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Epistemic and Temporal Disjunctions: (Re)Mapping "Suicide Risk" Epigenetics Through Birth Cohorts

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The McGill Group for Suicide Studies (MGSS) has garnered significant attention for its epigenetic models of suicide risk. These models suggest that early life adversity may set people on pathways of neurobiological vulnerability and, ultimately, suicide risk, which are correlated with distinctive epigenetic traits. While the core of this epigenetic and neuroscientific research is carried out on the donated brains of people who have died by suicide, some MGSS researchers explore pathways to suicide risk through other means, including clinical and birth cohort research. These clinician-researchers pair psychiatric epidemiology with clinical studies of teenagers to investigate the links between socio-environmental/familial indicators (from low birth weight and socio-economic status [SES] to parental mental illness) and peer victimization (e.g. cyberbullying) as factors in the development of neuropsychiatric risk and mental distress.

One of the birth cohorts they study is the Quebec-based QLSCD (Quebec Longitudinal Study of Child Development), a longitudinal study of people born in 1998 with the motto "I am, I'll be: The survey on the future of a generation." Attention to this cohort research highlights epistemic and temporal disjunctions in the characterization of suicide risk within the multiple research programmes at the MGSS: the places (home/school), people (family/peers), timelines (early years/early teens), and the data sources (clinical/epidemiological) correlated with neurobiological vulnerability and suicide risk differ in substantial ways. Mapping prospective and retrospective research across these disjunctions raises critical questions about the nature(s) and pathways of neuropsychiatric risk and vulnerability. Analysing cohorts as a specific "technology of the biosocial," we examine how suicide risk is reconfigured as an object of research with distinct traits from those found in epigenetics research shifting the focus from "I was" to "I am, I'll be."

In this piece, we explore two central questions in the epigenetics of neurobiological vulnerability and suicide risk: *What is a "suicide completer" vs. a "potential suicide completer," as studied in retrospective environmental epigenetics research vs prospective cohort research, respectively? What characterizes the lives and deaths of the people within these two research agendas?* In doing so, we explore two interrelated angles on risk within environmental epigenetics versus developmental and longitudinal cohort research: the times and spans of neurobiological and suicide risk and the spaces, both epistemic and material, of risk.

The lifespan of neurobiological risk

At the core of MGSS environmental epigenetics research, which has been the focus of our longitudinal multi-study project since 2013, is the argument that early childhood adversity (ECA) is associated with structural and functional changes of the brain that are considered to alter behavioural development and increase the vulnerability to psychopathology via epigenetic (dys)regulation (Lutz et al., 2017; Turecki 2016). This research, carried out on the frozen brain tissue of people who have died by suicide, focuses on and portrays a very specific form of vulnerability in which "traits" are seen to be impressed on a young person's brain. This early canalization is believed to set youth on a trajectory of negative mental health

and psychosocial outcomes, and disadvantageous decision-making (Jollant et al. 2010). As we have argued elsewhere, what is striking about the research at the MGSS "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 programmes 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, behaviours, and risks" (Lloyd and Larivée forthcoming). The model proposed by the MGSS is thus consistent with broader narratives in neuroscience research of early childhood brain plasticity (Champagne 2010 McEwen & Morrison 2013). These "plastic" understandings (Meloni 2019) of the relationships between life experiences and psychopathology have become anchored in research agendas ranging from studies of the developmental origins of health and disease (DOHaD) to "trauma-informed care" (Edwards, Gillies and White 2019). Within these agendas, the lifespan takes centre stage as the core analytic to conceptualize how experiences at different points in the lifespan shape people's life trajectories (Lappé and Landecker 2015; Pentecost and Meloni 2020).

The environmental epigenetics research at the MGSS is carried out *retrospectively*. As a result, researchers have limited entry points – coroners' reports, history of pharmaceutical use, and psychological autopsies – to gain an understanding of the person who has died by suicide. Among these tools, psychological autopsies provide the greatest insights into the lives of people lost to suicide. In these autopsies, MGSS researchers use the proxy-based interviews questionnaire, the "Childhood Experience of Care and Abuse" (CECA), to assess the adversity experienced by a person based on reported experiences (Bifulco et al., 1994). The results determine where a person's profile fits in the MGSS's typology of "suicide with or without abuse." This retrospective epigenetics research portrays suicide later in life as the result of these early experiences of adversity, with intervening life experiences and precipitating events eclipsed in terms of their etiological weight (Lloyd and Larivée, forthcoming).

The parallel clinical and developmental research at the MGSS draws on *prospective and longitudinal* experimental designs such as cohort studies and longitudinal research (Geoffroy et al., 2018a; 2016; Séguin et al., 2014; 2011) to study suicide risk. This research focuses on identifying multiple, specific types of childhood and adolescent adversities, such as neglect and peer victimization, as distinct predictors of suicidality later in life, as well as mapping additional risk variables, such as the frequency and severity of abuse, types of abuse, and the relationship between the victim and the abuser as potential moderators of psychopathology. As a whole, these studies are interested in the potential cumulative and interactive effects of life events that are considered specific to youth and young adult suicide trajectories (Séguin et al., 2014, 2011).

Central to this lifespan approach (c.f. Lappé and Landecker 2015) is an effort to reconstruct and illuminate the sequence of difficulties experienced between early life (e.g., physical and sexual abuse, neglect and tension) and other variables emerging during teenage years (e.g., conduct and behavioural difficulties, social and school difficulties, etc.) or those emerging during young adulthood while also mapping differential *suicide profiles* across detailed biographical encounter (Seguin et al., 2014; 2011). In the move from "variable-oriented" to "person-oriented" research (Séguin et al., 2014: 124), the narrative methodology demands more than just simple facts and isolated attributes, but requires thinking "in terms of conditional and cumulative probabilities and contextual factors" (*ibid*. 125). Overall, these studies aim to identify risk and resilience factors involved in suicidality among youth and in mental health outcomes (Geoffroy et al., 2018a; 2018b; Orri et al., 2018; Perret et al., 2020); in other words, the dynamics that move people onto and off of trajectories of neurobiological and suicide risk. Among the MGSS's psychiatric epidemiology research are studies of multiple cohorts, with a focus on distinct suicide risk factors ranging from birth-related factors (e.g., low birth weight, young maternal age at birth; Geoffroy et al., 2014) to ongoing (or more recent) factors in the longitudinal cohorts (e.g., bullying or cyberbullying; Geoffroy et al., 2016; Perret et al. 2020). These are population-based cohorts who are not tracked for a

specific profile; rather they are monitored for changing situations, behaviours, and attributes (e.g., psycho-social, developmental) at different points of their lifetimes. Researchers who study the cohorts thus claim "to observe subjects rather than influence them through exposure to certain influences or research variables" (Lamoreaux 2016: 199).

For our present argument, then, we are less interested in a specific identity (e.g., biological citizenship [Aarden 2018]) within the group and more interested in the specific forms of knowledge that are produced through cohort research, as a "technology of the biosocial" (Gibbon and Pentecost 2019). Emerging from these longitudinal studies is a particular *image of* the identity of "potential suicide completers" whereby cohort data is enrolled to supplement, though not always mapping easily onto, the profile of the suicide completer in brain-based epigenetics research. That the cohort data does not necessarily map onto the suicide completer profile demonstrates the challenges posed in "lifespan" research, as researchers attempt to track how a multitude of shifting developmental traits (as measured by psychological and biological studies) interact with one another.

Time, space, and at-risk subjectivities

The lifespan, then, is read through multiple lenses at the MGSS: according to the type/nature of study, different time ranges/points and different spaces are framed as potentially critical junctures in a person's life. Through these studies, a picture emerges of complex interactions between and accumulations of traumas over the lifespan as well as across spaces. While in epigenetics research, the predominant focus is on family (or early intimate) experiences, in cohort and clinical research the scope is broadened to include educational and social spaces in which youth circulate and are potentially exposed to other forms of adversity.

In order to develop a portrait of these temporal and spatial landscapes of neurobiological vulnerability, the psychiatric epidemiology research carried out at the MGSS, and specifically their analyses of cohort studies, consider a wide variety of risk factors identified over multiple time points and within multiple environments. For instance, through assessment of data from the 1958 British Birth Cohort (i.e., the National Child Development Study [NCDS]), a group of MGSS researchers has identified prenatal risk factors (including higher birth order, low birth weight, and young maternal age), developmental risk factors (such as externalizing behaviours in males), and the number of childhood adversities as associated with suicide later in life. Based on these factors, MGSS researchers make a case for a *gradient of suicide risk*. While each of these factors are thought to increase risk of suicide later in life, and to imply different "hazard ratios" and co-factor interactions some stood apart (such as prenatal factors) as "unaffected" risk factors that might operate through relatively independent pathways (Geoffroy et al., 2014: 1250).

Moreover, some factors were deemed particularly salient in the development of suicide risk, such as multiple experiences of adversity early in life (i.e., before the age of 7) and externalizing problems among male participants. These factors are described in terms of their population attributable risk fraction, which refers to the proportion of suicides in the study that the authors believe could be prevented if these factors were abolished (Geoffroy et al., 2014: 1247). These findings lead the authors to conclude that while "[s]uicide is often considered to be caused by mental disorder and adverse events such as job loss and relationship difficulties around the time of death, (…) trajectories leading to suicide in adulthood have roots in early life" (Geoffroy et al., 2014: 1250). Thus, consistent with findings from other Adverse Childhood Experiences (ACEs) studies (Dube et al., 2001; Anda et al., 2006; Chapman et al., 2007), the authors identify "a dose-response association between the number of emotional adversities and suicide, with the highest suicide risks among those experiencing three or more adverse experiences, independently of other factors examined" (*ibid*.: 10). These factors are believed to have concrete effects, with cumulative experiences appearing associated not only directly with suicide but with the development of internalizing disorders (i.e., anxiety/depressive disorders) and with suicidal ideation at age 45. This association suggests that "adult internalizing disorders may be on the pathway linking some (though not all) early-life factors with suicide mortality" (Geoffroy et al., 2018b: 122).

Beyond the early years, adolescents and adolescent risk factors are of keen interest within lifespan research— and at the centre of both MGSS clinical care and their psychiatric epidemiological research. In terms of the latter, the Quebec-based birth cohort, QLSCD, which is a cohort of 2120 people born in 1997-1998, has been studied by MGSS and MGSS-affiliated researchers. While the initial interest of the QLSCD was child development, social adjustment, and academic performance, its scope has been considerably broadened over the years, in some respects shifting with the age of cohort members and, in other respects, reflecting the interests of researchers who study the cohort, scientific committees, and the priorities of funding agencies. For example, MGSS researchers are interested in the contribution of childhood irritability and depressive/anxious mood symptoms to suicide risk, developing "multi-trajectory models" that trace the longitudinal-developmental course of suicidality in relation to these personality traits and experiences in different settings, at different points in time (see Orri et al., 2018: 469). By focusing on experiences of peer-victimization or bullying (Geoffroy et al., 2018a; 2016), the MGSS research focus shifts to public and educational spaces, wherein the chronicity and severity of at-school experiences/exposures are described as pivotal to suicide risk. In a recent paper (Geoffroy et al., 2018a) that compares three trajectories of peer-victimization – low, moderate, and severe from ages 6 to 13 years – MGSS researchers report that adolescents who were more severely victimized *throughout* their school journey are "at greater chance of suicidality in adolescence than less severely victimized children, even accounting for a plethora of confounders assessed throughout childhood" (Geoffroy et al., 2018a: 41). The notion of being (or becoming) at-risk implies, in this purview, a particular neurobiological profile of suicidality that is said to be modulated by a set of adverse experiences and exacerbated, later in life, by continuous exposures that may manifest in the form of mood disorders, school problems, and negative personality traits.

From this very perspective, adversity, vulnerability, and neurobiological risk are compounded by critical junctures and transitions over the lifespan (e.g., from early childhood to the school-age and into adulthood), which are context-dependent. Being "at-risk" is therefore contingent on experienced *spaces of risk and life circumstances* that are intersubjective, spatiotemporally situated, and epigenetically transmissible. As critical social research has suggested, moreover, "being at-risk" intersects notions of (un)healthy trajectories, developmental risk, and cognitive potential (Meyers 2013, Filipe 2015; Gillies et al. 2016) that inhabit very different epistemic spaces. Given this, multiple and sometimes even competing models of trajectories of neurobiological risk trajectories emerge, which have to be rendered coherent by researchers within the same group endeavour to render their findings coherent with one another. The ways in which these constructs and epistemic spaces of risk can be connected is through a lifespan perspective on neurobiological risk that is instrumental to global DOHaD research and local environmental epigenetics — a spatio-temporal perspective that has been "made relevant", moreover, through the biosocial technologies and methodologies of longitudinal, cohort research. This research perspective thus calls for the theorization of biosocial research methodologies and of epistemic spaces that are jointly inhabited by epigenetics and cohort research, as well as the reconceptualization of what environmental transmission (c.f. Roberts and Sanz 2017) means and how it "gets done" in that space.

Epistemic and temporal disjunctions

Thus three main discrepancies emerge from an analysis of prospective and retrospective models of "potential suicide completers and/or ideators" and "suicide completers" in terms of how these people's lives are perceived. ***First***, the retrospective prospective vs MGSS researchagendas vary in terms of *their interest in temporal qualities of adversity,* with the *timing* of early events during critical windows of neuroplasticity seen as most significant) v*s. a dose-dependent approach,* in which the effects of stress and negative experiences are seen as *cumulative over the lifespan*. ***Second***, they also focus on *different aspects of a person's life*: depression and irritability in prospective research as opposed to impulsive-aggressive research in retrospective research (with the assumption that the former transitions to the latter throughout the lifespan). ***Third***, a focus on *different types of life*

experiences perpetrated by *different groups of people:* in prospective research the attention is on early adversity and neglect by close family members as well as peer victimization and bullying during adolescence, whereas in retrospective research, attention is oriented toward early adversity typically within the familial context. Added together, these differences point to *a disjunction between epistemic approaches and temporal foci* on suicide risk in brain tissue-based epigenetics vis-a-vis psychiatric cohort research agendas.

In *both* agendas, however, researching suicide risk comes down to profiling "who" and mapping "why": that is, who will (or who might) commit suicide and why. The cohort research and the environmental epigenetics research – seen as different tools of the biosocial – offer different, yet non-mutually exclusive, answers. First of all, tensions and apparent contradictions between findings (e.g., about the role of early adversity) are circumvented through synecdochic reasoning in which both sets of findings about the effects of negative experiences, albeit at different points in the life span, are seen to stand in for an as-yet not fully understood neuropathophysiological process (see also Young 2001). As such, both research agendas are able to imply that life experiences, even different ones, (translated into biomarkers) explain the "why." The narratives of the two research agendas diverge to some extent when the question of "who" is posed. In environmental epigenetics research, anyone can be seen as becoming vulnerable and tracked toward becoming a "suicide completer" given specific environmental risk factors. Birth cohort studies map a different set of biosocial experiences, in which the "who" is characterized by an essential risk factor (genetic or epigenetic) that establishes vulnerability but which must be modulated by a second "dose" of negative experiences, such as bullying, later in life.

To these considerations of "who" and "why", we would like to consider their framing of "when." Through environmental epigenetics research, the suicide completer is seen as someone who reacts poorly to stress and whose experiences immediately surrounding suicide attempts — that is, precipitating events — are considered unexceptional. "When" was always going to arrive for this group people; suicide was the foregone extreme conclusion of their trajectories. By contrast, in cohort research, there is space for considering sources of "vulnerability" and their effects later in life, alongside factors that might lead to "resilience" (with all the complex and problematic issues to be assessed related to the use of these terms). There remains space for "if" in the models of these people's lives. So, if the people characterized in the two bodies of research are considered to start from the same point of shared vulnerability, for one group this vulnerability is considered enough, it is essential, whereas for the other group vulnerability brought on by early adversity is seen as only one point on a still-unsettled trajectory – a finding that renders conditional the QLSCD motto: from "I am, I'll be" to I am, I might be(come).

"Being at-risk": from image to practice and experience

Mapping prospective and retrospective research across these spatio-temporal and epistemic disjunctions raises critical questions about the presumed nature(s) and multiple pathways of suicide risk. Overall, these two research agendas (i.e., epigenetic vs. clinical and cohort research) share the same research aims and foci yet set forth different approaches to (i) the *mapping the profiles, nature, and meaning of suicide risk* and (ii) the *remapping of its dynamic interplay with "resilience" to early adversity, exposure, and abuse*. Both speak to the dynamics of acquired risk; yet environmental epigenetic studies of neurobiological risk tend to focus on "essential risk" and how people stay on a track of adversity leading to suicide, whereas cohort and clinical research turn attention to "gradients of risk" and "resilience windows" into consideration, which trace how and at what points in time people not only get "in" to but also "out" of that presumed suicidal trajectory.

As is well-documented in other clinical and anthropological studies, for example, of PTSD, and Alzheimer's (Young 2001; Serafini et al., 2016; Lock 2013), the image of neurobiological risk emerging from epigenetic (dys)regulation holds explanatory power for only a very small percentage of the population in which the "extreme end point" of epigenetic and mental ill-health vulnerability takes shape, e.g. in the form of suicidality. Alternatively,

biomarkers for risk as found in epigenetics and developmental psychiatry could become stronger predictors of future risk as they incorporate a lifespan perspective and new social constructs derived from longitudinal cohort research. Yet the literature suggests that such markers may continue to be misleading and retain low predictive value due to the application of group data to transitioning population subgroups (e.g., adolescents and young adults) in which certain traits may or may not manifest (c.f., Singh and Rose 2009, Patton 2009, Sawyer et al. 2018). While this suggestion has important conceptual and methodological implications for how we frame and approach the lifespan of neurobiological risk and suicidality, it raises substantive ethical questions as to (i) how scientific images of "being at-risk" of suicide are enacted and experienced by youth; and (ii) whether they may contribute to the development of *negative* risk subjectivities and identities that exacerbate vulnerability (Rose 2010, Gillies et al., 2016, Pitts-Taylor 2019).

For all these reasons, the intersections between environmental epigenetics and longitudinal cohorts may increase the relevance of both kinds of research to contemporary models of suicide and neurobiological risk, yet may fall short from addressing their broader ecosocial dimensions (i.e., the structural conditions that set people on so-called pathways of risk and/or trajectories of resilience). Equally, questions arise as to whether this form of biosocial research can guide innovative and effective public health interventions in the field and what are the future implications, as conventional health prevention and promotion strategies shift toward risk reduction and early intervention. By highlighting these intersections as well as substantial disjunctions between methodologies and technologies of the biosocial, we can better understand the multiple ways in which these complementary agendas are mapping, modelling, and re-envisaging the lives and afterlives of neuropsychiatric risk.

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This article is part of the series: Excavating and (re)creating the biosocial: Birth cohorts as ethnographic object.