EPIGENETICS AND PRIMAL THERAPY

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Abstract

Epigenetics is focused on the study of how experience changes an individual's genetic expression, previously considered inalterable. Recent pioneering research indicates that epigenetics is revolutionizing the study of mental health and challenging the belief that DNA is destiny. Moreover, in terms of the history of science, the new developments augur the convergence of previously distinct fields, psychology and biology. There is a growing understanding that mental illness has a crucial physical component and may cause disturbances of mind and body which strongly emphasizes a crucial role of psychotherapy focused on early stressful experiences mainly during critical developmental periods including prenatal and perinatal traumas.

Key words: Hypnosis; Hypnoanalysis; Age regression; Implicit memory; Phobia; Borderline personality disorder

1. INTRODUCTION

In one report regarding research that shows a link between early-life adversity and changes in a person's genetic make-up, McGowan & Szyf (2010) come to this sweeping conclusion: "Epigenetics could serve as a bridge between the social sciences and the biological sciences, allowing a truly integrated understanding of human health and behavior." (McGowan & Szyf, 2010, p. 71)

In short, there is a growing understanding that mental illness has a crucial physical component and that maintained neurosis is a disturbance of mind and body. Because of that in our treatment, both mind and body must be involved for a cure. Now, science is showing us how that is possible at a cellular level. Unlike genetic mutations, the researchers state, "epigenetic alterations are potentially reversible" (McGowan & Szyf, 2010, p. 66). And that is the most promising finding of all.

These alterations turn the gene on or off, and thus help set in what seem like genetic changes. And there are different chemicals that accompany the epigenetic events, methyl and acetyl groups, for example. The critical work in this field shows how imprints can be passed down through generations – from parent to child and grandchild – primarily through the biochemical processes known as methylation and acetylation. We need to differentiate, however, between healthy and unhealthy methylation. Under normal conditions, methylation is a necessary and naturally occurring process that helps regulate the expression of an individual's genetic make-up (McGowan & Szyf, 2010). But excess methylation becomes

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pathological and leads to a disease. The process goes awry when the individual suffers physical or psychological trauma, especially in the womb and in infancy. It seems that for each and every pain we endure during gestation and at birth there is a change in the chemicals that enhance the repression of pain. When the pain or adversity is prolonged, the system is overtaxed and we now have the mechanism of leaky gates; that is, repression begins to falter due to an overload of chronic pain (McGowan & Szyf, 2010; Janov, 2013).

Does it mean that by examining our womb-life in detail we can often predict our future: our sexual problems, the possibility of later cancer, psychosis, heart problems, Alzheimer's disease, and a whole host of afflictions? (Johnstone & Baylin, 2010; McGowan & Szyf, 2010; Janov, 2013)

One study suggests that the biological underpinnings of bipolar affective disorder are not primarily genetic, but are epigenetic (Rutten & Mill, 2009; Janov, 2013). Even an individual's tendency toward violence, once thought to be a brain disorder, is being shown to have epigenetic roots. In research with rats, investigators at Switzerland's Ecole Polytechnique Fédérale de Lausanne found that animals subjected to trauma in childhood showed changes in two parts of the brain – the orbitofrontal cortex and the amygdala (Márquez et al., 2013). Those changes taken together combined to lower the threshold of aggressive impulses and weaken the ability to control them. (A report summarizing the findings was also released by the Swiss research university under the title, "Childhood Trauma Leaves its Mark on the Brain."¹). The results were surprisingly similar to changes found in the human brains of traumatized children, who grew up to be violent adults. In addition, the researchers also measured changes in genes known to be associated with aggressive behavior. Researchers were able to reduce the levels of aggression with antidepressants, specifically an MAOA gene inhibitor. In short, childhood stress produced epigenetic changes that heightened violent tendencies. Drug treatment later tamped down the violence, reversing the long-term impact of early trauma. In our own work, we have found that the deeper patients descend down the levels of consciousness the more likely there can be rage and violence (Rutten & Mill, 2009; Janov, 2013).

Until recently, the role of epigenetic mechanisms in transmitting trauma across generations has been demonstrated in animals but not in humans. A new study, involving Holocaust survivors and their children, shows for the first time how the epigenetic impact of stress, the cellular changes, can also be passed down among humans, from one generation to the next (Yehuda et al., 2015). Researchers found that children of Holocaust survivors frequently gave birth to anxious children. At first, they thought it was because the parents told horrible stories to the children, but later they discovered that the anxiety came down through the genetic chain, as we shall see in more detail shortly. The point is that the genetic effect of wartime stress had descended from the mother's physiology through epigenetics (Yehuda et al., 2015; Rutten & Mill, 2009; Janov, 2013).

I will discuss clinical implications of the research in the second half of this article. Suffice to say for now that pharmacological treatment may not be the only way to reverse epigenetic changes. Most likely the effects of methylation – as an agent of repressive chemical processes – can be reversed using some psychotherapeutic method (for example the "Primal Therapy" and some other psychotherapy approaches that enable reliving of past experiences), which revisits and resolves the traumatic events that likely may trigger the repressive chemical process to begin with. The real revolution lies in the possibility that people no longer have to live with their genetic inheritance but can actually take charge and change it. If it is life experience that caused changes in the biochemistry and neuronal circuitry, then it is not a fixed entity. It can be altered; the way this is done is by retrieving and reliving key imprints, as I will explain.

¹Childhood trauma leaves its mark on the brain. (2013, January 15). Ecole Polytechnique Fédérale de Lausanne. Retrieved from <http://www.sciencedaily.com/releases/2013/01/130115090215.htm>

2. HEREDITY TURNED ON ITS HEAD

Let's make sure we understand this notion of epigenetics because in the coming years it will likely be one of most important areas of scientific research. As I've mentioned, one reason for its preeminence is that many of the serious diseases we think are genetic are actually epigenetic and, therefore, environmentally caused, and possibly treatable. That is now an established fact in human development. However, early discoveries in the field a short decade ago were so startling that they even surprised the scientific world.

We now understand that the imprint is aided and abetted by the process of methylation, in which the chemical methyl group is added to the genome to restrict its expression. In other words, the imprint is laid down, in part, by a change in the cell, as certain chemical reactions are taking place – hydrogen removal, methyl infusion, and so on. Methylation leaves a heritable imprint, one that can be passed down even from grandparents to their grandchildren, as research has shown. So what we always thought was genetic may well be the result of very early experiences diverting the genetic legacy (Meaney, Aitken, Bodnoff, Iny, & Sapolsky, 1985; Janov, 2013). In short, the experiences of our forbearers can endure and be passed down the epigenetic chain – the inheritance of acquired characteristics. This is something science thought impossible not long ago.

In a key experiment Meaney et al. (1985) compared two groups of rats: one group consisted of the offspring of normal mothers, who frequently licked their babies but had subjected the offspring to stress during pregnancy, with a second group of pups who were also under stress but experienced no licking (Meaney, Aitken, Bodnoff, Iny, & Sapolsky, 1985). Not surprisingly, those babies who were heavily licked turned out to be the most normal and well adjusted. What is a surprise, however, is how much womb-life counts; what the results have shown is that the right amount of licking and grooming early on, left offspring less responsive to stress hormones as adults. It is what we all know: that early love makes us stronger and less anxious. But it turns out that if the mothers were licked and groomed early on in their lives, that experience could be passed on too; the stress hormone genes of their offspring could be modified by the methyl group (and also other chemicals) in a beneficial way.

Almost every animal form that is loved and licked has grown up pretty healthy with no serious disease; and in my therapeutic experience, those children who had bad and traumatic births with unhealthy gestations are the ones who suffer the most as adults. Too often, catastrophic early life equals catastrophic disease later in life. For example, there are epigenetics studies that document "the intergenerational effects of the Holocaust" by showing that "the atrocities altered the DNA of victims' descendants." Armed with that knowledge, activists called for "a mental health provision to treat inherited trauma."²

Implications of these findings seem to be clear. "The gene changes in the children did not appear to be mediated by adversity experienced during their own childhood but could only be attributed to Holocaust exposure in the parents," said Yehuda³: "Environmental influences such as stress, smoking or diet can affect the genes of our children."

4. THE URGENCY OF EARLY LOVE

What is important is that we can begin to zoom in on a specific answer to the question that eludes so many in the study of mental illness – why? Based on recent research a damage is reflected by heavier methylation. But what is that great damage? The answer is simple – early lack of love and stress. It takes many forms in humans: poor nutrition, abuse, neglect, lack of

²West, J. (2015, August 3). Holocaust survivors' grandchildren call for action over inherited trauma. *The Guardian*.

³Holocaust survivors pass on trauma to their children's genes. (2015, August 25). Retrieved from <http://www.mpg.de/9375728/holocaust-trauma-epigenetics>

touch (licking in animals). Methylation seems to be an important marker for lack of early love, both in animals and in humans. The new research indicates that many diseases are affected by methylation, including multiple sclerosis, diabetes and heart disease. Again, these are stress-related and the great stressor seems to be a simple lack of love, meaning deprivation of basic needs. Not surprising in the rat study was the fact that heavy methylation occurred in the limbic/feeling structures such as the hippocampus which has to do with feeling/memory. The upshot is that rat pups that are unloved are more susceptible to later stress, while those that do get loved (licked) do much better later in life, becoming more adventurous and curious (Janov, 2013).

Animal experiments have shown that separating a baby sporadically and unpredictably from its mother causes severe stress, and the same applies to a human baby who is not touched right after birth and in the first weeks of life, as we find in babies raised in incubators. Another study showed that early trauma produced heavy methylation in those children who grew up in orphanages (Drury et al., 2011). And that process then affected much more in terms of brain and neuronal development. The effects of this stress become heritable from the epigenome. This imprint then affects many aspects of our biology, including the memory system. That may mean that a condition such as Alzheimer's disease may get its start from birth, and not show up for another sixty years. Research by Lahiri and Maloney (2010) for instance, indicates how this all might work— first by the imprint and then, they suggest, by the methylation that carries on the imprint.

Other research has shown important findings about the so-called emotional synchrony, i.e. the non-verbal communication between mother and child (Waters, West & Mendes, 2014). In a phenomenon they dub "stress contagion," the baby is learning how to manage the incoming stress of the mother. They did studies of several different mothers who gave a talk with a different audience – one approving, one neutral and one not approving. Guess what? The 14-month old babies reflected what happened. There were differences in heart rate and a greater stress response in those children of mothers who had disapproval. The children "learned" through some kind of osmosis. They were inculcated by the mother's emotional state. As lead researcher Sara Waters stated in an article on the website of the Association for Psychological Science, which published the research: "Your infant may not be able to tell you that you seem stressed or ask you what is wrong, but our work shows that, as soon as she is in your arms, she is picking up on the bodily responses accompanying your emotional state and immediately begins to feel in her own body your own negative emotion."⁴ Now imagine that the baby and mother are one, where the baby lives inside the mother. The influences are far more impactful.

So what gets transmitted? Odor, facial expression, lack of feeling, body movements and on and on. All of the parent is transmitted to the child. Even food preferences can be imprinted in the womb and passed on through generations. If you love sweets and cannot resist, it could be due to womb-life. In other words, the mother's compulsion becomes your destiny. This can explain a good percentage of obesity in children. Bad eating habits begin in the womb, as do so many other compulsions. For the most part, people only see the visible manifestation of these hidden forces. So they ask, for example, "Why does this person eat so much?" We know that it is not current culture that is the sole cause; it could also be because the mother was compulsive. While in the womb the baby is learning about his/her world and what to expect from it; hence lots of food is to be expected from a mother who indulges. More evidence is piling up to show how this early start can predict the early onset of disease and a shortened lifespan.⁵

⁴For Infants, Stress May be Caught, Not Taught. (2014, February 3). *Association for Psychological Science*. Retrieved from <http://www.psychologicalscience.org/index.php/news/releases/for-infants-stress-may-be-caught-not-taught.html>

⁵See the work of Keith Godfrey, Professor of Epidemiology and Human Development, and others at the University of Southampton in England.
http://www.southampton.ac.uk/medicine/research/groups/human_development_and_physiology_research_group.page

Other findings show that even the memory of a specific smell can be inherited (Dias & Ressler, 2013). In this research male mice were trained to associate the smell of cherry blossom with an electric shock, making them fearful of it. They then impregnated females with the sperm of these mice and found that the pups were also fearful of the cherry blossom aroma. Even the grand-pups inherited the fear of that specific smell. How did this olfactory trait get passed down through generations? Researchers attribute it to epigenetics, noting that DNA from the grandfather mice and their pups revealed epigenetic marks on the gene encoding the receptor for that specific smell, known as M71. In other words, this inheritance came through experience, not just genes. Like their traumatized grandfathers, the grand-pups were more sensitive to the aroma of cherry blossom because their receptors were also acutely attuned to it, more than control mice. The research "provides some of the best evidence yet that memories or developed traits can be inherited," according to a report on the experiment published in *New Scientist*.⁶

According to Ressler^{7,8}: "Knowing how the experiences of parents influence their descendants helps us to understand psychiatric disorders that may have a trans-generational basis, and possibly to design therapeutic strategies".

So are we born fearful? Could be. We can be jumpy, nervous and erratic, all due to epigenetics. It seems so early as to be genetic, but it is more likely to be epigenetic, the condition of the mother (and father) while carrying. So you say to yourself, "Did I inherit my mother's craziness?" The answer could be, yes... but not in the usual sense of inheritance. Rather, who she was while carrying - hyperactive or depressed and down - left you with a neurotic inheritance that still shaped your life. This should teach us something about memory; for memories while being carried can last decades and drive and/or channel behavior. We do not simply "grow out of it."

There is a recent study which may provide an insight, this study suggests that those who had combat fatigue generally had more trauma growing up (Berntsen et al., 2012). In that study researchers interviewed a group of 746 Danish soldiers before, during and after their deployment to Afghanistan. They wanted to trace the causes of PTSD and find out why some soldiers developed the disorder while others did not. They found that the vast majority of subject soldiers handled the war experience with little or no psychological harm. Surprisingly, for those men who did develop serious stress symptoms, the cause was not found to be connected to battlefield trauma. Instead, the strongest predictor of PTSD was extreme childhood abuse, not combat experience. Researchers found that the PTSD sufferers were more likely to have been victims of severe beatings, burns and broken bones, or to have witnessed family violence as children. In addition, these soldiers had past experiences that they were unable, or unwilling, to talk about with the investigators.

However, in an unexpected twist on conventional wisdom, researchers found that some of the already stressed soldiers, about 13 percent, actually felt better after being sent to the battlefield. These were men who exhibited stress symptoms, such as major anxiety and frequent nightmares, *before* their deployment. But once in the war zone, their stress temporarily improved, only to reappear once they were safely back home. The question is: Why would they feel better when suddenly plunged into an unfamiliar and threatening situation? The answer, as this study suggests, is that being sent away to war allowed them to briefly escape their own private battlefield - the family.

"In other words, they showed improvement as soldiers only because they were in such poor psychological condition in civilian life," concludes an article about the research published in *Scientific American*. "Army life -even combat- offered them more in the way of

⁶Geddes, L. (2013). Fear of a smell can be passed down several generations. *New Scientist*, 220(2946), 10. doi:10.1016/s0262-4079(13)62827-4

⁷Mice can inherit learned sensitivity to a smell. (2013, December 2). Retrieved from http://news.emory.edu/stories/2013/12/smell_epigenetics_ressler/campus.html

⁸<https://www.youtube.com/watch?v=gz2yDSsSOkg>

social support and life satisfaction than they had ever had at home. These soldiers were probably benefiting emotionally from being valued as individuals for the first time ever and from their first authentic camaraderie – mental health benefits that diminished after they once again returned to civilian life.”⁹ Similarly, when a baby or fetus is traumatized it leads to increased sensitivity to later stress (Fagundes, Glaser, Malarkey & Kiecolt-Glaser, 2013).

5. EPIGENETICS AND BRAIN DEVELOPMENT

Although the study of epigenetics can get fairly complex, one of the keys to proper understanding lies in accounting how the brain develops during the fetal period. The thalamo-cortical (thinking/feeling) circuits are established very late in gestation (Janov, 2013). There was a study reported by Garcia, et al. (1999) in which the investigators noted that when babies are under threat the amygdala sends a signal to the prefrontal cortex, triggering the expression of fear in behavior (Garcia, Vouimba, Beaudry & Thompson, 1999). The cortex becomes the “decider,” as it were, planning for action. As part of the study, the researchers trained mice to associate a tone with an accompanying shock delivered whenever the tone was issued. Each time the mice heard the tone, there was commensurate brain activity in the prefrontal area, signaling a threat. But when the amygdala was surgically removed there was no longer any prefrontal activity; the former could no longer signal fear to the top level. The same is true when we drug that structure or tranquilize it: we thereby diminish the force that mounts in the prefrontal area.

What seems to be most important from my perspective is that when the brain is marked by trauma, serotonin supplies is depleted; and when that happens, we have what I call “leaky gates” for a lifetime. We are then less effective in our efforts to repress. Pain roils the brain. We are disturbed and cannot concentrate or learn. And later in life, we are more susceptible to mental illness. This was found by Booij et al. (2012), who measured the serotonin synthesis capacity of 26 healthy adult males, recruited from a 27-year longitudinal study. The results were then correlated with reported birth trauma, especially a delivery where the fetus showed signs of physiological distress. The study concluded that “perinatal stressors may contribute to increased vulnerability for psychiatric disorders in which serotonin plays a major role.”

Recently, researchers have found that children with OCD, Obsessive-Compulsive Disorder, are much more likely to have suffered a birth trauma than controls (Geller et al., 2008). And the question is, why does this reaction get imprinted and last so long? Because it is essential for survival that we remember what is dangerous and how to react to it. We need to have the capacity to feel terror and get galvanized to react immediately. Part of this is that the secretion of noradrenaline affects the amygdala and elements of the brainstem, which are mobilized. We become hyper-alert and ready for action, and this alertness interacts with the memory system to direct our efforts.

In one interesting experiment Meuret et al. (2011) attached mobile monitors to panic sufferers and recorded round-the-clock readings of vital signs. What they found was that subjects were completely unaware of physiological symptoms that could have signaled an impending panic episode, the biological precursors to manifest symptoms such as chest pain, dizziness, trembling or hot flashes. Patients were oblivious to these “waves of physiological instability” for at least an hour after the symptoms had started. Suddenly, as if on a time-delay, the patient becomes aware that he is having a full-blown panic attack (Meuret et al., 2011). It is as though the pain/terror is on the rise and we are not aware of it until it engulfs our consciousness. (The experiment is also explained in a YouTube video posted online and

⁹Herbert, W. (2012). Embattled Childhood: The Real Trauma in PTSD. *Scientific American Mind Sci Am Mind*, 23(5), 74-75. doi:10.1038/scientificamericanmind1112-74

featuring the study by Alicia Meuret¹⁰). Because the terror is set down so early, in the beginning months of gestation, and imprinted so deep in the brain, we have no idea where it comes from. Terror surely begins its life in the brainstem and in archaic parts of the limbic system (amygdala). These data seem to be in agreement with evidence provided by Perna et al. (2014), who indicate that panic patients tend to have abnormal brainstem activation to emotional stimuli when compared with healthy controls.

Beyond gestation and birth, early childhood is important, as well, when attempting to identify the origins of later life problems, as evidence of imprinting can be seen in the experiences of very young children. There is a study by McGowan et al. (2009a), who found when child abuse exists there is a change in the gene NR3C1 (glucocorticoid receptor gene) that affects how the child will deal with the abuse. Measures of the gene's function were much lower in abuse victims who eventually took their own lives. It appears that childhood abuse had changed the gene's structure, making the gene less active. And these modifications endured throughout the children's lives. Epigenetics had affected the function of the stress apparatus, what is called the hypothalamic-pituitary-adrenal axis (HPA), a complex part of the neuroendocrine system that controls reactions to stress and influences many body processes, including digestion, the immune system, mood and emotions, energy storage and expenditure. A possible implication of these findings is that the changes are more or less permanent; they alter the gene's activity, leading to later illness and suicidal tendencies. When the NR3C1 gene is less effective, it cannot produce the kind of alerting, galvanizing chemicals that help one fight through things. (Clearly, such trauma also diminishes an individual's adaptive capacity, as I will discuss below.) As a result, the body behaves as though it were constantly under stress. Moreover, what this research group believes is that mothers can affect the fate of their children even before they are born. Epigenetic changes passed on during gestation may contribute to depression and suicidal thoughts later on. So what looks like genetics, in reality, is a much more complex interaction between biological and environmental factors, an intricate "if-then" sequence which spans generations.

What this may mean is that my notion of the imprint has to be wound way back. A more accurate framework holds that the experience of the parent leaves an imprint on the sperm and egg. Ng et al. (2010) reported data from experiments with male rats that were fed a high-fat diet. Their sperm seemed to change — that is, many of their babies had adult-onset disease, even though the mothers were normal. The children had a greater frequency for deviated insulin and glucose resistance, hence a propensity for diabetes, even though the fathers had no previous history of the disease. So what looks like pure heredity is actually a molecular memory of the experiential effects on that heredity. These research animals had defects with their on/off switches. This imprint endures and affects our physiology for perhaps a lifetime, sowing the seeds for later adverse effects on the kidney, liver, or heart. For humans, this may mean the tendency to be fat derives, in part, from what a father had before conception. If that father over-ate as a child, his offspring have a much greater chance of being fat and developing diabetes.

It seems, we need to change our focus in order to understand who we are. Our idea of what is heredity is rapidly changing. There are all kinds of intriguing possibilities. In one recent experiment, some animals that were raised in an enriched environment and appeared smarter had offspring who seemed to inherit that intelligence (finding their way through mazes more easily) even though they were not raised in an enriched milieu.

¹⁰Meuret, A. (2011, July 26). SMU: Out-of-the-blue panic attacks aren't without warning. Retrieved from <https://www.youtube.com/watch?v=x3SLGt8smTw>

6. WOMB-LIFE AND THE MYSTERY OF TWINS

Recently Murgatroyd and Spengler (2010) published that life events can induce long-lasting changes in our brain, physiology, and behavior. Early life stress can cause over-secretion of the stress hormone cortisol, which in turn weakens our ability to remember things clearly and cope with stress. (The article includes a detailed explanation of the long-duration effects of methylation). In their study of mice, the researchers and their colleagues found that periodic infant-mother separation just after birth was a major cause of anxiety. The published results from the study end with a rather bleak assessment: "Adverse events in early life can leave persistent marks on specific genes that may prime susceptibility to neuroendocrine and behavioral dysfunction." Yet further evidence that early events have a profound effect on later life.

In addition to altering metabolic function and reshaping our personality, traumatic experiences in the first years of life may weaken the disease-fighting ability of the immune system and reported data for example by Shirtcliff, Coe & Pollak (2009) indicate that children who had an abusive early life or spent time in an orphanage showed a compromised ability to defend against disease. Even after the children were removed from the adverse environment, damage was still apparent. As part of the study, the investigators used the body's ability to control latent herpes viruses as a measure of immune competence. People with an intact immune system can usually keep these viruses under control. Those who are neglected and unloved cannot. Thus, such afflictions as the herpes virus, which often lie latent, are more likely to be activated in those who have poor immune control. In this case, traumatized patients had higher levels of herpes antibodies, indicating their immune systems were compromised against the herpes virus. Those later living in a stable environment still showed higher levels of herpes antibodies.

There is little question now that stress and chronic anxiety of the mother affects the baby's HPA axis, as we have seen. Thus, stress sets the stage for later anxiety in the offspring, partly accomplished by methylation. It heightens cortisol levels, and chronically high stress hormones affect so many functions later in life, not the least of which is thinking and memory (Radtke et al., 2011).

Some studies with twins provide fertile ground for demonstrating the far-reaching impact of womb-life on who we turn out to be and what we suffer from years later. In one study, researchers investigated the perplexing case of identical twin girls born with vastly different physical conditions. One girl was normal while the other had severe birth defects, born with two vaginas, two colons and a spinal cord that split in two towards the bottom of her back. "So how could twins who shared the same genes be so different?" asked the author of a report on the epigenetic research, "The Third Factor: Beyond Nature and Nurture," in *New Scientist*.¹¹

In another study Gordon et al. (2012) found great differences in the methylation patterns even between identical twins. Investigators looked at umbilical cord tissue, cord blood and the placentas of newborn twins and found differences that play an important role in individual development. And here is their important conclusion: "This must be due to events that happened (in the womb) to one twin and not the other," thought Jeffrey Craig of Australia's Murdoch Childrens Research Institute, in a press announcement from the research laboratory.¹² So although twins share a womb, what happens to each of them can be quite different. The study, published online in *Genome Research*, has "for the first time shown that

¹¹Pilcher, H. (2013). The third factor: Beyond nature and nurture. *New Scientist*, 219(2932), 44-47. doi:10.1016/s0262-4079(13)62149-1

¹²Cold Spring Harbor Laboratory. "Differences between human twins at birth highlight importance of intrauterine environment." *ScienceDaily*. ScienceDaily, 15 July 2012. www.sciencedaily.com/releases/2012/07/120715193843.htm.

the environment experienced in the womb defines the newborn epigenetic profile.”¹³ And, no surprise, the authors believe that womb-life events may have a more profound effect than previously thought.

7. REVERSING METHYLATION THROUGH RELIVING

Can we reverse or undo methylation? Can the imprint of trauma be removed at a cellular level? The good news seems to be that, unlike pure genetics, methylation can be reversed, at least through chemical means (Cheishvili, Boureau & Szyf, 2015). Thus, epigenetic-caused disease may be normalized at last. As we mentioned earlier, Meaney et al. (1985) found that deprived animals, when later raised by a more loving mother, experience a partial recovery as higher-level brain processes override some of the effects of early imprints. The neo-cortex can provide compensations for the pain, masking the imprint, but cannot eradicate it. Meaney's rats, deprived and damaged early on by a disappearing mother, were later put into an enriched environment where they seemed happier and played well together. But their stress hormone level was still high; they still suffered, as do humans under similar conditions. Masking the pain is not the same as resolving it, and we may die prematurely from that masking. You might call it a devil's bargain: if we mask the pain we may die early, but if we don't we suffer. However there is a third possibility: relive and integrate the pain, and thus be done with it. There seems to be a window of opportunity before methylation sets in when the imprint can be partially reversed.¹⁴ But it is a narrow, short-lived window. After that the imprint remains for a very long time.

8. HOW TRAUMAS GET EMBEDDED

A recent report by Jovasevic et al. (2015) suggests that some traumatic memories, such as chronic child abuse, are so painful that they get buried deep in the brain and become difficult to access. Those memories were created in a certain mood/feeling or state of arousal and “can best be retrieved when the brain is back in that state.” In addition it seems that each new harmful experience that remains un-integrated at lower levels is later re-represented in a higher level of the nervous system. When a trauma exists later on, it may “resonate” with earlier imprints and set off the whole memory intact. It is here that there may be inordinate responses to the most banal of events. We are winding down again to the originating sources, the base of the feeling. That is how we relive purely physiologic brainstem responses without ever acknowledging them. It is how we get to preverbal events automatically. When the “Primal imprint” sends its message higher up, feelings are added to the impulse, and then later ideas and comprehension are included. Together these form a complete feeling. It may be that specific brain frequencies tie these events together. Figuratively, what is going on is much like the stone thrown into the pond: a ripple effect in the way the neurons connect to each other in mirrored progression. When there are certain kinds of triggers, the brain conjures up its related history, intact, kindling like-minded feelings and their physiology together.

Recent research is showing that being unconscious of pain may work as a survival mechanism and a protection against overwhelming input. But the imprinted memory stays and continues to do its damage over a lifetime. In short, it is not inert. It has a force that

¹³Differences between human twins at birth highlight importance of intrauterine environment. (2012, July 16). Retrieved from <http://genome.cshlp.org/site/press/Announcements.xhtml>

¹⁴In our forthcoming research on demethylation, with Dr. Justin Feinstein of the Laureate Institute for Brain Research, we hope to find more answers. Our hypothesis is that if we take a certain sample (lymphoblasts) from the bone marrow and see how it grows into white blood cells, we can measure demethylation. It is a preliminary theory, but there may be a way, in the near future, to see what effects our therapy or other chemicals may have on methylation.

threatens our ability to adapt, as I explain in detail shortly. The deeper we go down in the brain the more powerful the force of memory. Hence the more unconscious it has to be... for protection. When the trauma is too great or too prolonged, the ability to adapt becomes more feeble and less flexible. This is especially true of long-term neglect and abuse. Here the memories seem to dig in and solidify, impervious to change. And as conscious/awareness lessens, the harm begins, both mental and physical. We are then partially unconscious and are unaware of the damage. Fear-memories are joined by multiple pathways and then are engraved. In short, certain kinds of severe memories need to be tuned properly to receive painful messages. I would put it differently, but we can agree that traumatic memories are stored in the brain, where they remain and cause damage.¹⁵

9. ON THE BREAKDOWN OF OUR ADAPTIVE CAPACITY

In a recent study, entitled "Epigenetic changes in the developing brain: Effects on behavior" has been discussed how methylation works to stamp in painful memory and imprint it (Keverne, Pfaff & Tabansky, 2015). The researchers noted that there are adverse effects on the feeling/hippocampus areas. In short, chronic unrelenting pain overtaxes the native ability to adjust, and we see the results. On the feeling level the person claims, it is all too much. Another related study "Disorder in gene-control system is a defining characteristic of cancer,"¹⁶ Their conclusion: "The behavior of a cancer cell is dictated not only by genetics – by the particular set of mutated genes within it – but also by epigenetics, the system for controlling the expression of genes". Recent findings suggest methylation is one of the defining characteristics of cancer (Landau et al., 2014). Epigenetic science can help explain this. Methylation is the agent for repression, which in turn prevents the person from putting away the pain and moving on (Janov, 2013).

It seems that reliving might be important for the therapy. That is, there is pain and denial of need that overwhelms normal functioning and causes a diversion. This happened because pain installed itself and forced change. And this likely can be measured; the amount of methylation can be observed and changed. That is meaningful progress. It informs us about altering neuroses. In a way, the levels of methylation can be a marker for having been loved early on, or not having been loved.

Neurochemistry may be a more reliable indicator because it has no reason to lie, or rather, no way to lie. It can be a marker for post-traumatic stress or how much repression exists in ADD, or how much pain/repression there is in Alzheimer's disease. We already have some information in this regard because autopsies on depressive/suicides found them to have been heavily methylated in the hippocampal (feeling=memory) area. The more abuse as a child in these cases the more methylation is produced. When we add this to our future research on telomeres and cortisol we will begin to have precise measures of the pain in us. And we will know when a drug is too dangerous for us, particularly the drugs like marijuana that tend to open us to ourselves, to our feelings and pain. Finally we will have a marker for the efficacy of certain psychotherapies. Does the psychotherapy undo the past? Does it help relieve repression and therefore depression? Is there great first line pain in anxiety states? I already can answer that in the affirmative, having treated person after person with anxiety.

The best way to reverse the imprint likely would be through the slow, methodical process of therapy where the least pains can be integrated first, finally descending to the great early traumas and then measuring the results. What is most important is that stress in the mother

¹⁵Paul, M. (2015, August 18). News. Retrieved from <http://www.northwestern.edu/newscenter/stories/2015/08/traumatic-memories-hide-retrieve-them.html>

¹⁶"Disorder in Gene-control System Is a Defining Characteristic of Cancer, Study Finds." Dana-Farber Cancer Institute | Boston, MA. December 8, 2014. <http://www.dana-farber.org/Newsroom/News-Releases/Disorder-in-gene-control-system-is-a-defining-characteristic-of-cancer-study-finds.aspx>.

compromises the repressive system in the fetus, so that later it will be difficult to mitigate surging feelings. Low-level imprints from womb-life burst through the repressive barrier, overloading the system, and – in the absence of a cohesive cortex – result in difficulty focusing and concentrating, and problems learning. The prefrontal cortex becomes overwhelmed as it is pressed into service to counteract and hold down painful feelings.

Why are those early imprints so critical? Because almost every key adverse event in the womb can be life-endangering: low oxygen, inadequate nutrition, too much agitation, flooding by drugs or alcohol, etc. they all affect vital organs and change the system of the baby accordingly. I will never omit smoking, which is deadly to the maturation of the baby. Imagine being in the womb while a mother ingests all kinds of toxins hour after hour, every day of the year. Who can survive that?

There is a beginning to personality development and we must not immediately ascribe it to genetics. Epigenetics is possibly more important. Life circumstances wrap themselves around the gene, and alter who we are and what we become. It is those days in the womb that form the crucible for personality type; they all accommodate life circumstance. They pivot around the imprint; and when we take patients down deep we find the little nugget, the key imprints that forced all that accommodation. And when those early imprints are relived and all the vital signs move as an ensemble down lower, we know we have struck gold. We have found Nirvana, the core of the pain. Remember, there is no suffering in the pure state of Nirvana.

The wonder is how we all manage to keep deep pain stored away, never once acknowledging it. The body does, however, and gets sick. And it makes us sick on the deep cellular level, the level where the early imprints lie. All the pressure to keep pain stored puts the cellular development at risk; eventually we find serious illness, which should not be a mystery but a foregone conclusion. For example, Ma et al. (2013) found that the origins of leukemia may be prenatal. The researchers there delved deeper into the disease process by looking at cases where both twins had developed acute lymphoblastic leukemia, a cancer of the white blood cells. They studied the DNA inherited from both parents, completing a full genome study on their subjects. They found that womb-life was a culprit in the development of the disease. They believe that the mutations accounting for it must have come from the womb. Other mutations may have come after birth. Similarly, Julie Sharp quoted by the BBC News, said, "Studies like this could reveal new ways to target the very roots of cancer and help us better understand how the disease develops over time. Survival rates have increased significantly over the past decades thanks to research, but there is still more to do to make treatments better with fewer side-effects."¹⁷

There are more existing studies that correlate parental abuse with later cancer. One study from Purdue University found that adults who were emotionally and physically abused as children had a much greater likelihood of cancer as adults (Morton, Schafer, & Ferraro, 2012). The more intense the abuse, the more likely the cancer. Imagine now if we have left out of the mix one of the greatest risks of all: constant abuse while in the womb – a drugged or depressed mother, or one who is chronically anxious or tense and angry. Add that to it all and you have one of the great causes of later cancer. The mechanism for this may well be the pattern of methylation that "seals in" the trauma – cancer is characterized by "methylation imbalance" (Baylín, Herman, Graff, Vertino & Issa, 1997).

¹⁷Some findings suggest leukaemia's origins 'back to the womb' (2013, April 9). Retrieved from <http://www.bbc.co.uk/news/health-31622341>

10. CONCLUSION

Recent findings suggest a question, how could we ever get to the bottom of cancer, heart disease, migraines and high blood pressure without a theory of psychological pain? I believe we can reverse some of it in our therapy, but I also believe that the earlier and stronger the imprint the more difficult time we will have to reverse it. The evolution of the genes has been rerouted. Epigenetics reigns. That is crucial; experience cannot change it. There is no way out of the biologic fact of the critical period, the time and space where love must be received or forever more becomes an imprint.

I have written about the irreversibility of early trauma, gestation and birth. The worst-case scenario is a traumatic birth followed by a loveless childhood later on. That compounding can be a person's undoing. It sets up insurmountable emotional problems that create damaged individuals. But having said that there is some hope. A certain level of trauma in-utero and at birth can be ameliorated by mitigating factors, namely plenty of early love. It never erases those traumatic imprints, but it does hold them at bay. I think that part of a good childhood can block the effects of first line early pain. For example, after a terrible birth, hugging and kissing a lot can minimize the damage. The same hugs six years later will not have that effect. That is why a father who leaves home for years and comes back needing acceptance will not get it. The pain is installed and working in the child. The child wants to love but the pain is blocking him. These findings and clinical experiences provided basic data for the theory of critical developmental periods (Andersen & Teicher, 2008).

Maselko (2010) observed the interactions between mother and child at the age of eight months. Those mother-child exchanges were then classified as high- or low-loving interactions. Decades later, the children were studied again as adults. The mothers who were judged most loving produced offspring who were low on anxiety, hostility and general distress. There was more than a seven-point difference in anxiety scores between loved and unloved children, and a three-point differential in hostility scores. Unloved offspring are more hostile. In brief, the higher the mother's warmth, the lower the score in distress. Similarly Maselko wrote: "These provocative findings add to the growing evidence that early childhood helps sets the stage for later life experiences and provide support for the notion that biological 'memories' laid down early may alter psychological and physiological systems and produce latent vulnerabilities or resilience to problems emerging later in adulthood."¹⁸

Doesn't that tell us a great deal? And it means that very early love is so, so important. Without it we have a damaged soul, someone more likely to fall ill and who has poor social skills. That lack of love makes us unable to interact lovingly with others, decades later. Affection is all, even if we had first line pain. You cannot as a parent say, "My children know I love them. I just can't show it." Sorry, that is not good enough. It is like saying I know my child is hungry but I cannot feed her/him.

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¹⁸Brauser, D. (2010, July 28). High Levels of Early Maternal Affection May Lower Emotional Distress in Adult Offspring. Retrieved from <http://www.medscape.com/viewarticle/725920>

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