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RESEARCH ARTICLE

Time, trauma, and the brain: How suicide came to have no significant precipitating event

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Argument
In this article, we trace shifting narratives of trauma within psychiatric, neuroscience, and environmental epigenetics research. We argue that two contemporary narratives of trauma – each of which concerns questions of time and psychopathology, of the past invading the present – had to be stabilized in order for environmental epigenetics models of suicide risk to be posited. Through an examination of these narratives, we consider how early trauma came to be understood as playing an etiologically significant role in the development of suicide risk. Suicide, in these models, has come to be seen as a behavior that has no significant precipitating event, but rather an exceptional precipitating neurochemical state, whose origins are identified in experiences of early traumatic events. We suggest that this is a part of a broader move within contemporary neurosciences and biopsychiatry to see *life as post*: seeing life as specific form of post-traumatic subjectivity.

Keywords: Suicide; environmental epigenetics; neurobiological risk; trauma; PTSD; anthropology

> Approximately 90 percent of individuals who die by suicide meet the diagnostic criteria for a psychiatric disorder in the last six months of life. . . . Cross-nosological depressive symptoms drastically alter the individual's problem-solving capacity and judgment [and] they may be interpreted as precipitants of suicidal crises. These depressive states associate with molecular changes that are likely to underlie proximal risk factors for suicide. (Turecki 2014a, 11-12)

From antiquity to the present, trauma has been a keyword used by clinicians and scholars to describe certain negative experiences and their aftermath (Ben-Ezra 2011; Berrios and Porter 1999; Birmes et al. 2003). Examined as a distinct object across a wide range of disciplines (Caruth 1996; LaCapra 2001; Leys 2000; Luckhurst 2008), trauma has been discussed and debated intensively. In recent years, humanities scholars and social scientists have explored trauma through a diverse set of situations of extremity and associated individual and collective experiences and responses (Bracken 2003; Das 2003; Fassin and Rechtman 2009; Mbembe 2006; Young 2007). Trauma, in these studies, is considered a multifaceted experience variously associated with illness and suffering, bodily experiences, and the moral politics of victimhood, blame, and accountability (Kirmayer, Lemelson, and Barad 2007, 4; Hinton and Kirmayer 2013; Kitanaka 2016; Fassin 2014; Rechtman 2000).

Contemporary psychiatric research on trauma has a distinctive history in which the concept has been serially reinterpreted as researchers attempt to characterize the specificity the origins of trauma and its consequences. The multiple historical transformations of the concept are inextricably connected yet equally characterized by intellectual disjunctions. The salient examples and prototypes of trauma within psychiatry have changed over time, as trauma theory has moved from surgical metaphors of injuries and healing in the late 1800s to domain-specific models based on

psychological and physiological processes in the 1990s and beyond (Leys 2000; Micale and Lerner 2001; Young 1995). This history is far from linear. As Judith Herman, a psychiatrist and trauma specialist, acknowledges:

> The study of psychological trauma has a curious history – one of episodic amnesia. Periods of active investigation have alternated with periods of oblivion. Repeatedly in the past century, similar lines of inquiry have been taken up and abruptly abandoned, only to be rediscovered much later. (Herman [1992] 2015, 7)

Throughout this history, the psychiatric profession has periodically been fascinated by the pathogenic nature of traumatic events, while at other times these professionals have called into question the relevance of personal accounts of trauma in the etiology of health and mental health problems (Van der Kolk 2007, 19). This article focuses on these shifting narratives of trauma, its effects, and the relationship of both to the development of psychopathology. We examine etiological narratives of trauma and most particularly shifts in the importance accorded to traumatic events at different points of a person's life and increasing interest in childhood trauma. If each narrative affects the significance accorded to personal accounts of trauma, we suggest that contemporary neuroscientific models of the embodied damage and neurobiological risk thought to follow early trauma consign later life experiences to etiological obscurity. Through attention to narratives of neurobiological risk emerging from psychiatric, neuroscience, and environmental epigenetics research, we explore how suicide has come to be seen as a behavior that has no significant precipitating event, but rather an exceptional precipitating neurochemical state whose origins are identified in distant experiences of early traumatic events.

While our research focuses on environmental epigenetics models of suicide risk emerging from the McGill Group for Suicide Studies (MGSS), these models are consistent with increasingly dominant etiological narratives in contemporary biopsychiatry more generally. These narratives are often seen as specific to the postgenomic era in which life is increasingly seen as characterized by different moments of instability and stability rather than being anchored by the "book of life" (i.e., a presumably stable genome) (Lappé and Landecker 2015). While the narratives we trace are closely linked to reasoning in the postgenomic era (Pentecost and Meloni 2020), we examine earlier narratives that contributed to contemporary etiological accounts of trauma and its effects. In particular, we draw on two narratives of trauma, each of which concerns questions of time and psychopathology, as narratives that had to be stabilized in order for MGSS models of suicide risk to be posited. We suggest that environmental epigenetics has provided a language and mode of reasoning to bring these two narratives together, drawing questions of time, trauma, and psychopathology into the heart of neuroscientific reasoning. In closing, we orient our attention to the question of suicide to consider how environmental epigenetics explanations of suicidal behavior reframe suicidal acts as "post": a post-traumatic subjectivity characteristic to contemporary neuroscience and biopsychiatry research.

Narratives of post-traumatic subjectivity

The introductory citation is drawn from an article "The Molecular Bases of the Suicidal Brain" by Gustavo Turecki, a psychiatrist and molecular biologist in Montreal (Turecki 2014a). Turecki is the director of the MGSS, where we have carried out ethnographic research since 2013.[1] The quotation reflects the MGSS's model of suicide risk, in which suicide is associated with psychopathology, which is in turn associated with specific, underlying neurobiological profiles. It is these

[1]This article focuses on the literature produced by the MGSS; however, it is informed by ongoing ethnography at the MGSS. Lloyd, who is also a principal investigator in the group, has carried out ethnographic research at the MGSS since 2013 and Larivée since 2017.

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brain-based molecular profiles that are thought to constitute suicide risk and the precipitating factors for suicide. The origins of these at risk molecular profiles are believed to be found in the experience of early childhood adversity (ECA). The lives of "individuals who died by suicide" (Chen et al. 2018), from this perspective, are seen as pathological trajectories set in place in the early years through experiences of adversity that enact durable epigenetic changes in the brains of youth. These people are considered susceptible to mental illness, poor decision-making, and a variety of "unhealthy" behaviors (Roth et al. 2009; Labonté et al. 2012; Jollant et al. 2010). Suicide is seen as the last, and most extreme, step in this trajectory (Turecki et al. 2012; Turecki 2014b).

Turecki's words are drawn from an article section entitled "Proximal or precipitating factors" of suicide (Turecki 2014a, 11). Subsections address a variety of molecular systems, from serotonergic alterations to inflammatory factors, considered to characterize the proximal or precipitating factors related to suicide. While the role of life events in triggering suicidal crises is acknowledged in this article and in MGSS research more generally, these events are typically described as quite common negative experiences, such as a rupture in a relationship or the loss of a job or status (Fiori and Turecki 2016; Turecki 2014b; Turecki et al. 2012). Given the relatively ordinary status of these events, it is neurobiological proximal states that are considered the significant precipitating factors in this model of suicide (Turecki 2014a, 33). This research seeks new ways of characterizing suicide risk, recognizing that decades of intensive efforts in fundamental and clinical research to reduce suicide rates have met with limited success (Turecki and Brent 2016). While the experience of certain forms of mental illness, such as depression, remain risk factors for suicide, research has equally made it clear that most people who experience depression will never commit suicide (Hawton and van Heeringen 2009; McGirr et al. 2008a). Research agendas like those at the MGSS attempt to identify who, among those people who share risk factors later in life (e.g., depression), are the ones most likely to develop suicidal ideation and behavior (McGirr and Turecki 2007).

What is striking about Turecki's argument is not its exceptionality among models of neurobiological risk, but its precise fit with contemporary neuroscience narratives. The MGSS models are consistent with a burgeoning set of research programs in neuroscience and epigenetics that aim to identify how specific life "exposures" (both material and social) lead to a range of mental health outcomes as well as a particular set of subjective experiences, behaviors, and risks. What these narratives share is attention to the ways in which the past is thought to invade the present. We will examine two of these. The first narrative concerns studies of ECA. Beginning with growing interest in the experience of ECA in the 1960s, we examine research on abuse in the last twenty years and the production of specific measures to quantify the experience and study its effects. We focus specifically on the measures of ECA used at the MGSS. Contemporary research on child abuse has demonstrated associations between early traumatic experiences and a wide range of mental health problems later in life (Briere and Elliott 2003; Heim et al. 2010; Scher et al. 2004). Attention within this research has been oriented to classifying the original experience and identifying its heterogeneous effects rather than on classifying a specific resultant psychiatric diagnosis.[2] As a consequence, research agendas focusing on ECA ran largely in parallel to the disorder-specific studies of post-traumatic subjectivity that have dominated psychiatric research on trauma since the publication of DSM-III (third edition of the Diagnostic and Statistical Manual of the American Psychiatric Association) in 1980.

The second narrative relates to the development and expansion of the diagnostic category post-traumatic stress disorder (PTSD). Post-traumatic stress disorder, while included in the DSM since 1980, was an exceptional diagnosis in the manual's symptom-based approach to mental disorders due to its presumed origins in the memory of a traumatic event (Young 1995). In comparison with

[2]Moreover, recognition of the co-occurrence of different forms of ECA has resulted in a shift from focusing on single type adversity to examining the associations between a number of adverse childhood experiences and psychopathology (McLaughlin, Sheridan, and Lambert 2014, 578).

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research on ECA, the DSM-III category PTSD situated traumatic events experienced in adulthood – and memories of these events – as playing an etiologically-specific role in the disorder. Over time, however, the etiological significance of traumatic events and their presumed effects shifted in PTSD research. Research on the prevalence, course, and comorbidity of PTSD turned from the exceptional character of traumatic events more proximate to the development of the disorder to predispositions to react pathologically to negative or trauma-level events later in life. The result is an understanding of PTSD in which the person's response to a stressor is determined not by the stressor itself but by the interaction between the stressor and the person's premorbid vulnerability (Shephard 2004, 54). Early life adversity is among the experiences considered capable of engendering this predisposition (Yehuda, Halligan, and Grossman 2001).

If historically these two areas of research were carried out with little overlap, with notable exceptions, over time the narratives within PTSD and ECA research have become more consistent with one another most specifically as they concern the neurobiological vulnerability thought to result from negative early experiences. As a case in point, many trauma specialists came to agree that the diagnostic criteria for PTSD, understood as a category tightly associated with the experience of early adversity, captured only a fraction of trauma-related psychopathology in children, invoking the need for an expanded PTSD diagnostic category (van der Kolk et al. 2005, 390). These "plastic" understandings (Meloni 2019) of the relationships between time and (psycho) pathology have become anchored in a wide range of influential research agendas ranging from studies of the developmental origins of health and disease (DOHaD) to "trauma-informed care."

Early childhood abuse: An etiological significant event without a diagnostic category

Studies of adverse childhood experiences or ACEs are a part of an increasingly substantial and influential body of research that has emerged in recent decades, alongside growing interest in developmental perspectives of risk. Emblematic of this fast-moving field of knowledge is attention to the putative role of early adversity on health and behavior, as demonstrated by foundational ACEs studies (e.g., Felitti et al. 1998) and the vast body of literature that followed (Anda et al. 2006; Briere and Elliott 2003; Dong et al. 2004; Dube et al. 2001; Edwards et al. 2003). While the history of psychiatry is punctuated by episodic attention to the effects of early adversity (Duché 1990; Dorahy, van der Hart and Middleton 2010; Bensel, Rheinberger, and Radbill 1997), it is only since the 1960s that early adversity gained a stronger foothold in psychiatric research, turning attention toward chronic social problems such as childhood abuse and maltreatment (Dorahy, van der Hart and Middleton 2010, 7-8; Hacking 1995, 58; Hacking 1991; DeMause [1974] 1995; Hendrick 1997; Myers 2008).

From this point forward, it was generally accepted that a history of severe or prolonged abuse, particularly in childhood, "fosters the development of a prodigious array of psychiatric symptoms," and appears "to be one of the major factors predisposing a person to become a psychiatric patient" (Herman 1992, 379). By the 1990s, there were so many possible effects and clinical manifestations attributed to childhood psychic trauma that psychiatrist Leonor Terr argued, "we must organize our thinking about childhood trauma, however, or we run the risk of never seeing the condition at all" (Terr 1991, 10).

In the 2000s, a working committee known as the Complex Trauma Task Force (CTTF) of the National Child Traumatic Stress Network (Cloitre et al. 2012) was brought together with the objective of consolidating research and clinical understandings of and responses to child abuse (Cook et al. 2003; Ford and Cloitre 2009; van der Kolk 2005). They proposed a new diagnosis provisionally called developmental trauma disorder (DTD) (Pynoos et al. 2009; van der Kolk et al. 2009; van der Kolk 2005) that aimed to bring attention to the ways in which "multiple exposures to interpersonal trauma, such as abandonment, betrayal, physical or sexual assaults, or witnessing domestic violence, have consistent and predictable consequences that affect many areas of functioning" (van der Kolk 2005, 406). Though proponents of the diagnosis argued for its addition

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to DSM-5 in 2013 (see Schmid, Petermann, and Fergert 2013; Sar 2011), the diagnosis has yet to be included in the diagnostic manual. It has nonetheless drawn increasing attention to ECA/ELA (early life abuse) and to the effects of multiple, cumulative, and/or chronic and prolonged childhood adversities from the early 2000s onward. According to the proponents of DTD, forms of ECA were believed to manifest themselves not only in post-traumatic stress psychological symptomatology, but also in the form of various medical illnesses and conditions which tended to be pervasive and multifaceted (Ackerman et al. 1998; Putnam 2003; van der Kolk 2000). These symptoms, and the biological reactions or responses to stress thought to underlie them, increasingly came to be understood through the lens of emerging neurobiological theories of trauma, a point that we will return to later in the paper (Cook et al. 2003; Perry et al. 1995; van der Kolk et al. 2009; Yehuda 2001; 2000).

Defining and measuring childhood adversity

In the present day, the association between ECA/ELA and mental and physical ill health is widely accepted in fundamental and clinical research (Anda et al. 2006; Dube et al. 2001; Dube et al. 2005; Edwards et al. 2003; Heim and Binder 2012). Among childhood adversities,[3] child abuse and maltreatment have been strongly correlated with adult psychopathology, psychiatric morbidity, and suicidality (Afifi et al. 2009; Brezo et al. 2008; Briere and Elliott 2003; Evans et al. 2005; Gilbert et al. 2009; Heim and Nemeroff 2001). These trajectories have been documented in clinical and developmental psychiatry using a range of screening tools and standardized instruments to retrospectively assess childhood adversities among both clinical and nonclinical populations (see Bailhache et al. 2013; Bethell et al. 2017; Milne and Collin-Vézina 2015; Roy and Perry 2004; Strand, Sarmiento, and Pasquale 2005; Thabrew, de Sylva, and Romans 2012; Tonmyr et al. 2011). Researchers have favored descriptive instruments to gather data on childhood adversities (Spinhoven et al. 2014, 718), which are then aggregated by, for instance, summing the number of traumatic experiences (ibid.) as in the case of the Adverse Childhood Experience Study (ACEs) (Chapman et al. 2004; Dube et al. 2001; Felitti et al. 1998) or classifying specific types of adverse experiences. In the latter case, researchers have sought to characterize types of trauma in terms of levels of psychological distress following a stressful life event (e.g., the Impact of Event Scale – revised version [IES-R] [Weiss et al. 1997]) or the specificity of the traumatic event such as exposure to crime, natural disasters, physical or sexual assault (e.g., the Trauma History Questionnaire [Hooper et al. et al. 2011] and the Life Incidence of Traumatic Events [LITE] [Greenwald and Rubins 1999]).

Drawing on the wide ranging accumulated evidence of the effects of childhood trauma on psychopathology, epigenetics, neuroscience, and developmental researchers at the MGSS have sought to identify whether an association can be identified between early abuse and specific profiles leading to risk of suicidal behaviors. In doing so, they identify specific groups and subgroups, attempting to delineate the particular types of suicide risk profiles associated with each of them. McGill Group for Suicide Studies environmental epigenetics researchers, for instance, have been particularly interested in what they refer to as impulsive or aggressive suicide risk, while clinical and developmental researchers at the MGSS have studied how irritability might play a role in this trajectory of neurobiological risk[4] (McGirr et al. 2008b; Orri et al. 2018; Zouk et al. 2007). In these

[3]Research on childhood adversity also includes neurobiological and epidemiological studies that assess cumulative, cascading, and multidimensional effects of chronic stress in childhood such as severe abuse (Bethell et al. 2017), maltreatment, and/or neglect (Cowell et al. 2015; Gilbert et al. 2009; Teicher, Anderson, and Polcari 2012), exposure to household and/or family violence, conflict and turmoil (Dube et al. 2001; Repetti et al. 2002; Lu et al. 2008), and childhood poverty (Conger and Donnellan 2007; Evans and Cassells 2014; Grant et al. 2003).

[4]McGill Group for Suicide Studies researchers have suggested that certain types of suicide risk may be more present among men than women. For instance, higher levels of impulsive and aggressive behaviors in men are associated with higher risk of suicide among those also affected by major depressive disorder. They add that this particular conjunction of traits appears to play a more significant role in younger cases of suicide (e.g., Dumais et 2005, 2121).

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models, the category of ECA is considered to have similar effects on a person's neurodevelopmental profile as trauma or stress, particularly chronic stress (Isingrini et al. 2016; Mahar et al. 2014; Mahar et al. 2017; Turecki and Meaney 2016). It takes the foreground as the earliest point in a trajectory whose extreme end point is thought to be suicide. Models of early adversity and its hypothesized biological consequences in the form of epigenetic traits imprinted on children's brains, enable MGSS researchers to envision a critical link between early-life adversity and development of negative mental health outcomes later in life (e.g., depression, personality disorders, suicide) (Turecki and Brent 2016).

To establish such correlations, researchers at the MGSS conduct clinically-informed interviews to collect detailed information about the lives of people who are referred to as "individuals who died by suicide" in MGSS and other suicide research (Ahmedani et al. 2017; Chen et al. 2018; Zhurov et al. 2012).[5] These post-mortem, biographical surveys, also called "psychological autopsies," are conducted with the deceased's relatives and loved ones, and collect specific information on traumatic events and episodes, personality traits, history of mental disorders, and a variety of other factors about the lives of people who die by suicide (Brent 1989; Cavanagh et al. 2003). These broader retrospective early-life history interviews are complemented by the use of the *Childhood Experience of Care and Abuse* (CECA), a questionnaire used throughout Canada, the United States, and internationally (e.g., Busso et al. 2017; Falgares et al. 2018; Fisher et al. 2011; Hart et al. 2018), that quantifies adversity based on reported experiences of care and abuse (Bifulco et al. 1994). The MGSS uses an adapted, proxy-based version of the CECA, in which people close to the deceased complete the questionnaire.

The CECA is based on categorical questions and a scale of four principal measures (including indifference/neglect, family tension and conflicts, physical abuse, and sexual abuse) covering the person's childhood experiences[6] (Bifulco et al. 1994, 1422). Compared to self-reported measures for assessing childhood trauma (e.g., Bernstein and Fink 1998; 2003; Greenwald and Rubin 1999; Hooper et al. 2011), retrospective interview methods for appraising adverse childhood experiences such as the CECA place particular weight "on clarifying the time order of critical experiences" to encompass the breadth and timing of experiences (Bifulco et al. 1994, 1422). The answers are scored and ranked according to intensity and/or severity providing what is described as "factual" measures[7] of abuse and neglect to determine where the participant profile fits in the group's typology of "suicide with or without abuse" (Lloyd, Filipe, and Larivée 2018). Intensity and/or severity is rated on a four-point severity scale ("1-marked", "2-moderate", "3-some/mild", "4-little/none") with a global severity rating (Bifulco et al. 2002; Brown et al. 2007; Bifulco and Moran 1998; Moran et al. 2002). Severity levels are determined by frequency, pervasiveness and/or intensity of the experience – for sexual abuse, relationship to the perpetrator and coercion/secrecy are equally important (Smith et al. 2002, 573). At the MGSS, researchers consider adversity as severe when reports of major physical and/or sexual abuse up to age fifteen include cases with the

[5]Until recently, these people were referred to as "suicide completers" in much research on suicide. The term is still used in some literature. In 2017, the MGSS ceased use of the term "suicide completer" to avoid connotations that suicide could be seen as a "solution" and because it is seen as a pejorative term.

[6]The different topics covered in the CECA interview include: sociodemographic factors, household arrangements and parental figures (including parental loss), parental care, familial and social context, physical abuse of children by their parents or other household members, psychological abuse by parental figures or other household members, sexual abuse by parental figures or other household members, different aspects of life and social contact with peers, networks and social support provided by adults and peers, cognitive patterns (e.g., feeling lonely, helpless, stigmatized), and circumstances related to leaving home (i.e., the parental house) (e.g., fugue, expulsion, voluntary departure) (Bifulco 2006, 21-22).

[7]These measures are described as "factual" because, for example, indifference/neglect items inquire into *behavior* rather than *feelings* about parents (e.g., were they interested in your school activities and your hobbies, did they show you a lot of affection or tenderness?). Similarly, abuse items are formulated in terms of examples of specific behaviors involved, such as being hit repeatedly rather than relying on respondents' interpretation of abuse (Bifulco et al. 2005, 576).

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maximum severity ratings of 1 and 2. Histories of abuse are validated with available reports from medical charts, coroner files, or youth protection services reports (Lutz et al. 2017, 1186).

The abuse measured with the CECA is considered to set people on "negative life trajectories". The degree to which adverse experiences *per se* serve as independent determinants of negative outcomes – as compared to the contribution of the dynamic interplay of early childhood adversities and circumstances (Boivin, Hertzman et al. 2012, 24) – remains beyond the scope of this epigenetic and neurodevelopmental research. Without being able to account for dynamic changes over the life,[8] trauma is instead studied as associated with trajectories, but trajectories with a wide range of possible outcomes. In adopting this approach, researchers focus on correlations rather than causal links. Thus, while ECA/ELA refers to specific measures and definitions of environmental circumstances or events, what these particular experiences lead to is far from clear, both within MGSS research and developmental research more generally. These studies nonetheless suggest that the experience of ECA is likely to require "significant psychological, social, or neurobiological adaptations from children who experience them" (McLaughlin 2016, 363-364), adaptations that are considered to make people, for instance, more reactive to stress. Consequently, in the MGSS models, while the effects of abuse might be modulated by events later in life that effectively move people off of negative trajectories, for a subset of the people who experience early abuse, the acquired neurobiological risk is thought to represent the origins of a diathesis that leads, for example, to impulsive or aggressive suicide, with specific neurobiological traits as the key correlating factor (Diaconu and Turecki 2009; McGirr et al. 2008b). These precipitating (neurobiological) profiles or states take center stage as the origins of a person's vulnerability to suicide risk.

Post-traumatic stress disorder: A diagnostic category with an exceptional etiological event

> When the term 'psychiatric trauma' is used today, it is generally in connection with the malady called *post-traumatic stress disorder* (PTSD). The idea that underlies psychiatric trauma is that disturbing experiences can create painful, persistent, and intrusive memories. (Young 2001, 661)

Since the 1980s, much of the discussion of trauma in psychiatry has been in connection with PTSD (post-traumatic stress disorder). When PTSD entered the DSM-III in 1980, it sat in discord with the rest of the manual in several ways. One of these is the temporal nature of the disorder: its symptoms are believed to emerge from *memories* of past traumatic events which enact damage on a person's present. The experience of trauma, in this perspective, refuses to be represented as part of the past, and is instead perpetually re-experienced in a painful, dissociated, traumatic present (Leys 2000, 2). Furthermore, while the extirpation of Freud and the concept of neurosis was a central goal of the manual – which was meant to set etiology aside and focus instead on symptoms – anthropologist Allan Young has argued that an exception was made in the case of the category PTSD. This allowed traumatic neurosis, in which the memory of an event plays an etiologically-specific role in a disorder, to live on in "post-Freudian psychiatry" (Young 1995, chap. 3; Young 2001, 663).

As Young explains, this was neither an oversight of the DSM committee nor an accident. Rather, it was a response to the political imperative to construct a category that would provide veterans of the Vietnam war with a legitimate diagnosis for their psychological difficulties and enable them to access services and disability pensions. Therefore, the category had to locate

[8]The tissue specificity of epigenetic traits hinders research on the dynamics of epigenetic changes related to early abuse throughout the life span because peripheral samples (e.g., blood) are not considered reliable indicators of brain-based processes. However, peripheral samples are nonetheless studied to provides insights into changes resulting from the treatment of depression with antidepressant medication (Fiori et al. 2018).

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the origins of their problems during their time in service – many years in the past – rather than a pre-existing condition or a post-service experience. This created several challenges for the committee and their solution was to identify the memory of an event that took place during their service years as the origin of veterans' problems (Young 2001, 664). The delayed effects of the "etiological experience[s]" were considered to make themselves known in the present through chronic polymorphous adaptations to anxiety. According to historian and trauma specialist Ruth Leys:

> the invention of PTSD had been propelled by the widespread desire to obtain formal acknowledgement of the idea that severely traumatic events could have prolonged psychological consequences in anyone, regardless of the individual's prior history or personality. In this regard, the PTSD diagnosis necessitated a shift away from the previous approach to trauma, which concentrated on the ways in which stressful life events were mediated by the subjective interpretation of the victim. (Leys 2006, 138)

For Leys, this meant that the previously, largely psychoanalytic emphasis on the effect of the subject's prior experiences on the traumatic reaction, especially his or her early libidinal history, was replaced by a focus on the role of the external environment (Leys 2006, 138).

This version of PTSD was narrow, conceived of to acknowledge how trauma – the specific atrocities soldiers experienced during the war – affected them in the present. According to Young, this is how Freud's traumatic neurosis gained an exceptional status in DSM-III. At this time in the disorder's history, it became:

> simply taken for granted that time and causality move *from* the traumatic event *to* the other criterial features and that the event inscribes itself on the symptoms. Because the traumatic event is the cause of the syndromal feelings and behaviours, it is logical to say that it precedes them .... The discovered memory is now the explanation (post hoc) for the onset of his symptoms. (Young 1995, 115-116, emphasis in original)[9]

In this "traumatic neurosis," the specific nature of these events and the memories associated with them were important: the events were considered sufficient to cause "significant distress to anyone, anywhere" (Young 2001, 668). It was these events and their related intrusive memories that both bound together and rendered distinct the broad range of symptoms of PTSD, which overlapped substantially with other disorders such as major depression and generalized anxiety disorder (Young 2001, 677).

For the purposes of our argument, we are most concerned with several specific elements of the diagnosis PTSD and its changes over time. We will devote the remainder of this section to these elements. We will first focus on the diagnosis in terms of the depiction of the relationship between traumatic events and psychopathology in early descriptions of the category. We will then examine changing understandings of the disorder since, with particular attention to the ways in which PTSD research has come to frame the disorder as the reproduction of the stress process in which it is thought to originate. In effect, that the stressful or traumatic events – and the psychological and biological effects of these events – at the base of PTSD, are considered to produce a certain kind of person: someone whose subsequent reactivity to stress is seen as the key pathological element of the disorder. We are particularly interested in how the temporal particularity of etiologically-significant traumatic event has shifted over the years.

In the original formulation of the disorder, the occurrence of the significant traumatic experience during war experiences was essential to its diagnosis. Yet, like many psychiatric classifications, PTSD – and understandings of trauma as the etiological event responsible for the

[9]For a more detailed account of the exceptionality of PTSD within DSM-III see Young 1995.

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disorder – is a moving target (Hacking 2007). After its inclusion in DSM-III in 1980, PTSD shifted quickly both in terms of its patient profile (with corresponding changes in its diagnostic criteria) and the trauma associated with the disorder. Allan Young noted, in 2001, that "in less than a decade [after its inclusion in DSM-III], PTSD had acquired a contagious quality, manifested variously in 'transgenerational PTSD' . . . 'vicarious PTSD,' [or] 'compassion fatigue'" (Figley 1995; Motta et al. 1997; Solomon et al. 1988; Yehuda, Beirer, et al. 2000, cited in Young 2001, 662). Likewise, he suggested there had been "a corresponding extension in the effects attributed to PTSD" (Young 2001, 662). Symptoms expanded from dreams, intrusive images, and phobias, to a variety of physical ailments including lung disease, obesity, and diabetes. PTSD was often diagnosed as comorbid with a variety of other mental disorders ranging from personality disorders to schizophrenia (Pynoos et al. 1999, cited in Young 2001, 662).

At the same time, the definition of the traumatic event at the heart of PTSD began to shift (Young 2001, 668). Whereas in DSM-III (1980) it was defined as an event that would cause "significant distress to anyone, anywhere" (Young 2001, 668), by the time DSM-IIIR was published in 1987, the traumatic events considered significant to PTSD shifted from being "out-of-the-ordinary" to "quite ordinary (but awful) experiences" (ibid.). While these changes are substantial, Young notes that they "simply codified what had become a convention by the end of the 1980s" (ibid.) in terms of the application of the diagnostic category. Nonetheless, the textual changes to PTSD significantly and formally broadened the range of people who could be diagnosed. PTSD diagnosis has since expanded to encompass an increasingly wide range of populations and presentations, regardless of background, history, or personal sensitivity (Summerfield 2001). This potentially includes "anyone who presents a psychological affliction following an unusual event, whether he is the victim or the perpetrator" (Rechtman 2002, 790, our translation).

Other post-traumatic syndromes conceptualized prior to PTSD such as the "rape trauma syndrome" (Burgess and Holstrom 1974) or the "battered women's syndrome" (Walker 1984) were subsumed under the new description of the diagnosis in DSM-IIIR, despite the fact that the different syndromes had originally been described with considerable variations from the eventual definition of PTSD (Van der Kolk et al. 2005, 389). These studies of physically and sexually abused children, as well as of women who were exposed to prolonged interpersonal violence, played a major role in establishing an integrated, post-Vietnam War-specific approach to trauma (Herman 1992, cited in Leys 2000, 5). In the process, forms of trauma were conceptually merged. Concomitantly, epidemiological studies of the era suggested that exposure of the American population to "trauma-level events" might be as high as 90 percent (Breslau 1993, cited in Young 2001). Trauma and responses to trauma needed to be reconsidered in light of this, and they were. Consequently, at this point in its history, PTSD became not about a normal response to an abnormal experience, but the opposite. Research was reoriented to identifying why, in response to relatively common (if awful) experiences, some people developed PTSD while others did not (Young 2001, 672). Molecular and genetic research was seen as a potential means of answering this question through the identification of a pathogenesis distinct to PTSD, setting subjectivity, memories, and differential diagnoses aside.

Diatheses and vulnerabilities: Environmental epigenetics and the unfurling of the past in the present

It is at this point in history that narratives of child abuse and suicide, and trauma and PTSD begin to more consistently share a number of common pathways that fit into parallel transformations in other research agendas and funding interests as they concern the Developmental Origins of Health and Disease (DOHaD) (Ngo and Sheppard 2015; Penkler et al. 2019) as well as the later phases of ACEs research (e.g., Sun et al. 2017; Thompson et al., 2015). Central to the shared reasoning in these narratives is a distinct etiological association between early trauma and a wide variety of specific forms of (psycho)pathology and (neuro)biological risk later in life.

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PTSD, cortisol dysregulation, and the origins of vulnerability

Throughout the 1980s, a growing body of research focused on the molecular biology of PTSD and attempted to ascertain the specific impact of trauma on the body as a means of explaining its enduring effects on certain people (Mason 1986, cited in Young 2001, 273). Research emerging from these studies suggested that traumatic stress leaves its mark on the brain in the form of literal "eidetic" or iconic "imprints" (van der Kolk et al. 1985, in Leys 2000, 250) and would be expected to exert profound changes over a wide range of biological structures and functions in a way that is theorized as standing "outside all ordinary stress response[s]" (van der Kolk 2000, 13). Cortisol emerged as a potentially significant hormone involved in the structural and functional profiles associated with the disorder and substantial research was dedicated to situating PTSD in relation to this hormone and its regulation via the hypothalamic-pituitary-adrenal (HPA) axis (Young 2001, 671-672; Young 2004, 14). Complicated findings resulted, without clear associations emerging even as to whether PTSD was associated with hypo- or hypercortisol excretion (Young 2001, 672, 677).

An essential part of the cortisol story changed in the mid-1990s, and this is the part of the story on which we will focus. Specifically, PTSD was reconceptualized as the consequence of a diathesis, similar to models of diathesis-stress interactions involving life stressors and predispositional factors thought to be involved in the development of schizophrenia and depression (Monroe and Simmons 1991, 406-407). While understandings of the effects of trauma as part of a diathesis had been in circulation since the days of Charcot, it was only at this time that they became a part of the PTSD research narrative (Young 2001, 678), with eventual implications for the interpretation of a wide range of other forms of psychopathology and neurobiological risk. Cortisol remained central to the story, but it became a story of "dysregulation" rather than "down regulation." The diathesis model, Young argues, "shifts PTSD's centre of gravity from the traumatic event to the traumatized patient" (Young 2001, 677). The origins of his/her/their current problems, in this model, relate to an interaction between a predispositional vulnerability and a stress caused by life experiences. The "vulnerability" or "predisposition" of the veterans could have its origins in, for instance, negative early life events or specific genetic profiles. While "pre-existing" conditions were seen as a potential challenge to the legitimacy of veterans' PTSD in 1980, by 1995 the disorder had become sufficiently anchored in American psychiatry and beyond that the diathesis model was no longer considered to undermine the credibility of veterans' suffering (ibid., 676-7). Considerations of predisposition and vulnerability to PTSD multiplied during this period and drew on studies of pre-war weakness before and after WWI (Scott 1993; Shephard 2001; Young 1999), WWII-era predictions of which soldiers would "break down" in combat based on their psychiatric histories (Grinker and Spiegel 1945; Kardiner and Spiegel 1947), and multicausal models of PTSD supported by post-Vietnam research regarding acute stress reactions and their possible origins in a wide variety of relatively ordinary yet severe stressful events (Blank 1985, in Shephard 2004, 53-54). Overall, studies of prevalence, course, and comorbidity of PTSD raised questions concerning the role and magnitude of proximate stressors as primary etiological factors (Breslau and Davis 1987), challenging the idea that PTSD was a typical and inevitable outcome of exposure to traumatic stress (Yehuda and McFarlane 1995, see Shephard 2004, 54–55 for details).[10]

Biomarkers specific to this diathesis model of PTSD vulnerability were sought in both gene-environment interaction (GEI) and epigenetic studies (Comings et al. 1996, 368–369, cited in Young 2001, 678). The glucocorticoid receptor, or GR, and its regulation of cortisol became central to these models of vulnerability. In the epigenetic models in particular, based on studies of rodent model organisms, early stress was seen to induce vulnerability and reactivity to future experiences of stress. "In other words, *the epigenetic marks reproduce the stress process in which they originate*" (Young 2012, emphasis in original). Yet despite an increasing number of molecular

[10]As argued by trauma experts Rachel Yehuda and Alexander McFarlane in a pivotal article published in 1995, "the observation that trauma is not a sufficient determinant of PTSD raises the possibility that there may be risk factors that account for a given individual's vulnerability to developing this disorder" (Yehuda and McFarlane 1995, 1708).

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studies of PTSD, across broad and varied datasets, these studies have yet to offer evidence of a molecular pathogenesis distinct to pure PTSD (in its common form it is presented with comorbidities), which would enable researchers to distinguish it from other disorders with similar symptoms (Young and Breslau 2016). In the end, or at least to date, it is only an indelible or "iconic" memory that distinguishes a diagnosis of PTSD from a diagnosis with other conditions with which PTSD shares a large number of symptoms and potentially neurobiological processes (ibid., 150; see also McNally 2009; and Weathers et al. 2014). Consequently, diathesis-stress models of PTSD, rather than identifying the molecular specificity of PTSD, instead contributed to a portrait of neurobiological vulnerability more broadly. Specifically, these models describe processes that have become core features of contemporary psychiatric models of risk and vulnerability in which early stress or trauma, via epigenetic changes of the GR,[11] are correlated with a wide variety of forms of psychopathology (Radtke et al. 2011; Turecki and Meaney 2016; Tyrka et al. 2012; Binder et al. 2008; Katz and Yehuda 2006; Heim et al. 2010; Heim and Binder 2012; Roth et al. 2009).

To a certain extent, these emerging understandings of mental illness recall earlier critiques of the limits of classificatory systems based in categorical approaches to psychopathology, such as those found in the work of Hempel (1965) and Eysenck (1964 and 1967) (see Demazeux 2008 for details). At the same time, they situate mental illness in ways that are consistent with orientations of the National Institutes of Mental Health's (US) Research Domain Criteria (RDoC) initiative, in which the origins of mental illness are thought to be found in a variety of dimensional traits (domains), from cognitive to arousal/modulatory systems, that cross-cut categories in current diagnostic systems (Sanislow et al. 2019). In these studies, while genetic vulnerability remains a key etiological consideration, researchers now look for additional sources of vulnerability given that decades of research which sought to identify the "gene for" a number of common forms of mental illness met with inconclusive results. Genetic studies identified the potential and partial role of many different genes and, in some cases, the role of a very wide range of common genes carried by the majority of the population (Kendler and Donovan 2014; Border et al. 2019; Banerjee, Morrison, and Ressler 2017). Continued efforts to identify origins of vulnerability have been described as the search for "missing heritability" "in which heritability estimates are significantly higher than the variance explained by known genetic variants" (Mayhew and Meyre 2017). The search has led researchers to increasingly examine the role of the environment in the development of vulnerability in the form of gene-environment interactions, but especially epigenetic mechanisms. This is the case not only for research focusing on neurobiological risk for psychiatric conditions, but also for a wide range of research agendas in the postgenomic era that focus on the plasticity of human bodies and in which nurture is increasingly seen as indissociable from nature (Meloni 2019; Fox-Keller 2010). As an environmentally-informed understanding of human development has begun to eclipse pre-genomic models, attention has turned to the moments at which people are considered to be most "plastic" or susceptible to environmental influence. Youth, childhood, and in utero experiences are now seen as not only relevant but crucial targets for research. In a context where scientists must explain why someone would respond more negatively than someone else to an "ordinary" adverse experience in adulthood, the source of exceptionality (i.e., vulnerability) is increasingly sought in adversity in the early years. Attention to time and trauma shifts in these accounts, with increasingly limited consideration of the etiological significance of the iconic memories historically associated with PTSD. In the process, it is the origin of the diathesis that has become the focus of research in molecular biology including environmental epigenetics.

[11]Postmortem studies of PTSD brain tissue, although limited in number, have provided evidence for dysregulation of GR sensitivity and signaling, gene expression, innate immune response, and inflammation pathways (Holmes et al. 2017; Matosin, Halldorsdottir, and Binder 2018; Young et al. 2015).

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Diathesis and/or Complex PTSD

In parallel with studies of diatheses, the search for biomarkers specific to pure PTSD, and studies of potential molecular pathways for a wide range of psychiatric conditions thought to be associated with the experience of early adversity, another history was unfolding, that of complex PTSD, or CPTSD. First proposed by Judith Herman in 1992, the disorder was conceived of to recognize more chronic and coercive forms of trauma. The diagnosis PTSD, Herman argues:

> derives primarily from observations of survivors of relatively circumscribed traumatic events: combat, disaster, and rape. It has been suggested that this formulation fails to capture the protean sequelae of prolonged, repeated trauma. In contrast to the circumscribed traumatic event, prolonged, repeated trauma can occur only where the victim is in a state of captivity, unable to flee, and under the control of the perpetrator. ... The psychological impact of subordination to coercive control may have many common features, whether that subordination occurs within the public sphere of politics or within the supposedly private (but equally political) sphere of sexual and domestic relations. (Herman 1992, 377-8)

Complex PTSD, as formulated by Herman and later by others, was meant to account for those types of trauma that did not fit with the existing criteria for PTSD. In particular, the disorder sought to describe the pervasive effects of prolonged trauma on a person, including symptoms, character and personality, and the "survivor's vulnerability to repeated harm, both self-inflicted and at the hands of others" (Herman 1992, 379; van der Kolk 2001).

While research on the diathesis model of PTSD turned attention to the potential for early life factors to render someone vulnerable to mental illness in response to trauma later in life, CPTSD framed the effects of post-traumatic stress more explicitly in a developmental perspective (Roth et al. 1997). Specifically, post-traumatic stress was described as resulting in an "impairment in developmental processes related to the growth of emotion regulation and associated skills in effective interpersonal behaviours" (Cloitre et al. 2009, 400). It is the experience of child abuse in particular that is considered by some researchers to be the key factor that distinguishes CPTSD from PTSD (Cloitre et al. 2013, 8; van der Kolk et al. 2005). This explicit association between early abuse and (C) PTSD, one that had circulated in terms of possible etiological explanations of PTSD but that had not been expressly put forward as a developmental process, produced a narrative continuum between early trauma and traumatic experiences later in life. Researchers further argued that the distinction between PTSD and CPTSD provided a "conceptual coherence to the multiple, diffuse, and apparently contradictory symptoms of complex PTSD" (Cloitre et al. 2009, 400) through attention to vulnerability caused by and the heterogeneity of adaptation to trauma (Resick et al. 2012). Complex PTSD was elaborated as a diagnosis alongside discussions of DTD. Complex PTSD and DTD share many features and both correlate early abuse with a wide range of psychiatric conditions as well as an array of "cognitive, language, motor, and socialization skills." (Van der Kolk 2005, 404-5) Van der Kolk and others consider early abuse and its after effects to be pervasive in society, arguing that they are leading to a "silent epidemic of neurodevelopmental injuries" (Kaffman 2009, cited in Ford et al. 2013). The results of these injuries, according to emerging narratives in developmental research, would include CPTSD.

Complex PTSD was not included in DSM-5 in 2013. Arguments against its inclusion involved its frequent comorbidity with PTSD and Borderline Personality Disorder (BPD) (Resick et al. 2012).[12] However, given that that CPTSD was included in ICD-11 in 2018 (the World Health Organization's *International Statistical Classification of Diseases and Related Health Problems*, 11th edition), there is reason to believe it will be included in future editions of the DSM.

Through the development and formalization of the classification CPTSD, we see the emergence of a model of trauma and its effects that is highly consistent with environmental epigenetics models of

[12]Some researchers argue that BPD should be reconceived of as CPTSD (Resick et al. 2012).

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suicide wherein trauma becomes a part of a person's future. Depending on the person and his/her/their later life experiences, this trauma might be seen as entering, shaping, and never leaving the body.[13] Moreover, this body of research frames childhood as a period in which specific types of adversities, such as neglect and peer victimization (Geoffroy 2018; idem 2016), under-resourced health services (Renaud et al. 2014), and poor nurturing environments (Bethell et al. 2017) appear endemic, placing children and youth at risk of negative developmental outcomes. The weight of claims emerging from molecular models of trauma have already resulted in shifts in the focus of ACEs research in recent years from the study of health outcomes in adult life to the study of neurobiological and psychosocial effects of abuse believed to already be tangible during childhood and adolescence (Sun et al. 2017; Thompson et al. 2015). In this context, there have been calls for health policies and services (e.g., psychiatric, social work) that focus on "trauma-informed care" (Edwards, Gillies, and White 2019) to promote "resilience" in children through interventions that aim to mitigate the potentially durable, negative effects of ECA and prevent them from entering stable, long-term negative trajectories (Davidson and Carlin 2019; Larkin, Felitti, and Anda 2014; Logan-Greene et al. 2014; Lang et al. 2019; Bethell et al. 2016; Ranjbar and Erb 2019).

Constructing the etiology of suicide

The environment epigenetics models of suicide risk at the MGSS emerged from the same diathesis-stress models that were at the base of PTSD, and later CPTSD, research. The origins of the MGSS epigenetics research program, for instance, can be found in the same rodent models of stress and epigenetic modifications of the GR that informed PTSD diathesis models, models that have had an immense impact on contemporary environmental epigenetics research. Researchers at the MGSS were the first scientists to translate these exceptionally influential studies on the effects of early adversity on stress reactivity later in life, carried out on model organisms (Meaney 2001; McGowan et al. 2009; Weaver et al. 2004), into human populations in the form of studies of people who experienced early abuse and who died by suicide later in life.[14]

Researchers at the MGSS argue that ECA leads to specific epigenetic profiles, at least some of which are sufficiently stable to be found in the brains of people who die by suicide (Labonté et al. 2012; Lutz and Turecki 2014; Lutz et al. 2015). They consider these epigenetic traits, identified in postmortem brain tissue, to constitute a molecular basis for suicide risk. In the words of Gustavo Turecki, ECA "lead[s] to the development of these maladaptive trajectories. . . . So, suicide risk is, perhaps the most severe negative end point of those psychopathological conditions that are in turn predicted by these negative trajectories."[15]

While their highly influential research is oriented toward the identification of a model for suicide risk, we suggest that their primary contribution to contemporary narratives of neurobiological risk is different. The core of their research program focuses on ascertaining the biological nature of the origins of the diathesis and identifying the biological changes during childhood that are thought to occur in response to early abuse (e.g., Brezo et al. 2010; Diaconu and Turecki 2009; McGirr and Turecki 2007; Turecki 2014a). So, if PTSD researchers reoriented attention from the traumatic event itself – initially seen as abnormal – toward a person's abnormal reaction to a normal traumatic event by recourse to a diathesis-stress model, the MGSS researchers are reorienting attention toward characterizing the origins of this vulnerability.

[13]This reasoning parallels that described by sociologist Hannah Landecker in her work on epigenetics and metabolism (Landecker 2011).

[14]Key examples of this research include experimental studies by the research group led by Michael Meaney, who since the 1980s has been studying the effects of early maternal care of rat mothers on their offspring, in particular stress responsiveness that may lead to long-term health changes (Meaney and Stewart 1979; Meaney et al. 1985). Meaney and his team have, more recently, considered these effects in terms of "epigenetic programming" (McGowan et al. 2011).

[15]Interview by Lloyd.

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Within this research program, a specific story of the lives of people who die by suicide is beginning to emerge, in which negative early life events set people on pathological life trajectories whose extreme end point is suicide. This conceptualization of suicide as the product of a diathesis is stabilized through recourse to temporal arguments about the presumed indelibility of early experiences on the body. Suicide in this view is bookended by two precipitating events: one in the past and (probably) "abnormal," the other more recent and "normal."

The biological embedding of time

Epigenetic traits are generally described as unstable precisely because they are considered responsive to environmental cues. As Turecki and the highly influential epigenetics researcher Michael Meaney acknowledge, recent evidence in epigenetic research supports the hypothesis that "epigenetic plasticity is sustained in the brain throughout adulthood, potentially as a mechanism to cope with the evolving demands of the environment, yet there are clear moments during development when plasticity is heightened, and these may be more strongly associated with the establishment of life-long epigenetic modifications" (Auger and Auger 2013, in Turecki and Meaney 2016, 7). Researchers at the MGSS explain the enduring effects of ECA in terms of, first, "critical windows of neuroplasticity" and, second, severity of abuse, as measured with the CECA (Lutz et al. 2017; Turecki 2016, 168; Fiori and Turecki 2016). In the first case, events that occur earlier in life are considered to have more significant consequences for subsequent development than events that occur later in life (Turecki 2014b, 144; Turecki et al. 2012).[16] Furthermore, the early abuse is considered to correspond to the timeframe of the stabilization of a specific epigenetic trait, CH methylation, in the first fifteen years of life. This form of methylation is thought to be present in higher proportions in the brain than other parts of the body (Barnett-Burns et al. 2018; Lutz et al. 2018; Lutz et al. 2017, 1186). Thus, MGSS researchers expect that the biological consequences of an experience during the first fifteen years of life, if significant enough to affect CH methylation, might be of greater significance and potentially more durable given that it stabilizes relatively early in life, as compared to other epigenetic traits (Lutz et al. 2018). Second, the severity of the abuse is considered sufficient to unleash biological consequences for anyone (Brezo and al. 2008). This is how MGSS researchers describe the origin of the diathesis, or developmental processes, underlying suicide risk – how one moment in time, a traumatic experience, becomes indelibly impressed on a person with enduring consequences for the rest of that person's life.

A life of various psychopathologies and personality traits (e.g., impulsivity, bad decision-making) is considered to follow early abuse and methylation: in these models, epigenetic marks reproduce the stress process in which they are thought to originate, as in the PTSD research Young studied (Young 2012). In MGSS models, the remainder of a person's life and the events preceding his/her/their later death by suicide appears to carry less weight than early traumatic experiences in terms of the suicide risk they confer. MGSS researchers consider the negative events proximate to suicidal behavior to be common problems (e.g., loss of a job, interpersonal conflict leading to rejection and isolation, stressful life events) (Jollant et al. 2011, 320; Turecki 2014a, 9; Turecki 2014b, S145; Zouk et al. 2006, 200, 202[17]). In this vision of time, trauma, and psychopathology,

[16]Examinations of the sustained effects of environmentally induced epigenetics modifications – for instance, in transcriptional activity of post-mitotic cells such as fully differentiated neurons in the brain (Maze et al. 2015; Riccio et al. 2010) – suggest that histone acetylation and DNA methylation may serve as a "cellular memory" of these experiences (Champagne 2010, 570).

[17]"Our findings support speculation about a developmental cascade that may begin with a biological predisposition, negative early life stressors and history of abuse, resulting in the expression of higher levels of impulsivity and aggressive behaviors. These behaviors in turn, may increase the risk of developing a cluster B personality disorder, which could subsequently lead to an increased risk of substance abuse/dependence (Johnson et al. 1999). The interplay of the above three factors could explain in part a developmental mechanism that may lead to increased predisposition to suicide, particularly following a triggering stressor and onset of depressive illness with suicidal ideation." (Zouk et al. 2006, 202)

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produced in the afterlife of a person who died by suicide, the events surrounding his or her death are considered almost an afterthought when compared to early life events.

Many questions remain. In the epigenetics studies of suicide, ultimately researchers do not know when these epigenetic traits or vulnerabilities were established – the epigenetic profiles identified in post-mortem brains could reflect a change near the time of death – but this is not the narrative that has been stabilized. In the PTSD studies, if researchers initially had to justify the persistence of a memory against research that showed memories to be highly malleable processes rather than "timeless photos," epigenetics researchers have to explain the apparent formation and persistence of what might otherwise be a highly changeable biological trait. These parts of the puzzle have yet to be answered.

The correlation of ECA/trauma first to an epigenetic trait and, second, to a variety of "negative mental health outcomes" could be described as the reduction of the complex or multiple to the simple or singular. Yet what we're interested in, beyond reductionisms or neoreductionisms[18] (Lock 2015), is what is elaborated in these studies: the stabilization of a particular narrative of time, trauma, and psychopathology. This is a story of recurrence, of the re-enactment of an original stress. It is about the nature of what stays and affects the rest of a person's life. It is a story about which experiences in our lives are considered significant.

Yet this is not a story that is specific to suicide or even the broader narratives of neurobiological risk at the MGSS. It is one of many incremental contributions that have led to current understandings of neurobiological risk and risk of ill health more generally. While much attention has been given to narratives of plasticity and vulnerability as characteristic of the postgenomic era, we suggest that these narratives of vulnerability can be traced back further in time. One precursor to current understandings of plasticity can be found in narratives of PTSD and, particularly, shifts in attention from the traumatized soldier to the vulnerable soldier. This narrative shift can now be seen as coherent with more recent research trends including, for example, the RDoC initiative, which in many respects represents a return to etiology in biopsychiatry after it had been banished from official psychiatric nosology only forty years ago. Moreover, this is a return to specific etiological narrative, one that traces significant etiological events to youth, reconfiguring how we account for their effects and people's subsequent subjective experiences.

The MGSS environmental epigenetics research on suicide risk and recent research on PTSD focusing on the origins of the diathesis are producing new narratives of vulnerability. In current narratives, with a focus on the GR and other biomarkers, a new timeline – a new sense of time – is created: a biologically embedded time. What, in other models, might have been considered indelible memories of early childhood abuse (Janet 1889; Breuer and Freud 1893) become past traumas stamped onto post-mitotic cells in a child's brain. This biologically embedded time is thought to live on in a person, recreating the stress out of which it was born, explaining why a person might respond "abnormally" later in life to a "normal" traumatic event. New etiologically significant events are created. And along with them, universal vulnerability – given a particular set of early negative environmental factors – to these conditions and behaviors.

It is in this way that, as in the case of PTSD, models of suicide provide insights into psychiatric reasoning about "the past invading the present" in the form of past abuse, diatheses, and development (Young 1995, 7; Young 2006). Narratives of the people who die by suicide allow molecular traits to be associated with specific moments in time, and further, allow them to be seen as indelible when they might otherwise be seen as fleeting. Ultimately, both research programs are ever-changing stories of time, trauma, and how we come to understand trajectories, futures, through people's pasts, even if, in the case of suicide, these are constructed in their afterlives.

[18]Though strategic reductionisms are certainly involved as they concern the experimental systems in MGSS research.

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When suicide no longer has a significant precipitating event

In these models, the scope of vulnerability changes. Researchers have moved beyond an interest in the exceptionality of proximate traumatic or stressful events (and their memories) as the cause of present disorders or behaviors, and (largely) beyond gene-environment interactions as a source of vulnerability in which case it would only be a certain, stable, and limited subpopulation that would be at risk (i.e., people born with a genetic predisposition). The meaning of wartime traumatic events involved in the original formulation of PTSD shift from exceptional to unexceptional, as predispositions and vulnerability established in youth become the key etiological factors that allow normal negative events later in life to be seen as sufficient to trigger pathological stress reactions. In environmental epigenetics models of suicide risk, potential vulnerability becomes universal: given the right (which is to say wrong) environment or traumatic experience early in life, anyone can be placed on an "unhealthy trajectory."

The temporal nature of risk and vulnerability in these models changes as well. The important event is not something later in life or closer to death, but early in childhood. As a result, perceived windows of therapeutic opportunity, origins of vulnerability, and evaluations of risk change. In some areas of research, these essential windows roll back even further than childhood to perinatal or in utero experiences and the preconception period. In such a context, the answer to the question "When is the beginning?" (Pentecost and Meloni 2020, 8) reveals an increasing schism between biographic and biological perspectives (Lloyd and Larivée forthcoming). Pathology comes to be seen as the result of not only experiences during one's own lifespan, but also inter- and transgenerational life experiences, conceived of as able to shape biological traits and personal trajectories.

For the remainder of this paper we will focus on the ways in which shifting understandings of vulnerability and risk have resulted in historically specific neurobiological models of suicide. Through these models, we have come to a point where suicide can be studied as an action without a significant precipitating event, only a significant precipitating biochemical state as a result of early-acquired neurobiological risk. This neurobiological state is considered to be the key precipitating factor for suicide. The common negative experiences that often precede suicidal acts are reconceived as triggers (Jollant et al. 2011; Turecki 2014a, idem 2014b; Zouk et al. 2006). According to MGSS neuroscience researcher and psychiatrist Fabrice Jollant:

> The triggering of the suicidal crisis often has external causes. Environmental events are frequently put forward in notes left by people who die by suicide or reported by suicide attempters themselves (Heikkinen et al. 1994). However, these reasons are in most cases very common problems including marital difficulties and separation or job problems and loss (interestingly, these reasons may be universal as shown by anthropological studies in pre-industrialized populations [MacDonald 2006]). In addition, suicidal acts may sometimes take place without any clear external triggers. Therefore, these environmental events are apparently not sufficient to totally explain the occurrence of a suicidal crisis. As external triggers of suicidal acts are often social by nature – they either involve other people (e.g., difficulty with one's partner) or one's place in society (e.g., loss of social status) – *individual differences in the way people experience social relationships, perceive oneself in society and respond to the social environment should be central to understanding the suicidal process*. (Jollant et al. 2011, 320, emphasis added)

Thus, while devoid of exceptional qualities, triggers are considered sufficient to set in motion affective responses and suicidal behavior among individuals who are vulnerable to pathological stress reactions.

The absence of a significant precipitating event in these understandings of suicide, replaced by triggers and affective states, raises questions about the cognitive and emotional aspects of people's

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suicidal behaviors. Ruth Leys is among a number of social scientists and humanities researchers who have critically analyzed neuroscientific explanations of affect as a neurobiological state (e.g., Blackman and Venn 2010; Connolly 2002; Hemmings 2005; Massumi 2002; Papoulias and Callard 2010; Thrift 2004). She argues that this framing of affect has significant implications for the ways in which people's actions are understood:

> there is a gap [in these explanations] between the subject's affects and its cognition or appraisal of the affective situation or object, such that cognition or thinking comes "too late" for reasons, beliefs, intentions, and meanings to play the role in action and behaviour usually accorded to them. The result is that action and behavior are held to be determined by affective dispositions that are independent of consciousness and the mind's control. (Leys 2011a, 443)

Leys suggests that these models have the effect of replacing "the idea of one's intention[19] with regard to objects or of the meanings those objects might have" with the idea of affect as triggered neurobiological states "operating outside the domain of consciousness and intentional action" (ibid., 465). These models, she writes, "posit a constitutive disjunction between our emotions on the one hand and our knowledge of what causes and maintains them on the other, because according to them affect and cognition are two separate systems" (ibid., 437). In other words, affects are only *contingently* related to objects in the world, defined in non-intentional terms: "they operate blindly," Leys adds, "because they have no inherent knowledge of, or relation to, the objects or situations that trigger them" (ibid.). This understanding of affect within the neurosciences is thus often aligned with models of self-organizing networks and bodily systems as processual and dynamic entities that attribute the origins of action to "the neural *infrastructure* of consciousness and *not* to our experience of the lived present" (Papoulias and Callard 2010, 47, emphasis in the original).

Debates over the nature and limits of intentionality and non-intentionality of affect thus speak to questions of whether affect requires (or not) a subject in order to understand or produce its effects (Blackman 2012, 12). This has a direct bearing on the extent to which personal experiences across time, social contexts, and relationships, are seen as tightly bound-up with human meaning-making (Dragojlovic 2015; Walkerdine 2010) and associated with neurobiological processes (Blackman 2010; Clough 2009).

The intersection between these debates and contemporary reasoning about a wide range of psychopathologies – including PTSD and the forms of mental illness considered associated with suicidal behavior, but extending well beyond them – are held together through narratives of diatheses and early trauma. Within these models, once an early neurobiological profile has been established, subsequent decision-making is seen as relatively impaired, with greater reactivity toward normal negative events, which are now conceived of as triggers. The content of these events is seen as relatively unimportant. In the case of suicide, this conceptualization of triggers speaks directly to the role of precipitating events, reasons for suicidal behavior, and the meaning of suicidal acts.

A number of explanations of suicide are incompatible with these models of vulnerability. Suicides of resolve, in which a person makes a reasoned decision to commit suicide, as documented by anthropologist Junko Kitanaka do not fit (Kitanaka 2011, see chap. 7). Kitanaka describes a process in which suicide came to be interpreted, if not as a result of specific neurobiological risk, almost exclusively as a result of mental illness, the result of impaired reasoning. Suicide as "way of belonging," when, in regions characterized by high rates of suicide, all you have

[19]Leys uses the term "intentionality" to refer to "states of mind that are directed towards an object and that include beliefs, judgements, wishes, and cognitions" (Leys and Goldman 2010, 669). Intentionality, she writes, "carries with the idea that thoughts and feelings are directed to conceptually and cognitively appraised and meaningful objects in the world" (Leys 2011b, 802).

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left is your connection to the dead is equally incompatible with emerging models of vulnerability that focus on early events and predispositions rather than temporally proximate reasons for suicidal behavior (Niezen 2009). Suicide as a shared experience under the sway of personal ties, regimes of desires, imagination and remembrance, and daily struggle with life and death, as documented by anthropologist Lisa Stevenson based on her experiences listening to suicidal youth in Nunavut, is an action that is not easily understood as set off only by a trigger. Suicide, in this instance, is seen as being deeply a "part of their everyday fabric of life" (Stevenson 2014, 9). Suicide as a protest to political or economic upheaval (Widger 2015; Imberton 2012) sits uneasily with environmental epigenetics explanations of suicidal behavior, in which precipitating chemical states replace precipitating social, economic, and political circumstances. Suicide bombers, as examples of late twentieth century reframings of suicidal acts as "weapons" (Hacking 2008), further complicate and distribute intentionality into the realms of social and political violence and imagination in which notions of class, gender, and ethnicity become intertwined (Brunner 2007; Jaworski 2010; Victor 2003). These contextualized experiences are pushed to the side when suicide is no longer seen as having a significant precipitating event or being the result of a rational decision. When neurobiological risk established in youth takes the foreground, other factors considered associated with distress and suicidal acts later in life recede to the background. Viewed from this perspective, the MGSS account of suicidal behavior and the wide-ranging neuroscientific literature that draws on and develops similar narratives of neurobiological vulnerability appear to sideline many of the considerations that are central to other accounts of suicide.

As sociologist of the life sciences Maurizio Meloni argues contemporary bioscientists' task "is not so much to map whether a disease is the product of gene or environment, or an interaction of the two, but how environmental exposures in critical windows of plasticity are biologically embedded and reproduced through a mixture of bodies-biohabits-culture" (Warin et al. 2015, cited in Meloni 2018, 6). Currently, in environmental epigenetics research on suicide, some of these windows receive more attention than others. Understandings of youth as a period of elevated vulnerability to the impact of negative events is stabilized in MGSS research through recourse to psychological tests and neural markers that act as proxies for proof of childhood trauma. Identification of this trauma is the basis of an explanatory model in which biomarkers identified in molecular research are understood as embodied evidence of abuse in the brains of people who have died by suicide or who are considered at risk of suicide. These preliminary hypotheses, which can also be seen as proxies, about how and under what circumstances negative experiences become part of one's cerebral constitution guide scientific models and, increasingly, priorities for intervention (White and Wastell 2017; Gillies, Edwards, and Horsley 2016). While proxies might be understood as interim solutions that "carry us through a period of ignorance until we find the underlying causes" (Duster 2005, 1050), as sociologist Troy Duster points out, these proxies can have powerful consequences. While new scientific findings and narratives will ultimately emerge and amend or displace prevailing hypotheses, Duster warns that a proxy can be "such a dominant category in the cognitive field that the 'interim solution' can leave its own indelible mark once given even the temporary imprimatur of scientific legitimacy by molecular genetics" (ibid.). In this sense, proxies play powerful roles in scientific narratives and must be considered more than simply place-holders. While details of the MGSS model shift over time, perhaps the durable impact of this narrative of neurobiological risk, along with those widely circulating in other areas of research, might most specifically concern time. When the diagnosis PTSD was initially described in 1980, it was "simply taken for granted that time and causality move from the traumatic event to the other criterial features and that the event inscribes itself on the symptoms. Because the traumatic event is the cause of the syndromal feelings and behaviours, it is logical to say that it precedes them . . . . The discovered memory is now the explanation (post hoc) for the onset of his symptoms" (Young 1995, 115-116). If many elements of the diagnosis have changed since 1980, this conceptualization of time, trauma, and causality have not. While the relevant time at which the pertinent trauma is thought to have occurred has shifted

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from adulthood to childhood, time and causality are considered to move just as surely from the traumatic event to the symptoms. This is how trauma at one point in time has come to be seen as having specific etiological consequences. Through the logic of narratives of plasticity and critical windows of neuroplasticity, this notion of how trauma leads to symptoms has taken a place at the heart of neuroscientific and biomedical reasoning more broadly.

Media and cultural studies specialist Lisa Blackman (2012) has argued that what lays in the background of concerns about neuroscientific models of psychopathology is a history in which, for the most part, psychologists and neuroscientists consigned threshold experiences and "immaterial process" to the category of pathology or viewed them as irrational (Blackman 2012, see chaps. 7 and 8). For Blackman, though, this does not necessarily reflect an attempt to dismiss the meaning or intentionality associated with these experiences. Indeed, iterative reinterpretations of experience have allowed assumptions about volition, consciousness, and intention to be actualized and transformed in different ways (Blackman 2014). The neuroscience and epigenetics research we discuss in this article, then, might be perceived not as necessarily implying the absence of a relationship between "the objects or situations that trigger them" and psychological subjects "that elicit them" (Frank and Wilson 2012, 874-75), but rather as providing an historically particular way of understanding the relations between affects and life. From such a perspective, these etiological explanations might be seen as an addition to rather than an erasure of preexisting and complementary understandings of experiences and their effects.

Indeed, at other research units and in parallel research activities at the MGSS, researchers draw attention to the effects of personal experiences across the lifespan (Nemeroff 2016; Provençal and Binder 2015; Roberts et al. 2018).[20] These models speak to flexible and complex timelines of the development of specific forms of subjectivity that might lead to suicide risk. This research intersects with broader neuroscientific visions of life concerned with the ways in which personal experiences articulate with events and traces of the past, while also being a part of a person's future and ongoing day-to-day life (Blackman 2014, 7; Lloyd and Larivée Forthcoming). In these models, clear boundaries between "pre" and "post" significant etiological experiences, for example, become difficult to identify and render unproductive attempts to either limit or rescue intentionality from one of these two poles. From this perspective, an approach that only focuses on "pre" or "post-abuse" would be a limited conception of life experiences, reproducing the idea that there might be a developmental point before which biology is uncompromised or brought under the sway of affects. Consequently, rather than trying to identify a point at which people – materially and experientially – might be considered to have a pure and unadulterated form of intentionality, this approach instead considers all subjectivities 'in the post' and as possible sites of entanglements (Wilson 2015, 39, 164). Interest in characterizing and understanding *life as post*, in which life is seen as shaped by early traumatic events, is, then, not only a concern of contemporary neuroscientists, but also of social science and humanities researchers who attempt to understand and document the experiences of people in conditions ranging from conflict or post-conflict environments to natural or human instigated disasters or persistent toxic exposures and the effects of these experiences on their lives and those of their families or descendants (Lester 2013; Kirmayer et al. 2010; Moghnieh 2017; Roberts 2017; Yamaguchi 2018). These accounts depict intentionality or volition as fluid states always anchored in personal trajectories within specific environments eschewing explanations that permit experiences at one moment in life (i.e., childhood) casting all others into the etiological and experiential shadows. This leaves a fertile space within which one might be able to consider suicide as both an act with a significant precipitating biochemical state and a significant precipitating event.

[20]Parallel to this brain-based epigenetic research, a subset of MGSS clinical and developmental researchers focus, for example, on identifying multiple, specific types of childhood adversities, such as neglect and peer victimization (Geoffroy et al. 2018; 2016) or unmet health services needs during childhood and adolescence (Renaud et al. 2014; 2009), as distinct predictors of suicidality later in life.

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