5 the autism spectrum is also codified by known genetic conditions, biomarkers, although only a small percentage of cases have known genetic markers.

Despite the move towards sub-grouping, there is still investment in discovering the genetics of autism across the whole spectrum. Some research groups aspire to create a genetic test for autism that could be administered before birth, and some commercial laboratories offer parents a non-invasive pre-natal test they claim can screen for mutations in a range of genes, including some related to autism.[74] This claim has provoked an outraged reaction from the autistic community. In 2005, the autistic activist Meg Evans created the *Autistic Genocide Clock* as part of her *Star Trek* fanfiction website, *Ventura33*. Evans became mobilised after joining the autistic forum, *Aspergia*, and later the chatroom *Aspies for Freedom*, founded by Amy and Gareth Nelson, who also published a declaration that autistic people should be recognised as a minority group.[75] The *Autism Genocide Clock* was a ten-year countdown in the image of a clock; it responded to and resisted a pronouncement in 2005 that genetic research on autism could lead to a genetic test

within ten years. Evans's point was that a pre-natal genetic test for autism could lead to abortions of foetuses that test positive for autism – in her view, a form of genocide. Writing in a collection of stories about autistic activists released as part of our *Exploring Diagnosis* project,[76] she described her timer clock as a reaction to autism discourse that, as she puts it, says 'the world should not have autistic people in it'. Evans took the clock down in 2011.

Certainly, the work towards pre-natal testing positions autism as a suitable rationale for abortion. Presumably such a test would be accompanied by genetic counselling for parents who chose to take it, to support them to decide whether to abort a baby with autism. Having been through such a scenario myself (when I was pregnant, my daughter screened positive for being at risk of Edwards's syndrome; it turned out to be a false positive), I know both how stressful this process can be for parents, and how powerful and potentially life changing the medical concepts can be in practice.

Evans's argument parallels those made by members of the disability rights movement, that pre-natal genetic tests are a form of eugenics, leading towards the elimination of people like them, and that allowing abortion on the grounds of disability is discriminatory.[77] Others argue quality of life is important to consider. Edward's syndrome leaves babies with heart, respiratory, kidney and gastrointestinal conditions, with 87% dying before one year old. The *Autistic Genocide Clock* illustrated the tension between a newer progressive, affirmative model of autism-as-identity and an older model of severe autism with co-morbidity and complications in a medical frame. Evans's strong language has parallels with historic resistance to the elimination of other minority groups.[78]

The twin processes of pushing back age of diagnosis into infancy and defining infants as 'at-risk' may have both contributed to the rise in autism observed in Chapter 1, if in a minor way. Earlier diagnosis contributes directly as a younger cohort is eligible for diagnosis. 'At -risk' status may contribute indirectly through widening 'what counts' as autism. Yet a more seismic shift in diagnostic practice occurred at the life stage covered in the next chapter: childhood.

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