

May 24, 2011

**Epigenetic transmission of Holocaust Trauma: Can nightmares be inherited?**

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**ABSTRACT**

The Holocaust left its visible and invisible marks not only on the survivors, but also on their children. Instead of numbers tattooed on their forearms, however, they are marked epigenetically with a chemical coating upon their chromosomes, which represent a kind of biological memory of what the parents experienced. As a result, many suffer from a general vulnerability to stress. Previous research assumed that such transmission was caused by environmental factors, such as the parents' child-rearing behavior. New research however, indicates that they may have been also (epi)genetically transmitted to their children. Integrating both hereditary and environmental factors, epigenetics adds a new and more comprehensive psychobiological dimension to the explanation of transgenerational transmission of trauma. A general theoretical overview of epigenetics and its relevance to research on trauma transmission is presented.

*A drowsiness hanged over him; a kind of paralyzing spell. As so many times before, he found himself again in the Ghetto, being chased by soldiers and looking for a hiding place. The nightmares were vivid and emotionally draining and he yearned for peace and relief. He tried to put shape and give meaning to the frightening images that kept haunting him, but he found nothing of the sort. Just a life-long struggle with a past terror that was not his own, with a tragedy that he, himself, had not survived and with a conviction that catastrophe would surely strike again. How long would it go on?*

Many children of Holocaust survivors have had such terrible nightmares in which they are chased, persecuted, tortured or annihilated, as if they were re-living the Second World War over and over again. At these times, they suffer from debilitating anxiety and depression which reduce their ability to cope with stress and adversely impact their occupational and social function. It seems that these individuals, who are now adults, somehow have absorbed the repressed and insufficiently worked-through Holocaust trauma of their parents; as if they have actually *inherited the unconscious minds* of their parents.

Apparently, not only children of Holocaust survivors, but offspring of other PTSD parents are also vulnerable to such a burdensome legacy, including descendants of war veterans (Dekel & Goldblatt, 2008), survivors of war trauma and childhood sexual abuse, refugees, torture victims and many others (Danieli, 1998). Moreover, the transmission may continue beyond the second generation and also include the grandchildren, great grandchildren and perhaps others as well.

This process of *transgenerational transmission of trauma* (TTT) has been repeatedly described in the academic literature for more than half a century (Kellermann, 2009a). Generally speaking, TTT refers to the process in which a trauma that happened to the first generation was passed on to the second generation. Such a process is deeply connected with the general theme of *heredity* – the transmission of characteristics from parents to their offspring. Despite more than 500 studies published, however, we are still unable to sufficiently explain exactly *how* the *unconscious* trauma of a PTSD parent can be *genetically* transmitted to a child and to verify this idea with sufficient empirical evidence. Such a notion evades any simple and logical explanations. How can a repressed memory be passed on from one person to another? Can a child really 'inherit' the unconscious mind of a parent? Is it possible for a child to remember what the parent has forgotten? Will we ever be able to produce neuro-biological 'hard' evidence of such far-fetched and preposterous assumptions and perhaps *see* traces of the unconscious trauma of a PTSD parent in a blood specimen or an MRI scan, of the child? Probably not. But even though we still know very little about the level of specific inheritance of trauma, new research indicates that traumatic experiences of parents may indeed lead to a general disposition to PTSD in the offspring. Family and twin studies have found that risk for PTSD is associated with an underlying genetic vulnerability and that more than 30% of the variance associated with PTSD is related to a heritable component (Skelton, et al., 2011). This heritable component can be observed in the epigenetic marks that affect gene expression patterns in the nervous system.

Epigenetics introduces a promising new and comprehensive explanatory variable of transgenerational transmission of trauma. Since it includes both hereditary and environmental factors, it adds a significant psychobiological dimension which may confirm clinical observations with empirical research. After a presentation and critique of the four prevalent major theoretical approaches to understanding trauma transmission, I will give a general overview of what epigenetics can teach us about TTT and discuss existing and future empirical research in this field. Finally, I will show how the inclusion of epigenetics may explain some of the discrepant findings from previous research on the transgenerational transmission of Holocaust trauma.

### Theory of TTT

Four major theoretical approaches to understanding trauma transmission have been earlier suggested by Kellermann (2001a): (1) psychodynamic relational models; (2) sociocultural and socialization models; (3) family systems and communication models; and (4) biological or genetic models. According to these theories, trauma transmission was assumed to be a function of unconsciously displaced parental emotions, inadequate parenting behavior, family enmeshment, and/or a hereditary predisposition in combination with specific aggravating and mitigating circumstances (Kellermann, 2009). Various manifestations of trauma transmission could thus be explained as being determined by any or all of these factors or by an *ecological* combination of them. For example, the recurrent Holocaust nightmares reported by the child of survivors in the first paragraph could be understood, first as a manifestation of the displaced unconscious fears of the parents. The child was experiencing what the parents themselves could not perceive and express. Second, it could be explained as the result of a specific kind of social learning and parenting. The child responded to the anxieties indirectly expressed in deleterious childrearing behavior. Third, it could be the result of family enmeshment and tacit communication. The child was trapped in a closed environment where survival mechanisms and the rules of war still reigned. Finally, the disorder of the parent would be genetically transferred to the child who then would become predisposed to certain biological stress responses.

At first glance, this integrative view of trauma transmission seems to make perfect sense. Upon further inspection however, they are too *general* to actually explain the *specific* process of how the impact of trauma can cross generations. Children are of course influenced by their parents in a variety of ways, either through heredity or upbringing, or through both (Maccoby, 2000). Studies of human heritability are plentiful and decades of social science research have established clear correlations between social, educational, behavioral, and economic qualities of parents and children. Clearly, there is nothing illuminating about such theories and they could be equally well applied to explain the transmission of traits such as child abuse, criminality or intelligence. Basically, they only confirm the well known saying that “an apple does not fall from the tree” or “such father, such son”, which do not sufficiently explain *how* TTT actually works.

First, psychodynamic models emphasize how children are detrimentally affected by a failure to find gratification of their various relational needs. But even though these theories may be *generally* accepted, they fail to sufficiently explain how the unconscious of the parent can be transmitted to the child when these needs are not met. We still do not understand how the repressed traumatic past experiences of a parent can enter into the mind and soul of the child. Second, while socialization obviously has enormous effects, TTT is not necessarily a function of inadequate child rearing, as shown by a study on parenting in this population (Kellermann, 2001b). ‘Good-enough’ Holocaust parents are also transmitting their past trauma to their children. Third, family systems and communication models of transmission have produced similar ambiguous research results. While too much talk about the Holocaust would obviously lead to a terrible burden being passed upon the offspring, many parents did not share their traumatic experiences with their children, but the children still absorbed much of their trauma. How do we explain that trauma transmission occurred also in harmonious families with plenty of opportunities for separation-individuation? Fourth, constitutional factors are of course highly potent influences but even though the Human Genome Project has mapped out about 3 billion letters of DNA, it cannot explain how TTT works. As emphasized by Jablonka & Lamb (2005), genetic mechanisms alone cannot explain how some cellular traits are propagated and heritable changes in gene expression and regulation that have little to do with DNA sequence seem to be more relevant to explain TTT.

In fact, evolutionary developmental biology may provide a more comprehensive approach to the study of TTT than the above mentioned models. In their book *Evolution in Four Dimensions*, Jablonka and Lamb (2005) suggest that intergenerational transmission may occur not just through the genes per se, but through heritable variations at four levels: Firstly, at the established physical level of *genetics*. Secondly, at the *epigenetic* level involving variations in the “meaning” of given DNA strands, in which variations in the expression of genes during developmental processes are subsequently transmitted during reproduction. The third dimension includes the transmission of *behavioral* traditions. There are for example documented cases of food preferences (cf. hang-ups about food in HS parents) being passed on, by social learning in several animal species, which

remain stable from generation to generation while conditions permit. The fourth dimension is *symbolic inheritance*, which is unique to humans, and in which traditions are passed on through language and culture.

Thus we may conclude that any theory explaining transgenerational transmission of trauma must somehow take into account the powerful *hereditary variations* which explain how parental trauma may be biologically passed on to the child before birth. These theories should explain how children *who have not themselves been traumatized* tend to manifest inherited emotional problems. As such, they must involve the conceptualization of some kind of *indirect* transmission mechanism in which a child is a (secondary) recipient of a parental trauma. From a strictly scientific perspective, such TTT must be caused by *some kind of hereditary factors* that are independent of the behaviors of the parent towards the child. For example, if a tormented war veteran PTSD father abused his son or daughter and the child responded with emotional problems, that child would in effect be a (primary) survivor of childhood abuse, but not (only and specifically) a TTT client. Since most PTSD parents probably affect their children through emotional contagion, these children would be survivors of such primary trauma themselves, rather than survivors of secondary trauma; over-protective mothers would most likely make their children anxious, depressed fathers would probably sadden their offspring and nervous parents would almost certainly produce fearful children, and so on. There is nothing inherently transmitting in such *direct* parent-child influences. What would be a sign of trauma transmission, however, and more difficult to explain, would be a situation in which children inherited the pain of their traumatized parents even though they were treated well throughout childhood. Such theories would explain, for example, the reverberation of trauma in a child with loving and harmonious war trauma parents or the sadness of a child of a depressed biological mother who had grown up in an affectionate adopted family with a happy childhood.

Epigenetic transmission seems to be able to explain such a specific process of TTT.

### **Epigenetic Transmission**

More than two centuries ago, the founder of evolution Jean-Baptiste Lamarck, suggested that acquired characteristics may be transmitted from one generation to another. Ever since, evolutionary developmental biology has continued to study this assumption and recent advances in the field of *epigenetics* are now revealing a molecular basis for how heritable information other than DNA sequence can influence gene function (Bernstein, Meissner & Lander, 2007; Eccleston, et al., 2007). These advances may add greatly to our understanding of trauma transmission and may even establish a promising new research paradigm in the field, as recently pointed out by Yehuda & Bierer (2009); “Epigenetic modifications, such as DNA methylation, can occur in response to environmental influences to alter the functional expression of genes in an enduring and potentially, intergenerationally transmissible manner. As such, they may explain inter-individual variation, as well as the long-lasting effects of trauma exposure. Although there are currently no findings that suggest epigenetic modifications that are specific to posttraumatic stress disorder or PTSD risk, many recent observations are compatible with epigenetic explanations” (p. 427).

Epigenetics is typically defined as the study of heritable changes in gene expression that are not due to changes in the underlying DNA sequence. Such heritable changes in gene expression often occur as a result of environmental stress or major emotional trauma and would then leave certain marks on the chemical coating, or *methylation*, of the chromosomes (Meaney & Szyf, 2005). The coating becomes a sort of ‘memory’ of the cell and since all cells in our body carry this kind of memory, it becomes a constant physical reminder of past events; our own and those of our parents, grandparents and beyond. “The body keeps the score” (van der Kolk, 1994), not only in the first generation of trauma survivors, but possibly also in subsequent ones. Because of their neurobiological susceptibility to stress, children of Holocaust survivors may thus easily imagine the physical suffering of their parents and almost ‘remember’ the hunger, the frozen limbs, the smell of burned bodies and the sounds that made them scared. This kind of epigenetic cell memory can possibly explain how ‘elements of experience may be carried across generations’, as described by Perry (1999);

“*Memory* -- the capacity to bring elements of an experience from one moment in time to another is the unique property of life forms. This remarkable property - the carrying of information across time - is the foundation of every biological process from reproduction to gene expression to cell division – from receptor-mediated communication to the development of more complex physiological systems (including neurodevelopment). To some degree, all of the organ systems in the human body have “memory.” This ability to carry elements of previous experience forward in time is the basis of the immune, the neuromuscular and neuroendocrine systems. Through complex physiological processes, elements of experience *can even be carried across generations*. Elements of the collective experience of the species are reflected in the genome, while the experience of the individual is reflected in the expression of that genome.”

In the same way as parents can pass on genetic characteristics to their children, they can also pass on all kinds of ‘acquired’ (or epigenetic) characteristics, especially if these are based on powerful life-threatening experiences, such as survival from starvation, torture or persecution. Such environmental conditions would leave an imprint on the genetic material in eggs and sperm and pass along new traits even in a single generation. This was metaphorically described by Klein (2008): “My parents are Holocaust survivors and, in all likelihood, had their epigenetics deeply impacted by that experience. In a very real sense, therefore, the Holocaust was imprinted into every one of my cells, at birth, and the same is true for Hannah, my daughter. Our experiences can thus ricochet through the generations at a deep biological level: a sobering thought for a parent.”

Such an explanation of TTT may be brilliantly expressed in computer terminology in which the genome would represent a kind of hardware that remains fixed, while the *epigenome* would represent the variable software with all the memory files. The epigenome thus would function like a ‘switch’, which has the inherent ability to turn certain functions ‘on’ or ‘off’. From such a point of view, offspring of trauma survivors would be somehow ‘programmed’ to express a specific cognitive and emotional response in certain difficult situations. In effect, these children of PTSD parents would be suffering from a kind of ‘software bug’; an error in a computer program or system that produces incorrect or unexpected results, or causes it to behave irrationally. This bug would for example switch on a panic attack and instruct the genes to prepare for ‘fight and flight’ when triggered, as if the individual was thrown into a Nazi persecution manuscript of catastrophic proportions, even in a relatively non-threatening situation. Metaphorically, such an epigenetic coating would affect the child of survivors in a way which is similar to a computer infected with a malicious *virus*; a malware that can copy itself and inflict harm at certain unpredictable points in time.

Any such explanation in epigenetic terms of how the Holocaust trauma can ‘run in families’ must first show that the PTSD parent was somehow ‘damaged’ with some kind of brain short-circuit or constitutional ‘PTSD bug’ and then demonstrate that the child was born with this same ‘bug’. Among children and grandchildren of Holocaust survivors as well as offspring of other traumatized populations, this ‘bug’ would be manifested as a latent susceptibility to (secondary) PTSD and would cause increased vulnerability to stress under certain conditions, such as when a new stress becomes the trigger to a past traumatic event. At such times the epigenetic switch would turn the survival strategy ‘on’ and activate a specific neuro-biological response. Initially, most affected offspring would not be aware of its origin or even of its existence until a new trauma occurs, and then be surprised that some old trauma of the parents would suddenly be surfacing. Schützenberger (2007) described such hidden elements – or unfinished business – in the generational histories as “hot potatoes burning people’s hands, which is quickly gotten rid of and passed down over and over from generation to generation” (p. 157). An excellent 2005 BBC-program depicted such epigenetic transmission as “The Ghost in Your Genes”.

The allusive quality of epigenetic transmission was apparent in a study on Holocaust survivor parenting (Kellermann, 2001b) in which children reported that they had inherited a heavy Holocaust-burden, even though their mothers and fathers were seen in general as ‘good-enough’ parents. They emphasized that a *legacy of suffering* and a kind of *dreadful shadow* had kept dominating their emotions throughout life, but that the origins of these vague sensations had remained largely unconscious for many years. A child of survivor exclaimed: “These tears and these fears suddenly appear ‘from nowhere’ as if some radioactive invisible force had evoked them. Then, they gradually disappear again as if they had only been a part of a dream or a fantasy. The tragic and terrorizing undercurrent is probably there all the time, lurking behind a dark screen, even though I cannot prove its existence...” “It is precisely because of this allusive quality that epigenetics makes so much sense as a suitable explanatory variable for TTT. It’s something latent, but not always manifested, since it may be turned on or off. It’s a kind of disposition, but it is not fixed. You are born with it, but you can also change it. You feel it, but you cannot really be sure where it comes from. You kind of remember it, but then you realize that you never really experienced it in the first place.

We are now in a better position to attempt to answer our original question about the prospect of inheriting the nightmares from our parents’ unconscious. In order to do this, however, we must re-examine psychoanalytic theory with emerging neurobiological research findings (Yovell, 2000), and try to reformulate psychoanalytic concepts in light of emerging biological data, as recommended by Freud (1920): “The deficiencies in our descriptions would probably vanish if we were already in a position to replace the psychological terms by physiological or chemical ones” (p. 60). Regarding TTT, such a reformulation was also suggested by Volkan (1997) who wrote that trauma can be transmitted from parents to their children ‘almost as if psychological DNA was planted in the personality of the younger generation through its relationships with the previous one’ (p. 44). Even if, or perhaps because, the trauma was denied or “forgotten” in the first generation, it would sooner or later find some expression in the emotional distress of later generations. Even though many factors contribute to the origin of bad dreams, we may assume that nightmares that resemble parental trauma suggest the presence of epigenetic transmission. It would be an

indication that the child has absorbed at least some of the parent's PTSD. Thus I propose that the *general* sensitivities of the unconscious of our parents may be inherited through the epigenome and that it has a continual powerful effect on us.

If any *specific* past memory can be epigenetically transmitted or not, however, must be left open to speculation and we should be careful not to slip from reasonable assumptions to fantastic and unsupported scenarios. While a general tendency for having frightening nightmares may well be epigenetically transmitted, and the persecution nightmares of children of Holocaust survivors may be colored by their Holocaust imagery, we are obviously still unable to show that the content of a specific nightmare is affected by epigenetic marks transmitted in a reproductive cell or in the womb.

### Epigenetic Research

The field of epigenetics is becoming increasingly more accepted by the scientific community and there has been a large increase in studies conducted during the last decade. A comprehensive review of more than hundred studies of trans-generational epigenetic inheritance was compiled by Jablonka & Raz (2009) who described the phenomena in a wide range of organisms, including bacteria, plants and animals. These studies included various kinds of adverse conditions, early stress and 'emotional trauma' of the 'first generation' which altered the gene expression in the subsequent generations. Reik, Dean & Walter (2001) also reviewed what is known about reprogramming in mammals and discussed how it might relate to developmental potency and imprinting. More recently, Franklin, et al. (2010) showed that chronic and unpredictable maternal separation induces depressive-like behaviors, not only in the first generation of mice, but also in their offspring. Empirical evidence of epigenetic transmission in human beings, however, is very scarce because of the difficulties in gathering relevant data from human as compared to animal subjects. Some of these will be summarized here briefly.

One of the first epigenetic studies on human beings were carried out by Bygren, et al. (2001) in Överkalix in Northern Sweden. He found that overeating as a youngster could initiate a biological chain of events that would lead one's grandchildren to die decades earlier than their peers did (Cloud, 2010). Thus it was shown – perhaps for the first time – that a famine or over eating at critical times in the lives of the *grandparents* could influence the life expectancy of the *grandchildren*. In their efforts to replicate this astounding finding, Pembrey, Bygren & Golding (2006) conducted another transgenerational study which showed that sons of men who smoke in pre-puberty were found to be at higher risk for obesity and other health problems than sons of non-smoking fathers. Much later, a series of unique post-mortem studies on the brains of men who had committed suicide in Canada (McGowan, et al., 2009), found that the chemical coating on genes seem to have been influenced by exposure to childhood abuse.

The complex mechanisms by which transgenerational transmission of stress responsiveness occur are rapidly becoming a focus of investigation (Matthews & Phillips, 2010). Rachel Yehuda and her team from the Mount Sinai School of Medicine, has been at the forefront of this research for more than a decade (Yehuda et al., 2009). Having found that parental PTSD appeared to be a relevant risk factor for the development of PTSD in adult offspring of Holocaust survivors with PTSD, Yehuda & Bierer (2008) summarized recent neuroendocrine studies in offspring of parents with PTSD. These studies indicated that offspring of trauma survivors with PTSD had significantly lower urinary cortisol excretion and salivary cortisol levels as well as enhanced plasma cortisol suppression than offspring of survivors without PTSD. In all cases, neuroendocrine measures were negatively correlated with severity of parental PTSD symptoms, even after controlling for PTSD and other symptoms in offspring.

Though the majority of their work focused on adult offspring of Holocaust survivors, more recent observations in infants born to mothers who were pregnant on 9/11 demonstrated that low cortisol in relation to parental PTSD appears to be present early in the course of development and may be influenced by glucocorticoid programming in unborn children. Lower cortisol levels were found in mothers who developed PTSD after exposure to the WTC attacks on September 11 compared with similarly exposed mothers who did not develop PTSD (Yehuda, 2002). Pregnant women, who had been close to the World Trade Center during September 11th 2001, gave birth to babies who had elevated levels of stress agents in their saliva (Yehuda, et al., 2009; Chemtob et al., 2010; Sarapas, et al., 2011). This data suggests that effects of maternal PTSD on cortisol can be observed very early in the life of the offspring and highlight the *in utero* effects as contributors to biological risk factor for PTSD (Yehuda, Engel, et al., 2005). Since low cortisol levels are particularly associated with the presence of maternal PTSD, the findings suggested the involvement of epigenetic mechanisms. In a more recent study on combat war veterans with and without PTSD, this line of research was continued and the PTSD+ group again showed greater cortisol and ACTH suppression (Yehuda, et al., 2010; Yehuda, et al., 2011).

In an early study of maternal Hypothalamic-Pituitary-Adrenal Axis (HPA-axis) functioning, Schechter et al. (2004) measured maternal salivary cortisol within a clinical sample of mothers before and after a mother-child interaction protocol involving

separations and reunions. The study showed modest, but significant associations between pre-separation cortisol as well as cortisol reactivity with the severity of maternal PTSD, dissociative symptoms, and atypical care giving behavior. Later studies of gene environment interactions focused on environmental stressors such as interpersonal violence and the regulatory effects of the serotonin transporter gene and other genes with which it is known to interact on the HPA axis (Kochanska, Philibert & Barry, 2009).

Apparently, parenting itself may be epigenetically transmitted from parent to child. A fascinating study of gene-environment interaction, Beaver & Belsky (2011) recently found a significant interaction between parenting quality and cumulative genetic plasticity in the prediction of parental stress during adulthood. Depending on genotype, parenting quality was thus shown to differentially affect future parental stress. Exposure to maternal parenting was measured prospectively when respondents were adolescents and parental stress was measured when they were parents themselves, some 14 years later. Some genes that seem to affect neural plasticity were shown to be involved and the variation in these genes affected parental behavior and the response to stressful parenting.

Finally, a range of different neurotransmitters have been investigated, from serotonin and dopamine to neuropeptide Y, brain-derived neurotrophic factor, and the glucocorticoid receptor in the predisposition to PTSD. In their review of molecular genetic studies relating to PTSD, Broekman, Olf & Boer (2007) found inconsistent results among 8 major genotypes: serotonin (5-HTT), dopamine (DRD2, DAT), glucocorticoid (GR), GABA (GABRB), apolipoprotein systems (APOE2), brain-derived neurotrophic factor (BDNF) and neuropeptide Y (NPY). According to Binder, et.al, (2008), several single-nucleotide polymorphisms (SNPs) in FK506 binding protein 5 (FKBP5) interact with childhood trauma to predict severity of adult PTSD. These findings suggest that individuals with these SNPs who are abused as children are more susceptible to PTSD as adults (cf. Newton, 2008). Recently, Ressler et al. (2011) found that a particular hormone-like molecule called PACAP (pituitary adenylate cyclase-activating polypeptide), which is known to affect stress response on the cellular level is significantly very high in women with PTSD.

As can be seen from the above examples, the potential for creative research in this field are huge. However, though it is widely accepted that epigenetic factors can play an important role in the development and transmission of PTSD, “there have been no empirical demonstrations of epigenetic modifications per se in association with PTSD or PTSD risk” (Yehuda & Bierer, 2009, p. 430). Uncovering the heritable biomarkers that are involved in TTT would thus be an important task for future research. What is required to reach this goal? What kind of basic research would help us get one step further? Which gene environment data is necessary to improve our understanding of how epigenetic TTT actually works and how psychosocial factors may cause psychiatric disorders (van Praag, 1979)? Apparently, any or all of the following four assumptions would have to be confirmed in order to answer these questions:

1. That a stressful event was experienced by the parent but not by the child,
2. That this event had an observable neuro-biological effect on the parent, who would be then diagnosed as suffering from PTSD,
3. That it would be leave an observable ‘mark’ on DNA methylation or other epigenetic factors in the parent, and
4. That this same mark would be observable also in the child, and shown to lead to susceptibility to stress.

Many years of brain research has shown that human beings are ‘hard-wired’ for stress through an intricate pattern of neural pathways designed for the fight-or-flight response. Research also suggests that chronic stress appears to destroy brain tissue, specifically the hippocampus and much of research on the fear response in humans has focused on the activating of the amygdala in subjects with PTSD (Ressler, 2010). On the basis of previous findings (Skelton, et.al, 2011), such neuro-biological research would study the (a) neuroendocrinology with biochemical analysis of for example the HPA function at baseline and with stressful challenge in media samples of serum, EDTA and Heparin plasma, urine, saliva and tissue culture cortisol levels; (b) the neurocircuitry interconnecting the limbic system and frontal cortex with neuroimaging techniques, e.g. the hippocampal structure with MRI and the hippocampal function with PET; and (c) the locus coeruleus-noradrenergic system.

According to Yehuda & Bierer (2008), intergenerational effects related to PTSD and HPA-axis stress reactivity are also likely via epigenetic mechanisms. New techniques are investigated to search the genome or gene modifications that have been identified as epigenetic risk factors. But while the majority of the initial investigations into main effects of candidate genes hypothesized to be associated with PTSD risk have been negative (Segman & Shalev, 2003), promising avenues of inquiry into the role of *epigenetic modifications* have been proposed and future studies of PTSD *epigenotypes* may help to elucidate the neurobiology of inherited PTSD. Epigenomic studies that look at patterns of methylation in many loci and particularly on

candidate genes are presently conducted in various places. Similar to the Human Genome Project (Wise, 2008), a public/private collaboration has initiated a Human Epigenome Project which aims to “identify, catalogue and interpret genome-wide DNA methylation patterns of all human genes in all major tissues.” (HEP, 2011). These institutions search for a particular form of a gene variation on a specific chromosome which makes some people more likely to develop PTSD than others. While simplified biological models may not properly capture the complex etiology of PTSD (Vidlock, et al., 2008), and though studies of genotype may only present a limited picture of the molecular biology of this disorder, there seems to be a clear rationale for examining genetic factors in PTSD in conjunction with environmental factors, such as trauma exposure. According to Yehuda, Koenen, Galea & Flory (2011), the examination of epigenetic mechanisms together with gene expression will help refine models that explain how PTSD-risk and recovery are mediated by the environment.

### **Conclusion**

Presenting such verifiable data of TTT would have far reaching consequences. First of all, it would continue to reinforce the paradigm shift in scientific thinking that underscore the impact of stressful events on the physiology not only of the trauma survivors themselves, but also of their offspring (Matthews & Phillips, 2010). Furthermore, improved understanding of epigenetic transmission of PTSD in children of trauma survivors allow for more accurate diagnosis, improved prevention and more targeted treatment interventions of such clients, leading to a sort of ‘epigenetic medicine’ (cf. Church, 2009). Specific epigenetic therapies hold promise for a wide range of biological applications, from cancer treatment to the development of induced stem cells (Hamm & Costa, 2011), as well as for TTT. Finally, any such verifiable data of trauma transmission may have legal consequences for generations of trauma survivors who want to claim reparation for their epigenetically inflicted wounds.

Most importantly, however, new epigenetic data has the potential to settle some controversies from previous research. A recent overview of such research (Kellermann, 2011) concluded that the contrasting forces of vulnerability and resilience were both present in many Holocaust survivors and their children. But how did the first generation of survivors achieve so much, and how can their children function so well? And how can we understand that offspring who came to psychotherapy complained so much from various kinds of secondary traumatization effects, while epidemiological studies repeatedly failed to show that they were any different from comparable populations? Clinical observations and controlled research were consistently divided in their assessment of this population for many decades. With the added use of epigenetics, however, this dispute has become much more reconcilable. Epigenetic transmission models make the discrepant findings regarding the presence or absence of specific psychopathology as well as the simultaneous presence of both frailty and hardiness in this population much more explicable. Because from the point of view of epigenetics, any inherited (genetic) dispositions can be either turned on or off, and thus activate either overwhelming anxiety or sufficient coping in the same person at different times, according to certain aggravating and mitigating (environmental) factors (Kellermann, 2009). As emphasized by Yehuda & Bierer (2009), “integrating epigenetics into a model that permits prior experience to have a central role in determining individual differences is also consistent with a developmental perspective of PTSD vulnerability” (p. 432).

Finally, epigenetics opens up a potentially more optimistic view of health and disease in offspring of trauma survivors. Since epigenetics conveys that human beings are not only predestined, but also highly malleable creatures, they are able to reverse the deleterious effects of trauma and find some closure to the endless multi-generational saga. This may be achieved either through a variety of established psychotherapeutic interventions or through new psycho-pharmacological drugs, or a combination of both. Even though such offspring might still be more or less influenced by their genes and despite their physiological predestination, they might realize that it’s up to them to decide what to do with all of it. Instead of succumbing to the emotional effects of the past tragedies, they might search and find some kind of personal transformation journey that gives new meaning to their legacy. After all, numerous studies on resilience in trauma survivors have confirmed the wide range of proactive strategies that traumatized people have utilized to cope successfully with insurmountable adversities (e.g. Seligman, 2011). The very same successful adaptive survival strategies described by Helmreich (1996) as enabling Holocaust survivors to overcome their difficulties ‘despite all odds’ (such as courage, initiative, flexibility, assertiveness, stubbornness and optimism), can thus be utilized also by their children (who in fact may have also inherited these characteristics).

The bad news with epigenetics is that we have to carry the load of our parents. The good news is that we apparently can do something about it. With an awareness of its existence and a better control of our inner ‘switch board’, we might choose to switch off the ancient horror images and switch over to another, more life-affirming channel. Perhaps this will give the next generation a somewhat lighter epigenetic burden to carry?

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