Well, hopefully our technological difficulties will be over for the day, but it’s such an honor and a privilege to be here today. And I have to say, when I first started bringing people together, it was really out of a luncheon that I had with Dave Corwin. And I just said, “This just can’t happen anymore; we have to do something about this.” And he said, “Well, why don’t you bring some people together and see what happens?” And they…definitely under the tutelage of Dr. Corwin, I have just learned an immense amount across the last decade or so. And especially, it’s been an exponential curve working with a multi-disciplinary group with the Trauma-Resiliency Collaborative. And I was just absolutely thrilled to have Carol Anderson be a part of that group, because where we have to start is really with the kids. And by addressing what’s happening in these younger years, we definitely can have a very significant impact and be preventative, as Jennifer Oxborrow mentioned earlier, and also Dave Corwin just mentioned.

[Slide: Speaker Intro] So these are my credentials; I don’t have any disclaimers. I'm kind of like a free agent, and I am an assistant professor; now I am officially, sorry, an adjunct faculty at the University of Utah. I spent some...a number of years there, and now I am actually the director for what’s called the sort of, community treatment team. And the reason why I am pointing that out is, that team is—it’s an old idea, but novel to Utah—and that targets people with very severe mental illnesses who require a lot of supportive care. The reason, and especially I am bringing that up today, is because pretty much 99% of those people on our team have extremely high ACEs scores. And it makes me take pause thinking, what could have happened? What interventions could have been done for these folks; how could they have been identified earlier on and maybe not have the extreme adversities that they have faced life-long?

[Slide: Points of Discussion] So points of discussion today will be...I’ll be talking about the difference between stress versus trauma, and then the physiological changes. And that’s going to be, really, the bulk of the talk, and that is really on the whole college level, university-level course, but we’ll be just touching the tip of the iceberg on that. And then, what are those
behavioral responses that come because of those physiological changes? And then we’ll just wrap it up briefly about what are our responses to those behaviors that we see in other people.

[Slide: A Wonderful Life] So Alice Miller—she’s one of the...she’s really the grandmother of looking at childhood trauma. She has contributed significant works on childhood trauma from the physical, emotional abuse, sexual abuse. And I love this quote: When we treat children with dignity and honesty, then they grow up to be intelligent, responsive, and empathic. It makes sense. We are talking about common sense here. If we are treating an animal in an abusive manner, we are going to expect a very violent animal that is very reactive. We are animals; we’re no different than other species. And that is the appropriate type of interaction that a child should be exposed to. No child should ever be exposed to violence and/or to abuse.

[Slide: Structures of the Limbic System] So what are the different areas of the brain that we are going to be talking about? I’m, again, giving you a very brief overview of what is involved here neurobiologically. What we are looking at here is called the limbic system. I don’t know if any of you are familiar with the limbic system. The limbic system is our emotional engine. It is what drives all of our emotional responses, and it also branches out to pretty much all of the different regions of our brain, too. So it has an extremely important function here. And the areas that are highlighted, we are going to be talking about in a little bit more detail, [if this works. Let’s see.] Okay, so the amygdala; amygdala has been really the cornerstone structure that has been studied with regard to effects from PTSD, and I would say also anxiety disorders in general. One of the reasons why anxiety actually has a little higher prevalence by scientific thought currently, in women—higher anxiety in women—is because the amygdala is actually a little bit smaller in women, and perhaps that gives more of a vulnerability for anxiety in general, and then also, perhaps vulnerability for PTSD. But the amygdala is responsible for our sociability—how we are interacting with other people—and this is all a normal state. It’s also responsible for how we approach fear and our aggressive responses, and also how quickly we calm down from those. It’s responsible for those memories directly related to that fear response or that anger response, and it’s also highly involved in pleasure seeking, and very importantly, it is also responsible for pheromone processing. Why is that important? We will talk about that a little bit later when we are talking about trauma reactions; but when we think about early neurodevelopment for children, they can recognize parents’ smells. They can recognize those pheromones of their parents, and they have that...it assists with that bonding and soothing responses.

Another structure, this long green area—it’s called the hippocampus—and that is very essential for turning short-term memory—what we are experiencing right now in this room—and putting it back into our brain, locking it in for a long-term memory. Just an aside, that’s one of the first areas that’s affected with Alzheimer’s, and a lot more research is actually coming down the pathway now for hippocampal research. And it also helps us to navigate, and it doesn’t mean just navigating how we are getting from the hotel to back to our home, but navigating ourselves in the atmospheres or the worlds that we live in—so navigating how we are going to strategize, how we are going to interact with other people.
And then the cingulate cortex is another major structure in the limbic system, and this assists with cognitive flexibility. I don’t know if any of you are familiar with that term, but as a psychiatrist, what I am looking at there is, how rigidly does a person think? Can they extrapolate, or does everything have to be very concrete? Can they apply certain knowledge, and then expand upon that, so we can have a more creative approach to life and have more options. It also relates—as you can extrapolate from that cognitive flexibility—to our social adaptation. If Dave Corwin—I see what he looks like physically—if I see another person who looks like Dave Corwin when I am walking down the street, I can socially adapt and say, “Oh, that’s not Dave Corwin,” and he may be a very different person and a very different personality type. This becomes very important when we are talking about trauma reactions, which we will talk about a little bit later. And then it also is responsible for helping us to learn what to avoid. We don’t want to have consequences. We don’t want to repeat the same behavior over and over again that gets us into bad situations.

And then we have the hypothalamus. The hypothalamus is really implicated in regulating our body, regulating that connection from our brain to our body. And last time I checked, my brain was still connected to my body, which is a good thing. But often times in medicine we forget that the brain is connected to the rest of our body, but it has very significant impact then whether we are having a healthy lifestyle or whether we are having a negative lifestyle. And then the hypothalamus connects to the hippocampus; so it goes back to the hippocampus, which is important to remember, because that little pathway there also intertwines then with what’s going on with that short- and long-term memory, and how does that short- and that long-term memory then affect what we are doing physiologically in our body. Does that all make sense? That’s a very quick overview of one big structure.

[Slide: Basic Neurotransmitters] So what are some of the basic neurotransmitters—these are the quintessential neurotransmitters that psychiatrists and primary care docs will talk about with their patients, in terms of those antidepressants, or in terms of mood stabilizers; so the primary ones are dopamine, serotonin, norepinephrine. And these have different types of trajectories or pathways across the brain and service different areas of the brain. They are also endogenous opioids—and that becomes really important when we are going to be talking about stress—but they’re also those good feelings, and so that helps to regulate that limbic system in a positive regard. And then hormones; hormones, when they are released in the body, they are automatically released throughout the entire body, so it’s what’s called a ubiquitous release. So it’s not just one little brain area that gets flooded with hormone, it’s our entire body that gets flooded with hormone pretty quickly, and that again becomes important when we talk about stress.

[Slide: Methylation Biochemistry] Dr. Corwin mentioned about nutrition. I am a big, big fan when it comes to nutrition and how that relates back then to our mental health. This is looking now inside the cell at what the cell is doing; what the cell is manufacturing in order to help us survive and be healthy. This is called the methylation cycle, and we will talk about that in a little bit more detail later. What I want to point out is, this methylation cycle is directly related to neurotransmitter metabolism. So going back to that dopamine, that serotonin, there is dopamine in this cascade, and norepinephrine—it comes right off of dopamine, and serotonin
is off of another shoot, and from there we have melatonin. Some of you might be familiar with melatonin. A lot of people will take melatonin to help them sleep. When there is anxiety, depression—a disregulation of that serotonin cycle—perhaps it’s because they are not sleeping very well, because there is a disregulation now of melatonin as well. You will not have a quiz on this afterwards, ok? I recognize there is a lot of dense information here, but I just want to give you kind of an overview of why these things are important.

So the folate cycle is part of that methylation cycle. This goes back directly to those nutritional principles. We have folate that is right in the heart of methylation cycle, and in the heart then, that productivity for these neurotransmitters. And then we have other vitamins here. There is B6 here, there, there, there, there. I think that was last one. Oh no, and then B12 becomes also important. So we are talking about basic nutrition. If we aren’t fed well, we cannot have the capacity to create what we need to function properly. It’s, again, common sense. And there’s our vitamin C that assists with dopamine into norepinephrine production, so don’t forget to have your vitamin C every day. And again, I want to bring your attention to methylation cycle. This becomes more important down the road.

[Slide: Exposure to Trauma] So exposure to trauma is defined by any stressful event that is prolonged, or that is overwhelming, or that is unpredictable. And this...I would say that this really should be an and/or in this. And as Dr. Corwin pointed out, a single event can cause very serious impact on the physiological changes in our body. And for the most part, what we do talk about with ACEs are a cumulative score of lots of different types of traumas. [Slide: Cicchetti Quote] So Cicchetti—if I am pronouncing that right—Dante Cicchetti started talking about, we need to look at these behavioral changes that happen after a stressful event, and then start looking at the anatomical structures that change, and then the physiological processes that change. So everything that we have talked about already—that we have glossed over already—with the limbic system, with the neurotransmitters, and also with that cellular function; all becomes very important.

[Slide: Exposure to Trauma: Epidemiology] So just a brief overview on epidemiology of exposure to trauma. Roughly about 8% of folks will have lifetime PTSD prevalence. When we look at what is the exposure to trauma, we are looking at 70% lifetime exposure, and exposure rate to trauma. That is roughly about a third of people every year will have some kind of traumatic event happen. However, it’s only a smaller percentage—that 10% to 20%—who actually develop PTSD. Probably, that PTSD starts to develop with different vulnerabilities, whether it would be genetic vulnerabilities, whether it would be the ACEs—the cumulative ACEs scores that are now mounting—but it’s a smaller percentage...a much smaller percentage of people who will have that PTSD response.

And then, I also want to put forth to my colleagues [Kristen?]...to my colleagues in the therapy world that we have the big trauma and we have the little trauma. The big trauma gets to be diagnosed as PTSD. The little trauma, the little “t” trauma—those are lots of cumulative stresses that don’t necessarily get to meet that criteria, but we as therapists and as healthcare providers, we recognize that there is a subsyndromal subset that is equally as impaired, and therefore has dysfunction and is at risk for all of these physiological changes.
What are those different types of risk to develop PTSD? It’s the severity of the event, the type of event, the chronicity—how frequently that’s happening in a person’s life—the proximity. Is it a person that we know? Because we certainly know that that has more of an impact if a trauma is caused directly by someone that we love versus some random event. What is that genetic loading? What is the developmental stage for that individual or for that child? Is that birth to five years old, where we know that there is an exceptionally high risk than for other psychiatric outcomes. And then, what’s that community at large? Are they constantly living in fear just going outside their home? Can the kids get to school without having a fear that they are going to be shot? These are real-life events that happen on a daily basis for a lot of people in our community. And then, there’s that risk factor—again, women. Just want to point out again, it’s a smaller percentage that develop the PTSD. And then what Dr. Corwin brought up, and also Jennifer Oxborrow, that this is preventable. We can’t change, necessarily, IQ, but we can help kids develop other aspects of resiliency in order to combat adversity that they are facing.

[Slide: Exposure to Trauma: Post Traumatic Stress Disorder] These are just the criteria—I’m going to brush through very quickly—for PTSD. [Let’s see; let me go back to that for a second.] So again, it’s the response to a traumatic event that produces fear, helplessness, or horror. These are again DSM criteria. It’s persistently re-experiencing phenomenon, which can include things like intrusive memories, flashbacks, nightmares. In children, however—younger children—they may just look more disorganized or agitated. They can’t really articulate, necessarily, what those memories are. [I keep doing this; sorry.] And that’s persistent avoidant behaviors. Are people avoiding anyone who looks like a perpetrator they have had? Are they staying in their home, because they can’t fathom leaving, because they are so afraid? However, children may just show social withdrawal within school. And then we have persistent negative behaviors and cognitions, and that may be, like, “Gosh, you know, life is never going to change for me,” or “I just expect that I am going to die young, and that’s just how it’s going to be.” Children, however, may show constricted play. They can’t get out of their...out of a single role play that they do repetitiously, where it previously wasn’t there. And then, children may show very severe temper tantrums because of hyperarousal behaviors that occur. And what we typically see in older children and adults, that we see anger reactivity, we see the hyper-vigilance, where they are constantly on guard. And some people will say that that’s paranoia; that’s not paranoia, that’s not psychological paranoia. That is something very different, but it becomes a very important distinction when we are looking at treatment for people who have been traumatized versus people who are psychotic, and sometimes there’s a blurring of both.

[Slide: Exposure to Trauma: Uncomplicated Trauma] Exposure to trauma—now we’re going to start getting into some of the neurobiological effects. The physiological response to—this is just for stress, initially—we get an acute cortisol release and that causes that fight-or-flight response. And we also get an acute opioid—endogenous opioid—release, which has that blunting effect. So when we think about our drive to work—I actually saw an accident on the way in here—but that instant reaction, that is that pulse of adrenaline that causes that fight-or-flight response. And then, in comes to save the day this blunting effect to help me calm down and reregulate through that hypothalamic portion in my limbic system.
This is my little ninja, and he is here just to illustrate that what is happening with those physiological responses—that fight-or-flight response, and also the blunting effect. Starting from the top we have CRF—corticoid-releasing hormone, or factor, and that goes down to the pituitary gland, and that releases something called POMC. Now POMC breaks off into this product called ACTH, and that goes right to our adrenal glands. We all know what adrenaline does, right? That’s that “whoop”—we get on edge. That’s what’s happening when we are in just normal stress, and that releases corticosteroids that goes back up to that center to help us calm down, and to help quiet down that circuitry. So that’s the very basic mechanism of the hypothalamic-pituitary-adrenal axis, and that allows us to have a normalized response to stress.

Now, in chronic trauma or toxic stress exposure—exposure to early stress—we know, we have clearly documented proof now, that yes, early stress does alter neural development, and it prepares that adult brain to live in a dangerous world. Who wants to sign up for that? Not me. And yet this is what is happening on a daily basis to so many people. And we have ways that we can help to prevent it, both societally and also at that intervention level through therapy. Different types of traumas or exposures to toxic stress—that same process is happening now—that cortisol release, and the opioid release, and that blunting affect that is trying to stop that fight-or-flight response. Does that all makes sense?

Alright, so what is happening now more globally in the body? We have trauma, trauma, trauma, trauma happening; lots of ACEs stuff, or maybe the same type of trauma that’s repeated over and over again. We start seeing that endocrine system or the hormonal system take root, which we just discussed, with the HPA. This is now going to start altering how our genetics function, and how the direct effect of the HPA...what the direct effect has on our hematological system—our blood system—and our immunological factors. And that gets really deep very quickly, but immunology is responsible, in a nutshell, for keeping us healthy. And then, the effects that it has on our nervous system. So you can see, trauma has this very global effect on all these different body systems.

So what’s happening with this person who is now very sad, very depressed already? He’s had lots of trauma, and now he is off kilter; he can’t keep balance any more. Same system: CRF is producing in mega doses; it goes down, produces mega doses to POMC, and then we have the negative feedback loops that are trying to shut this process down. What ends up happening...because evolutionary principles say we want to live conservatively, right? We don’t want to keep expending and expending and expending all this energy to produce all of this system. What ends up happening over a time is a dampening effect of the concentration of these hormones, but we have a hypersensitivity then at the receptor level in those limbic system areas. Does that make sense? So we don’t want to have to expend all this energy, we are trying to conserve, and then those structures way up north here [pointing to brain], those become hypersensitive to these glucocorticoid releases. And BAM!—something happens, and we are on edge instantly. And we saw that in the video. I loved that video—I was going to start crying in that—we saw that in that video that Carol showed us at the
very beginning of that girl getting a dress, a nice gesture, but bam! It went right to her limbic system, and she was on fire, and she could not see past that. So we have the altered response.

[Slide: Brain Diagram] So this is where those structures live, and this is just to reinforce what is happening...what’s the cascade now that’s coming down from the limbic system when we have the traumatic stress? And this just stratifies what those different pathways are that we talked about in that other slide.

[Slide: Structures of the Limbic System] So here we’re back to the same slide. We already have learned, and I know you have it all memorized, what these different structures are doing and what they are responsible for. Now let’s look at what happens with chronic stress, and how these are affected. With the amygdala, we start having fear reactivity. Bam! We are instantly going to...we are instantly thinking that we are going to be attacked. So what do we do? We have aggression, we have lots of impulsivity going on, which can look like ADHD for some of those kids, but it’s not ADHD. It’s driven by a very different process. And then we have, again, that hyper-vigilance. It’s not paranoia, but it is on the prowl and looking to make sure that nobody is going to hurt me. These are adaptive responses. These are normal responses, as Jennifer brought up. These are normal responses to the environment in which they are surviving.

In the hypothalamus we start to have psychosomatic symptoms that emerge. We have gastrointestinal problems, so lots of tummy aches. Depending on the stage of development, there’s some literature to suggest really early on, that if there is a lot of abuse that has incurred early on, that there is a lot of gastric motility dysfunction. Makes sense; the body remembers. And as van der Kolk talks about, the body keeps score. We are going to have impact on our physiological systems. Cardiovascular events start happening—much higher risk—and actually...it’s in a later slide, but Dr. Corwin already mentioned about higher ACEs being independently seen as a risk factor for cardiovascular events. We see decrease of immune function. The body just can’t simply keep up with this, and it...we start having autoimmune problems because of immune dysfunction. We start having higher rates of cancers because of immune dysfunction.

And then the endocrine system; it’s not just the HPA that gets affected, but it is a multitude of hormones, and we’ll briefly touch on some of those. And then the hypothalamus is perhaps involved with some of those dissociative features, where people can’t feel their body. They physically can’t feel their body, and they are so emotionally numb they can’t feel. And what do they end up doing oftentimes? They end up hurting themselves, because they want to feel something. [Oh, that’s kind of hard to see, I think], but within the singular cortex, what starts happening is [gosh, I can’t even read that from here] cognitive rigidity, and what do we start seeing when a person has been traumatized a lot? We start seeing, perhaps, even some OCD symptoms. They have to do something a very certain way, because if they don’t, all hell may break loose. They have to protect themselves, so they’ve got very concrete about what has to happen and not. They start misreading social cues. Someone looks at them funny; they can’t socially adapt. They can’t extrapolate and think, “Oh well, maybe that person just had a bad day; maybe they stubbed their toe.” No, they are interpreting that this person can’t stand them. What starts happening? We get a limbic firing, and all of a sudden, now we have
someone who is hyper-aroused, going back to that amygdala, and we have a fight that breaks out because of how someone looked at them.

And then with the hippocampal impact, we start having difficulties laying down memory. We can’t learn effectively. Makes sense, right? When we are hyper-stressed—well, we are not talking about toxic stress—but when we are hyper-stressed, it’s really, really hard to learn. It’s really hard to remember what we’ve done. At least I know, when I am trying to organize my kids, and I feel so stressed, I can’t remember anything. My kids are like, “Mom, we just talked about this.” That’s just a normal effect from stress, from toxic stress. It becomes more pronounced, and kids end up looking like they may have learning disabilities. No, they have some pretty significant stuff that needs to get cleared up. And then we can become very stagnant. We can’t do that navigational piece anymore. We can’t figure out how to—if I have to leave this door, and I have severe PTSD, how I am going to get out that door if I am really afraid? I can’t navigate around this, and I become stagnant in my behavior patterns. Does that all make sense?

Oh, and actually, one other piece I just want to mention—the hippocampus. In the parentheses it says: enhanced by strong emotion. When we have that strong emotional response, we are going to have a very heavily embedded memory. Emotions are the first things that we experience, but when we are not aware of what we are feeling—when we’re not aware of what those emotions are—it’s really hard for us to discern anything other than anger and rage. And on the flip side, when things are normal—I mean, we are just having normal stress response—like we are studying for a test, and we’re under the gun, and we got to get all that information done. So in a normal stress—I failed to mention at the very first sight of this [slide]—that that actually can be adaptive in enhancing our memory. So, test cramming: we can remember a whole lot more when we are under the gun, but when that system gets overloaded, then we can’t really lay down those memories other than from severe emotion.

[Slide: Behavioral Changes] So, van der Kolk I mentioned before, and he described—actually, he has just been talking about the impact of stress back since the ’70s—but he said the loss of the ability to regulate the intensity of feelings—so the inability to regulate the limbic system—is the most far-reaching effect of early trauma and neglect. It makes sense, doesn’t it: now that we have looked at those anatomical structures, it makes complete sense why it’s so far-reaching. It goes way beyond what we are looking at psychologically; this has far-reaching effect for not only our physical body, it has far-reaching effect into our society and what we are paying for.

So what kind of brain do you guys want to work with, one that’s on fire or one that has been calmed down? I put this slide in for my dear colleague, Krista Warnick, who is an amazing EMDR therapist; that we clearly see that there is a dampening effect when people can deal with their trauma.

[Slide: Altered Neurotransmitters] So it starts happening with altered neurotransmitters. I mentioned before about hormones—that the hormone cascade starts altering. Well, what’s happening? That CRH—I love that little neutron effect—that the corticoid-releasing hormones start having this very significant impact, and it really starts altering, especially, these
hormones that have now been identified. I am sure you are able to see a whole lot more, and we clearly know when there is a lot of stress that menstrual regulation is all disorganized, and also spermatogenesis. So it’s far more reaching in than the impact of just these hormones, but these are ones that have been studied and we have some really interesting data to support this. They mention about leptin; there is just this fascinating rat study, and maybe it’s from that same group you are talking about, because I don’t ever remember names. But they put rats under stress models, and then gave them choices of what to eat. And what started happening is the rates who were non-stressed continued to grow in a healthy fashion, predictable fashion, and their coats remained normal. The rats that were stressed became obese, and they chose very different things to eat, and their actual coat—their fur coat—changed. That’s what we see societally. We don’t have fur, but we definitely have a very different and pronounced shift with body mass index when there has been stress. And one of our colleagues, Brooks Keeshan, will be continuing his studies in that area as well.

We also get some alteration in the balance of the endogenous opioids, and one in particular is dynorphin. Dynorphin is actually implicated in the addiction field, and raises that...how can I put this...it imbalances that normative response, and it puts us at risk for that repetition, compulsion, and of addiction—of seeking out addictive substances—and that gets into the literature pretty deeply and quickly. [Oops, wrong way; sorry.] And then why is this all happening? Going way back to the top, going back up north here, POMC is responsible, at the very beginning of this train, for all of these alterations.

[Slide: Methylation Biochemistry] Back to that...to the methylation cycle—I hope that I have started to explain to you why all of these pieces are so important. In order for us to have normative...or to normalize now—go back to normal, as normal of a balance possible—we have to have that nutritional piece in order to produce what we need.

[Slide: Regulated by environmental stimuli] And that ties directly into something called epigenetics. Are any of you familiar with epigenetics, with that term? So just a few of you. I find this absolutely fascinating, and I think this is the kind of stuff that I geek out at every night. Just reading more and more in depth about what scientists are now starting to understand about the genetic influence, or the influence on our genes so...and this ties into methylation, which you’ll see in just a moment. So in normal circumstances, methylation regulates what’s happening in our environment, and it also records molecular memory. So what we are experiencing in this room right now—our body is absorbing that, and actually using that methylation process that was up in this slide. It is using that methylation process to talk to our genes, and to say, “Oh, I need a little bit more of this protein right now, because I have to stay more awake, so I am going to produce now a little bit more norepinephrine,” so my genes have to unwind and produce a little bit more of that protein. If I am really stressed, that’s not going to happen so much. [Slide: Epigenetic changes are essential for normal development] But epigenetic changes are methylation on the DNA itself, and also something called histone modification, and also non-coding RNA. Lots of complicated stuff, but the basic message here is that...that this process translates past stressful events into our genetic expression—how that affects our HPA, how it affects our neural circuitry; and especially if we’re a little baby and we have lots of neural circuitry to develop, it’s going to have a very significant impact, and it’s
also then going to affect what our future behaviors are. [Slide: What is the epigenome?] [So I am going to hope that this...let's see. We don't have our IT guy. This is a little video. Let's see. I don't know how to get my slide to go. Oh, there it is, OK. There it goes.]

[Video] But DNA is only half the story. The DNA in our bodies is wrapped around proteins called histones. Both the DNA and histones are covered with chemical tags. This second layer of structure is called the epigenome. The epigenome shapes the physical structure of the genome. It tightly wraps inactive genes, making them unreadable. It relaxes active genes, making them easily accessible. Different sets of genes are active in different cell types. The DNA code remains fixed for life. But the epigenome is flexible. Epigenetic tags react to signals from the outside world, such as diet and stress. The epigenome adjusts specific genes in our genomic landscape in response to our rapidly changing environment. [Music]

[Slide: Learn. Genetics] So that’s a very, very quick overview of just a tremendous body of research, but I think it gives such a quick picture about how important it is to be aware, to...going back to those trauma-informed principles. Why it’s so important for us to reduce whatever kind of allostatic or stress load that we possibly can in our environment.

[Slide: Why Epigenetics Can Be Multigenerational] And this also goes back to that multigenerational piece that Jennifer brought up, and it’s not just that parent/child interaction. We are actually...there is a lot of data coming down the pipeline now to look at three generations that stress affects at that epigenetic phenomenon. Because when that mom is now pregnant, her epigenome is being affected; that fetal environment is being affected, because guess what? The uterus itself produces its own CRH, so that top stream of chemical from, or hormone from, the HPA—the whole uterus produces its own CRH to be protective for the fetus. But when the mom is pregnant, and she is going through life stresses that are more than normal, that whole uterus is now going to be bathing that fetus and neural development with altered ratios of those hormones, and we are going to see a different child that is fresh out of the womb. And those epigenetic alterations are now embedded in the germ line, or in the reproductive organs, of that growing fetus.

[Slide: Behavioral Changes: Chronic Trauma] But this is all preventable, right? So what does chronic trauma cause? From a shrink’s perspective, it causes a lot of problems, and we may have genetic underpinnings. We may be prone to depression. We may be prone to bipolar disorder. We may be prone to psychosis. But that doesn’t necessarily mean that we will have that disease process emerge. It could emerge even without ACEs, but putting ACEs on top of that, through the epigenetic phenomenon, we are guaranteeing that we are going to unleash what is already embedded in our genes. Does that make sense? And going back to what I said at the very beginning about the members on my ACT team, I am sure that they had the genes already that predisposed them to schizophrenia, to schizoaffective disorder, to severe depressions. But would their depression have been as bad, or their psychosis have been as bad, if those ACEs never were there in the first place? Very important that we start looking at this, and how does that effect children with learning disorders, and also the disruptive behavior disorders in that classroom setting.
I’m nearing the end of my talk here, but I just wanted to present a couple of cases. These are… I have changed the information and the data so none of you can recognize them, but this is a child from my days at Odyssey House. So his mother was in the program, and she is in the parent/child program, and he was just a very sad little boy. And he was under the age of five, and his mother described this: *He has seen terrible situations with police, drugs, violence, raids. I wasn’t around very much, and he was neglected also at my mom’s house* [so kinship placement]. *There may also have been some sexual abuse by my ex, [her ex-boyfriend] and, oh yeah, a friend of the family hung himself while he was around.* So under the age of five: extreme trauma. What were his diagnoses? Of course, post-traumatic stress disorder—rule out ADHD—probably because his limbic system is so on fire. And it’s really hard to get that quieted down when you have such a severely traumatized kid who can’t do anything except stare at a blank wall, which we also saw in that video. She is staring out to the ocean, but just incapable of really interacting with the environment. And then oppositional defiant disorder; childhood anti-social behavior. I detest putting that onto a list of diagnoses, because I believe in the ability of kids and adults to change, but there are some people who become so damaged that that kind of diagnosis is really merited, when their aggression becomes so over and they can’t get beyond it. They get that cognitive rigidity, and they can’t navigate beyond that. And then, also had in-utero exposures, including interpersonal violence; so that again, that uterus was now bathing that kid. What kind of chance does this kid have? I don’t know, and again, for those educators here looking at those ADHD symptoms in your classroom, maybe it’s not ADHD. And these are just a list of all of the exposures that he had to different abuses and then…[Excuse me, I have to get a tissue. I am having some allergy issues.]

So what were his exposures? He had prenatal exposures to substance use, to domestic violence, interpersonal violence that he both witnessed and was a victim of. He was neglected. He had physical and emotional abuse, and parental drug use. We clearly see that correlation with the ACEs studies. Parents who were both imprisoned at different points in his life, kinship foster placement that didn’t turn out to be so good; and the list just goes on and on. He was placed at nine years old… I think he was nine—in his latency years—as a sex offender, because of pretty much lies that people were saying about his behavior, and it was actually behavior caused by the adults in his life. And he was put in one program after another, and this made me so sad. He said he was never allowed to talk about his trauma. He had to just accept that he was a perpetrator, and was never allowed to talk about his trauma. He is now in his 20s, and he is ready to talk about that, but guess what, he suffers from so much psychosis. We have a lot of work to do in order to get him to a stable place. So I am not going to say that he was absolutely innocent in everything, but we have to look at these issues very differently. And what are his diagnoses? There you can see for yourself—lots of bad stuff, and probably a lot of preventable stuff. With all my work as an addiction specialist, I have yet to meet someone who has told me, “I love my addiction, and I set out to have my addiction.” Never met one person
who has said that, and by and large—especially with the folks that I see at Odyssey House—by and large, they have huge ACEs scores that have racked up and have really driven very significant addiction histories. Are there exceptions? Yes, absolutely. I want to bring your attention down to under AXIS 3 [on slide]. This person in his 20s—hypertension; hyperlipidemia, which means lots of cholesterol and fats swimming around in his bloodstream; obesity. These are the ACEs in living data. This is the effect of the hypothalamic impact on our body and how the limbic system affects us. And also for the educators in here, I want to point out that he has this diagnosis of provisional ADHD. I actually think that he really does have bona fide ADHD, but I have to sort out lots of other things, and treat lots of other things first, before I can actually start treating that.

[Slide: Psychopharmacology] So psychopharmacology—I just threw this slide in to make two points. One is that when we are treating people with severe PTSD, these are the clusters of symptoms that we are looking at, and the types of medications. However, my favorite day is saying minimal medications as much as possible. This is not a treatable illness with medication. The real antidote here is—maybe some medications to address symptoms that are so severe—but the real impact is with intervention. [Slide: Mindfulness Upgrades the Physical Anatomy of Your Brain] And that is with learning how to quiet down that limbic system, learning how to start doing the self-regulation that is absolutely essential, and actually is restorative, to those brain tissues. Without doing that, when we have that cognitive rigidity and we are doing the repetition compulsion—that psychobabble term that just means we repeat compulsively what we don’t understand. And that’s why we see people go back into abusive cycles over and over and over again—different partner, different face, same situation—it’s that repetition compulsion. When we start to raise that awareness, and become mindful, and we start looking at, what am I doing here? When we work with a therapist who is mindful and trauma-informed, we start to see very significant changes in behaviors, sometimes and very often times, not even with medications. My favorite day is to get people off medication, but I won’t do that until I know, or I feel like, they are ready to do that, and when I start seeing improvements in different psychiatric symptoms. Wouldn’t it be a great day that all of our kids in our schools started off, or had time during their day, to do mindfulness, to have time for reflection? Wouldn’t it be great that they had great nutrition in our school system, or these—maybe for those kids who are at most risk and have little access to food—maybe they get that one really good meal that’s full of appropriate nutrition. Maybe they will have a little bit different outcome, and maybe they will actually be able to do the mindfulness work, and they don’t need other medications to help to quiet that limbic system down.

So Jennifer had this question: What happened to you? instead of What’s wrong with you? [Slide: Interplay of mental and physical illnesses] And by asking that question, we start reshaping the way we think, and we start looking at that interplay of mental and physical illnesses, and we start addressing that disrupted development that was caused way long ago, that was no fault of their own. Validating that kid or that adult who experienced all that stuff, and saying, “You know, who is responsible for this? This is not... You are not responsible for this. Yes, you had a lot of behavior choices that you’ve made, but let’s look at their root here. So we can start reshaping those brain structures, and have an impact on the neurobiology, and get you to a place of better functioning.”
These are just some data from the ACEs...I'm sorry, this is the Utah population data that Dave actually mentioned. And this is the TANF data, and Dave had mentioned earlier about [I don’t think I have a pointer here], but about the differences in the TANFs scores. I want to bring your attention, not to the top of the slide, but down to the bottom of the slide, where it has the ACEs scores. For general population, about 40% of the population here in Utah has a zero for their ACEs. Greater than five ACEs, it’s about 10%. When we look at the TANF data—those kids and those adults who are most at risk—we see the exact flip. We see that 8%, almost 9%, have a zero ACEs score, but greater than five we see 46%, and we could even take it down to...I think the cut-off starts happening at four, where we start getting into those higher 40s percentages.

[Slide: What the ACE Study Is...] [I think my time is almost up here. Yep; okay.] [Slide: Response to Behaviors] So resiliency—these are just little pieces. This is just looking at the school impact—that...what we can do in school, looking at that TIC—trauma-informed care—what can we do at that academic level? And I know that we are going to have some great discussion later in the afternoon with the panel, so I am not going to bore you with details, [Slide: Response to Behaviors] but what do we have to create here? It is a trauma-informed care perspective change. We have to create environments—wherever we go—you are safe here. You are welcome and you are safe, no matter if it’s at school, at work, out in our community. We may not be able to control what happens behind closed doors, but we can have such an impact on what happens in the community, and communicating, “You are not alone in this.” Dave Corwin brought that up, that this is a community-wide effort. It’s not just the schools; but we can have such an amazing impact starting with the schools, and starting to correct the behaviors that are so predominant.

[Slide: Wrap-Up] So as a wrap-up, we briefly addressed what stress...what a normal stress response is; how that’s different in a trauma-adaptive response; the physiological changes that occur due to trauma, but are predictable and also detrimental; and then the behavioral responses due to those physiological changes, again, adaptive—they are having a normal response for what they have experienced. And then, how can our responses change to those behaviors we observe? [Closing Slide] So I invite us all to continue in this progress, and be the change that we want to see in our world. Thank you.