**Lecture 3: Memory-Emotion Interactions**

This is lecture number 3 on Memory-Emotion interactions. This lecture gets to the core of what this whole story is about. A main point that will take a lot of time today is that there are multiple types of memory that interact with emotion. This is kind of the basic science underpinning of differential psychotherapeutics. We'll spend a fair amount of time talking about the scientific foundation and evolution of cognitive and behavioral therapies. Cognitive Behavioral Therapy (CBT) has a distinct advantage relative to psychodynamic psychotherapy because of their claim about a strong scientific foundation, and, in fact, they do have a strong scientific foundation. I want to tell you what that is and contextualize where psychoanalysis is in relation to that. We will then delve a bit into evidence for memory reconsolidation in animals and in humans. Afterwards, we will talk about interactions between emotion and different kinds of memory, particularly episodic memory (including flash bulb memories and trauma memories). We will touch a little bit on this area that I’m trying to define as the foundation for psychoanalysis and psychodynamic psychotherapy, which is schematic memories and their interaction with emotion. I then will tell you about some research that we're doing to advance knowledge. Finally, I want to finish by bringing in a psychoanalytic perspective about memory and emotion. That involves picking up on what Freud talked about in terms of memory re-transcription and corrective emotional experiences, which we highlight as the second step in our 3-step model. One of the starting points for this whole undertaking in terms of understanding common mechanisms of change is the fact that there are probably 500 different forms of psychotherapy, the question remains, how do you choose? Moreover, when is psychoanalysis or psychodynamic psychotherapy indicated?

Psychoanalysis is not for everyone. When I was an undergraduate, 50 years ago, one of my beloved professors who taught my first course in psychoanalysis said, providing psychoanalysis for all mental health problems is like trying to solve the transportation problem by giving everyone a Rolls Royce. That was my perspective for a long time. Is psychotherapy like an automobile? There are hundreds of makes and models, some are faster, some are slower, some people are city drivers, some want to go off road or have electric cars, and some people don't mind using gas. However, don’t all cars basically work the same way, and don't all cars help you to get where you're going? The answer is yes, perhaps they do. But maybe it matters what your goals are. People aren't all starting from the same place, and they're not all going to the same place. The nature of the problem and what you're trying to achieve matters. Now we have online approaches to psychotherapy which is starting to get close to the issue of public transportation, and certainly has a role, and it can be great. It's great here in Vienna that's for sure. One of the ways that the field has grappled with the complexity and diversity of different psychotherapies is to talk about common factors in psychotherapy. I’m quoting from a review article from 2021. Common factors include: a strong therapeutic alliance, higher ratings of therapist empathy, positive regard of the therapist for the client, there is genuineness and more favorable outcome expectations are related to improved treatment outcomes. That's very well established and transcends modalities. However, debate continues regarding whether psychotherapy outcomes are most strongly determined by these common factors, or by factors specific to the type of therapy used. This review paper said an integration of the two perspectives and a shift toward evaluating mechanisms is a way to move the field forward. To figure out what's specific about different therapies and how they work. I did a little thought experiment about pharmacology in general. Think about medications for asthma, hypertension, pain, and bacterial infection. You find common factors, no matter what class they're in. You must have a certain amount of bioavailability. A good drug has few side effects, and you need a reasonable half-life. These are common factors. The specificity matters when you're targeting specific path of physiology. That’s what we might be after with psychotherapy. I was thinking about psycho pharmacology in psychiatry. I looked at these common factors and thought to what extent do they help determine outcome of medication management for depression and anxiety? Therapeutic alliance, higher therapist empathy, positive regard, genuineness, and favorable outcome expectations, influence outcome. Therefore, one of the challenges is demonstrating that psychotropic agents of specific efficacy above and beyond placebo and all these factors are clustered into what placebo is. Placebo is real. There are real brain changes associated with placebo. What we're after is trying to look at specific mechanisms, not just common factors.

Jeffrey Smith is a psychiatrist in New York who is the head of the Psychotherapy Caucus of the American Psychiatric Association, and incoming President of Society for the Exploration of Psychotherapy Integration. He's very interested in defining common factors and common mechanisms. We've talked about different kinds of psychotherapy. There may be a common infrastructure to all therapies. Dr. Smith says all these therapies and facilitating factors on the you might think about as common factors: arousal regulation, motivation, safety, relationship, having a good therapeutic alliance, and these three key steps you must reactivate, revise, and reinforce new learning. The key point is, there are these three final common pathways that all involve memory. According to this, you have three choices. One choice is that you can keep the old learning intact but develop new learning and practice it so it out competes the old learning (but the old learning is still there). The second one is, you can suppress the old learning, the old learning is still there. That's extinction but you know it can provide relief sometimes for a sustained period. Memory reconsolidation is the only one that really involves changing the memory, and therefore that's the idea of enduring change less prone to relapse. I've emphasized memory reconsolidation is a critically important mechanism for enduring change. Especially as it applies to psychoanalysis and psychodynamic psychotherapy. It’s important for you to know that there is criticism of this by Meryl Kindt and her group from Amsterdam. So, by the way, if you talk to friends and colleagues, and you say, “hey, memory reconsolidation is really it.” they'll say, hey, what about this alternative perspective, you should know about it. The review article says it’s premature to invoke memory reconsolidation as an explanation for change in psychotherapy until it's been demonstrated. They have tried very hard in Amsterdam to demonstrate it, and they haven't quite been able to meet all the criteria for proof. It's not yet been possible to demonstrate change eliminating symptoms as an outcome in anxiety disorders based on a classical conditioning model which is the closest human analog to animal research where memory reconsolidation has been demonstrated. So, explaining what's already being done in therapy based on memory reconsolidation is a major error. What I'm proposing is not simply explaining what's already being done based on memory reconsolidation, but I think memory reconsolidation has implications for how you should do the therapy.

There are 4 different criteria to demonstrate that memory reconsolidation has happened. 1) You must have a reactivation by memory interaction. 2) You must demonstrate time dependency, you must intervene in that window of 4 to 6 hours after memory's been activated.3) You must show memory specificity, that you're manipulating one memory and not another, and the one you manipulate changes, and the other one doesn't. 4) You must demonstrate dissociation of immediate and delayed effects. You should not see an immediate effect, but then, after a night of sleep you should see the change, and you should also see the change in long-term follow up. That is super difficult to demonstrate, and that's why we don't have absolute definitive proof in humans.

I am going to list 7 different kinds of memory that interact with emotion. We have classical conditioning: the associative pairing of unconditioned stimulus and condition stimulus. We have associative learning: emotionally neutral content plus unrelated emotional arousal. Reinforcement learning or operate conditioning: the affective consequences of an action change the value of the action. Procedural memory I've included here, after our reading of the Boston Change Process Study Group where we're talking about how different kinds of actions have emotional consequences, and as a behavioral form of emotion regulation. Moving into more complex kinds of memory, the kind that people can recall and put into words, episodic memories with strong emotional content versus those without trauma memories would be included here. We have semantic memory, schema being one example of that with inherent emotional content. Shout out to Bruce Ecker, who is the first person to talk about memory, reconsolidation, and psychotherapy in a book over 10 years ago. Finally, the internal working model of the social world, which would be a combination of schemata. So, about the Meryl Kint criticism, let's say you could demonstrate anxiety disorders from a classical conditioning model change with memory reconsolidation. Would that really get us where we want to go about psychoanalysis and psychotherapy? I would say no, because we're talking about more complex forms of memory that involve complex interactions between situations, behaviors and mental representations of people and emotions. Is animal research relevant to psychoanalysis? Are we rats passing levers for awards? No. Don't, we have free will, are we self-aware, aren't we interested in the meaning of our experiences unlike other creatures? Yes. I’m going to invoke Freud here. A starting point for Freud is that humans are animals with basic drives. How can these basic jobs be reconciled with the demands of civilization? The learning mechanisms that we've retained through our evolutionary history are relevant and important.

I was a psychology major in college and received a PhD in experimental psychology. When people talked about classical conditioning and operant conditioning I glazed over. I felt this wasn’t relevant to what I'm interested in. Now I can tell you, I find this fascinating because we're really trying to figure out about memory and emotion interactions as the basis of all psychotherapy and trying to figure out how psychoanalysis and psychodynamic psychotherapy fit in relation to these other modalities. Also, what about the claims that they have such a strong scientific foundation? I’m not convinced that they do, and I will tell you why. It puts it in perspective as to where we are and what we need to do to advance psychodynamic psychotherapy.

So classical conditioning and operating conditioning. Classical conditioning is really about the stimuli. Specifically, recognizing or learning the association between a neutral stimulus and the association with danger. Whereas operant conditioning really has more to do with the response. For example, we see this dog, and it's been trained. The whistle is the conditioned stimulus and it's been paired with the food. When you blow the whistle, the dog will salivate. That's classical conditioning described by Pavlov in 1897. We're going to see the historical progression. Freud wrote basically around the same time. Then there is operant conditioning, for example, when a rat gets a reward for pressing a lever. It learns to do that repeatedly. This is the reward, this is the pairing, and this is the reward value of actions. BF Skinner described this in 1937 and developed very elaborate explanations for human behavior based operant conditioning. 10 years later there's Mowrer’s 2-Factor Model of Avoidance Learning Mowrer was interested in integrating these two different theories of learning (i.e., classical conditioning and operant conditioning). He was interested in trying to understand how these would be relevant to anxiety disorders. Classical conditioning alone wouldn't typically explain an anxiety disorder because you must keep repeating the pairing intermittently to keep the conditioning going. If you don't do it, the learning extinguishes. How can classical conditioning explain clinical anxiety? Operant conditioning includes both positive reinforcement (e.g., you get the food if you press the bar) or negative reinforcement, (e.g., when you avoid something negative that's reinforcing. For example, if you've previously been assaulted in a parking lot and you think about going somewhere in a parking lot, that will raise your anxiety and may make you think, ‘okay, I'm not going to go there, I can skip it.’ That thought becomes relieving and reinforcing. That avoidance reinforces a connection between the conditioned and unconditioned stimulus. That can explain the perpetuation of the initial conditioning. Avoidance as a basic mechanism in psychopathology, and the need to overcome that is described by David Farlow and unified protocol for reading anxiety, and depression. This is the basis for it.

The combination of operant and classical condition can explain clinical anxiety. To summarize extinction: we're carrying the bell with the food, then you keep ringing the bell, and then you salivate. After a while you stop salivating. Once you have a model, you can develop it further to enhance it for clinical purposes. So here are some examples of how extinction can be enhanced. You can increase the number of trials. Let's say people were assaulted, and they developed a conditioned response to parking lots. You could present parking lot stimuli repeatedly. You could also switch contexts to avoid context specificity. You could go to the original parking lot but then you could go to other parking lots. Presenting the aversive outcome or a non-extinguished queue, together with an extinguished queue. You could habituate or extinguish the original parking lot, but then you might show a picture of someone who was like the one who assaulted you. That would enhance the extinction and make it more generalizable. You could replace the aversive outcome with a novel, non-aversive outcome. For example, you go to the parking lot, and win a prize. That'll change your association. These adjustments can enhance the strength and generalizability of extinction. This illustrates the benefits of a validated model for improving and refining clinical interventions. I think it would be nice if psychodynamic psychotherapy had a similar model where you could say, okay, this is really the fundamental mechanisms. Now we can tweak it this way in that to enhance it. We don't have that yet.

In 1958, systematic desensitization was defined by Wolpe. His work was inspired by the idea that if you have conditioning maybe you can counter condition by pairing relaxation with anxiety or fear at the same time. It turned out that didn't work. The reason it didn't work, he said, was because individuals cannot be both relaxed and anxious simultaneously. So, he developed systematic desensitization which did work. You develop a hierarchy of the patient's fears, and the patient is systematically exposed to increasingly threatening stimuli. Here we have a young lady looking at a picture of a spider far away. Then the picture gets closer. Then we have a real spider far away, and gradually move the real spider closer. And now she's inviting the therapist to bring the stimulus closer, now we have the spider closer, and now it's very close to her face, and then finally she's touching it. This is kind of a counter conditioning where a relaxation response is introduced with the phobic stimulus. As a result, patients improve their distress tolerance. They learn to tolerate and improve functioning in anxiety provoking contexts. The initial pairing doesn’t work, but the systematic desensitization does.

The cognitive revolution and the advent of CBTs in the 1950s and 60s. To provide perspective, psychoanalysis focused on unconscious determinants of behavior which are difficult to observe, measure, and test. Learning theory (i.e., classical conditioning and operant conditioning) focused on environmental determinants of behavior which are much easier to observe, measure and provide scientific support. Now in the 1950s and 1960s, there's a new observation. The way we interpret stimuli influences our emotions and behavior. The mind matters again. Albert Ellis developed rational emotive therapy, Aaron Beck established cognitive behavior therapy. Aaron beck was a psychoanalyst and noticed that his participants kept having negative thoughts, and he thought those negative thoughts were contributing to their mood. He thought, why not change your thoughts, that might improve your mood? That's the starting point for CBT. In CBT, patients identify irrational beliefs and negative thought patterns and change them to improve mood and behavior. There's no question that this helps some people, maybe it helps a lot of people. There's a question about how long it lasts, but it's It brings a lot of relief. CBT isn't for everyone, nor is psychoanalysis.

The bio-informational theory of emotional imagery by Peter Lang in 1977 showed that imagery, imagining things (not just thoughts), can evoke an emotional response. The image in the brain is indicative of a conceptual network controlling specific, somato-visceral patterns constituting a prototype for overt behavioral expression. Now we're getting into complexity with bodily physiology, with motor behavior networks in the brain, and emotions. Emotional imagery may elicit similar physiological responses in both peripheral and central nervous systems as would be evoked during actual experience. This advance permitted the advent of behavioral fear extinction through imaginal exposure therapy. I do want to say here that I know Peter Lang, and he was very influential in my own career. When I got this scientist development award in the nineties, we were among the first to do functional brain imaging of emotional states, and Peter Lang was extremely helpful. We used his international affective picture system to evoke emotional responses in the scanner. The title of my PhD dissertation was a functional neuro anatomy of pleasant and unpleasant emotion, based on that work and other work. As a further testament to this idea that images activate complex patterns in the brain, subliminal exposure to images of feared stimuli can induce extinction to conditioned fear. It's a way around presenting people with nasty images or nasty memories consciously and trying to bring some relief. We keep talking about anxiety disorders. That's a narrow domain, and in general psychoanalysis, psychodynamic psychotherapy we are interested in symptoms, but also particularly personality disorders.

Emotion processing theory in the eighties. Rachman described emotion processing as the process by which emotional disturbances are absorbed and dissipate, including experiencing and expressing the distress. Edna Foa and Kozak argued that exposure therapy alone doesn't work simply by inhibiting the conscious or unconditioned stimulus associations. An essential ingredient in exposure therapy is the experience of safety. You must be exposed to the public stimulus. and then experience safety to get better. She would say, it's the introduction of corrective information during exposure and additional cognitive processing is imperative for success. The concept of corrective experience has really gained currency in academic psychotherapy circles. It's now widely seen as useful across a variety of psychotherapy modalities. Notice I am saying corrective experience. I'm not saying corrective emotional experience, because there's no consensus about how corrective experiences work. There is debate, is emotional experience necessary? That's debated. You now know something about priors and prediction errors. Are predictions that update priors sufficient. You don’t necessarily have to have the emotion. That's where the field is. Consider about what Edna Foa was talking about with exposure therapy. Exposure therapy is kind of like the leading treatment for PTSD these days, and it's close to these three essential ingredients from enduring change in psychotherapy. You must activate old memories and old feelings, without awareness of their connection to the past. Concurrently engage new emotional experiences that change old memories through reconciliation. And then you reinforce the strength of new memories and the semantic structures by practicing new ways of behaving and experiencing the world in a variety of contexts. There’s a kind of convergence going on here, and I'm claiming that the same principles apply to psychodynamic psychotherapy and psychoanalysis.

Now I want to transition talking more about memory reconsolidation and interactions between memory and emotion. Lynn Nadel, my colleague at the University of Arizona, as well as senior editor with me of the book and co-author of our paper in 2015. He's really been at the forefront of memory research in the past half century. He was very involved in what can come to be known as memory reconciliation. He and Moskovic in the late nineties describe multiple trace theory. Previously the hippocampus was only thought to participate in the creation of new memories. The role of the hippocampus in memory formation and memory involvement was thought to end with consolidation, the transfer from short term to long term memory. That fit with the observation that amnesia typically is characterized by an inability to make new memories so called anterograde memory (i.e., from the time of the injury forward, you can't make new memories). However, they observed that patients with amnesia showed unexpected deficits in details of remote memories (retrograde memories). This suggested that remote memory was changing over time. They hypothesized that the role of the hippocampus was not time limited. They went on to do functional neuroimaging to support that. Multiple trace theory was a forerunner of memory consolidation. It redefined memory as undergoing constant revision. The idea that retrieving a memory itself puts the memory in a labile, transformable state. What I’m going to describe here are hippocampal cortical complexes or memory and how it changes. So hippocampal traces are distributed ensembles. The hippocampal traces act as an index to the distributed cortical regions that represent various aspects of the episode. A memory has visual information and auditory information. That's why we think that memories involve networks and multiple brain areas and the hippocampus kind of organizes it. Kind of indexes it. Now the hippocampal cortical complex is the episode memory trace. The hippocampal trace not only binds elements in the cortex but it's also the repository of critical episodic components for contextual detail. Okay, that's part of what the hippocampus does. What happens with reconsolidation? Basically, you're adding one new element to the hippocampus, and the other elements coalesce into a new trace. Reactivation retrieval occurs in a different context and results in a new, sparsely distributed ensemble. The idea is that when you recall a memory, say in therapy. you're in a different context from when the memory happened originally. That information of the new context gets added in right to update the memory. This was prior to the animal studies showing memory reconsolidation.

A breakthrough study in 1997 involved rats learning a maze task. NMDA receptors play an important role in experience dependent neuroplasticity. Blocking NMDA receptors interferes with synaptic changes associated with learning. The study examined the timing of intra peritoneal injection of an NMDA antagonist in rats after various stages of learning a maze task. They did it at different times to assess at what point these receptors are playing a role in learning. They observed that for a task that was already well learned, injection up to three hours after a new trial, injection of this agent disrupted performance 24 hours later, suggesting that the memory was made labile by reactivating it. We've previously talked about the Nader and Ladeu study. This is the one that's gotten the most attention. It was published in a higher Profile Journal in Nature. But what did it involve? It involved a replication and extension of the previous study. It used a different kind of learning. Instead of maze learning, it involved conditioned fear, a different drug, anisomycin instead of NMDA antagonist, it had more anatomical specificity, and there are also many more controls. It was a more definitive extension and replication that really convinced people that this is a real phenomenon. What kind of evidence do we have for memory reconsolidation in human beings and healthy volunteers? This is work done at the University of Arizona with Lynn Nadel. There was a first session you brought people in, and they learned new information, they learned lists and they heard stories. They came back for a second session a couple of days later and they were divided into two different groups. Half the subjects were given a reminder of the first session because they were brought to the same room, they had the same experimenter, and were even asked the question about what it was like to reactivate the memory. The other half of subjects didn't have any reminders at all. Then they learned a new set of information, new lists, and stories. What we're interested in is what happens when you learn new information having had the previous information reactivated, put into a labile state, or no reminder, no reactivation, not putting the information into a labile state. They came back two days later and were tested for their memory information in the first session. First sessions are important because in one group it was made labile, and in the other group it was not made labile. This is what they found with recall from the first session. The no reminder group and the reminder group. The number correct recall from the first session was quite comparable. But there was a big difference in intrusions. That is, to what extent did information and the list from session to, get incorporated into a recall of session one lists and stories. What they found was that there were many more intrusions when the first session information was made labile right. The other information that was being learned could infiltrate, whereas if it wasn't made labile, it was much less likely to happen. Now, this doesn't meet all of Meryl Kint’s criteria, but It's interesting. Initial criticisms were, maybe people were confused, they didn't remember exactly what you were asking. There’s now a meta-analysis of this, and dozens of studies have been done to show that there is an overall effect. They show that episodic memory does change when you activate memories, and then add new information simultaneous. Meta analysis shows an overall effect with an effect size of .29, which is considered small and is highly, statistically significant. There are several different studies that were done obviously with different designs. They found larger effects with older memories. That would mean, memories that maybe are not particularly well rehearsed, and may be more fragile or narrative memories that had more elements to them that could be updated. There was a large effect for the number of intrusions.

This is from that review article from Meryl Kindt, James Elsey, and Vanessa Van Ast. It's kind of a nice way of summarizing the evidence for memory reconsolidation in humans in different contexts. We have declarative memory, retrieval extinction, behavioral interventions, pharmacological interventions, procedural memory. We're simplifying psychotherapy by talking about memory-emotion interactions. As we get into it, we realize that variations are illuminating, and it gets kind of complicated. What are the alternatives to memory Reconsolidation? Well, we talked about extinction as one. Another one is enhanced retrieval competition. You could have new learning that out competes old learning, but you could also have retrieval of pre-existing memory that's enhanced as a way of explaining improvement. And then this one was a little tricky, impaired retrieval of problematic memory. There's a good example of that coming up. We'll talk about one of the approaches to dealing with traumatic memories is to give people propranolol and you give that to them before you reactivate the memory. That does have the effect of reducing PTSD symptoms over 6 sessions. There's this interesting observation that propanol inhibits retrieval of conditioned fear. The conditioned fear may still be intact, you just have trouble reactivating it in the context of propanol. The issue is that the memory is still there, and you’re trying to change the memory. Within the domain of memory reconsolidation there are various things that can happen. We talk about updating. I think that's the most reasonable thing that you can do clinically. You're adding new content, you're revising old Content. Erasure is what everybody wants to do right for bad memories. That could have some bad side effects, unintended consequences, but it only can be done in animals, it's not happening in humans and we're not close to that. You can weaken memories through reconsolidation, but you can also strengthen them. I think the old habit of having people reactivate painful memories and just having them dwell in painful affect, and then the sessions up and you see them later can strengthen the pathological memory. That's why I think it's important to have a safety experience or a corrective experience of some kind. This corresponds to the evidence regarding reconsolidation that I mentioned. Episodic memory, single episodes, are easiest to update. It stands to reason the semantic memories which are distillations of episodic memories are harder to update and procedural or habitual memory are the hardest and may not even be possible to update at all.

Now, we're going to talk about emotions. Synaptic plasticity, which is the molecular basis for encoding memories, is enhanced by the neurotransmitters hormones norepinephrine and cortisol that are activated by emotional arousal. This study by Larry Cahill was very interesting and was the basis for what inspired Elaine Brunet to develop propanol assisted memory consolidation therapy for PTSD. Larry Cahill at the University of California, Irvine in 1994 had participants take propanol or placebo, prior to hearing a story that had neutral details, or another group story that had both emotional and neutral details. Two days later they return for a surprise memory test. The placebo group had more accurate memory for the emotional details. The propanol groups recall of the emotional details was blunted and accuracy for the emotional details was comparable to recall for the neutral details. It was only the placebo group with the emotional content that had recall of the emotional content.

Emotion enhances encoding and retrieval of episodic memories, and that's based on a lot of evidence in the chapter that was assigned. In general, this phenomenon of mood congruent memory was well established. That is, when you're in a certain mood state, it's much easier to recall other sad memories. If you're happy, it's much easier to recall other happy memories. The emotion is facilitating retrieval. Emotional details of an episodic memory are selectively retrieved relative to peripheral details. Emotional content amplifies certain aspects of a memory more easily recalled. What about encoding? Various studies have looked at the effect of emotion on neutral stimuli. Emotion enhances memory for simultaneously presented neutral stimuli. Emotion enhances memory for subsequent neutral stimuli. You are emotionally activated and then you hear some neutrals stimuli, and the emotion arousal carries over. Emotion enhances memory or previous neutral stimuli. The conclusion is emotion strongly influences both encoding and retrieval of episodic memories. I'm trying to lay the evidence-based foundation for arguing that emotion is very important in psychotherapy if you're trying to change memories. Behavioral tagging, published in Nature 2015 describes that subjects look at pictures of tools and animals. Why, Tools and Animals? Animals are natural and biologically prepared stimuli, Tools are man-made. A short time later a shock was delivered while viewing a tool or an animal not previously seen. You're exposed to tools and animals, you encode them, then later you get shocked as you see the tool or an animal. This results in an improved recall only for the items in the same category as the shock that had you been exposed to previously. This effect occurred 6 to 12 hours later than the task, suggesting that consolidation was necessary. Strong encoding with the shock enhances learning of related items that were previously only weakly encoded. Dopamine released by the shocks may be a mediator. The idea is that actual memory formation takes a while, and that the neurochemical milieu makes a difference. Remember how we talked about how the reconsolidation window is open, after the session ends for another 4 hours. It's relevant what happens emotionally after the session.

Flashbulb memory is a vivid long-lasting memory about a shocking event that happened in the past. Subjects were interviewed a few days after 9/11, 11 months later, 3 years later, and 10 years afterward. They were asked to write questions, personal details, about where they were, what were you doing at the time, and where was President Bush, what airline was it etc...? There was a surprising decrement in personal detail recall 11 months later. Interestingly, people were very sure that their memory was accurate, even though it had changed. Memory, stabilized at 11 months and didn't change that much after 3 years and 10 years. The point is that even strong memories are malleable. Now let's talk about trauma memories. If arousal level is too high, the hippocampus cannot adequately encode context. I've mentioned this before. This can lead to over generalized sensitivity to threat queues, and any complex cognitive function declines around the levels too high. Hyper arousal impairs medial prefrontal amygdala encoding of specific emotions. Improvement of PTSD symptoms with CBT involves top-down modulation by the prefrontal cortex. We see extinction being mediated by connections between the prefrontal cortex and the amygdala. What about the use of propranolol, developed by Elaine Renee, inspired by Larry Cahill’s work. This is a treatment for PTSD. In successions you have people take certain amount of propranolol an hour before each session. In the first session you create a detailed narrative, a couple of paragraphs long, of all the sights, sounds, thoughts, and experiences about happened in the trauma. You do that repeatedly for 6 sessions. This is a placebo-controlled trial, half of the subjects received the placebo before they did this recall, and the other half got propranolol. There's a more rapid decline in PTSD symptoms with propanol compared to Placebo. There’s a slight decline in the placebo group and that may have to do with extinction with repeated exposure of the traumatic stimuli. There have been meta-analyses that have been done, and some meta-analyses clearly support the effectiveness of propranolol relative to placebo and other met analyses don't support it. I'd say the preponderance of the evidence does support propanol relative to the placebo, but that doesn't prove memory, reconciliation. With trauma, you have hyperactivation of the amygdala and inhibition of the hippocampus. With this intervention you have the reverse; the amygdala is inhibited, and the hippocampus is intact, so that the memory can reconsolidate without that high level of emotional input. The theory makes sense, but that doesn't prove it's reconsolidation. We have extinction, and, as I mentioned before, propanol impairs retrieval of conditioned learning. So it could be that, in fact, people have less distress. It’s not because the memory is changing. It's because they're not able to activate the memory. A very important point that Cahill gave propanol before the initial encoding. Now we're trying to change something that has been encoded again. That's the whole idea of reconsolidation, and that may account for some of the difference.

More posterior structures in the hippocampus and temporal lobe have more detailed cues, like remembering the tenth birthday party and the cake at the party. More anterior hippocampal structures have more gist features; mom’s house and the tenth birthday party in general. The prefrontal cortex is where we're getting into abstraction. That's where we have schemas and more generic memories, like a house in a party, and so a house cue might reactivate the whole memory. This whole area of the neurobiology of schematic memory is relatively new. The whole area of emotion updating schematic memories is even newer. That is why I’m teaming up with people to do research on us. I was talking to Lynn Nadel, 4 years ago. How can we possibly move this forward with imaging and the scanner. What we're really interested in is the internal working model of the social world. He said, oh, why didn't you say that before? There’s this nice paradigm developed by Daniella Schiller and her group at Mount Sinai in New York, involving social navigation in the human brain that's basically tracked by the hippocampus. This is published in neuron in 2015. The basic idea is that there are two fundamental dimensions of social interaction: power and affiliation. They created a video game where people arrived in the new town, and they had to find a job, find a new place to live, and they interacted with people. They set up the interactions to give people choices that you could track where people were on these two dimensions of power, dominance, affiliation, or friendliness. Here is an interaction in the game. The boss says I need you to stay later, we need to have it ready in two days. You have a choice. You could say, well, whatever you need boss, or you could say, I’m putting my best efforts in this project, can I get paid extra time? You answer these questions, and then you, reliably get placed on a dimensional grid where you fall between power and affiliation. Well, it turns out that the hippocampus is tracking that. It's kind of a social map if you will. We have spent the past several years trying to take this paradigm and create emotional learning. For example, you have an interaction, say with the boss, and he's kind of nasty. Then, we come back the next day, and we have new interactions with him where he's apologetic, nice, and very helpful. You're trying to update a negative memory with corrective emotional experience. After a series of 4 or 5 studies, 1000 subjects, we have been able to show behaviorally that the affective response to certain people changes and you can play a trust game, that behaviorally demonstrate more trust.

So now we've developed the paradigm so we can do it in the scanner. We can ask, can schematic memories be updated by new emotional information? Does this updating increase trust in the other person, and does such updating involve interactions between the amygdala, hippocampus, and prefrontal cortex? We want to apply that to clinical material. With clinical material it's not just new learning that you're changing with another new learning, it's deep learning that you're trying to update.

My last section here has to do with applications to psychoanalysis. Where does psychoanalysis stand in relation to all this? Freud’s concept of “Nachträglichkeit.” In 1895, Freud described memory retranscription (also translated as deferred action), as the pathogenic effect of a dramatic memory occurring in childhood, manifesting retrospectively when the child reaches a subsequent phase of sexual development. For example, a young child might be sexually molested and doesn't really know what that means, but then reaches puberty, and realizes this is horrible. Retroactively, this experience is reinterpreted as a terrible thing that wasn't recognized as such. Freuds theory of deferred action can be simply stated that memory is reprinted in accordance with later experience. He used the concept again in 1918, but he never offered a definition, much less a general theory of deferred action. It's generally accepted that Freud muted as part of his conceptual repertoire, but it wasn't developed. French psychoanalysts, however, thought that it was very important. Lacan considered the real implication of the nachtraglichkeit has been ignored. It was there all the time, only to be picked up, and Laplanche, a French analyst, who was analyzed by Lacan, Freud's concept of deferred action contains both richness and great ambiguity between retrogressive and progressive directions. Analysts have written about this concept to understand the transformative, effective interpretation on previous understandings and personal narratives. It has not yet been used to describe a general model of enduring change within psychoanalysis, which is what posing here.

Corrective emotional experience. This idea was put forward by psychoanalysts Fronze Alexander and Thomas French. The corrective emotional experience was the fundamental therapeutic principle of all etiological therapy, and their definition meant to re-expose the patient under more favorable circumstances to emotional situations which he cannot handle in the past. The patient, to be helped, must undergo a corrective emotional experience suitable to repair the traumatic influence of previous experiences. Intellectual insight alone is not sufficient. Well, historical objections to the concept of corrective emotional experiences by psychoanalysis. This was the predominant view that it was not a good idea. It was rejected by mainstream psychoanalysts for a variety of reasons. First, it was thought to be contrived. It was misunderstood what Alexander had in mind. They were thinking, you have to kind of create an interaction that wouldn't otherwise occur, that was corrective. It’s not part of the natural process. It maybe had unrealistic characteristics, but most importantly, it involved a de-emphasis on interpretation and insight and unneeded emphasis on emotional experiences. It was thought that in the interaction, the transference relationship with that there's plenty emotionally potent. You didn't have to put any special emphasis on emotion. Moreover, this idea did not adequately consider the transference relationship with the therapist. I have thought about this and asked, what about corrective emotional relationship? It retains the primary focus on the transference as the focal point of therapeutic interaction and psychoanalysis. It bypasses conceptual baggage of corrective emotional experiences. It captures and highlights, abundant, relevant, implicit, as well as explicit emotional processes, and the therapeutic interaction. It provides repeated emotional responses and experience inconsistent with expectation. Old memories and all feelings are activated entirely consistent with how memory reconsolidation works. It provides a plausible explanation for how emotion and schematic memories that are older, are stronger and can be slowly updated over time.

In conclusion, psychotherapy across a range of modalities addresses maladaptive, emotional learning that is memory emotion Interactions. I hope I've convinced you that across all these different modalities. A variety of types of memory interact with emotion contributing to a range of disorders and a range of treatments. CBT and related modalities have a strong tradition of basing interventions on validated scientific principles. Psychoanalysis and psychodynamic psychotherapy have fallen into disfavor because of the history of rejecting objective research as useful, and therefore lag. Psychoanalysis and psychodynamic therapy have demonstrated effectiveness, but the mechanisms of enduring change remain to be elucidated. Schematic memories capture more complex patterns of dysfunction comprising recurrent maladaptive patterns that transcend specific situations and time periods. Classical conditioning is not going to explain what we're interested in. Neurobiological understanding of schematic memories and they're updating through reconsolidation of new emotional experiences in its infancy. This framework potentially provides an opportunity to validate time-honored aspects of psychoanalysis and psychodynamic psychotherapy. Improve their effectiveness and efficiency addressing experiences versus an understanding and establish a unique niche among the range of psychotherapy modalities available.