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SCIENTIFIC FORUM: RESPONSE

Validity of early identification and early intervention in autism spectrum disorders: Future directions

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Abstract
The papers on early identification and early intervention for autism spectrum disorders (ASD) in this scientific forum (published in volume 16(1) *International Journal of Speech-Language Pathology*) raise many important points, including describing the substantial progress made to date as well as analyses of current gaps and weaknesses in the existing evidence base. It is humbling to see the collective expertise of the distinguished authors contributing to this scientific forum including interdisciplinary perspectives and it is not surprising that there is ongoing debate on this important topic. In addition to discussing the points raised by these authors, this paper considers the implications of the new diagnostic criteria for ASD and for social communication disorder (SCD) in the Diagnostic and Statistical Manual of Mental Disorders-5 (DSM-5) in the US. Differential diagnosis of ASD and SCD will be paramount in testing early intervention for ASD and the expertise of speech-language pathologists in identifying SCD in infants and toddlers will be a central feature of discovery for both early identification and for early intervention in the decades to come. Finally, a biomedical example on testing early intervention on a spectrum disorder, derived from diabetes, is presented to illustrate both the promise and the pitfalls in testing interventions in the absence of well-validated assessment and intervention paradigms.

Keywords: *Autism spectrum disorder, early identification, early intervention, autism spectrum disorder screening, autism spectrum disorder treatment.*

Introduction

In a recent review appearing in the prestigious *New England Journal of Medicine*, Baker (2013) discussed the implications of the new Diagnostic and Statistical Manual of Mental Disorders–5 (DSM-5) (American Psychiatric Association, 2013) classification system for autism spectrum disorders (ASD). He notes that the new guidelines are designed to yield more accurate diagnoses, and that ASD is now viewed as a dyad of conditions that include restricted interests and stereotyped behaviours AND social communication deficits. Baker indicates that the revised criteria are, in part, a response of the growth in the autism

> … umbrella encompassing a tremendous range of patients, varying greatly in cognitive and social abilities as well as associated genetic or neurologic conditions. Determining what kinds of therapy work, and how much is needed, has become very challenging (Baker, 2013, p. 1091).

Importantly, for speech-language pathologists, he also points out that children who do not display the second feature of ASD, strict adherence to routines or stereotypes, should be diagnosed with social communication disorder. Asperger syndrome and Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS) have been removed from the DSM-5, so that socially awkward children should NOT be automatically diagnosed with ASD. The new APA DSM-5 guidelines have profound implications for ASD and studies of early identification and early intervention.

Differential diagnosis of social communication disorder and autism spectrum disorders: Implications for early identification and early intervention

Models of language and language disorders have long included the social use of language (pragmatics) as a key feature (e.g., Bishop & Norbury, 2002; Camarata, 1991; Leonard, 2000). That is, fundamental models of language usually include phonology (speech sounds), morphology (word endings and affixes), semantics (meaning), syntax (rules for combining words into sentences), *and* the social aspects of communication as a core element (see Prutting &

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Kirchner, 1987). Similarly, Bloom and Lahey's (1978) foundational discussion of language disorders viewed language as the integration of content, form, and use, with the latter construct referring to the social aspects of language. Moreover, they also included examples of disruptions in use (social skills) as a typology of language disorder.

From an even broader perspective, linguistics has long viewed the social aspects of communication a natural part of language. For example, Bloomfield's (1933) classic text on language includes a chapter on "The use of language" and another on "Speech-communities" describing the social milieu as a part of language study. The point herein is that social communication is inarguably a *language* construct and social communication disorder (pragmatic language disorder) is not *necessarily* a form of autism spectrum disorder.

That is, although a diagnosis of autism spectrum disorder naturally includes social communication disorder, additional symptomology outside language domains is required. As the DSM-5 indicates "Autism spectrum disorder is the primary diagnostic consideration for individuals presenting with social communication deficits. The two disorders can be differentiated by the presence of autism spectrum disorder over restricted repetitive patterns of behaviour, interests, or activities and their absence in social (pragmatic) communication disorder" (American Psychiatric Association, 2013, p. 49). As with differential diagnosis of autism spectrum disorder, this is generally much simpler in older pre-school and school-age children, and the DSM-5 indicates that social communication disorder is usually diagnosed at ~ 4 years of age. It is likely that of the children previously identified as on the autism spectrum with PDD-NOS or Asperger syndrome will likely be more accurately captured using the social communication disorder diagnosis. However, the overall accuracy and stability for identifying ASD in older children will likely remain high, even with the addition of social communication disorder to the DSM-5.

However, the challenge under consideration in the current scientific form is generating a credible evidence base in support of early identification and early intervention for ASD (Brignell, Morgan, Woolfenden, & Williams, 2014; Camarata, 2014; Charman, 2014; Crais & Watson, 2014; Koegel, Koegel, Ashbaugh, & Bradshaw, 2014; Samms-Vaughan, 2014; Taylor, Maybery, & Whitehouse, 2014; Trembath & Vivanti, 2014; Volkmar & Reichow, 2014; Webb, Jones, Kelly, & Dawson, 2014). Moreover, the more specific symptomology required for an ASD diagnosis coupled with refinement in the diagnosis of social communication disorders will likely have a much greater impact on early identification and, consequently, early intervention as well. Diagnosis of an ASD requires not only disruptions in social skills, but also restricted interests and/or stereotypies, which is a salient and useful diagnostic marker in 4- and 5-year-olds, and, in many cases even 3-year-olds. However, restricted interests can be much more difficult to identify in infants and toddlers, and Stone, Lee, Ashford, Brissie, Hepburn, Coonrod, et al. (1999) reported that deficits in language abilities and social skills are the most salient diagnostic markers in young children. As Crais and Watson (2014) note, restricted and repetitive behaviours (RRBs) are a key marker distinguishing ASD from other disabilities, especially other forms of DD. There can be no doubt that these RRBs are indeed readily identifiable and salient, even in toddlers with classic ASD (level 3 in the DSM-5). However, RRBs are more difficult to differentiate in toddlers with more marginal features of ASD. Also, because of this, it is not surprising that identification or assigning risk for autism spectrum disorder in infants and toddlers has relied heavily upon markers for social interaction and language delay. Going forward, social deficits and language delay should *not* be sufficient for an ASD identification, nor should such cases be employed to test the effects of early intervention, as least as these are *exclusive* to ASD.

If one accepts the premise that ASD includes *at least* the dyad of social skills symptoms and restricted interests/stereotypy, as Brignell et al. (2014) point out, researchers will be challenged to develop both behavioural and/or biological markers capturing these domains *simultaneously* in infants and toddlers. Taylor et al. (2014) are inarguably correct in calling for better integration of biomarkers with the behavioural data. Perhaps, recent advances in imaging neural centres associated with stereotypy and in imaging reward centres showing potential differences in ASD will yield important contributory biomarkers to augment the behavioural data employed to identify ASD in infants and toddlers. However, the current diagnosis is founded on behavioural observations in semi-structured assessment settings such as in the toddler module of the Autism Diagnostic Observation Schedule (ADOS-2, Lord, Rutter, DiLavore, Risi, Gotham, & Bishop, 2012).

The authors of the ADOS-2 are properly cautious in describing the limits and pitfalls of assessing toddlers (Lord et al., 2012). They note that, although the lower limit of the module is 12 months, the child must be walking and have sufficient motor capability to participate in the play activities that form the basis of the assessment. In addition, they note limitations on employing the diagnostic framework to children with hearing loss or other primary sensory (e.g., blindness) or motor (e.g., cerebral palsy) deficits. One could argue that the ADOS-2 represents the current state-of-the-art for generating risk estimates in ASD in toddlers. This does not mean that severe and salient deficits in social communication that cannot be reliably detected in children are not good candidates for assessment with the ADOS-2, rather, these diagnoses and judgements of risk for developing ASD rest even more heavily upon subjective

interpretation. Stated simply, this translates into more variability in the observations, which makes it that much more difficult to conduct credible studies examining the effects of early intervention and the reliability of early identification. Notice that the overwhelming majority of the high-risk symptoms and target behaviours in the review by Webb et al. (2014) relate to the social domain of ASD. Future studies are needed to determine what extent these markers and target behaviours overlap with social communication disorders.

Confirmatory diagnosis vs differential diagnosis of ASD

One of the foundational challenges in testing the validity of early identification and, ultimately, early intervention is current practice in the US wherein primary diagnostic responsibility for ASD rests with autism specialists who are very well trained indeed in autism and related ASD conditions, but who may have less training and experience with language disorder, social medications disorder, intellectual disabilities, or other conditions that share diagnostic features with ASD, particularly in early childhood. As Crais and Watson (2014) discuss, in infants and toddlers, current measures are reasonably accurate for detecting some form of developmental delay, even if it is not ASD. However, because of this, in practice, it may be difficult to conduct a *differential* diagnosis in these young children, especially for marginal cases. Given the challenge for differential diagnosis and the research and community factors that may bias questionable or marginal cases, it is not surprising that clinicians may watch for symptoms that *confirm* the presence of an ASD.

Conducting confirmatory diagnoses of this nature will yield higher incidence rates for ASD, while also confounding attempts to evaluate the effectiveness of early intervention because diverse disability typologies will be conflated with ASD. For example, a preschooler in the Vanderbilt University speech-language pathology clinic (Bill Wilkerson Center) had an older brother with classic autism and so was evaluated as an ASD sibling at high risk. The evaluation completed by the autism specialist confirmed that this younger sibling should be placed on the spectrum. To be fair, I did not assess this child until ~ 6 months after the ASD specialist had completed their evaluation, so it is possible that there were significant autism symptoms present in that initial evaluation. I also note here that this child did not receive intervention after the initial ASD diagnosis due to waitlist constraints on receiving services. So the differences in the previous confirmatory ASD assessment and the subsequent differential diagnosis assessment could not be attributed to intervention. The differential diagnosis indicated that this younger sibling had a moderate-to-severe speech sound disorder that did not meet the diagnostic criteria for ASD. He was enrolled in a speech-language pathology intervention that did not include intervention for ASD symptoms and reached normal functioning for speech in ~ 18 months. Now, this is not conclusive evidence that the early intervention for speech sound disorder was effective, as proving this is always problematic. However, using a single subject design with multiple baselines on various speech sounds indicated that there was a functional relationship between the treatment on specific speech targets and improvements on these targets.

If this child had been enrolled in a follow-up study for ASD following the original confirmatory diagnosis, he would have been a "hit" for recovery and an optimal outcome. After all, by the time he started school at the age of 5 all symptoms of autism had disappeared. Also, there can be no doubt that the autism expert conducting the original ASD evaluation was well qualified and very experienced. However, because this child was difficult to test and would tantrum at times, even when participating in the differential diagnosis, it is understandable that an ASD was confirmed. However, subsequent assessment and treatment on speech sound disorders only cast reasonable doubt on whether an ASD diagnosis was appropriate in this case. A strong recommendation going forward is that all children enrolled in early identification and early intervention studies be assessed using differential diagnosis rather than confirmatory diagnosis for ASD.

It is important to bear in mind that there are numerous educational, financial, and societal factors coalescing to support confirmatory diagnostic practices. In the US, higher levels of support are available for children eligible for ASD services relative to other eligibility typologies. Also, state guidelines often are fairly broad in terms of establishing ASD eligibility. Similarly, there are higher reimbursement rates for insurance coverage or to even get insurance coverage requires ASD eligibility. Again, there's a completely ethical but understandably high motivation on the part of the clinician and also on the part of the family to confirm an ASD. Also, families that have an ASD diagnosis may be eligible for government assistance in the form of disability payments. The goal of this paper is not to criticize these practices and terms of service delivery, insurance eligibility, or disability subsidies. Rather, from a scientific viewpoint, it is important that differential diagnosis be completed in fair studies of early intervention and early identification of ASD.

Evaluating early intervention in autism spectrum disorders: A biomedical example

In order to illustrate the challenges for evaluating early intervention in a spectrum disorder it may be useful to draw a parallel from the biomedical literature. Perhaps the most lucid example can be drawn from diabetes, which is indeed a spectrum disorder.

This medical condition includes "pre-diabetes", which is defined as fasting blood glucose levels that are elevated relative to norm-referenced values, but below the diagnostic cut-off value for a diabetes classification. The "diabetes spectrum" also includes people with relatively stable but chronically elevated blood glucose readings and, in a parallel to classic autism, people whose blood glucose readings vary but are consistently dangerously high. This latter group, which represents ~ 5–10% of the diabetes spectrum population in the US, has full-blown "type I" diabetes, which is also known as juvenile diabetes. The people who have chronically elevated blood glucose levels but not to the degree seen in "classic" diabetes are classified as "type II" or sometimes "adult onset" on the diabetes spectrum, and those with elevated, but not clinical levels of blood glucose would be considered "at high risk" for developing either type II or type I diabetes.

For the purposes of this example, let's now assume that an early intervention program is implemented in toddlers who are "at risk" for being on the diabetes spectrum. This group would include pre-diabetics, latent type II diabetics, and some children who have already manifested type I symptomology and latent type I diabetics who will go on to develop this form of classic diabetes later on in childhood or early adulthood. This latter group could be considered as a parallel to "child disintegrative disorder" in the autism spectrum. Because more than 80% of individuals in the US on the "diabetes spectrum" symptoms are associated with two primary markers: obesity and a secondary lifestyle, and because a large proportion of the people on the diabetes spectrum show improved blood glucose readings if an intervention for diet and exercise is adopted, there is no question that an early intervention for diabetes spectrum that included parent education and behaviour modification for nutrition and for increasing exercise would be effective both in improving outcomes for those already on the diabetes spectrum and for reducing the incidence in "at risk" individuals.

However, alas, there is no glucose meter for identifying ASD, so we are left with behavioural proxies for high blood sugar when attempting to diagnose autism spectrum disorder. To be sure, there has been progress on both fronts. As seen in the review by Taylor et al. (2014) in this scientific forum, Herculean efforts are underway among neuroscientists and geneticists to develop accurate biomarkers for identifying autism spectrum disorder. Indeed, in the US alone, tens of millions if not hundreds of millions of research dollars have been invested in this quest. As the Taylor et al. (2014) review reveals, there are a number of promising leads, but it does appear that developing a biomedical "glucose meter" remains somewhere off into a bit of a murky future discovery path. Stated simply, current researchers attempting to evaluate the effectiveness of early intervention are left with behavioural "glucose meters" for identifying and measuring progress in autism spectrum disorders.

On this latter front, the Volkmar and Reichow (2014) review and the Charman (2014) review in this scientific forum clearly indicates that much progress has been made on more valid and reliable behavioural "glucose meters" for examining the autism spectrum. Volkmar and Reichow (2014) also remind us of the difficulties encountered along the way, as well as chronicling the discovery path for diagnosis of autism as well as the ongoing debates about what should, and what should not be included. Also, as these outstanding papers indicate, there has been substantial progress on defining ASD as a construct and on developing more refined behavioural measures for diagnosing autism spectrum disorder.

Perhaps the most salient example of this progress is the recently released "toddler module" of the ADOS-2 (Lord et al., 2012), and there can be no doubt that this instrument represents a significant advance in the ability to diagnose ASD in young children. Indeed, reading Volkmar and Reichow (2014) was a poignant reminder that, prior to the development of the ADOS and other instruments such as the Screening Test for Autism at Three (STAT, Stone et al., 2004), an ASD diagnosis was founded *solely* on clinical judgement. Of course, clinical judgement remains the gold standard for diagnosing ASD, but the advent of instruments such as the ADOS and the STAT have provided a standard context for gathering information on young children including toddlers and pre-schoolers upon which to form a clinical judgement. This alone represents a significantly advance in "glucose meters" for behavioural diagnosis of ASD.

However, even instruments such as the toddler module of the ADOS-2 et al. cannot be employed to evaluate the accuracy of early identification and the children described in the Webb et al. (2014) review in this scientific forum. That is, the ADOS-2 toddler module requires a child to be ambulatory and display sufficient motor development to participate in the toys and materials included in the instrument. Although the manual indicates that the lower age limit is 12 months, practically speaking, because even many typical children do not begin walking until after 12 months, and children with disabilities may extend gross and fine motor development much further than this, including children who do not have ASD, the lower limit is more likely to be 20 months or even older in many children. Although the ADOS-2 is a useful instrument, it will no doubt undergo considerable refinement in future years as larger scale validity studies are conducted.

In order to scientifically test the theoretical perspectives described in Webb et al. (2014), the first, and necessary, step in the discovery path would be to complete longitudinal studies with relatively large sample sizes so that the long-term diagnostic markers for ASD can be identified and measurements

with reasonable sensitivity and specificity can be developed. In this case, one could argue that long-term means following the children until 36 or even 42 months of age. This upper limit is suggested because (a) there are a plethora of studies showing that intervention delivered after this age can be effective; (b) although spontaneous recovery could still occur, the proportion of the sample doing so would likely be much lower; and (c) differential diagnosis for other conditions such as global intellectual deficits, language disorder, speech sound disorder, and social communication disorder can be completed with much higher sensitivity and specificity.

Returning to the diabetes spectrum parallel, in addition to having a glucose meter to validate identification, the same marker can be used to evaluate progress in intervention studies. Even better for the diabetes spectrum, there is another global biomedical measure, glycosated haemoglobin (Hb1AC) that essentially summarizes the average amount of glucose in the blood over the previous 6 months. This can be used to chart progress as well. Alas, there are literally no parallels for charting growth in the autism spectrum. The ADOS-2 is not well suited to measuring progress, so clinician scientists must rely upon standardized measures and an associated domain such as cognitive abilities and language abilities as well as qualitative measures of social skills and repetitive behaviours.

However, these are not unique to ASD and, thus, cannot directly test whether early intervention is effective for ASD as a condition rather than language disorder or other non-ASD disability. The Keogel et al. (2014) review correctly points out that a single subject design is very well suited to charting learning these individual behaviours, and there is no doubt that the literature is replete with plenty of validated intervention strategies for individual children with ASD at all disability levels. Also, it is inarguable, as Koegel et al. (2014) report, than many developmental correlates of ASD including language, social skills, disruptive behaviour, and pre-linguistic skills can be improved in individual children using these techniques. However, single subject designs, by nature, are not well suited to detecting latent change (see Yoder and Symons, 2010), in part because relatively rapid changes in the target behaviour are needed for scientists to detect functional relationships between the intervention and the dependent measures. Also, even in relatively severe disabilities, ambient growth rates, albeit shallow, would result in unstable baselines that extended many months or even years.

As a further extension of the diabetes spectrum example, consider the following theoretical design to address early intervention *without* access to glucose meters or measures of glycosated haemoglobin. That is, using only risk factors and behavioural correlates of diabetes spectrum disorder. As mentioned above, the highest risk factor for being on the diabetes spectrum is 2-fold diet and exercise. It is also possible using observations to determine not only which children, but also which families are at high risk for developing diabetes. This is a parallel to the autism siblings logic, as described by Webb et al. (2014), that families with older siblings and/or parents documented on the diabetes spectrum could be enrolled in the intervention. Because so many people on the diabetes spectrum benefit from a diet and exercise program, the early intervention delivered would be based on educating parents/parent training on how to properly feed their children in accord with diabetic diet guidelines, and would include weekly or even more intensive home-based exercise.

In direct accord with the Koegel et al. (2014) discussion for ASD, there is a high face validity to this approach and there is no doubt that this early intervention would have a positive impact for many of the children either on or at risk for diabetes spectrum disorder. However, an evidence-based review of this approach to testing the effectiveness for early intervention in diabetes spectrum disorder would yield, at best, a "weak" rating, because the design is confounded. That is, such a study or series of studies would not be conclusive because the proportion of at risk infants who actually ended up developing diabetes, or even the broader diabetes spectrum disorder, would be unknown. Without these data, it is simply not possible to directly test whether the disease was prevented or improved significantly. Even comparison treatment designs, without a representative control group, are vulnerable to diagnostic drift and uncontrolled variance in development. Perhaps this contributed to the Warren, McPheeters, Sathe, Foss-Feig, Glasser, and Veenstra-VanderWeele (2011) review failure to find credible evidence for early intervention in ASD.

Indeed, there are parallels in the ASD reviews by Webb et al. (2014) and Koegel et al. (2014) to diet and exercise approach to early intervention in diabetes spectrum disorder, because the techniques described in these reviews are indeed effective, but are not exclusive to treating ASD. That is, parent–child interaction and direct instruction techniques for teaching language and improving behaviour are effective across many disability typologies and can accelerate learning in typical children as well. For example, in the word learning arena, the groundbreaking work by Keith E. Nelson and his colleagues (see Nelson, 1989 for a review and see Camarata, Nelson & Camarata, 1994) has demonstrated over the last 40 years that responding to child initiations with linguistic models will dramatically accelerate linguistic development in typically-developing as well as children with a range of disabilities.

Koegel et al. (2014) have shown that responding to initiations is a highly effective approach, with appropriate adaptations, in autism (see Koegel, O'Dell, & Dunlap, 1988). Moreover, one cannot under-estimate the importance of these discoveries; prior to the development of the Natural Language Paradigm (Koegel, O'Dell, & Koegel, 1987), treatment for autism was

largely restricted to discrete trials approaches, so Koegel et al.'s work was literally a breakthrough for autism intervention. Both the original "natural language paradigm" and the more recent pivotal response training have reinforcing attempts as a core intervention strategy (Koegel & Koegel, 2006). The hundreds of studies they have published leave no doubt that this is indeed effective for teaching children with ASD a wide variety of skills, including language. However, from an early intervention perspective, this treatment is not differentially effective for ASD. The challenge is that nearly any child receiving this intervention, regardless of whether they had ASD, would likely show differential growth in response to the intervention. However, the relative resistance to intervention for children with classic autism is problematic when analysing the current evidence base on whether early intervention for ASD is effective.

In the diabetes spectrum parallel example, this is akin to the problem regarding diet and exercise as an early intervention and full-blown, type I diabetes. This is problematic because there are different mechanisms for classic diabetes and other forms on the diabetes spectrum. So the problem is 2-fold: (1) nearly everybody in the population would benefit from a diet and exercise early intervention and (2) the unique biological mechanisms for classic diabetes means that diet and exercise are not an effective intervention. That is, although pre-diabetes in many forms of type II diabetes will be significantly improved or even "cured" by diet and exercise, classic, type I diabetes requires insulin injections.

Now, let's follow this example to its logical conclusion: one could expand the original definition of severe, classic diabetes, and expand it to the broader diabetes spectrum. One could then conduct a generic, non-specific early intervention consisting of diet and exercise. Using single-subject design one could plausibly show improvements in individual children, and using group designs with random assignment could show that some, and perhaps even a significant proportion of people on the diabetes spectrum, were significantly improved or even "cured" using this early intervention. Indeed, because more than 85% of the diabetes spectrum includes people who are not type I or "classic" diabetics, there would be fairly convincing evidence that the early intervention was in fact effective. However, this design in these results would obscure the fact that core or classic diabetes did not have positive outcomes and there would be many people who showed gains in the proxy measures who do not have diabetes and who never would have gone on to develop diabetes. Worse, the classic type I diabetics would continue to be morbid at precisely the same rate with and without early intervention, Thankfully, in classic autism, the long-term morbidity is not death, as is the case in type I diabetics. Nonetheless, ASD is a very serious condition so that studies must be completed ethically.

I hypothesize that, as in the diabetes spectrum example, classic autism has an altogether different mechanism from a biomedical perspective than the broader autism spectrum, and that current researchers are in precisely the same situation as physician-scientists more than 100 years ago before Banting, Best, Collip, Campbell, and Fletcher (1922) discovered that a digestive hormone, insulin, secreted by the pancreas is an effective treatment for classic, type I diabetes. Ironically, the broader diabetes spectrum was born when Himsworth discovered (in 1935) that there were diabetic patients who were not responsive to insulin treatment. These type II (insulin resistant) patients now form the overwhelming majority of diabetic patients in the US.

It is noteworthy that, prior to the discovery of insulin and subsequently of type II diabetes, there were a plethora of questionable and even quack treatments for diabetes. Several of these are chronicled in Sattley (2008), and some are eerily similar to fringe autism treatments, such as highly restricted diets, special substances (such as broken red coral and sweet almonds), and activities such as horseback riding. If a patient suffered from type II diabetes and the diets, no matter how strange, were associated with weight loss, and the exercise, such as horseback riding, generated at least some cardiovascular benefits, then these rather strange "cures" may have had some indirect benefits. However, they would have been useless for "classic" type I diabetes. As with the "diabetes spectrum", credible tests for the effects of early intervention must be constructed in such a manner that the specific treatment can be tested, and the particular symptoms within the spectrum can be fairly isolated, especially if, as with diabetes, multiple biomedical mechanisms are converging to create the autism spectrum.

This analogy directly relates to the brilliant review by Trembath and Vivanti (2014) on individual differences. Essentially, everyone on the diabetes spectrum ultimately must work with their physicians and dieticians to craft an individualized treatment plan for the kind and severity of diabetes they have. Some only need to lose weight and get a bit more active, others will require more restricted diets, others require all this plus oral medication, and some will require insulin shots. Trembath and Vivanti (2014) are correct in arguing that a key focus of research on the ASD must be on *what* procedure works for *whom* and *why* it works. This is precisely the approach that has yielded such dramatic improvements for *everyone* on the "diabetes spectrum", and in fact was the research framework that led to the discovery of type II diabetes as distinct from type I.

Lessons from the secretin trials

Secretin is a digestive hormone that was originally reported as an effective treatment for ASD (see Perry

& Bangaru, 1998). Also, indeed, there were reports of individual children with ASD demonstrating positive effects when treated with secretin (e.g., Horvath, Stefanatos, Sokolski, Wachtel, Nabors, & Tildon, 1998). As an aside, the parallel to the diabetes spectrum is intriguing because secretin was actually the first hormone discovered and, like insulin, is a digestive hormone. Regardless, Krishnaswami, McPheeters, and Veenstra-Vanderweele (2011) published a comprehensive review of the literature on secretin trials for children with ASD in the American Journal *Pediatrics*. As most clinicians and clinician scientists are aware, it was ultimately clear that clinical trials of secretin indicated that it is not an effective treatment for ASD. This conclusion was based upon multiple studies that compared secretin to a placebo. Importantly, the scientific approach and the nature of the treatment permitted a double-blind paradigm: neither the clinician administering the treatment nor the patient was aware of which treatment had been delivered. However, although the failure of secretin to treat ASD is now widely known, another outcome from these studies has received far less publicity. Specifically, there were quite a number of studies that yielded improvements in the outcome measures for ASD symptomology for secretin and for placebo. That is, there were improvements for the placebo condition!

To be sure, significant placebo effects are hardly unique to ASD. However, the Krishnaswami et al. (2011) review illustrates that the promising discovery paths and research findings for early intervention described by Koegel et al. (2014) and Webb et al. (2014) require more extensive testing and validation studies that adequately control for potential confounds and can be compared to "placebo" type treatment effects.

Summary and conclusions

There has indeed been significant progress in defining and diagnosing ASD at ever younger ages, which is, of course, a pre-requisite to testing whether early intervention is effective. However, it seems that the call for early intervention continues to push the limits of the ability to accurately diagnose ASD. For example, the recently released toddler module for the ADOS-2 (Lord et al., 2012) has a published floor age of 12 months, but practically speaking is likely to be more accurate in 20-month-old's or even older children. Yet, as in the Webb et al. (2014) paper, there are calls for testing or delivering early intervention in infants who are at "high risk" for ASD. As this review shows, the current state-of-the-art for identifying ASD in infants and toddlers makes it problematic to fairly test the effects of early intervention. It is clear that the last several decades have seen important refinements in treatments designed to improve the symptoms for ASD. However, these interventions are not differentially effective for ASD. So, as with the diet and exercise early intervention for diabetes spectrum example illustrates, additional discovery is needed to yield a stronger evidence base for early identification in ASD. Moreover, everyone helping children with ASD should recognize the important contributions of the groups at the University of Washington (Webb et al., 2014), University of California, Santa Barbara (Koegel et al., 2014), and the University of North Carolina (Crais & Watson, 2014), and many other clinician scientists for the decades long leadership in developing and testing new interventions for ASD. These centres have inarguably established that people with ASD can learn when effective intervention is provided; families of children with ASD and researchers building on these discoveries owe these labs a debt of gratitude for what has been accomplished. The next step is to conduct studies that can plausibly yield strong, minimally confounded tests of whether early intervention is differentially effective in toddlers and pre-schoolers with ASD.

Finally, the inclusion of social communication disorder in the DSM-5 presents a unique opportunity for speech-language pathologists to play a key role in the earlier intervention and early identification research going forward. Indeed, as this review describes, speech-language pathologists are perhaps in a unique position to perform differential diagnosis of ASD and social communication disorder. Such differential diagnosis will represent a key advance in our ability to test the intervention because this practice should replace the current predominant "confirmatory" diagnosis model in ASD. However, this opportunity is not guaranteed. It is sobering to know that both PDD-NOS and Asperger syndrome have been dropped from the DSM-5 due to diagnostic instability. There are no guarantees that social communication disorder will survive into the DSM-6 or even the DSM-5TR if our field does not take the initiative to develop instruments and procedures for accurate differential diagnosis of ASD and social communication disorder and to conduct intervention studies to improve social skills in this condition. It behooves us to refine current paradigms for identifying and treating social communication disorders and to substantially expand the literature on this important diagnostic typology.

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