SUPPLEMENT TO

CHAPTER 9, V1[1]:

CLINICAL APPLICATIONS OF PRINCIPLE 1:

UNCONSCIOUS PROCESSING

Warren W. Tryon, Ph.D., ABPP

Cite as: Supplement to Chapter 9, V1: Clinical Applications of Principle 1

from Tryon, W. W. (2014)

Cognitive Neuroscience and Psychotherapy: Network Principles for a Unified Theory

New York: Academic Press

FREUD'S LEGACY

All considerations of the clinical implications and applications of unconscious processing necessarily include Freud. Freud is, continues to be, and will always be an exceptional figure in the history of clinical psychology because he was responsible for creating the big bang of psychotherapy (Tryon, 2014, p. 370) in September of 1909 with his five lectures at Clark University that: a) presented a theory of psychopathology, b) presented a method of its treatment, and c) presented case study evidence of therapeutic effectiveness. The forces that Freud's big bang set in motion have largely shaped the look and feel of contemporary clinical psychology as surely and completely as the astronomical big bang formed our universe and world. His theories[2], related theories by other authors that were based on and derived from his theories, and the many critical reactions to all psychodynamic theories by other psychologists have strongly shaped the contemporary theory and practice of clinical psychology. This is Freud's legacy. Freud's legacy does not depend upon the accuracy or inaccuracy of his theories. No one should expect that Freud got everything right as he was working alone in a brand new area at a time when neuroscience was in its infancy. From our modern perspective it is far easier for us than for him to appreciate his limitations and mistakes. It is the tremendous impact that Freud has had, both directly and indirectly, on psychological science and the professional practice of clinical psychology that is his

[1] V1 stands for Version 1 which implies that subsequent chapter updates will become available.
[2] I say theories because Freud changed his views during his professional career.

legacy and it will endure until the end of recorded history. Nothing that anyone can say about him or his theories will, or ever can, diminish this legacy in any way.

FREUD BASHING

Some readers may be confused and distracted by the fact that a substantial portion of Tryon's (2014) Chapter 9 concentrated on criticisms of Freud and the psychodynamic theory of psychopathology and symptom formation because this material does not positively present any clinical implications of unconscious processing as promised by the chapter title. The recommendation here would be to delete this material on the basis of irrelevance and get directly to the main point of the chapter. However, this material is relevant and was included in order to impede or prevent the wholesale reflexive uncritical inclusion/adoption of extant psychodynamic theorization about the clinical implications of unconscious processing after accepting unconscious processing as Principle 1. Considerable teaching experience has consistently revealed a tendency of students, especially those with a strong psychodynamic background and/or clinical orientation, to take my arguments for the need for psychological science to recognize and include unconscious processing as vindication and validation of essentially everything that psychodynamic theorists have ever written about unconscious processing. The strength and consistency of this motivation requires strong push back to create space and time to seriously rethink the clinical implications of unconscious processing. That is the purpose served by this critical material.

A possible misperception of my criticisms of Freud's theory and professional conduct is that I have been unfair to the point of "Freud bashing". The term "bashing[3]" injects an emotional tone that indicates that we have moved beyond rational discourse. My main concern with being categorized in this emotional way is that this label will be used to justify ignoring my call to seriously rethink the clinical implications of unconscious processing and worse, to use the empirical evidence that I provide for unconscious processing as support for every form of psychodynamic theory that has been proposed over the past century or will ever be proposed.

[3] The "define:bashing" command reveals the following definitions: (1) physical assault: mugging or violence, especially when directed at a particular group of people. (2) criticism: hostile comment directed at a specific person or group. (3) excessive use: the exposure of something to repetitive or prolonged use. Synonyms include battering, pounding, knocking, beating, and pummeling.

I am also concerned that being categorized as a "Freud basher" will alienate some readers who might otherwise be sympathetic to my overall effort at theoretical unification and psychotherapy integration. The emotional tag of "basher" constitutes a scarlet letter[4] for all to see, revile, and reject.

Criticism For All

The following two paragraphs demonstrate that I have not singled Freud out for harsh criticism. They show that I have strongly criticized cognitive behavioral therapy, the empirically supported treatment movement, and psychological science in general. This context shows that I have not treated Freud differently from how I treat contemporary psychologists.

In Chapter 1 I criticized Castonguay and Beutler (2006) for how they used the term principle in their book entitled *Principles of Therapeutic Change That Work*. I criticized Beck and Ellis for taking key ideas from Hartmann's book on ego psychology without citation. I criticized Tee and Kazantzis (2011) for presenting Carl Roger's ideas as their own. I criticized the entire empirically supported treatment movement for supporting "purple hat" therapies. I criticized CBT for being too cognitive, presenting emotions as secondary to cognitions, and for not formally recognizing unconscious processing. I criticized the Association for Psychological Science and Wargo (2012) for publishing about psychological mechanisms when there is no evidence that such things exist. I criticized the BioPsychoSocial model as a list of ingredients that explains nothing and by implication criticized all those who support and work from this model. Likewise, I criticized box and arrow and structural equation models and by implication all those who support and work from these models. I specifically criticized Monshower et al. (2012) for claiming that logistic regression provides mechanism information. I criticized Skinner and his followers for ignoring the need to provide mechanism information. I criticized all psychologists for their double denials; i.e., a) denying that psychological mechanisms are entirely mental, and b) denying that psychological mechanisms are entirely biological. In my supplement to Chapter 1 I criticized Young (2014) for mischaracterizing evidence of dependency as explanatory mechanism information.

In Chapter 2, I criticized all of contemporary brain imaging as modern phrenology and by implication criticized all investigators who contribute to this research. I criticized all of psychological science for its lack of replication. In the supplement to Chapter 2, I specifically

[4] The scarlet letter was a capital A for adultery. It was a sign that fidelity in a monogamous relationship was breached. Here I am concerned about psychoanalytic fidelity.

criticized Walton (2014) for basing his introduction of a "new science of wise psychological interventions" on extreme and unreplicated findings. In Chapter 3, I criticized most of contemporary psychological science for its conscious-centric orientation and presented evidence that requires a transition to an unconscious-centric orientation. In Chapter 7, I strongly criticized Marcus's (1998) analysis of connectionism. Likewise, I criticized Chomsky (1971, 1975, 1980, 1986), Fodor and Pylyshyn (1988), and Marcus (1998) for their explanation of language development. In Chapter 8, I criticized Judith Beck (2010) for failing to provide a suitable theoretical basis for CBT. In Chapter 9, I undercut Kazdin's (1982) arguments in defense of symptom substitution. In sum, I have seriously criticized many psychologists and much of psychological science to the point of vigorously recommending a paradigm change.

Curiously, the term "bashing" has never emerged in response to any of these serious criticisms of specific individuals or of psychological science. Psychologists are often quite critical of each other, but the term "bashing" only seems to occur in reference to Freud. One explanation is that use of this term reveals greater admiration than I have expressed above; perhaps even an ideological loyalty on the order of that which Freud demanded of his followers. I consider that my treatment of Freud and the psychodynamic theory of psychopathology and symptom formation is not harsher than my treatment of cognitive psychology and psychological science has been.

Sympathy for Freud?

The charge of Freud bashing assumes that Freud was a good guy who has been unfairly maligned; treated far more harshly than is warranted by his behavior. We tend to protect and defend innocent people who are being picked on for little or no reason. After all, is what he did or did not do really so different from the rest of us that we can't just let these matters go and move on and get back to business as usual? I suggest that the information provided below will show that my criticisms are well deserved.

All theory construction problems that were present at the time of Freud's big bang also propagated around the world along with his new theory and therapy. His ideas became foundational for many other psychologists. The false premises in Freud's work seriously compromise all subsequent theories built upon them. While one may accidentally reason from false premises to true conclusions, one cannot consistently reach true conclusions from false premises. Hence, it is important to examine how psychodynamic theories got started. The extent

of these problems is what required spending a third (18 of 54 pages) of Chapter 9 mostly on two main issues. The first issue concerns the causal role of phantasy as articulated in the Oedipus complex. The second issue concerns the role of symptom substitution as a necessary consequence of the psychodynamic formulation of symptom formation.

Oedipus Complex[5]

Freud began his psychoanalytic career by studying the origins of what was then known as hysteria in 18 women. He concluded that all of them had been sexually abused at a young age. Outrage at his findings by his medical peers led Freud to repudiate the clinical evidence that he so carefully collected from all 18 of his female clients and claim that their problems were entirely the result of their own sexual phantasy. They were accused of unconsciously wanting to be sexually abused. The clinical evidence that they were sexually abused was repudiated. What would we think of a contemporary clinician who behaved in this way?

Anna Freud claimed that without the Oedipus complex psychoanalysis would not have existed because the Oedipus complex is the prototypical illustration and clinical proof of the causal role that psychoanalysis claims that phantasy plays in both normal and abnormal mental life. Freud's original clinical observations continue to contradict what subsequently became a conceptual pillar of psychoanalytic theory. Abnormal psychology textbooks continue to teach the Oedipus complex as fact.

Scientific Misconduct

Freud's effort to curry favor with his medical colleagues reviewed above was not an isolated incident. Tryon (2014, p. 402) reported two specific instances of scientific misconduct by Freud. They are: a) his published, and subsequently publically retracted, false claim that he cured a patient of morphine addiction using cocaine without addiction to cocaine, and b) his false claim that psychoanalysis cured Anna O as empirical support for the efficacy of psychoanalytic treatment

[5] The following link provides an alternative interpretation of the Greek play Oedipus Rex than that provided by Freud http://www.psychologytoday.com/blog/the-art-flourishing/201205/the-real-oedipal-complex. I did not know that Oedipus did not lust after his mother nor did he fear is father as per Freud's Oedipus complex.

when she died uncured in a Swiss sanitarium. Scientific misconduct breaches professional credibility and casts doubt and suspicion on all that a person has or will ever publish.

Freud engaged in other questionable scientific activity. Tryon (2014, p. 400) systematically avoided citing authors who discussed unconscious processing so that he could claim that he would receive full credit for this discovery which, in his view, warranted recognition as a scientific contribution equal to that of Copernicus and Darwin (Tryon, 2014, p. 400). Such behavior calls the scholarly quality of his work into question.

Science or Cult?

Freud was a neurologist who contributed scientific evidence regarding evolution. Psychoanalysis derived from his medical practice. It would therefore seem that his clinical practice was an extension of his scientific training. But Freud did not support open inquiry and debate regarding psychoanalysis. Instead he required loyalty and obedience. According to Grosskurth (1991), Freud issued gold rings to his disciples, demanded their absolute loyalty to him, and required their total and uncritical acceptance of his theories. In effect, Freud functioned as "lord of the rings". He dismissed disciples when they deviated from orthodoxy by thinking for themselves[6]. Freud's secret committee governed the psychoanalytic movement from 1912 to 1927. Its two main purposes were: a) to monitor the activities of Carl Jung who Freud once named as his successor but subsequently expelled from his inner circle and b) to insure that Freud alone continued to dominate psychoanalysis. This history helps us to understand why psychodynamic clinicians have such a long history of orthodoxy even in the face of contradictory empirical evidence.

It is also true that Pythagoras ran a carefully controlled cult of sacred geometry known as Pythagoreanism. Their creed was "like the Theorem or get boiled alive!"[7]. Isaac Newton devoted many hours to the numerological analysis of biblical scripture. He also was very interested in alchemy and constantly tried to transmute lead into gold. Why then are Pythagoras and Newton viewed differently today than Freud? Like Freud, Pythagoras and Newton started new schools of thought. They differ from Freud in that Pythagoras's theorem held up to subsequent scrutiny as did Newton's Principe. Freud's false claim that he alone discovered the unconscious, his false

[6] See http://en.wikipedia.org/wiki/Inner_circle_(psychoanalysis)
[7] See (http://the-random-dreamer.deviantart.com/art/Pythagoras-Killing-Spree-244509294)

claims of cure, and his mistaken Oedipal views including the causative role of phantasy[8] have not held up to subsequent scrutiny. Consequently, Freud receives more criticism than Pythagoras and Newton combined. Another reason why Freud has been harshly criticized by behavioral and cognitive behavioral psychologists is because behavior therapy was so strongly opposed by psychodynamic clinicians that they were forced to fight for professional acceptance.

Naïve?

Some readers may have interpreted my use of the word "naively" in following statement to mean that Freud should have known better and therefore constitutes Freud bashing:

> What he did not realize is that cocaine creates vivid dreams dominated by sexual and aggressive themes. Naively, Freud generalized his and his patient's cocaine fired dream findings to everyone, apparently unaware that he mistook a drug effect for normal psychology (Tryon, 2014, p. 402).

I think that Freud really was naïve about the effects that cocaine had on dream content because he was using this drug so recently after it was discovered and became available. However, the question of whether or not Freud should have known about the effect of cocaine on the dream content of his patients from his own personal experience with this drug is beside the main point here. The scientific relevance of this matter is that from our modern psychopharmacological perspective we can confidently conclude that cocaine over represented dream themes of sex and aggression in his psychodynamic theories and consequently did so in the theories that some other psychologists built based upon Freud's work. To ignore/excuse these drug effects and retain Freud's emphasis on sex and aggression would be a mistake. The serious criticism that would be leveled against anyone attempting to publish a theory of unconscious processing today that ignored drug effects would not be deflected and dismissed as author bashing.

[8] Jonathan Lear (1998) reported that Freud tentatively reversed his view on phantasy and sexual abuse in 1917 and 1938 but by then Freud's ideas had become foundational for many of his followers who built their theories on views contradicted by Freud's own clinical evidence.

Conclusions

The fact that I have levied equally strong criticism against contemporary psychologists as I have against Freud means that I have not singled Freud out for unusually harsh treatment. I submit that Freud's professional conduct diminished his credibility. Blaming female victims for the traumatic results of sexual assault, publishing false claims of cure by cocaine and psychoanalysis, failure to cite work by others, demanding complete fidelity issuing gold rings to disciples to bind their loyalty, and overlooking drug effects are all reasons to pause and completely rethink the clinical implications of unconscious processing.

We now turn to two hard and risky predictions of psychodynamic theory that have failed spectacularly. They both suggest that substantial portions of psychodynamic theory concerning unconscious processing need to be completely rethought. They are the necessary condition thesis and symptom substitution.

NECESSARY CONDITION THESIS

Tryon (2014) discussed this topic in Chapter 7 but it is also pertinent to Chapter 9. Adolph Grünbaum[9], a physicist, mathematician, and philosopher, formulated a "Necessary Condition Thesis" on the basis of his analysis of psychodynamic theory that he presented in his 1984 book entitled ***The Foundations of Psychoanalysis: A Philosophical Critique*** as follows:

> The assumptions that Freud actually invokes in this context can be stated as the conjunction of two causally necessary conditions as follows: (1) only the psychoanalytic method of interpretation and treatment can yield or mediate to the patient correct insight into the unconscious pathogens of his psychoneurosis, and (2) the analysand's correct insight into the etiology of his affliction and into the unconscious dynamics of his character is, in turn, *causally necessary* for the therapeutic conquest of his neurosis. I shall refer to the *conjunction* of these two Freudian claims as his "Necessary Condition Thesis" or for brevity, "NCT"" (pp. 139-140; italics in the original).

[9] See http://en.wikipedia.org/wiki/Adolf_Gr%C3%BCnbaum

> Clearly, NCT entails not only that there is no spontaneous remission of psychoneuroses but also that, if there are any cures at all, psychoanalysis is *uniquely* therapeutic for such disorders as compared to any *rival* therapies. In view of the importance of NCT, I have dubbed it "Freud's Master Proposition" (p. 140; italics in the original).

The NCT makes the hard and risky prediction that only psychodynamic therapy provides lasting and effective therapeutic results. This master psychoanalytic proposition justified the superior position that psychodynamic clinicians took for several decades relative to all other therapies.

The advent of behavior therapy provided a major empirical test of the NCT because it presented a radically different and contradictory approach to treatment. The many studies of the effectiveness of behavioral (Applied Behavior Analysis), cognitive therapy, cognitive-behavioral therapies, dialectical behavior therapy, and psychopharmacotherapy published to date have consistently and clearly falsified this master proposition. There really is more than one way to effectively treat psychological problems. Curiously, the psychodynamic community has taken little if any notice of how these empirical findings seriously undercut and falsify their theoretical orientation.

SYMPTOM SUBSTITUTION

Tryon (2014) raised the symptom substitution issue because it concerns a hard and risky prediction that stems from the psychodynamic understanding of psychopathology and symptom formation that has been consistently falsified. Symptom substitution was a sufficiently important topic in clinical psychology in 1982 that the *Psychological Bulletin* published Kazdin's (1982) defense of it. Symptom substitution was still of sufficient interest to clinical psychologists in 2008 that the *Clinical Psychology Review* published Tryon's (2008) overview of it. When I joined the Fordham faculty in 1970, psychodynamic clinicians frequently characterized behavior therapy as bordering on malpractice due to the near certainty that symptom substitution would lead to other and potentially greater problems for their clients. That these claims are no longer made today does not mean that symptom substitution is no longer a hard and risky prediction that necessarily follows from the psychodynamic theory of psychopathology and symptom formation. The core issue here concerns how is it possible for psychodynamic theory **not** to predict symptom substitution given its theory of psychopathology and symptom formation? Grünbaum (1984)

presented the theoretical basis for predicting symptom substitution as follows (Tryon, 2014, p. 417).

> A neurotic symptom is held to be a compromise formed in response to an unresolved conflict between a forbidden unconscious impulse and the ego's defense against it. The symptom is held to be sustained at any time given time by a coexisting, ongoing unconscious conflict, which – as claimed by NCT – does not resolve itself without psychoanalytic intervention. Hence, if the repression of the unconscious wish is not lifted psychoanalytically, the underlying neurosis will persist, even if behavior therapy or hypnosis, for example extinguishes the particular symptom that only manifests the neurosis at the time. As long as the neurotic conflict does persist, the patient's psyche will call for the defensive service previously rendered by the banished symptom. Hence, typically and especially in severe cases, **the unresolved conflict ought to engender a *new* symptom**. And incidentally, this expectation qualifies as a "risky" prediction in Popper's sense, since such rival extant theories as behavior intervention disavow just that expectation (p. 162; italics in the original, bold emphasis added).

The consistent finding is that symptom substitution just does not happen when it should occur in virtually every case where a non-psychodynamic intervention is applied. It is easy to understand why psychodynamic theorists have long been reluctant to consider this risky prediction because the consistent negative evidence so completely falsifies their theory of psychopathology and symptom formation. Asking psychodynamic theorists and clinicians to accept the hard, risky, and falsified predictions required by the NCT and symptom substitution is rather like asking cigarette companies to accept the associated health risk evidence or asking the oil companies to accept the climate change evidence. That psychodynamic authors and externship supervisors do not discuss these matters is understandable but does not resolve them or make them disappear.

The absence of symptom substitution seriously questions whether unconscious processing works as Freud hypothesized. Perhaps psychological symptoms are not the result of unconscious impulses threatening to break through ego defenses. The term "symptom" reflects the medical model. Freud was a physician. He therefore believed that symptoms result from underlying illnesses. Repressed motives concerning sex and aggression were his prime causal candidates. I

recommend that we discard both the medical model and its psychodynamic variant by replacing the term "symptom" with the psychometric term "indicator" based largely on the work of Krueger (1999), Krueger and Markon (2006), and Krueger, Caspi, Moffitt, and Silva (1998) and Caspi et al. (2014).

Clients will sometimes present with a single issue; perhaps anxiety or depression. Subsequent to the formation of a good working alliance and progress towards identified therapeutic goals the client will reveal another problem such as an eating disorder. This is not evidence of symptom substitution. The client most likely had the eating disorder upon intake but did not reveal it out of embarrassment or caution since they did not know you well then. Such reasoning is consistent with the finding that many patients are comorbid. Those who do not appear to be comorbid probably are. That is a strong implication of the Caspi et al. (2014) finding of big p. This rival explanation needs to be rejected before the symptom substitution hypothesis can advance.

Unidimensional treatments that focus exclusively upon symptom reduction/removal tend to ignore other factors that support maladaptive behavior including reinforcement histories associated with family and other close relationships as well as distorted thinking which Caspi et al. (2014) demonstrated is an indicator of internalizing disorders that in turn are indicators of externalizing disorders. This is why the recommended broad-based Applied Psychological Science (APS) clinical orientation is so important.

CLOSING COMMENTS REGARDING PSYCHODYNAMIC THEORY

It seems to be true that no "hard core" psychodynamic theorist has ever formally recognized in print that either the NCT or symptom substitution are hard and risky predictions of psychodynamic theory. That no fully fledged psychodynamic theorist has proposed or endorsed either the NCT nor symptom substitution suggests that these concepts are not a formal part of psychodynamic theory but rather distortions cooked up by behaviorists as straw men who can be easily defeated. Fortunately, Adolph Grünbaum is a philosopher and not a psychologist because that takes him out of the direct line of controversy between behavioral and psychodynamic clinicians.

Adolph Grünbaum received a B.A. with twofold High Distinction in Philosophy and in Mathematics from Wesleyan University, Middletown, Connecticut in 1943. He obtained both his M.S. in physics (1948) and his Ph.D in philosophy (1951) from Yale University. He was the

Andrew Melton Professor of Philosophy and Research Professor of Psychiatry at the University of Pittsburgh when he published ***The Foundations of Psychoanalysis: A Philosophical Critique*** in 1984. I take these to be acceptable credentials. I believe that Grünbaum was seriously engaged in a scholarly analysis of Freudian thought and not constructing a straw man for use by behaviorists.

Contemporary acceptance of CBT and DBT by psychodynamic clinicians is a welcomed move towards psychotherapy integration. However, it does not address, let alone resolve, the logical difficulties associated with the psychodynamic theory of psychopathology and symptom formation. Without formal revision of psychodynamic theory such eclectic practice raises at least the following problem. CBT treats "symptoms". Using such treatments contradicts the theoretical basis for doing psychodynamic psychotherapy and therefore should not be effective; but it is effective in treating personality disorders. This contradiction can be removed by claiming that CBT and DBT effectively treat the hypothesized underlying repressed issues that psychodynamic and psychoanalytic clinicians have long focused on as crucial causal factors thereby avoiding symptom substitution. Or one can admit that the psychodynamic formulation of symptoms is wrong. DBT is a very direct conscious-centric intervention. Hence, the more likely option is that the psychodynamic formulation of how symptoms form is flawed.

The final take home message of the portion of Chapter 9 that was critical of Freud is that there are enough theory construction problems with how psychodynamic theory got started and incorporated into subsequent variations that the time has come to completely rethink the clinical implications of unconscious processing. The remainder of Chapter 9 is an initial attempt to do so.

AUTISM

This section extends the brief discussion of autism by Tryon (2014, p. 440) in Chapter 9. The network cascade that constitutes unconscious processing depends directly upon neural architecture; how the brain is wired. Abnormal wiring can cause the problems with processing social information that people with Autism Spectrum disorder (ASD) are known to have.

Pelphrey, Adolphs, and Morris (2004) reviewed evidence that the amygdala is involved in social cognition. This view is consistent with a report by Brothers (1990) that included the superior temporal sulcus and gyrus and orbitofrontal cortices along with the amygdala as the neurobiological basis of social cognition. Adolphs, Tranel, and Damasio (1998) reported that

bilateral damage to the human amygdala impairs social judgments of trustworthiness and approachability by increasing apparent trustworthiness and approachability. Pelphrey, Adolphs, and Morris (2004) also reviewed evidence that the superior temporal sulcus region and the fusiform gyrus, are impaired in ASD.

A much more general problem was identified by Minshew and Williams (2007). They reviewed research demonstrating that ASD is a disorder of intrahemispheric under-connectivity of the associative cortex that has a broad impact on cognitive functioning that requires the integration and coordination of multiple neural networks. Substantial empirical support exists for this view. Pelphrey (2011) reported that the following **structural** neuroimaging studies of older children, adolescents, and adults with ASD have found brain under-connectivity: Barnea-Goraly et al., (2004), Cheung et al., (2009), Fletcher et al. (2010), Keller, Kana, and Just (2007), Kumar et al. (2010), Sahyoun, Belliveau, Soulières, Schwartz, and Mody (2010), Sivaswamy et al., (2010). Pelphrey (2011) also reported that the following **functional** neuroimaging studies of adolescents, and adults with ASD have found brain under-connectivity: Castelli, Happé, Frith, and Frith (2000), Just, Cherkassky, Keller, and Minshew (2004), Kana, Keller, Cherkassky, Minshew, and Just (2006, 2007), Kleinhans et al. (2008), Koshino et al. (2005, 2008), Mason, Williams, Kana, Minshew, and Just (2008), Monk et al. (2009), Noonan, Haist, & Müller (2009), Villalobos, Mizuno, Dahl, Kemmotsu, and Müller (2005), and Wicker et al., (2008).

Pelphrey (2011) concluded that:

> The appeal of the underconnectivity notion lies partly in the fact that it appears to offer a systems-level model of brain dysfunction that purports to account for the specific symptoms of ASD as well as the heterogeneity of etiology, behaviors and cognition (Geschwind & Levitt, 2006) (p. 632).

Pelphrey (2011) noted the following limitation with this view:

> However, it is not yet clear how the underconnectivity perspective accounts for the specific patterns of dysfunction in individuals with ASD. That is, how might this perspective explain what is common among individuals with ASD and what separates ASD from other neurodevelopmental disorders that also feature underconnectivity? (p. 632).

A partial answer to this question comes from connectionist simulations of schizophrenia where underconnectivity has been studied via simulations. Synaptic pruning is a normal developmental process that physically sculpts the brain by reducing the number of synapses so that it can more efficiently and effectively specialize in the developmental tasks required of a particular person in a specific environment at a given time in history. Tryon (2014, p. 305) presented neurobiological evidence of this normal process in Chapter 6. Normal developmental synaptic pruning ends between 18 and 22 years of age. Symptoms of schizophrenia appear if this trend towards under-connectivity continues past that time as discussed by Tryon (2014, p. 304). The inability to form certain associations characterize both ASD and schizophrenia. This does not mean that ASD is a form of schizophrenia. What it does mean is that connectionist simulations have helped us to better understand that reducing the degree of connectivity of a neural network below a critical level reduces its ability to function properly. Put otherwise, connectionist simulations can help explain why under-connectivity matters. Connectionist models of ASD should be done in order to better understand how ASD deficits arise from underconnectivity.

REFERENCES[10]

Adolphs, R., Tranel, D., & Damasio, A, R. (1998). The human amygdala in social judgment. *Nature, 393,* 470–474.

Barnea-Goraly, N., Kwon, H., Menon, V., Eliez, S., Lotspeich, L., & Reiss, A. L. (2004). White matter structure in autism: Preliminary evidence from diffusion tensor imaging. *Biological Psychiatry, 55,* 323–326.

Brothers, L. 1990. The social brain: A project for integrating primate behavior and neurophysiology in a new domain. *Concepts in Neuroscience, 1,* 27–51.

Caspi, A., Houts, R. M., Belsky, D. W., Goldman-Mellor, S. J., Harrington, HL, Israel, S., Meier, M. H., Ramrakha, S., Shalev, I., Poulton, R., & Moffitt, T. E. (2014). The p factor: One general psychopathology factor in the structure of psychiatric disorders? *Psychological Science, 2* (2), 119-137. doi 10.1177/2167702613497473

[10] References not included in this list are in the reference section of the Tryon (2014).

Castelli, F., Happé, F., Frith, U., & Frith, C. (2000). Movement and mind: A functional imaging study of perception and interpretation of complex intentional movement patterns. *NeuroImage, 12,* 314–325.

Cheung, C., Chua, S. E., Cheung, V., Khong, P. L, Tai, K. S., Wong, T. K., Ho, T. P., & McAlonan, G. M. (2009). White matter fractional anisotrophy differences and correlates of diagnostic symptoms in autism. *Journal of Child Psychology and Psychiatry, 50,* 1102–1112.

Fletcher, P. T., Whitaker, R. T., Tao, R., DuBray, M. B., Froehlich, A., Ravichandran, C., Alexander, A. L., Bigler, E. D., Lange, N., & Lainhart, J.E. (2010). Microstructural connectivity of the arcuate fasciculus in adolescents with high functioning autism. NeuroImage, 51, 1117–1125.

Geschwind, D. H., & Levitt, P. (2007). Autism spectrum disorders: Developmental disconnection syndromes. *Current Opinion in Neurobiology, 17,* 103–111.

Grosskurth, P. (1991). *The secret ring: Freud's inner circle and the politics of psychoanalysis.* Reading Mass: Addison-Wesley.

Grünbaum, A. (1984). *The foundations of psychoanalysis: A philosophical critique*. Berkeley, CA: University of California Press.

Just, M. A., Cherkassky, V. L., Keller, T. A., & Minshew, N. J. (2004). Cortical activation and synchronization during sentence comprehension in high-functioning autism: Evidence of underconnectivity. *Brain, 127,* 1811–1821.

Kana, R. K., Keller, T. A., Cherkassky, V. L., Minshew, N. J., & Just, M. A. (2006). Sentence comprehension in autism: Thinking in pictures with decreased functional connectivity. *Brain, 129,* 2484–2493.

Kana, R. K., Keller, T. A., Minshew, N. J., & Just, M. A. (2007). Inhibitory control in high functioning autism: Decreased activation and underconnectivity in inhibition networks. *Biological Psychiatry, 62,* 198–206.

Kazdin, A. E. (1982). Symptom substitution, generalization, and response covariation: Implications for psychotherapy outcome. *Psychological Bulletin, 91,* 349-365.

Keller, T. A., Kana, R. K., & Just, M .A. (2007). A developmental study of the structural integrity of white matter in autism. NeuroReport, 18, 23–27.

Kihlstrom, J. F., Barnhardt, T. M., & Tataryn, D. J. (1992). The psychological unconscious: Found, lost, and regained. *American Psychologist, 47*(6), 788–791.

Kihlstrom, J. F. (1984). Conscious, subconscious, unconscious: A cognitive perspective. In K. S. Bowers, & D. Meichenbaum (Eds.), *The unconscious reconsidered* (pp. 149–211). New York: Wiley.

Kihlstrom, J. F. (1987). The cognitive unconscious. *Science, 237*, 1445–1452.

Kleinhans, N. M., Richards, T., Sterling, L., Stegbauer, K. C., Mahurin, R., Johnson, C. L., Greenson, J., Dawson, G., & Aylward, E. (2008). Abnormal functional connectivity in autism spectrum disorders during face processing. *Brain, 131,* 1000–1012.

Koshino, H., Carpenter, P. A., Minshew, N. J., Cherkassky, V. L., Keller, T. A., & Just, M. A. (2005). Functional connectivity in an fMRI working memory task in high-functioning autism. *NeuroImage, 24,* 810–821.

Koshino, H., Kana, R. K., Keller, T. A., Cherkassky, V. L., Minshew, N. J., & Just, M. A. (2008). fMRI investigation of working memory for faces in autism: Visual coding and underconnectivity with frontal areas. *Cerebral Cortex, 18,* 289–300.

Kumar, A., Sundaram, S.K., Sivaswamy, L., Behen, M.E., Makki, M.I., Ager, J., Janisse, J., Chugani, H.T., & Chugani, D C. (2010). Alterations in frontal lobe tracts and corpus callosum in young children with autism spectrum disorder. *Cerebral Cortex, 20,* 2103–2113.

Lear, J. (1998). *Open minded: Working out the logic of the soul*. Cambridge, MA: Harvard University Press.

Mason, R. A., Williams, D. L., Kana, R. K., Minshew, N. J., & Just, M. A. (2008). Theory of mind disruption and recruitment of the right hemisphere during narrative comprehension in autism. *Neuropsychologia, 46,* 269–280.

Minshew, N. J., & Williams, D. L. (2007). The new neurobiology of autism: Cortex, connectivity, and neuronal organization. *Archives of neurology, 64,* (7), 945-950.

Monk, C. S., Peltier, S. J., Wiggins, J. L., Weng, S-J., Carrasco, M., Risi, S., & Lord, C. (2009). Abnormalities of intrinsic functional connectivity in autism spectrum disorders. *NeuroImage, 47,* 764–772.

Noonan, S. K., Haist, F., & Müller, R-A. (2009). Aberrant functional connectivity in autism: Evidence from low-frequency BOLD signal fluctuations. *Brain Research, 1262,* 48–63.

Pelphrey, K., Adolphs, R., & Morris, J. P. (2004). Neuroanatomical substrates of social cognition dysfunction in autism, *Mental Retardation and Developmental Disabilities Research Reviews, 10,* 259-271.

Pelphrey, K. A., Shultz, S., Hudac, C. M., Vander Wyki, B. C. (2011). Research Review: Constraining heterogeneity: the social brain and its development in autism spectrum disorder. *Journal of Child Psychology and Psychiatry, 52* (6), 631–644 doi:10.1111/j.1469-7610.2010.02349.x

Sahyoun, C. P., Belliveau, J. W., Soulières, I., Schwartz, S., & Mody, M. (2010). Neuroimaging of the functional and structural networks underlying visuospatial vs. linguistic reasoning in high-functioning autism. Neuropsychologia, 48, 86–95.

Sivaswamy, L., Kumar, A., Rajan, D., Behen, M., Muzik, O., Chugani, D., & Chugani, H. (2010). A diffusion tensor imaging study of the cerebellar pathways in children with autism spectrum disorder. *Journal of Child Neurology, (10),* 1223-1231. doi: 10.1177/0883073809358765

Villalobos, M. E., Mizuno, A., Dahl, B. C., Kemmotsu, N., & Müller, R. A. (2005). Reduced functional connectivity between V1 and inferior frontal cortex associated with visuomotor performance in autism. *NeuroImage, 25,* 916–925.

Tryon, W. W. (2008b). Whatever happened to symptom substitution? *Clinical Psychology Review, 28*, 963-968.

Tryon, W. W. (2014). *Cognitive neuroscience and psychotherapy: Network Principles for a Unified Theory*. New York: Academic Press.

Walton, G. M. (2014). The new science of wise psychological interventions. *Current Directions in Psychological Science, 23* (1), 73-82. doi 10.1177/0963721413512856

Wicker, B., Fonlupt, P., Hubert, B., Tardif, C., Gepner, B., & Deruelle, C. (2008). Abnormal cerebral effective connectivity during explicit emotional processing in adults with autism spectrum disorder. *Social Cognitive and Affective Neuroscience, 3,* 135–143.