SUPPLEMENT TO

CHAPTER 10, V1[1]:

CLINICAL APPLICATIONS OF PRINCIPLE 2:

LEARNING AND MEMORY

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FUNCTIONAL RELATIONSHIPS VS. THEORIES

A possible misreading of Chapter 10 is that it maintains that classical behavioristic psychology is essentially correct but just needs some neuroscience spiffing-up. This perspective mistakenly conflates, the **functional relationships** discovered by behaviorists with the explanatory system of **behaviorism**. This defensive position has been taken by many psychodynamic and cognitive clinicians in an effort to reinforce their respective schools of thought by criticizing and dismissing behavioral theory and research. Psychodynamic and cognitive theorists eagerly dismiss all of the functional relationships demonstrated by behaviorists because of faults they find with behaviorism.

Many behaviorists have made a similar mistake. They tend to dismiss all of the randomized controlled trial (RCT) research that demonstrates that psychodynamic treatments are effective because of faults they find with Freud and/or with psychodynamic theory. Many conservative behaviorists, like those who prefer applied behavior analysis, tend to dismiss CBT interventions because of faults they find with cognitive theory. Skinner (1977) was very clear concerning his reasons for rejecting cognitive psychology in his article entitled "Why I am not a cognitive psychologist".

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[1] V1 stands for Version 1 which implies that subsequent chapter updates will become available.

Many psychodynamic clinicians make the same logical mistake. Most all of them reflexively dismiss the extensive single subject research design evidence derived from applied behavior analysis and all of the RCT studies that support CBT because of faults they find with behaviorism and cognitive psychology.

In all these cases, serious errors of judgment are made. The proverbial baby is thrown out with the bath. These conflation mistakes arise from the logical error of affirming the consequent. This logical error has the following form: If A then B, B, therefore A. We make this logical mistake when we conclude that behavioral therapies validate behaviorism, when we conclude that cognitive therapies validate cognitive theory, and when we conclude that psychodynamic therapies validate psychodynamic theory. **Clinicians can, and often are, right for the wrong reasons.** The take home message here is that empirical findings sometimes validate the theory that gave rise to them but they do not always or necessarily do so. This logical error appears to be emotionally motivated which explains much of the passion that loyal supporters demonstrate. Readers are referred to Principle 7 of Tryon (2014, p. 235) concerning Consonance and Dissonance and pertinent hot cognitions. This illogical reasoning has formed the foundation of the competitive schools and camps that have formed over at least the last half century of psychological science generally and clinical psychology particularly. This illogical reasoning has nurtured the divisive attitudes that impede efforts to explain the replicated results that each school/camp have developed.

The intended contribution of Chapter 10 is to consider the clinical implications of learning and memory when understood in terms of the neuroscience mechanisms that modify our neural networks in ways that enable memories to form and learning to occur. The resulting **Bio↔Psychology Network (BPN)** theory provides **behavioral psychology** with some of the required missing mechanism information. It provides an explanation that **Skinner** would approve of because: a) the theory requires on to think in physical rather than mental terms, b) the network cascade is a deterministic process, c) the theory explains behavior using the principles of variation and selection, d) the theory rejects the computer metaphor of memory as storage and understands memory as changing the organism, and e) all operant and respondent conditioning phenomena can be simulated from this perspective. The BPN explanatory system delivers, at least in part, an explanation that **Pavlov** would approve of because: a) it is a neural network explanation, and b) Pavlov was a biologist. The synthesis of psychology and neuroscience provided by the

Bio↔Psychology Network theory is fully consistent with **cognitive psychology** because: a) Cognition is formally represented by the middle layer of processing nodes where stimuli are transformed into latent constructs by Principle 3 and b) behavior is a transformed result of these latent constructs. The Bio↔Psychology Network theory is consistent with **psychodynamic psychology** in that it calls for a paradigm shift from a conscious-centric to an unconscious-centric psychological science. It also recognizes the crucial importance of very early experiences that have long-lasting epigenetic effects. This theoretical synthesis of psychology and neuroscience authorizes a formal integration of cognition and conditioning that is fully consistent with CBT, CT, and ABA and the remaining Big Five clinical orientations.

THE COGNITIVE REVOLUTION

Behaviorism is a scientific methodology as well as an explanatory system. The functional relationships discovered using behavioral methods are among the most robust findings that psychological science has to offer. Examples include positive reinforcement, extinction, stimulus control, schedules of reinforcement, and the matching law among many other phenomena that have been carefully studied under highly controlled laboratory conditions and applied successfully in thousands of clinical cases. Skinner explained behavior using Darwin's functional principles of variation and selection. However, Skinner could not explain why behavioral variation occurred or how selection, reinforcement, work. Darwin faced a similar opposition from biologists when he published his *On the Origin of Species* in 1859. This was before population genetics was known and therefore Darwin could not explain why organisms differed nor could he explain the mechanisms by which selection occurred. He was said to have just a functional theory. The lack of mechanism information made Darwin's theory incomplete and consequently unacceptable to the scientific community. Darwin's theory became widely accepted only after population genetics provided the required missing mechanism.

On the other hand, the explanatory system known as behaviorism is a **functional theory** and like all functional theories it cannot explain why the functional relationships that it discovers exist and work as they do. Behaviorism was correctly said to have an explanatory **black box** because stimuli went in, responses came out, and no one had a clue as to why any of this worked the way did. One might argue that behaviorism was really a form of behavioral biology and not a psychology of any kind precisely because it could not explain anything that happens inside the

black box. Skinner refused psychological explanations. His 1977 article entitled "Why I am not a cognitive psychologist" provided multiple reasons for his decision to categorically reject cognitive psychology. Instead, Skinner left these explanatory issues to neuroscience who he said had the proper tools to conduct the required studies.

The learning and memory neuroscience mechanisms that constitute Principle 2 provide important missing mechanism information that is fully capable of explaining why the functional relationships discovered by behaviorists work as they do. All, or almost all, of the functional relationships that constitute operant and respondent conditioning have connectionist simulations. Principle 2 provides behaviorists with the ability to explain for the first time their functional relationships. Principle 2 fills behaviorism's black box with neuroscience facts and mechanisms. Principle 2 does for Skinner what population genetics did for Darwin. This is way more than spiffing-up unless what population genetics did for evolutionary theory constitutes spiffing-up Darwinism with some genetics.

The cognitive revolution in psychology (Bandura, 1978; Dember, 1974; Gardner, 1985; Mahoney, 1974, 1977; Meichenbaum, 1977) promised to fill the behaviorist's black box with causal mechanism information. Clinical psychologists were promised new treatments based on cognitive science that would be superior to anything that came before it included the extensively empirically supported interventions derived using applied behavior analysis. We would finally be able to understand why psychological treatment worked.

Unfortunately, the **behavioral black box** was replaced with a **cognitive black box** that contained box and arrow diagrams where the arrows imputed causal connections but did not provide any causal mechanism information. The cognitive revolution exchanged one black box for another. "We went from **not** having one kind of theory to **not** having another kind of theory" Tryon, 2014, p. 395; emphasis in the original). We replaced a functional theory of one kind with a functional theory of another kind. All functional theories lack mechanism information which is why the cognitive black box does not, and never will, offer better explanations than the behavioral black box did. However, the cognitive black box provides an illusion of understanding. Readers are referred to Tryon's (2014) discussion of these issues in Chapter 1. Psychologists readily accepted, and continue to accept to this day, this illusion without noticing that they are being duped.

Serious clinical consequences followed from the logical mistake of conflating functional relationships discovered by behaviorists with behaviorism as mentioned in the first paragraph. One such consequence was abandoning clinical assessment by functional analysis and endorsing the Diagnostic and Statistical Manual (DSM) series. A corollary consequence was to accept the medical disease model that all DSMs are based on as a replacement for the behavioral model. Another consequence was to accept manualized treatments as a replacement for customizing interventions to individual needs based on operant and respondent conditioning principles. Empirically Supported Principles of operant and respondent conditioning were replaced by Empirically Supported Treatments that in the beginning did not yet have empirical support while the first studies were conducted.

SEMANTICS AND JARGON

Another possible misreading of Chapter 10 is that it mainly deals in semantics and jargon. Fussing with semantics and/or jargon seems like a waste of time or at least a pedantic academic exercise that has little or no practical value. Would that everyone considered the theoretical differences involved as mere semantics and jargon because then such differences would be unimportant, trivial, and without much effect on the way that psychologists think and behave. If these differences were this trivial then they would not have divided and polarized psychologists in the way that they have. Experience has clearly shown that words carry important meanings that can drive very different understandings to where psychologists form competing schools and camps. That the cognitive revolution over threw behaviorism in psychological science and altered clinical practice clearly demonstrates the conceptual power of words and the theoretical positions that they inspire and support.

CONTRIBUTIONS OF BIO↔PSYCHOLOGY NETWORK THEORY

The synaptic plasticity, epigenetic and glial experience-dependent plasticity mechanisms that mediate learning and memory formation and change provides at least some of behaviorisms missing mechanism information. Operant and respondent conditioning are two ways to activate these mechanisms that have been intensively studied by psychological science. The behavior change technology known as behavior modification has received extensive empirical support across a wide range of clinical disorders. The Bio↔Psychological Network theory is a cognitive

theory that is fully compatible with behaviorism as discussed above. The following issues help to clarify what this theory entails.

Learning vs. Conditioning

Introductory psychology textbooks discuss conditioning as learning. I prefer to understand conditioning as one way to activate the experience-dependent plasticity mechanisms that enable learning to occur via memory formation. One benefit of this view is that it helps us to better understand that extinction entails the formation of new memories rather than the removal of old ones. A fire goes out when it is extinguished. This is not the case when behaviors are said to have been extinguished.

Cognition vs. Conditioning

Anrep made a serious mistake when he mistranslated Pavlov's (1960/1927) word "conditional" as "conditioned". While the two words differ in just their last two letters, their meanings could hardly be more different. The term "conditioned" implies fixed and rigid. It implies something that once done cannot be undone. Pavlovian conditioning was once understood to be the result of a spinal reflex. Conditioning implies that people are unthinking creatures that behave only on the basis reflex. Upon this view people passively respond reflexively to stimuli without any ability to modify or control their behavior. This is the antithesis of a thinking person who develops expectations. Clinicians happily distanced themselves from this limited perspective.

The term "conditional" implies temporary, dynamic, and cognitive. Conditioning is an elementary cognitive process. Behaviors derive from expectations concerning the conditional relationships observed among stimuli and responses. These behaviors disappear if the conditional relationships between stimuli and responses are not maintained. Upon this view people can be seen to develop expectations on the basis of experience. Pavlov was interested in how the frontal lobes controlled behavior. Hence, he was the first clinical neuropsychologist. We presently understand that the frontal lobes mediate cognition.

Conditioning vs. CBT

Historically, contemporary cognitive-behavioral therapy (CBT) derived from behavior therapy (BT) including applied behavior analysis and systematic desensitization. However,

inspection of contemporary CBT publications reveals a consistent absence of operant and/or respondent conditioning principles. For example, Judith Beck's 2011 book entitle *Cognitive behavior therapy: Basics and beyond* does not contain the word conditioning in the index. I believe that this lack of coverage extends to virtually all other contemporary CBT books. The journal entitled *Behavior Therapy* and *Cognitive and Behavioral Practice*, published by the Association for Behavioral and Cognitive Therapies (ABCT), does not discuss conditioning. In short, The B has gone out of CBT leaving CT just as the H has gone out of ADHD leaving ADD. Applied behavior analysts were once the core of what was then called the Association for the Advancement of Behavior Therapy (AABT). But the cognitive revolution brought us ABCT as the applied behavioral analysts formed their own professional association called the Association of Professional Behavior Analysis[2].

Theory & Understanding

Theory is how science understands the world. Theory gives meaning to facts. This is why theory is so important. Behaviorism is a functional theory and like all functional theories cannot explain why its phenomena exist or work as they do. Hence, behavioral explanations were derided as a **black box**. Stimuli went in and behaviors came out but no one had the faintest idea why. Psychologists realized that behaviorism was not psychology but rather a form of behavioral biology. Skinner prophetically left all explanatory questions to neuroscience; i.e., he felt that questions like why a reinforcer is reinforcing were best left to neuroscience because they had the proper tools for answering such questions. Psychologists were unhappy with and refused to accept the explanatory black box that behaviorism provided. Psychologists wanted to know more. They engaged in the cognitive revolution in order to gain more. But this revolution largely abandoned the empirically supported principles of operant and respondent conditioning. Unfortunately, we went from not have a theory of one kind (behaviorism) to not having a replacement theory (cognitivism). What we got were box and arrow models that imputed causation but did not actually explain anything. We received a cognitive black box. Our desire that it should explain what we want it to explain has given rise to the illusion that it does even though it doesn't.

[2] See http://www.apbahome.net/

Theory and Evidence

Facts are just facts until theory is applied to see which ones make sense. Evidence that conforms to theory is especially credible. Replicated evidence that contradicts theory requires that the theory be modified or rejected. But how shall theory be modified? An alternative theory that predicts the anomalous result is required. Existing theory can survive intact in the face of contradictory evidence if no alternative understanding is available.

Investigators who cannot find evidence predicted by theory are likely to have their competence questioned. Take for example the high school or college student whose lab results do not come out as predicted. These data do not cause the instructor to doubt the theory upon which the lab assignment is based.

Recent discussions concerning whether or not priming is a replicable phenomenon illustrates the how theory interacts with data. Our Principle 1 concerning the network cascade holds that activations propagate through the network. Our Principle 2 concerning learning and memory and associated experience-dependent plasticity mechanisms requires that neurons that fire together will wire together. It follows necessarily that repeated subliminal activations can prime, biologically reinforce, particular processing pathways such that supraliminal activation will preferentially follow those pathways. Hence, priming is a hard risky prediction of the Bio↔Psychology Network (BPN) theory. This theoretical explanation and prediction also renders priming understandable. Positive evidence of priming strengths our belief in the BPN theory. Investigators who cannot find positive evidence of priming are likely to be carefully questioned because there are many ways to get things wrong and often just a few or but one way to get something right. Negative evidence of priming would be more persuasive if it was accompanied by an alternative theoretical explanation showing why priming should not occur.

The controversy over whether or not priming is a replicable phenomenon is currently at the "just facts" stage where negative and positive evidence appear to be equally likely; i.e., carry the same weight. The BPN theory increases the credibility of the positive evidence and decreases the credibility of the negative evidence thus giving the edge to the positive evidence that priming is a real phenomenon and therefore can be trusted. The advantage that theory provides here is that it diminishes the importance of failures to find positive evidence of priming. How is one to understand why priming should not occur? Such an explanation necessarily competes with an explanation of why priming should occur. The combination of a theory that explains why priming

occurs and the positive supporting evidence outweighs even repeated failures to find priming because there are many ways of not finding things and without a compelling rationale for why something should not occur, most scientists will side with the theory and positive supporting evidence. Whether priming is so easy and robust that it takes little skill to find it is a different question than does priming exist. Negative evidence requires a theoretical rationale to be convincing.

Thinking Physically; Not Mentally

The BPN theory promotes thinking physically, not mentally, about psychology and behavior. This necessarily means considering the biological mechanisms that mediate psychology and behavior. A common misperception that **biological means immutable** and may make people hopeless that psychology can do anything for them. The view here is that biological treatments are needed for biological problems. Missing from this perspective is our discussion of how experience-dependent plasticity mechanisms are activated by psychological interventions. This makes psychotherapy a biological treatment. The discussion by Tryon (2014) of OCD in Chapter 10 provides brain scan evidence to support this claim.

A related concern is that perhaps too much emphasis is being placed on biology; to the exclusion of psychosocial variables. My effort is towards a comprehensive explanation that includes psychosocial causes that activate the biological mechanisms that mediate psychological and behavioral effects.

Causes vs. Mechanisms

Mechanisms are what enables causes to have their effects. Kazdin (2008) clearly distinguished causes and mechanisms.

> The distinction between cause and mechanism is readily conveyed with the familiar example of cigarette smoking. Cross-sectional and longitudinal studies with humans and experiments with non-human animals have established a causal relation between cigarette smoking and lung cancer. Establishing a causal relation does not explain the mechanisms, that is, the process(es) through which lung cancer develops. The mechanism was shown by describing what happens in a sequence from smoking to mutation of cells into cancer (Denissenko et al., 1996). A chemical (benzo[a] pyrene) found in cigarette smoke induces

> genetic mutation at specific regions of the DNA that is identical to the damage evident in lung cancer cells. This finding conveys how cigarette smoking leads to cancer" (p. 151).

Life experiences are causal. Reinforcers activate dopamine mediated reward circuits that modify synaptic properties. Threatening and punishing stimuli activate adrenalin mediated amygdala circuits. Winning at sports increases testosterone and dopamine. Watching your sports team succeed increases testosterone. Both positive and negative family experiences exert their effects via biological mechanisms. In this sense it is not possible to rely too much on biological mechanisms. The more common explanatory mistake is in not telling the entire story by leaving out the experience-dependent plasticity and epigenetic mechanisms that link biology to the environment and therefore to psychology.

There are no psychological mechanisms per se because there is no psychological substrate for them to operate on. Only biological mechanisms exist. Experience-dependent brain plasticity EDP mechanisms are crucial for psychology. Psychotherapy works via EDP mechanisms. Effective therapists find ways to activate these mechanisms. We must therefore strive to think physical; not mental so that we can increase our awareness and knowledge of how psychological treatments actually work.

MEMORY MODIFICATION

Brian Pilecki's Dissertation[3] is entitled "The role of Memory Modification in the Treatment of Childhood Obsessive-Compulsive Disorder" is the first empirical study regarding a hard BPN prediction. It is based on BPN Principles 2: Learning and Memory and Principle 9: Memory Superposition. Principle 2 requires synaptic modification to store memories. Principle 9 maintains that memories are superimposed upon one another in the same neural networks. It follows directly and necessarily that new memories interact with old ones unless new memories are orthogonal to the existing memories which they almost never are.

Brian screened 18 children for OCD and found 13 who met inclusion and exclusion criteria. They were assessed at intake and again 1 month later. They were given an exposure and response prevention treatment and assessed again post treatment. Four of six hypotheses were partially

[3] Professor Dean McKay is Brian's doctoral dissertation mentor. He is coauthor of the Tryon and McKay (2009) article where the memory modification prediction first appeared.

supported. The complexities of developing a coding system capable to identifying ritual steps was a limiting factor. That participants were children further limited the investigators ability to determine what changed in the way their ritual was remembered post treatment. My suggestion for future research is to video the children while they engage in their ritual at pretreatment and again at post-treatment based on their memory for how they performed their ritual at pretreatment. Timing the ritual duration might be a better way to measure difference than the rating approach Brian took. Omitting steps should shorten the time to complete the ritual. It seems unlikely that new components of equal length to deleted components could be injected.

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