

Past, present and future perspectives of Holocaust trauma transmission

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Abstract

For almost seventy years, observational and empirical data have been collected on a unique population at risk because of their parents' war-time experiences, resulting in extensive and sometimes confusing findings on the intergenerational transmission of Holocaust trauma in survivor families. This paper presents a critical overview of past research, discusses the current shift from a psychosocial to a neurobiological focus, and delineates the essential variables in future research on trauma transmission. Such variables include *particular* parents who transmit *various* influences to *certain* individual children under *specific* circumstances and during *different* critical time periods. A more integrative conceptual model of trauma transmission is suggested from a psychosocial and neurobiological life-course developmental perspective that may guide future research in this field with a variety of populations. Offspring who were earlier regarded as being either vulnerable or resilient are now viewed as being more or less biologically susceptible to a more or less accommodating environment.

Can parental traumatization be transmitted to their offspring? This question has repeatedly been asked ever since the Second World War in connection to the offspring of Holocaust survivors (OHS). This population has regularly been studied almost since they were born; from infancy, childhood, adolescence, throughout their early adulthood and midlife ([Shrira, Palgi, Ben-Ezra, & Shmotkin, 2011](#)), to the present stage of mature adulthood and old age. For almost seventy years, qualitative and quantitative data have been collected, resulting in a sizeable cumulative database that provides a unique long-term perspective of a population at risk due to their parents' war-time experiences.

The knowledge gained from Holocaust trauma transmission (HTT) has substantial translational applications since it is relevant also for the offspring of survivors from a

wide variety of other traumatic events. This includes the offspring of survivors of other types of mass trauma ([Bezo & Maggi, 2018](#)), victims of Apartheid-era human rights violations ([Adonis, 2016](#)), Native Americans ([Brown-Rice, 2013](#)), natural disasters ([Juth et al., 2015](#)), as well as the offspring of combat veterans ([Creech & Misca, 2017](#); [Dekel & Goldblatt, 2008](#)), ex-POWs ([Costa, Yetter, & DeSomer, 2018](#)), survivors of terrorist attacks ([Weinstein et al., 2018](#)), and victims of torture and domestic violence ([Daud, Skoglund, & Rydelius, 2005](#); [DeGregorio, 2012](#); [Timshel, Montgomery, & Dalgaard, 2017](#)). Indeed, anyone who has experienced an adverse event may wonder how the impact on themselves may also have affected their children.

The question is not only limited to the transmission of anxiety disorders, and complex stress-related disorders such as PTSD, which are moderately hereditary ([Banerjee, Morrison, & Ressler, 2017](#); [Duncan et al., 2007](#)). It is also relevant to a wide range of mental disorders that can be passed on from parent to child. For example, the offspring of parents with severe mental illness are at increased risk for a range of psychiatric disorders ([Rasic, Hajek, Alda, & Uher, 2014](#)). However, despite substantial progress in other areas of psychiatric genetics, few risk factors have been identified ([Smoller, 2016](#)) and there are still many open questions regarding trauma transmission in general: *Who* is more likely transmit, *what* is transmitted, *who* is more susceptible, *where* and *when* is it more likely to happen, and *how* does such a transmission transpire? Since no definitive answers have been found to these questions, the purpose of this paper is to take a new look at the underlying conceptualizations and assumptions of trauma transmission. A summary of the currently available knowledge on HTT will enable us to draw general conclusions from separate psychosocial and neurobiological investigations that address similar or related assumptions. By focusing on the past, present and future perspectives, the review will critically evaluate the strengths and weaknesses in past research, highlight important issues that have been left unresolved and suggest how future research can produce new information on the relevant variables in the study of trauma transmission. In conclusion, a more comprehensive and integrative conceptual model of transmission will be suggested from a developmental systems perspective that can guide future research in this field with a variety of populations.

Past perspectives

During more than half a century, studies on OHS have advanced through the following simplified and roughly estimated six decades:

The 1960s: Initial case studies were based on clinical observations and anecdotal reports of adolescent OHS who seemed to be different from their peers (Rakoff, Sigal, & Epstein, 1966).

The 1970s: From similar observations of OHS, who had now reached early adulthood, psychoanalytic case studies were published. Preliminary empirical investigations were also carried out ([Sigal, 1971](#)), but later criticized for methodological flaws ([Solkoff, 1981](#); Albeck, 1994). When the book, *Children of the Holocaust* (Epstein, 1979) was published, it resonated well with OHS and gave them a distinct identity as the *Second Generation*.

The 1980s: Various conceptualizations of HTT were suggested, and the professional literature increased in scope (e.g., Bergmann & Jacovy, 1982). Concurrent with the launch of PTSD, and its increased application in the treatment of Holocaust survivors, clinicians started to question if this disorder could be intrinsically transmittable to offspring. Individual and group psychotherapies for young adult OHS were therefore provided in many countries. At this time many OHS became parents themselves and thus gained a deeper understanding of what it means to be a parent.

The 1990s: The number of controlled studies increased and also shifted to nonclinical samples (Felsen, 1998). Even though these studies did not find more psychopathology in adult OHS (e.g., [Major, 1996](#)), more wide-ranging study designs were used to investigate the interaction between parents and offspring in Holocaust survivor families ([Bar-On et al., 1998](#)). Different outcomes in these studies led to an attempt to integrate earlier findings of vulnerability and resilience, and the entire field became more diversified and multi-faceted. Initial results from studies on possible biological correlates also began to appear ([Yehuda, 1999](#)).

The 2000s: When OHS had reached mid-adulthood, a more integrative understanding of trauma transmission had evolved ([Kellermann 2001c](#)), with an increasing focus on the aggravating and mitigating factors ([Kellermann, 2008](#)). In comparative studies on representative samples, OHS were not found to differ in secondary traumatization (meta-analysis: [Van IJzendoorn, Bakermans-Kranenburg, &](#)

[Sagi-Schwartz, 2003](#)), nor in physical morbidity ([Levav, Levinson, Radomislensky, Shemesh, & Kohn, 2007](#)). Similar results were reported in a later review of studies on the impact of genocide on children's psychopathology in general ([Lindert et al., 2017](#)). The clinical subgroup, however, continued to present emotional problems and utilize mental health services. Possible epigenetic pathways of transmission were suggested for this population ([Yehuda & Bierer, 2009](#)).

The 2010s: As OHS reached late adulthood, the number of OHS studies declined, with a focus on the aging process ([Shrira, 2018](#)), and the biological basis for Holocaust trauma (HT).

A historical and life course approach is useful in order to understand the changes in OHS over time ([Hareven, 1994](#)) because simultaneously with the advance in research on OHS, the population also changed across time, through childhood, adolescence, young adulthood, adulthood, and old age. Each stage of development required a different coping strategy. Most importantly, their perception of the Holocaust and its impact on themselves gradually shifted. Findings from the 1970s, when OHS were young adults, were unlike those reported in 2015 when they were middle-aged or older. Younger OHS presented more identity problems than older OHS who had developed a more stable personality. Meta-analytic studies that did not consider such a time perspective may, therefore, have reached faulty conclusions on the population of OHS as a whole. Earlier studies should, thus, be understood from when the specific studies were conducted and the biographical time of the participants when the studies recruited and examined them.

Changes in post-war readjustment were also reported in contemporaneous studies on Holocaust survivors, completed with regular intervals ten, twenty, thirty, forty, fifty, sixty, and seventy years after the war ([Kellermann, 2018](#)). These studies surveyed together indicate that survivors gradually reversed the harmful effects of trauma instead of succumbing to the emotional effects of their past tragedies. The Holocaust survivor parents who transformed their lives by finding a new meaning to their legacy also helped OHS to find some closure to the multi-generational saga.

Present perspectives

The OHS who were born early after the war are now retired or approaching retirement age while those who were born later have reached mature adulthood. Some are caring for their elderly parents ([Shrira, Menashe, & Bensimon, 2018](#)), but many have already lost them. When becoming parents and grandparents themselves, their focus of attention has shifted to their own children and grandchildren. Numerous OHS have built successful careers and seem to have transformed the legacy of the Holocaust into post-traumatic growth. For them, the impact of the Holocaust has become less important in their lives, and they seem no longer to be “lost in transmission” ([Fromm, 2012](#)). The scientific debate about whether OHS suffer from more psychopathology than comparable populations is mostly settled. It is now widely acknowledged that parental trauma does not have a universal detrimental effect OHS in general. Only a few OHS meet the criteria for mental disorders and require mental health treatments from time to time. Many more are preoccupied with disorganizing experiences ([Scharf & Mayseless, 2011](#)) and have occasional Holocaust associations from time to time. Some also struggle with stress-related problems and a lack of emotional resources when faced with adversity.

The paradoxical coexistence of both vulnerability and resilience in this population was confusing to clinicians and researchers who were unable to understand how OHS could function so well and achieve so much while struggling with emotional stress symptoms throughout their lives. The possible role of epigenetic mechanisms ([Burggren & Crews, 2014](#); [Jablonka & Lamb, 2015](#); [Lehrner & Yehuda, 2018b](#); [Yehuda & Lehrner, 2018](#); [Youssef, Lockwood, Su, Hao, & Rutten, 2018](#)), offer an explanation of this observation because epigenetic “vulnerability markers” can lay inactive and do no harm for years, until being suddenly switched on in a threatening situation. Thus, it was suggested that OHS might have inherited such chemical markings upon their chromosomes, similar to the numbers tattooed on their parents’ forearms that lead to their “inherited nightmares” ([Kellermann, 2013](#)). These marks would make it possible for the children to “remember” in their bodies what their parents had repressed.

Epigenetics provided a plausible theory to explain how early life traumas can produce functional “scars” in parents ([Gröger, et al., 2016](#); [McEwen, 2017](#); [Skinner, Mohan, Haque, Zhang, & Savenkova, 2012](#)), and also transmit them to offspring in the form of epigenetic methylation marks; the reversible chemical modification to DNA that

typically blocks transcription of a gene without altering its sequence. During the last decade, exhaustive reviews have been published on transgenerational epigenetics and psychiatric disorders ([Franklin et al., 2010](#)), fear memory and biomarkers ([Maddox, Schafe, & Ressler, 2013](#)), epigenetic risk factors in PTSD and depression ([Raabe & Spengler, 2013](#)), epigenetic mechanisms in learned fear ([Zovkic & Sweatt, 2013](#)), neural fear network and PTSD ([Vukojevic et al., 2014](#)), early life stress ([Provençal & Binder, 2014](#)), the inheritance of learned behaviors ([Dias, Maddox, Klengel, & Ressler, 2015](#)), recent genetics and epigenetics approaches to PTSD ([Daskalakis et al., 2018](#)), and most recently on the inter- and transgenerational inheritance of behavioral phenotypes ([Jawaid & Mansuy, 2019](#)). Contrary to previous notions, ground-breaking research showed that DNA methylation could escape the “reset” mechanism or “reprogramming” in human germ cells ([Bohacek & Mansuy, 2015](#)). Because of these emerging findings, there was a paradigmatic shift in the study of HTT. While HTT was earlier investigated mostly within psycho-social disciplines, an increasing number of transmission studies include a measurement of (1) the neuroendocrine, (2) neuroanatomic, and (3) the epigenetic systems ([Bowers & Yehuda, 2016](#); [Klengel, Dias, & Ressler, 2016](#)).

(1) *Neuroendocrine*: Amongst such psychobiological correlates, altered HPA axis reactivity was the most investigated in OHS with parental PTSD ([Yehuda & Bierer, 2007](#); [Yehuda, Teichler, Seckl, et al., 2007](#)). OHS were found to have significantly lower cortisol, but better cortisol suppression in their blood, than offspring of survivors without PTSD ([Yehuda, 2009](#)). There is no consensus, however, in how to interpret these findings. Cortisol responses show large intra- and inter-individual variability ([Yehuda & Seckl, 2011](#)) and it is not clear if the contra-intuitive findings of low cortisol signified a preexisting vulnerability, a reaction to new exposure, or an attempt to regain an inner balance. There is also disagreement regarding baseline levels of cortisol observed in people with PTSD. The finding of low cortisol levels in trauma survivors and their offspring have, therefore, been the subject of much debate, and their current clinical application remains limited ([Olff & van Zuiden, 2017](#)). While reduced cortisol levels and enhanced GR sensitivity are still assumed to be a risk factor for developing PTSD ([Szeszko, Lehrner, & Yehuda, 2018](#)), other pathways of traumatization are increasingly investigated, such as neuroanatomical correlates and epigenetic methylation markers.

(2) *Neuroanatomic*: Exposure to excessive stress hormones also has an effect on brain structures involved in cognition and mental health ([Lupien, McEwen, Gunnar, & Heim, 2009](#)). The advent of noninvasive brain imaging techniques, such as fMRI and PET, made it possible to identify various brain circuits affected by traumatization. Studies have detected changes in brain structure and function in patients with PTSD ([Rauch, Shin, & Phelps, 2006](#)). Such changes were observed, for example in the hippocampus and in the amygdala, as well as in cortical regions including the anterior cingulate, insula, and orbitofrontal region ([Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008](#)), which are involved in the processing and regulation of emotion ([Sherin & Nemeroff, 2011](#)). Episodic memories of fear were found to be located in the dentate gyrus in the hippocampus ([Besnard & Sahay, 2016](#)). Abnormalities in processing involving the salience network, including the amygdala, anterior cingulate cortex, and insula, may also play a role in successful PTSD treatment response ([Szeszko & Yehuda, 2019](#)).

Neuroanatomic HTT would thus suggest that traumatic stress can cause, not only an irreversible effect on the parent's brain but also that the irregular brain-circuits can be inherited by OHS. The child would be born with a preprogrammed salience network and thus respond with excessive anxiety when faced with situations that resemble the war. There is an ongoing search for the location in the brain where trauma memories are located ([Sartory et al., 2013](#)). If these locations are found, they can be correlated with similar findings in offspring to determine a possible hereditary path.

(3) *Epigenetic*: The first actual findings of epigenetic transmission in Holocaust survivor parents and their offspring were published only a few years ago ([Yehuda et al., 2014](#)). It was found that Holocaust exposure affected FKBP5 methylation in parents and also in their offspring, a correlation not found in the control group and their children ([Yehuda et al., 2016](#)). These findings created a lively debate in the scientific community ([Yasmin, 2017](#)), and the study became known as the “most over-interpreted epigenetics study of the week” in a blog ([Birney, 2015](#)). In response to this critique, [Yehuda, Lehrner, & Bierer \(2018\)](#) claimed that their findings had been over-interpreted in the popular media and that methodological difficulties remain to confirm the epigenetic transmission hypothesis. For example, experimental biological data has come mostly from animal studies. There is a great deal of controversy within the study of

transgenerational epigenetics in humans ([Albert, 2010](#); [Heard & Martienssen, 2014](#); [Nagy & Turecki, 2015](#)).

The research on epigenetic HTT involves multiple stages. Proving the existence of a psychobiological predisposition involves showing, not only that the body of the parent actually can “keep the score” of trauma ([van der Kolk, 1994](#)), and how mental stress is physically manifested in biological correlates ([McEwen, 2018](#); [Nasca, Rasgon, & McEwen, 2018](#)), but also how such manifestations are inherited by the following generations. Several laboratories are searching for the biochemical origins to stress responses in non-exposed descendants ([McEwen, 2017](#); [Skinner, Mohan, Haque, Zhang, & Savenkova, 2012](#)). However, as for now, no valid and clinically applicable biomarkers for PTSD have been found (Schmidt & Vermetten, 2017), and there is insufficient evidence of the assumption that massive stress exposure in parents can influence the risk of stress-related mental problems in their children. As a consequence, there is no tangible evidence for the epigenetic inheritance of phenotypes in the etiology of HTT, and no “Geiger-counter” for Holocaust traumatization ([Kellermann, 2019](#)).

The interpretability of epigenetic findings of psychological phenomena is a complex undertaking ([Jones, Moore, & Kobor, 2018](#)). Obstacles are caused by methodological constraints and also because HT cannot merely be regarded as one specific and persistent disorder detached from the human mind. Difficulties in finding biomarkers are due to the reality that traumatization in itself (1) cannot be easily measured in human beings; (2) is not clearly identified; (3) tends to vary between individuals and populations; (4) is not constant over time; and (5) is the result of a failure to regain physiological homeostasis rather than a simple physiological response to stress ([Kellermann, 2018](#)). While these obstacles will make future research on psychobiological HTT a challenging undertaking, chronic stress in the first generation has documented effects on the development of physiological, neural, and behavioral phenotypes in the second generation ([Crews et al., 2012](#); [Gillette, Son, Ton, Gore, & Crews, 2018](#); [Hanson & Skinner, 2016](#)). An epigenetic perspective ([McEwen, 2019](#)) across the lifespan of OHS seems, therefore, to be the most promising avenue for future progress currently ([Petronis, 2010](#)).

Previous studies were based on descriptive, epidemiological and correlational data from retrospective cohort studies on small samples ([Lindert et al., 2017](#)). These

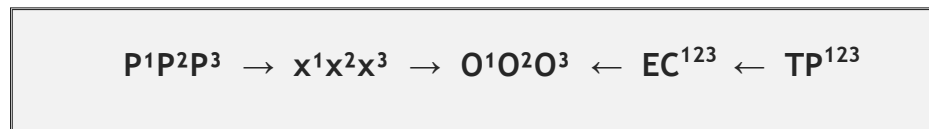
studies did not fulfill the essential methodological criteria that transmission research should meet, such as prospective designs, large representative samples, valid and reliable measures and different reporters for each generation ([Thornberry, Knight, & Lovegrove, 2012](#)). It is therefore not possible, at this time, to give a final assessment of the trauma transmission hypothesis. Even though much progress has been made, neither the past psychosocial nor the contemporary epigenetic perspective has produced a definitive answer to the question of whether Holocaust traumatization can be transmitted to OHS.

Major variables of HTT

Trauma transmission was initially hypothesized as psychological responses of offspring to parental behaviors that influenced the process of transmission under different circumstances ([Kellermann, 2001c](#)). This conceptualization has been expanded to include *particular* parents (P) who transmit *various* influences (x) to *certain* individual children (O) under *specific* environmental circumstances (EC) and at *different* critical time periods (TP).

The dependent variable - (x) - is a function of various extraneous or independent variables, creating the equation $f(x) = P+O+EC+TP$ (and possibly others) (cf. Figure 1).

Figure 1.



(P) = Parental diversity factors. (x) = The content (*what was transmitted?*).

(→) = The process (*how was it transmitted?*). (O) = Offspring diversity factors.

(EC) = Environmental factors. (TP) = Time periods.

After some brief remarks on the ambiguous terminology in transmission studies, I will discuss (1) conceptual issues within the different kinds of transmission (x), (2) parental diversity factors, (3) offspring diversity factors, (4) environmental diversity factors, (5) critical times of transmission, and (6) the process of transmission.

Ambiguous HTT terminology

It is often difficult to make an exact distinction of survivors, offspring, and grandchildren in transmission studies, and to clearly define to which group a specific person belongs. This ambiguity is caused by the fact that a variety of labels have been used to designate the major players in this multi-generational drama. Within transgenerational studies, the Holocaust survivors regularly belong to the F0 generation, even though they are not necessarily the first generation. They can also be the second or third generation considering that they may be the offspring of trauma survivors from World War One, or children of parents and grandparents who experienced pogroms, violent revolutions and economic hardships during the Great Depression. However, even when disregarding this ambiguity, and including only those who experienced the Second World War firsthand, nine vastly diverse groups of this first generation have evolved during the post-war years ([Kellermann, 2012](#)). These include persecuted Jews, displaced persons, war refugees, former camp inmates, victims of Nazi persecution, survivors of the Holocaust, war-witnesses, eligible survivors and hardship survivors. The designation Traditional Generation has most recently come into use, including Veterans, the [Silent Generation](#) or the Greatest Generation.

OHS are generally known as the Second Generation, the F1 generation within transgenerational research, or [Baby boomers](#) in post-war history studies and the popular media. They differ significantly from offspring of child survivors, who are not only younger than those born immediately after the war, but also grew up in a different period, and are called “[Generation X](#)” in popular media. Their diversity will be further discussed below.

The grandchildren of survivors are a highly heterogeneous age-cohort and have been lumped together as the “Third Generation,” even though it is unclear how many survivor grandparents they must have to belong to this group. They would constitute the F2 generation within transgenerational research, and “[Millennials](#)” or “Generation Y” in popular media.

The most important difficulty to separate the various generations, however, comes from the fact that transmission of trauma does not always represent a “true” transmission from exposed F0 to unexposed F1. Since abuse in itself tends to “run in families” ([Ertem, Leventhal, & Dobbs, 2000](#); [Montgomery, Just-Østergaard, & Jervelund,](#)

[2018](#)), it is often difficult to differentiate the exposed generation from the unexposed one ([Bowers & Yehuda, 2016](#)). Harmful child-rearing behavior by the F0 parent in effect transforms the offspring from being (unexposed) F1 to becoming (exposed) F0. OHS who became traumatized by growing up with abusive parents who had PTSD may, for example, be included in this group, and be regarded as both F0 and F1.

Because of these ambiguities, and in order to more accurately describe how environmental influences propagate across generations, it is essential to distinguish between “intergenerational” and “transgenerational” transmission ([Klengel, Dias, & Ressler, 2016](#)) of parental trauma. The influence of parents upon their offspring (F0 to F1) is called “intergenerational transmission” (between generations) while the more stable transmission to further generations (F0 to F1, F2, F3 and further) is called “transgenerational transmission” (across generations). For a transgenerational inheritance to be epigenetic, it must be transmitted through gestational exposure to epi-mutations in the gametes, and can only be confirmed with evidence from F2 male subjects or F3 females because the ovum of a granddaughter is present already in the ovum of the grandmother. To date, there is a growing body of evidence for intergenerational transmission of environmental exposures in animals and humans (see reviews mentioned above), but still little data on transgenerational inheritance in humans ([van Otterdijk & Michels, 2016](#)).

1. Different possible kinds of transmission (x)

The theory of HTT postulates that something (x) is passed on from parent to child. But what is it? Can it be anything or only something specific that is associated with the Holocaust? Should it focus on stress-related psychopathology? If so, should it primarily emphasize intangible or tangible aspects of traumatization? A more precise description of the contents of transmission is needed because as long as we have not defined what we mean by Holocaust traumatization in the first generation, it is not clear what we are trying to find in the second.

Studies on transmission consider whatever is transmitted (x) as the dependent outcome variable. It has mostly been described in terms of a mental disorder. However, there is a great deal of diversity among the clinical population of OHS, and a lack of consensus regarding the possible mental health manifestations of HTT. It is, therefore,

unclear if the assumed psychopathology in OHS should be described in terms of an already established diagnostic entity, or if it should be given a new label, such as transgenerational disorder, cultural trauma, or anything else.

When asking OHS what they have absorbed from their Holocaust survivor parents, a great variety of answers will be received. Some will talk about their identity conflicts, their cognitive dissonance of “knowing and not knowing” ([Laub & Auerhahn, 1993](#)), their persistent worries and sadness, their interpersonal difficulties, and their struggles to cope with stress ([Kellermann, 2001d](#)). Others will mention their (more or less adaptive) survival mechanisms, which can later be interpreted as resilience, or post-traumatic growth ([Calhoun & Tedeschi, 2006](#)). Can these self-reported characteristics be regarded as possible manifestations of HTT? Can any sign of distress found in the second generation be evidence of HT from the first generation? If so, should the classic symptoms of PTSD be a focus of study, with intrusion, avoidance, and hyperarousal, also in OHS, or should some “milder version” of this disorder, such as “post-traumatic stress symptoms” (PTSS) ([Danieli, Norris, & Engdahl, 2016](#)), or “post-traumatic embitterment disorder” (PTED) ([Lehrner & Yehuda, 2018a](#)) be the assumed outcome?

Many of these conceptual questions are open for discussion. OHS cannot be assumed to be aware of all the traits that they absorbed from their parents, and self-report studies will not provide all the answers. The tendency of some OHS to suffer silently and not to ask questions (also found in survivors of abuse who claim that “it does not hurt”), make self-reports very unreliable. Studies using close-ended questionnaires tend to minimize symptomatology while disclosing such manifestations after some more in-depth interviewing. As a result, and despite numerous reports on the manifestations of HTT, it is still difficult to fully delineate all possible kinds of transmission in OHS. What can be concluded, however, is that OHS obviously did not “inherit the trauma” of their parents. According to [Yehuda, Lehrner & Bierer \(2018\)](#), “terms such as ‘inherited trauma’ ... obfuscate rather than clarify what is being transmitted and how —indeed, how can an experience be inherited? It is clearer to frame the discussion around how the impact of trauma occurring to the parent can affect the offspring. Thus, the term ‘intergenerational trauma’ is misleading because it is meant to refer to the intergenerational manifestation of the effects of parental trauma” (p. 5).

Investigating the *impact* of trauma or its *effects* in OHS, however, is not less ambiguous than studying trauma in itself. There is little consensus regarding the meaning of trauma, and there is even more disagreement about its effects. Attempts to define stress disorders clearly, including PTSD, have been met with all kinds of objections for a long time ([Rosen, Spitzer, & McHugh, 2008](#)). These objections have been voiced because “stress” is not a monolithic concept but rather, an “emergent process” that involves interactions between individual and environmental factors, historical and current events, allostatic states, and psychological and physiological reactivity ([Epel et al., 2018](#); [Robinson, 2018](#)). Even if it is possible to show with some certainty that the war left an indelible impact on Holocaust survivor parents, it remains difficult to conceptualize this impact within the lives of the offspring. If, for example, parents feel “accumulated emotional pain from the past” ([Tolle, 1997](#)), or suffer from anxiety symptoms with complicated grief, it is not possible to infer with certainty that these same symptoms, also found in the offspring, is a result of transmission. They were possibly caused by other lifetime adverse events, rather than by parental trauma transmission, or by various visual & verbal representations of the ancestral trauma that were disseminated in social media.

Most theories of traumatization are based on the notion that time has stopped, and that painful memories from the past are coming back, like a broken record that is spinning around and around. The strange thing about trauma transmission, however, is that the most critical event in the lives of OHS happened before they were born. How can a past that is “coming back,” not in the form of one’s own memory, but in the form of the memory of someone else, be conceptualized? Researchers from psychology and psychoanalysis have struggled to answer this question for a long time and used different metaphoric terminologies to describe it. They have for example described the effects of the Holocaust upon offspring as “shadows” (Hass, 1996; Moses, 1993), “echoes” ([Wiseman & Barber, 2008](#)), “psychic holes” (Kogan, 1995; [Kogan, 2013](#)), “ghosts” ([Stein, 2014](#)), “knowing the unknown” ([Laub & Auerhahn, 1993](#)), “the presence of absence” (Berger, 1997), or “radioactive” (Gampel, 2010) and “projective” ([Rowland-Klein & Dunlop, 1998](#)) identifications. From an ethnographic perspective, [Hirsch \(2008\)](#) suggested calling the unconscious manifestations of parental repressed memories “post-memories” since they could be neither understood nor recreated by OHS. In cognitive

neuroscience, such memories are categorized as “non-declarative” or “implicit” because they make people perform thoughtless and automatic tasks, such as the sudden Holocaust association in OHS that may appear when faced with hunger, frost and physical pain.

While some of these concepts are useful in clinical and research settings, they remain fuzzy and imprecise and difficult to operationalize and measure objectively. Psychobiological research, therefore, attempts to conceptualize the manifestations of HTT in more concrete terms, as “scars,” “footprints,” or “psychophenes” (behavior genetics) that can be measured within endophenotypes, neuroendocrinological correlates, or within the neuronal circuits of the brain. These “embodiment” concepts are based on the assumption that thoughts, feelings, and behaviors are grounded in physical interactions with the environment, and that traumatic experiences influence body morphology, sensory systems, and motor systems ([Francis, 2018](#)). While such conceptualizations of trauma allow for a more precise definition and measurement, the drawback of using them is that they are too reductive and simplistic to answer the “hard question” (Chalmers, 1996) on how a mental process can create the experience of HTT. As a result, psychobiological studies may, therefore, study something that becomes irrelevant to traumatization itself. Perhaps this is the reason why it is difficult to translate the various experiences of OHS into tangible biomarkers.

Earlier studies have been unable to agree on a standard demarcation of what was passed down from parent to child. Utilizing the simple phrase “emotional residues” to delineate the x may incorporate much of what is known from earlier traumatology studies. This neutral construct will enable future elaboration of the contents of HTT in both psychological and biological terms as a memory, or a scar that remains after a harmful event that happened to someone else.

2. Parental diversity factors (P)

Holocaust survivor parents come in all shapes, and there is an almost endless number of parental characteristics that can influence OHS. The most crucial parental diversity factors, as reported in studies of Holocaust survivor parents, are gender, age, traumatic experiences, mental state, child-rearing, family functioning, communication patterns, coping styles, and positive parenting qualities.

Gender is perhaps the most crucial parental diversity factor. Even though there is evidence that both maternal and paternal stress can affect the neurodevelopment in offspring ([Chan, Nugent, & Bale, 2018](#)), some studies have identified different underlying mechanisms for transmission depending on parental gender ([Yehuda et al., 2014](#)). During pregnancy and intra-uterine development, the maternal impact will be critical, as well as immediately after giving birth. Later in life, women have a more significant investment in maintaining relationships with their children than do men ([Rossi, 1993](#)). Paternal influence, if not studied in the sperm, can be similarly investigated in paternal child-rearing practices (Lamb, 2002). If both parents were survivors, the impact of HTT was found to be more apparent ([Lurie-Beck, Liossis & Gow, 2008](#); [Scharf, 2007](#); [Van IJzendoorn, Bakermans-Kranenburg, & Sagi-Schwartz, 2003](#); [Weinberg & Cummins, 2013](#)). Worse subjective impressions of emotional and physical health were related to maternal but not paternal exposure to the Holocaust ([Flory, Bierer, & Yehuda, 2011](#)) and lower cortisol level in offspring were associated with maternal, but not paternal, PTSD ([Yehuda et al., 2007](#)). Maternal PTSD significantly enhanced the risk for PTSD in OHS, while paternal PTSD significantly elevated risk for depression ([Yehuda, Bell, Bierer, & Schmeidler, 2008](#)). Both fathers' and mothers' PTSS were positively related to offspring's PTSS in a prospective study of the intergenerational transmission of captivity trauma ([Zerach, Levin, Aloni, & Solomon, 2017](#)).

The *age* of parents during the war is an additional important diversity factor. Older survivor parents usually developed general resilience in most life domains ([Shmotkin & Barilan, 2002](#)), while those who survived the war at an earlier age were more vulnerable ([Danese & McEwen, 2012](#)). For example, OHS with mothers who were younger than 18 during the war and survived alone reported more symptoms of anxiety and depression ([Aviad-Wilchek, Cohenca-Shiby, & Sasson, 2013](#); [Cohenca-Shiby, & Aviad-Wilchek, 2018](#)). Traumatization adversely impacts young children's mental representations of self and others (e.g., [Schechter et al., 2007](#)), and a significant predictor of later psychopathology was the age at which children were separated from their parents during the Holocaust (Keilson, 1992). After liberation, younger parents who were still recuperating from extreme starvation were more likely to influence their offspring than those who had some time to create a new life for themselves ([Yehuda, Flory, Southwick, & Charney, 2006](#)). Any such adverse childhood experiences, early life

stress, childhood trauma ([De Bellis & Zisk, 2014](#)), childhood maltreatment ([Neigh, Gillespie, & Nemeroff, 2009](#)), and damaging attachment-styles have been found to be prevailing risk factors for many mental disorders ([Bosquet Enlow, Egeland, Carlson, Blood, & Wright, 2013](#); [Ehlert, 2013](#); [Hesse & Main, 1999](#); [Provençal & Binder, 2015](#)). Such life-changing defining moments at an early age seem to have made a fundamental difference, not only in how each responded on a long-term basis to the adverse events of the war, but also how they raised their children ([Bakermans-Kranenburg, van IJzendoorn, & Juffer, 2003](#); [Winston & Chicot, 2016](#)). The growing literature on offspring of such early maltreated mothers ([Brand et al., 2010](#); [Juul et al., 2016](#)) suggests that, if they also suffer from PTSD or depression, they are more likely to give birth to more vulnerable offspring.

Any *traumatic pre-war, war and post-war experience* that affected the parent's anxiety levels would also influence their parenting style since offspring would perceive such specific parental traumatic experiences differently. Parents who were hidden as children, would, for example, be distinguished from parents who survived in ghettos or camps, and parents with unresolved grief may have been particularly likely to transmit their trauma. Taking such particular stressful events into account will be central to understanding the trauma transmission process as a whole. Survivors who lost previous children during the war, and who related to their "post-war children" as "replacement children," might have been especially likely to pass on their unresolved grief to their offspring. How parents dealt with their past victimhood and their learned helplessness during post-war readjustment also influenced the kind of atmosphere that each family established on a long-term basis. When parents were unable to "let go" of the past, and mourn their losses, they tended to become more abusive towards OHS ([Starman, 2006](#)).

The *mental state* of the Holocaust survivor parent is an additional diversity characteristic since parents with some stress disorder are more likely to transmit their emotional burden than parents who are more balanced ([Bjorklund & Ellis, 2014](#); [Lambert, Holzer, & Hasbun, 2014](#); [Roberts et al., 2012](#); [Shrira et al., 2017](#); [Turner, Beidel, & Costello, 1987](#); [Yao et al., 2014](#); [Yehuda, Halligan, & Bierer, 2001](#); [Yehuda, Schmeidler, Giller, Siever, & Binder-Brynes, 1998](#)). Regardless of parental traumatization, however, it was found that children of parents with mental illnesses in general experience significantly more secondary trauma than children of non-ill parents

([Lombardo & Motta, 2008](#)). Since parental trauma increased the risk for mental health problems in offspring, the DSM-5 (American Psychiatric Association, 2013) included a stressor criterion related to learning that a close relative or close friend was exposed to trauma. If Holocaust survivor parents suffered from primary PTSD, it was likely that their offspring would have absorbed some secondary traumatic disorder ([Baranowsky, Young, Johnson-Douglas, Williams-Keeler, & McCarrey, 1998](#); [Juni, 2015](#)). Similar to “vicarious traumatization” ([McCann & Pearlman, 1990](#)), OHS would gradually absorb the distressful experiences of their parents during childhood and later. In addition, if the post-traumatic stress symptomatology of the parent increased over the years, it also led to higher rates of symptoms in OHS ([Nicolai, Zerach, & Solomon, 2017](#)), and PTSD was found to be more common among Israeli OHS who had experienced combat fatigue ([Solomon, Kotler & Mikulincer, 1988](#)). When survivor parents displayed more dissociation, cortisol levels in OHS decreased ([Van IJzendoorn, Fridman, Bakermans-Kranenburg, & Sagi-Schwartz, 2011](#)), which is a common find in psychobiological studies in this population. There is also evidence for the impact of parental depressive symptoms on offspring temperament ([Huynh, Finik, Ly, & Nomura, 2014](#)) and of other mental disorders that were transmitted through emotional contagion. However, it is important to remember that some survivors may have suffered from mental disorders before the war and that their children possibly inherited the same disposition regardless of the effects of the war.

Various *child-rearing factors* will have been found to influence HTT ([Maccoby, 2000](#)). Such factors include over-protective parenting, an enmeshed family system with “invisible loyalties” (Boszormenyi-Nagi, & Spark, 1973), parental authority (Baumrind, 1971), and emotional contagion ([Bachem, Levin, Zhou, Zerach, & Solomon, 2018](#); [Berger, 2014](#)). In one comparative study, OHS perceived their parents as more worrying ([Keinan, Mikulincer & Rybnicki, 1988](#)), and in another study OHS felt that they had absorbed the inner pain of their parents even though these parents were not different in affection, punishing and overprotection from other Israeli parents ([Kellermann, 2001a](#)). Walker (1999) found that victimized parents who had difficulties with trust and intimacy impacted their ability to develop a healthy attachment with their children and often led to a cycle of continued inter-generational violence. Probable PTSD, in conjunction with unhealthy behavior, such as smoking, alcohol consumption and lack of physical activity

was also found to be highly correlated in Holocaust survivor parents and OHS ([Shrira, 2018](#)). However, since parenting behaviors in themselves are both intergenerational (Steele & Steele, 1994), and also multi-determined (Belsky, 2005), there will always be a particular “transmission gap” between parental qualities and transmission outcomes, as exemplified in the divergence between maternal sensitivity and transmission effects ([van IJzendoorn & Bakermans-Kranenburg, 2018](#)). Decades of research has suggested that child-rearing is associated with a wide range of other contextual characteristics, such as ethnicity, cultural and religious affiliation, financial stress, social class, education, employment, and physical health ([Klahr & Burt, 2014](#)). Furthermore, various kinds of parenting habits, such as overly permissive parenting, difficulties in providing consistency and warmth and in responding empathically to a child in distress also affected the transmission process.

Deficient *family functioning* and a stressful family atmosphere have often been described in OHS families ([Fossion et al., 2015](#); [Letzter-Pouw, Shrira, Ben-Ezra, & Palgi, 2014](#); [Palgi, Shrira, & Ben-Ezra, 2015](#); [Rose & Garske, 1987](#); [Sigal, & Weinfeld, 1987](#)). OHS who for example grew up with parents who had constant marital conflicts and low positive emotional bonding were at higher risk to develop psychological disorders than those who grew up in harmonious surroundings ([Fonagy, 1999](#)). There were no other socialization experiences in closed family systems of isolated immigrant families. Some of these OHS became “parentified” ([DiCaccavo, 2006](#)) and assumed the adult role of caring for their parents, often acting as intermediaries (and translators) between their parents and the community. Any such PTSD-driven parent-child interactions will aggravate the effects of HTT ([Creech & Misca, 2017](#); [Danieli, Norris, & Engdahl, 2016](#); [Lang & Gartstein, 2018](#)). Some OHS changed their views of their parents after having children of their own and were able to forgive them for some of their earlier behaviors ([Wiseman & Barber, 2008](#); [Wiseman, Metzl, & Barber, 2006](#)).

Different *patterns of communication* in Holocaust survivor families have often been found to be a mediating factor of transmission ([Braga, Mello, & Fiks, 2012](#); [Lichtman, 1984](#); [Wiseman et al., 2002](#); [Wolf, 2019](#)). Some parents tried to find the most age-appropriate and non-frightening ways to talk with the children about their war experiences. Others repeatedly shared gruesome details of their tormenting experiences with their children. A few of them kept silent for their entire lives. Despite these early

findings in studies of communication styles, many Holocaust survivor parents modified their narratives as the Holocaust became more generally talked about in society and as the children grew up.

Different ways of *parental coping* is an additional diversity factor. Parents who were consummated by their loss and remained in a state of overt or covert depression, as well as those who expressed anger and resentment for their entire lives ([Wiseman et al., 2006](#)), were more problematic role models for their children than those parents who were able to function adequately. Highly charged parent-child relations, such as those found in Holocaust survivor parents with wounds that did not heal ([Teicher, 2002](#)), would produce increased anxiety in their children. Parents who continued to blame the Germans, the world, God, or themselves for the agony inflicted upon them, while others took a more conciliatory stance, and started to trust people again.

Positive parenting qualities are rarely mentioned in the study of transmission, but should also be included in the study of parental diversity factors. After all, some studies found that Holocaust survivor parents were fully functioning, without severe mental disorders and not overly occupied with the Holocaust by a group of clinical offspring ([Kellermann, 2008](#)). Such parents seemed to have adapted well to environmental challenges and helped their children to develop functional coping mechanisms in life. They established clear family structures and provided “good enough mothering” (Winnicott, 1973) to their children. These qualities contributed to the fact that most OHS became well-integrated members of their communities with successful professional achievements.

3. Offspring diversity factors (O)

A wide variety of offspring diversity factors have been shown to impact the likelihood of each child to absorb the emotional residues of their parents. Clinical observations suggest that many OHS are “highly sensitive persons” ([Acevedo et al., 2014](#); [Aron, Aron, & Jagiellowicz, 2012](#)). Since this trait is associated with greater responsiveness to the environment, OHS may, therefore, be more attuned to their parent’s emotional pain. As a result, *susceptible* OHS may be more likely to absorb their parents’ HT “radiation,” (Gampel, 2010) than resilient OHS ([Charney, 2004](#); [Yehuda, 1999](#)). A key challenge is to discover the cause of such *differential susceptibility* from

the point of view of genetic makeup, child-caregiver interaction, or temperamental characteristics ([Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011](#); [Russo, Murrough, Han, Charney, & Nestler, 2012](#)). Studies that attempt to explain how offspring respond, for better and for worse ([Belsky, 2016](#)), in various environmental contexts have been, and are still, conducted, but no decisive conclusion have yet been made. It is perhaps the most central question in all research on the origin of stress-related psychopathology in general. According to [Lurie-Beck, Liossis & Gow \(2008\)](#), the differential impact of demographic factors on HTT has been found to explain much of the variation of HTT on OHS. Except for the nature of parents' Holocaust experiences during the war, this includes time and place of birth, birth order, and gender ([Kellermann, 2008](#)), as briefly described below.

Time and place of birth: OHS born immediately after the war seem to have absorbed more parental traumatization than those born later. Being born in a displaced person camp was an additional aggravating factor. The country of origin, as well as being born before or after immigration, also had a substantial effect on HTT. Birth order: Being the first child seems to have resulted in more severe HTT for various reasons. For example, if this child was also a "replacement child" ([Schwab, 2009](#)), and received the same name as a child killed during the war (Wardi, 1992), it often led to a substantial additional burden. Accentuated sibling differentiations were also observed among OHS ([Felsen, 2018](#)). Gender: While studies on gender differences among OHS are scarce, a higher number of female OHS in most HTT studies, and the higher prevalence of PTSD in women ([Olf, 2017](#)), suggest that females are more vulnerable than males.

There are an infinite number of additional individual differences, based on personality, physical abilities, social class, culture, religious affiliation, political beliefs, and immigrant status, that also affect how each offspring dealt with his or her unique manifestations of HTT ([Gottschalk, 2003](#); [Prince, 1985](#)). For example, [Novac & Huber-Schneider \(1998\)](#) reported increased comorbidity in previously healthy OHS who were treated for anxiety and depressive disorders. A more personalized approach ([McEwen & Getz, 2013](#)) in the study of transmission is required to illuminate such differences. As found in psychoanalytic case studies (e.g., Kogan, 1995; Gampel, 2010), such an approach will emphasize within-individual (as opposed to group average) symptom clusters. The individualized approach will also be able to assess and control for possible

non-Holocaust related traumatic life-events, such as the loss of parents, serious accidents, wars or severe personal crises, which were either sufficiently stressful on their own or triggered HTT.

4. Environmental conditions (EC)

Various environmental conditions will also influence the likelihood of OHS to absorb the emotional residues of their parent. The social and cultural context in which OHS grew up, and the support systems present in the community, profoundly changed how much they absorbed the emotional residues of trauma from their parents. Even if OHS grew up with severely traumatized parents and were predisposed to suffer from stress-related problems, they may not have developed any symptomatology due to such protective factors in the environment (Cummings, Davies, & Campbell, 2000). Such gene-environment interactions ($G \times E$) may clarify why environmental risk factors have a significant effect on some persons but not on others, and why relatives who are genetically at risk for a disorder do not all get the disorder ([Vinkhuyzen & Wray, 2014](#)).

The impact of environmental circumstances that reduce distress should not be underestimated when trying to understand the process of transmission. For example, offspring who grew up in an accommodating Jewish community, with many survivors and immigrants, and had a close-knit peer network outside their own families, were more protected than those who grew up in isolation non-Jewish surroundings where they felt different and estranged. The different immigrant statuses in Israel, the US, Australia ([Weinberg & Cummins, 2013](#)), Brazil ([Braga, Mello, & Fiks, 2012](#)), Europe, or the former USSR were very large and had a substantial impact on OHS adjustment ([Weiss, O'Connell, & Siiter, 1986](#)). This was confirmed in the meta-analysis by [Barel, Van IJzendoorn, Sagi-Schwartz, & Bakermans-Kranenburg \(2010\)](#) who found that the effect of the Holocaust was stronger among survivors living abroad vs. those living in Israel, so it is possible that the effects of HTT were also greater abroad.

Various forms of “outside-the-home” socialization frameworks in the peer groups of childhood and adolescence ([Harris, 1995](#)) are especially important in mitigating the transmission effects of Holocaust survivor parents. In young adulthood, some OHS joined commemorative organizations and self-help groups, thereby defining themselves as a group of people with a lot in common ([Solomon, 1998](#)). Such groups have existed all over

the world for more than 50 years ([Fogelman & Savran, 1960](#)) and they have helped to mitigate the deleterious effects of HTT ([Fogelman, 1998](#)). As the level of emotional residues of HTT decreased, the framework of these groups changed to become “channels of memory” and “communal remembrance” within an ethnographic setting ([Kidron, 2009](#)).

5. Time periods (TP)

When are OHS more susceptible to HTT? At which critical periods in child development is the transmission of stress symptoms likely to occur? The “three-hit concept of vulnerability and resilience,” ([Daskalakis, Bagot, Parker, Vinkers, & de Kloet, 2013](#)) is a useful overall framework to answer these questions. This concept is based on the assumption that stress tends to accumulate, one upon the other, to increase the likelihood of absorbing parental traumatization in different ways and during different time periods. Offspring born with a genetic predisposition (first hit), to a mother who endured a difficult pregnancy (second hit), and who also experienced a troubling childhood and adolescence (third hit), will be more vulnerable to stress later in life. If these “hits” were predated by parental traumatization during the Holocaust, the likelihood of OHS to develop stress symptoms would be increased.

Different time perspectives are considered in any such analysis of HTT. From the perspective of biological theories, the transmission begins before birth and possibly even before conception. The delineation of “critical windows” of possible epigenetic alterations is crucial since it determines when offspring are particularly malleable to epigenetic changes due to environmental influences ([Burggren & Mueller, 2015](#)). The following time-periods have been delineated as more or less “critical” ([Bowers & Yehuda, 2016](#)): (1) Hereditary transmission via gametes, (2) Transmission during pregnancy (in utero), and (3) Transmission through early postnatal care. Accordingly, transmission is assumed to have occurred either through the sperm or ova, during pregnancy, or immediately after birth through breastfeeding and caretaking of the newborn child.

Prenatal periods are at particular risk to environmental insults. In pregnant mothers, placental signaling creates changes in inflammatory, nutrient-sensing, and epigenetic pathways ([Lindsay et al., 2019](#)). In fathers, transmission of preconception

paternal stress exposure is associated with changes in epigenetic marks in sperm, with a focus on the reprogramming of methylation, histone modifications, and small non-coding RNAs ([Bohacek & Mansuy, 2017](#)). Even though there is little data available on such effects in OHS, we may assume that such effects are substantial. Eitinger (1993) reported on a study from 1948 of newborn children of camp survivors with a higher percentage of congenital malformations. The challenging physical conditions which mothers endured during pregnancy, besides early childhood experiences, severely affected their offspring ([Keinan-Boker, 2014](#)). [Hazani & Shasha \(2008\)](#) postulated that maternal hunger and stress-induced high levels of maternal steroids during crucial stages of fetal development possibly exposed the unborn child to risks of increased cardiovascular morbidity and mortality in adult life. Future longitudinal, epidemiological, prospective genome- and epigenome-wide multi-generational studies ([Keinan-Boker, 2014](#)) may detect the endophenotypes (heritable phenotypes associated with illness risk) that are measurable independent of illness state ([Gottesman & Gould, 2003](#)). Such endophenotypes emphasize the genetic, environmental and individual preconditions for secondary traumatization in OHS, and the “sufficient cause” (Rothman & Greenland, 1998) for HTT to occur.

From the perspective of psychosocial learning theories, HTT starts from birth. Time factors have been traditionally emphasized in conjunction with the delineation of specific stages of child development. Such stages will be critical when interpreting earlier findings from OHS cohort studies since the developmental timing of trauma exposure will influence emotional dysregulation also in adulthood ([Dunn et al., 2018](#)). Observational and empirical studies on OHS found developmental arrests during various phases of child development. A brief summary of such milestones, during which trauma transmission was found to have occurred, includes the first toddler years of attachment and bonding ([Bar-On et al., 1998](#); [Brothers, 2014](#); [Fonagy, 1999](#); [Letzter-Pouw & Werner, 2012](#); [Sagi-Schwartz et al., 2003](#)), the early childhood and adolescent years of identity formation and separation-individuation ([Barocas & Barocas, 1980](#); [Brom, Kfir, & Dasberg, 2001](#); [Freyberg, 1980](#); [Quadrio, 2016](#); [Zilberfein, 1996](#)), and the young adult years when OHS had obtained a more fundamental understanding of the impact of their parents’ Holocaust past on their own lives ([Kellermann, 2001b](#)). Early life exposure to maternal distress was associated with poor psychological health in children ([Bale, 2014](#)). The

stages of puberty and adolescence ([Thapar, Collishaw, Pine, & Thapar, 2012](#)) were also considered to be particularly critical to the impact of HTT because, during this time, the child's emotional, social, and cognitive skills grew the most and became increasingly complex. In young adulthood, OHS continued to consolidate their understanding of their parents' war-time past and started to integrate it within their sense of self. When becoming parents themselves, this entire process was combined with a deeper understanding of parental roles ([Letzter-Pouw et al., 2014](#)). Now, the aging process among OHS has become the focus of some studies since it is again reshaping the intergenerational relations in Holocaust survivor families ([Shmotkin, Shrira, & Palgi, 2011](#); [Shrira et al., 2017](#); [Shrira, Menashe, & Bensimon, 2018](#)). The main conclusion from this brief overview of different time periods is that HTT gets a different meaning, and a disparate impact, on OHS depending on their age and life-cycle stage when they were studied. Even though the influence of parental HT was substantial during childhood, this influence appears to decrease (or change) in adulthood, similar to other kinds of heritability ([Bouchard & McGue, 2003](#)).

Defining a population at risk within a specific time perspective, however, is a continuing challenge because both biological and psychosocial risk factors contribute to our understanding of the transmission process. The first will be based more on heredity, while the second on psychology and socialization, reflecting the nature or nurture focus of the study design. While it is essential to include the earliest stages of life, beginning well before birth and immediately following to determine possible hereditary disposition and epigenetic pathways, the stages of puberty and adolescence cannot be overlooked from a psychosocial developmental perspective. During this latter period, a second separation-individuation phase ([Blos, 1967](#)) has been identified that determines if the possible predispositions of OHS will result in a curse or a legacy ([Kellermann, 2008](#)). Because even though epigenetic alterations may occur throughout life, these physiological and cellular processes will interact with additional cumulative experiences to either minimize or maximize the harmful effects of trauma transmission. From this point of view, transgenerational cycles are mediated by epigenetic mechanisms through environmentally driven neuronal and behavioral adaptations ([Konrad, Herpertz, & Herpertz-Dahlmann, 2016](#)). Such long-term effects of HTT have been shown to interact with family constellations and aging processes in general ([Shmotkin & Barilan, 2002](#);

[Shmotkin, Shrira, & Palgi, 2011](#)) and interdisciplinary and cross-cultural study designs are therefore required.

A suitable model to study HTT from a time perspective is the “life-course developmental theory” ([Adonis, 2016](#); Bengtson, Elder, & Putney, 2005; [Hareven, 2000](#)). With an emphasis on time and age, it incorporates the biological foundation and possible epigenetic alterations of early life experiences with later social patterns and environmental demands. All of these processes together help to understand how OHS think, feel and behave as they age over time. A life course perspective distinguishes significant life events over the lifespan and attempts to discover how social processes influenced the developmental paths of each person ([Kirmayer, Gone, & Moses, 2014](#)). Studying OHS from such a life-course perspective holds considerable promise because it allows a longitudinal perspective of individuals based on their patterns of stress adaptation. It covers a large variety of responses, such as chronicity, recovery, delayed onset, and resilience ([Galatzer-Levy & Bryant, 2013](#)). The lives of OHS can thus be described as a story of generational change (Berger & Berger, 2001; Sicher, 1998: [Stein, 2014](#)), with a beginning, a middle and an end; a universal story of endurance, adaptation, evolution, and transformation.

6. The process of trauma transmission (→)

How can traumatization be transmitted from parent to offspring? What does the term “transmission” actually mean? Is it similar to an infectious disease being carried over from one person to another, or to a hereditary trait being passed down from parents to their offspring? Which mechanisms are involved in the process of HTT?

Many theories have been suggested to answer these questions, either from a psychosocial or from a biological perspective. The psychosocial perspective includes psychoanalytic notions, behavioral conditioning models, principles of cognitive appraisal, attention bias/information processing, interpersonal coping styles, parent-child interaction schemes (e.g., contagion, modeling), socialization models and family systems perspectives ([Kellermann, 2009](#)). The biological perspective suggests a hereditary transmission of neuroendocrine, neuroanatomic, and epigenetic systems. All of these theories tacitly assume that there is a “mediating agent” between the transmitting parent and the absorbing child, similar to the one observed in the

transmission of a virus, in which a mosquito carries the virus from one person to another.

The psychoanalytic theory assumes that the unconscious may in itself be “infectious,” especially if it is disavowed. If the Holocaust survivor parents become aware of their loss and work through their repressed emotions, they will be less likely to pass them on to their children. Family system theory assumes that unhealthy communication is the main mediating agent. If the Holocaust trauma is talked about in a balanced manner, it is easier for the child to digest it. However, if it is talked about too much or too little, it will become malignant. Socialization theory assumes that parenting style is the primary mediating agent of trauma transmission. Inadequate parenting that leads to enmeshment will affect the general family atmosphere and have a detrimental effect on the child's behavior. Biological theories are based on the assumption that there is a genetic predisposition to a person's illness and that parents pass on “acquired traits” to their offspring through physiological pathways. Manifestations of HTT can thus be understood, first as displaced unconscious fears in parents. The children internalize what the parents themselves cannot handle. Second, it can be explained as the result of social learning and parenting. The child responds to the anxieties indirectly expressed in unhealthy child rearing behavior. Third, it can be the result of family enmeshment and tacit communication. The child is trapped in a closed setting where it adopts a threatening world view that regulates thoughts, feelings, and behavior. Finally, it can be the result of hereditary transmission of the parents' neural fear network, allostatic physiological stress-response system, and HPA-axis regulating system. As a result, OHS will become physiologically predisposed to vulnerability, even though they do not necessarily manifest psychopathology.

At first glance, these theories of the process of trauma transmission seem to make perfect sense. Upon further examination, however, they are too general to explain the specific process of how the impact of trauma can cross generations. First, psychoanalytic theories cannot fully explain how repressed traumatic experiences in parents can enter into the minds of offspring, even with concepts such as “projective identification” “transposition,” “internalization” and “role induction” ([Kahn, 2006](#)). Neither is the theory of a disruption of the dialogic self ([Bradfield, 2013](#)), sufficient to explain the process of HTT. Second, while deficient parenting and flawed socialization

had important effects on OHS, these factors cannot fully explain the process of HTT, since “good-enough” parenting was also found to transmit the emotional residues of the Holocaust to OHS. Third, family systems and communication models have produced a similar indefinite explanation of the transmission process. Emotional ties in Holocaust survivor families have consistently been reported as being strong, but there is also much ambivalence manifested in such families, and it is not clear how changes in family structures during their lives affected the impact of HTT, as both the parents and their offspring grew older ([Shmotkin, Shrira, & Palgi, 2011](#)). Also, while too much talk about the Holocaust would lead to a burden being passed upon the children, many parents did not share their traumatic experiences, but the children still absorbed much of their past trauma. Finally, HTT has also been reported in harmonious families with plenty of opportunities for separation-individuation. Fourth, while findings from animal studies have indicated that acquired traits from the past can be transmitted to offspring, there is insufficient evidence on the epigenetic transmission of such characteristics in humans ([Daxinger, & Whitelaw, 2010](#)), and fundamental questions remain regarding the transgenerational transmission of epigenetic alterations to future generations ([Grossniklaus et al., 2013](#)). Holocaust survivor parents suffered from malnutrition during the war, and also experienced extreme death anxiety that may have caused their cortisol to be elevated for long periods. However, there is still insufficient evidence of the assumption that the stress responses of Holocaust survivor parents became permanently dysregulated, and that OHS inherited stable constitutional tendencies or robust epigenetic alterations. It is, therefore, currently impossible to attribute transmission effects in humans to a single set of biological or other determinants ([Yehuda, Lehrner, & Bierer, 2018](#)).

Future perspectives

Where do we continue from here? Which new perspective of HTT is best suited to answer the question of whether parental traumatization can be transmitted to their offspring? Assuming that trauma can indeed be transmitted; how can the aggravating and mitigating factors that increase or decrease the likelihood of offspring psychopathology be further investigated?

It will be the task of future studies to specify the diverse parental, offspring, environmental, and time factors which increase or decrease the transmission of trauma from parents to their offspring. These variables together will explain more precisely how (→) various kinds of transmission occur from both a psychosocial and biological point of view. Since transmission tends to be highly volatile, it will also have to take the adaptability of parents and children into account, as well as the shifting environments and critical periods when the transmission is more likely to occur. A systems biology perspective may be needed to measure the quantity and connectivity of all these variables ([Ma'ayan, 2017](#)).

Future transmission studies will benefit from both neurobiological and psychosocial measures in conjunction with one another. Until recently, transmission was studied from either one of them; each with its own conceptualizations, methodology and scientific frame of reference. One would investigate the influence of various parent-child interactions, while the other would search for biomarkers within biological psychiatry. These two assumed pathways of HTT should no longer be regarded as mutually exclusive since the pathways of HTT can occur at multiple levels, including epigenetic alterations of stress responses, changes in individuals' psychological well-being, family functioning; community integrity and cultural identity ([Sotero, 2006](#)).

Various manifestations of trauma transmission can thus be explained as being determined by any or all of these factors or by an ecological combination of them ([Kellermann, 2001c](#)). Such a combination can be exemplified, in the amalgamation of neurobiology and psychoanalysis within neuro-psychoanalysis. It also appears in studies on the biosocial context of parenting ([Feldman, 2016](#)), on the telomere length in PoW ([Solomon et al., 2017](#)), and on the limbic-hypothalamic-pituitary-adrenal (LHPA) axis reactivity on separation-individuation in subjects three generations removed from the Holocaust ([Ullmann et al., 2018](#)). Both approaches were emphasized when explaining the possible parental transmission of a variety of disorders, including chronic pain ([Stone & Wilson, 2016](#)), and immune deficiencies ([Jonker, Rosmalen, & Schoevers, 2017](#); [Olf & van Zuiden, 2017](#); [Segerstrom & Miller, 2004](#)). The usefulness of such a broad-based approach was evident in a review of over a hundred studies on offspring with parents who suffered from PTSD ([Leen-Feldner et al., 2013](#)). The multitude of biological and environmental factors that influence individual development can thus be considered,

especially those factors that can be reliably transmitted ([Blumberg, 2017](#)). The challenge will be to integrate findings from both fields of research and include all the systemic and central biological and psychosocial mechanisms underlying stress-related disorders ([Nasca, Rasgon & McEwen, 2018](#)). This approach can pave the way for multi-disciplinary intervention strategies for offspring at-risk.

Genetic disposition and socialization factors brought together within a single framework contradict the nature-nurture dichotomy ([Cavalli-Sforza & Feldman, 1973](#); [Lewkowicz, 2011](#)). It shows how our understanding of transmission and heredity has changed through the development of new concepts within Mendelian genetics and neo-Lamarckism. It also corresponds to the appreciation of transmission in evolutionary biology, as described by Jablonka & Lamb (2005) in their book [Evolution in Four Dimensions](#). From this theoretical background, transmission occurs not just through genes per se, but through the heritable variations transmitted from generation to generation by whatever means. Such variations can occur (1) at the physical level of genetics, (2) at the epigenetic level, (3) at the level of social learning, and (4) at the level of culture. This complex environment-biology interface has shown promise as a possible route for the intergenerational transmission of the effects of trauma ([Lehrner & Yehuda, 2018b](#)). Within such an integrative bio-psycho-social explanatory model of traumatization, all the essential hereditary variations must be taken into account, rather than only a purely biological reductionist one ([Kendler, 2005](#); [Omid, 2013](#)). When trying to understand the causal pathways through which individuals show resilience or vulnerability in the face of adversity ([Bowes & Jaffee, 2013](#)), this explanatory model becomes particularly relevant.

Conclusion

Can parental traumatization be transmitted to offspring? The present overview of the literature suggests that traumatization in the first generation can indeed be a risk factor for adverse long-term outcomes in the second. Risk factors, however, are multi-determined and to specify and disentangle all the origins of transmission is a daunting task. A range of vulnerability and resilience factors, environmental pressures (and provisions), psychological outfit and subjective appraisal of the traumatic event will all influence the process of trauma transmission ([Qi, Gevonden, & Shalev, 2016](#)).

In the past, research of HTT sought to discover the general characteristics of OHS behaviors and suggested formulations of resilience in conjunction with specific vulnerabilities. A developmental psychopathology model is here suggested as an alternative. This model illuminates the numerous factors that yield different effects along various stages of transmission. It recognizes a “matrix of child-intrinsic factors, developmental maturation and experience, life events, and evolving family and social ecologies” ([Pynoos, Steinberg, & Piacenini, 2000](#), p. 1542). From this life-course developmental point of view, particular parents passed on various influences to individual children under specific circumstances and at different critical periods through a combination of biological and psychosocial mechanisms. A highly condensed summary of findings from earlier studies on HTT would thus imply that *Holocaust survivor parents who experienced adverse experiences in childhood were more likely to transmit their accumulated emotional residues to highly sensitive offspring during adolescence if a supportive social network was absent.*

This differential appraisal should also recognize that *any* child will somehow be affected by their parents’ war experiences. Rather than focusing on one or the other outcome of HT, it seems more relevant at this point to reflect on the journey of post-war adjustment as a whole. Even simple organisms learn fundamental survival skills and pass these on to their offspring, and it is not surprising that life-changing experiences in Holocaust survivor parents, which result in knowledge useful for survival, would be passed on to OHS. If survival is the name of the game of evolution, the post-war generation of Holocaust survivors and their offspring became masters in it. It has become part of their very nature and, as such, it will probably be passed on also to future generations.

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