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Letter To The Editor

The importance of getting Kanner's account right in debates over first descriptions of autism

Much of the debate over who first described autism has centred around the question of whether Leo Kanner was influenced by Hans Asperger (other suggested contenders for first description include Louise J Despert, Ida Frye and Suchawara). Kanner, in Baltimore, published his account of infantile autism in 1943. This was earlier than Asperger, in Austria, who published on autistic psychopathy in 1944. However, some have suggested that Kanner was influenced by a more basic account which Asperger published in 1938 (Chown 2010, p.2263; Lyons & Fitzgerald 2007, p.2022). In a slight twist of the tale, Chown and Hughes (2016) describe how Steve Silberman (2015), in his recent book *Neurotribes*, argues that Kanner would have known about Asperger's work. Silberman describes how Asperger's chief diagnostician, Georg Frankl, actually ended up working with Kanner.¹ With Kanner's help Frankl emigrated from Nazi Austria to the United States and ended up being employed in Kanner's clinic and therefore Kanner knew about Asperger's notion. Silberman suggests Kanner ignored Asperger's account or even suppressed it rather than adopting it. Following this argument, Chown and Hughes claim Kanner promoted his own account and consequently from the 1940s to the 1970s “Kanner's, in some cases inaccurate, original account of autism was far more influential than Asperger's (and the only game in town for many years)” (2016 p.2271). Chown and Hughes believe this had significant ramifications. They write that “[w]e believe that if Asperger's work and beliefs had been in circulation, instead of Kanner's, or even alongside, and in fair competition with Kanner's theories, it is probable that the lives of very many people with autism would have been improved” (2016, p.2271-2272). Specifically, “it would have been recognised much earlier that autism was not ‘just’ a condition of childhood, or one that affected lower-functioning children only, but could be seen in people of all ages, and at all levels of intellectual ability” (2016, p.2273).

However, I suggest that these descriptions of Kanner's account of autism are significantly inaccurate. It is, unfortunately, unclear what evidence Chown and Hughes rely upon when making these claims about Kanner since they only cite one of his papers. It seems possible that they are repeating the claims found in *Neurotribes*. If so, this would be problematic because essay length reviews of *Neurotribes* have suggested that Silberman's historical account of Kanner is flawed (Fellowes 2017; Harris 2016).

¹ Frankl was actually Asperger's teacher, not chief diagnostician (Harris 2016, p.732)

Chown and Hughes claim that Kanner thought his account of autism was restricted to childhood (2016, p.2273). However, Kanner regularly talks about autistic adults in later publications (for example, Kanner & Eisenberg, 1956; Kanner & Lesser, 1958; Kanner, 1973). Indeed, in a 1951 commentary on an article Kanner described a thirty-four year old woman as being autistic (Kanner in Darr & Worden, 1951, p.570). She would have been twenty-six in 1943, meaning that Kanner considered someone as autistic who had not been diagnosed as a child.

They also claim that Kanner thought autism only affected lower-functioning children (2016, p.2273). However, Kanner regularly demarcates between levels of functioning. He described different clinical pictures between those “relatively “successful” children” (Kanner & Eisenberg, 1956, p.559) who develop language by age 5, and those who do not develop language who often end up in institutions. This was a point he explicitly emphasised. For example, in a 1958 paper he purposefully provides two case studies with very divergent clinical pictures “to indicate the wide range of later development” (Kanner & Lesser, 1958, p.711).

Chown and Hughes describe how Kanner's autism had a very specific clinical picture which, unlike Asperger's approach, would have “effectively excluded” (Chown and Hughes, 2016, p.2271) from a diagnosis of autism many children whom Asperger's account would have covered. It is true that Kanner felt autism had a quite specific clinical picture and objected to attempts to broaden the clinical picture of autism. Whilst this point is correct it needs contextualising by recognising that Kanner, from around the late 1940s onwards, considered autism closely related to the much less specific diagnosis of childhood schizophrenia.

The various forms of childhood schizophrenia share with early infantile autism the loss of effective contact and autistic thinking. However, in other forms of childhood schizophrenia there is usually a later onset and a period of normal development preceding it. Communication and affective perceptions are not usually as deeply disturbed as in autistic children. In the broader schizophrenic group there may also be a wider variety of symptoms (Kanner & Lesser, 1958, p.728).

Children excluded from Kanner's autism might have been diagnosed with the much less specific childhood schizophrenia, a diagnosis which covered many symptoms of both Kanner's infantile autism and Asperger's autistic psychopathy.

They claim that Kanner contributed to notions that autism is caused by poor mothering (Chown and Hughes, 2016, p.2273). Whilst in 1960 he describe the mothers of autistic children as “just happening to defrost enough to produce a child” (Kanner in Anon 1960), there is much disagreement within the history of autism about whether, and in what manner, Kanner actually thought poor mothering caused autism (see Donovan & Zucker 2016, p. 89; Evans 2017, p.112; Eyal et al 2010, p. 93; Feinstein 2010, p.35; Harris 2016, p.733; Jacobsen 2010, p.440; Raz 2014, p.5; Silverman 2012, p.38). It would have been helpful to acknowledge the complexities of this debate.

Finally, whilst it is tangential to my purpose of correctly describing Kanner's account, it is worth noting that the account of Frankl's relationship with Kanner

which Silberman describes has also been challenged (Harris 2016; Robinson 2016).

It is important to correct misleading representations of Kanner's approach. Had Chown and Hughes account of Kanner's notions of autism been accurate then plausibly Kanner's approach could be dismissed as a mistake which is of little value today. However, Kanner's position is much more sophisticated and complicated than Chown and Hughes or Silberman portray. This is not the place to provide a full account of his notion of autism and to assess if it could compliment or be a credible alternative to DSM-5 approaches. Rather, I have sought to correct inaccurate portrayals of Kanner's account which I hope will encourage a fairer treatment to be given to Kanner's account than seems to often occur post *Neurotribes*.

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