

Natali Valdez

Center for the Study of Women's Gender and Sexuality, Rice University
Department of Women's and Gender Studies, Wellesley College
(E-mail: nvaldez919@gmail.com)

The Redistribution of Reproductive Responsibility: On the Epigenetics of "Environment" in Prenatal Interventions

The rapidly shifting field of epigenetics has expanded scientific understanding of how environmental conditions affect gene expression and development. This article focuses on two ongoing clinical trials—one in the United States and one in the United Kingdom—that have used epigenetics as the conceptual basis for testing the relationship between nutrition and obesity during pregnancy. Drawing on ethnographic research, I highlight the different ways that clinical scientists interpret epigenetics to target particular domains of the environment for prenatal intervention. Here I examine three environmental domains: the pregnant body, the home, and everyday experiences. In so doing, I show how different scientific approaches to epigenetics multiply concepts of "the environment," while also individualizing responsibility onto pregnant bodies. Ultimately, I argue that how the environment is conceptualized in epigenetics is both a scientific and a political project that opens up questions of reproductive responsibility. [epigenetics, environments, prenatal interventions, clinical trials]

Last spring, I gave a presentation at a workshop on how epigenetic science is a powerful tool for changing prenatal and maternal health policy—especially with regard to obesity during pregnancy. After my presentation, Anne, a mother of two in her mid-40s, approached me with a question. Anne told me that her first pregnancy was traumatic because of the fear and anxiety around her weight. While she was pregnant, the doctors kept telling her that she was gaining too much weight. In an effort to address the doctors' concerns, Anne monitored her weight, walked every day, and kept a strict diet. Anne gave birth to a healthy eight-pound baby. Fast-forward 17 years. Anne's first-born is struggling with her own weight. "She just eats sugar," Anne explained. "She cannot stop eating sugar." The mother then wondered out loud: "Did I do this to her? Is my daughter's insatiable desire for sugar and her weight gain a consequence of my own anxieties about sugar and my weight gain during pregnancy?"

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Anne's question reflects an interpretation of epigenetics that correlates her own eating and behavior during pregnancy with future health outcomes. Her feelings of responsibility and guilt for having "done this to her daughter" are connected to a historical, political, and scientific framework that targets women's bodies and behaviors as the only ones responsible for the health and development of their children (Ginsburg and Rapp 1991; Rapp 1999; Riessman 1983). In my response to Anne, I took care to emphasize that the potential epigenetic effects on fetal development during gestation are influenced by many different and *unpredictable* environmental factors such as toxic exposure, food production processes, past trauma, and poverty (Guthman and Mansfield 2012). I also stressed that the influence of one scale of the environment—such as the uterine environment—compared to another is still not well understood.

In my research, I found that pregnant women were increasingly targeted for nutritional interventions in the name of obesity and diabetes prevention efforts. To understand how pregnant bodies became environmental sites for intervention in the postgenomic era, I examined clinical trials that used epigenetics as the conceptual basis for testing nutritional interventions on ethnically diverse pregnant women who were deemed obese. In the case of obesity during pregnancy, epigenetic science bridges the decisions that women make about their weight and diet with the future health of their children and subsequent generations. Although epigenetic science claims that women who are obese during pregnancy have a higher risk of having children that are obese and diabetic (Barker 1992; Barker et al. 1993; Gillman et al. 2008), other interpretations of epigenetics also make clear that the risk is contingent and unpredictable (Niewöhner 2011). In what follows, I argue that the different biomedical frameworks used to translate epigenetics into prenatal clinical trials create certain environmental targets and interventions, which has consequences for the (re)distribution of reproductive responsibility.

The "Environment" in Epigenetics

As a result of the growing scientific and public attention around epigenetics (Meloni and Testa 2014), the environment has reemerged as a significant factor in genetic development and inheritance. I say reemerged because the role of the environment in genetic development has had a capricious history that can be traced in and out of evolutionary science (Jablonka and Lamb 1995, 2005). Presently, epigenetic science, as it appears in what is often referred to as a "postgenomic paradigm," reflects the renewed focus on studying the environment as an active agent in genetic regulation and expression (Rapp 2011; Richardson and Stevens 2015).¹

Broadly defined, epigenetics refers to the study of gene-environment interaction and genetic expression. However, the definition of epigenetics has changed over time. When Conrad Waddington first coined the term epigenetics in the mid-1900s, it was derived from the term epigenesis, or the process of development through differentiation (Holliday 2006; Hurd 2010; Szyf 2009). Waddington explained that epigenetics was the study of the interaction of genes and epigenesis (Waddington 1956). In current understandings, epigenetics can include a variety of mechanisms involved in genetic programming such as DNA methylation, histone modification, and noncoding RNAs, as well as the regulation of chromatin structure and gene

expression (Bird 2002; Feinberg 2007). In addition, epigenetics can also include genome–environment interactions, and epigene–environment interactions (Lappe and Landecker 2015). In what follows I use the general term epigenetics or “epigenetic science” because the trials that I observed did not specify any particular subfield of epigenetics, but still engaged with ideas from nutritional, molecular, and environmental epigenetics as well as developmental origins of health and disease (DOHaD). This ambiguity reflects the unstable boundaries in the (sub)disciplinary formations within epigenetic science and epigenetics as a way of thinking. In describing the contours of epigenetics as a phenomenon, I also risk using the term as if it is a stable coherent thing.

In my analytical approach, I situate ideas of the environment in epigenetics from within science and technology studies (STS). STS research on epigenetics shows how concepts of exposure, scale, space–time, and molecularization animate the environment (Pickersgill et al. 2013). In current epigenetic literature, the environment encompasses that which is both inside and outside the body. The environment is at once *exposure* from inside the body, including cells, organs, as well as the microbiome and *exposure* from outside the body, including the natural built environments (Landecker 2011; Landecker and Panofsky 2013). Importantly, though, epigenetic theories do not necessarily imply a division between inside and outside; rather, epigenetics emphasizes that the environment occurs on different *scales*, from the cellular level all the way to the atmospheric level (Shostak and Moinester 2015). Further, Niewöhner’s work highlights how environments in epigenetic research are “spatio-temporal contexts” (2011, 290). For instance, Lappe’s work on autism highlights how the timing of environmental stimulation is a key component to understanding epigenetic modifications (2014).

Another key theme across STS literature on epigenetic science is the molecularization of the environment. By interviewing scientists who work on gene–environment research, Darling et al. (2016, 58) found that scientists conceived of the environment as “anything non-genetic.” In the context of environmental health research, Shostak (2013) highlights how the molecularization of the environment dramatically shifted understandings of exposure to the molecular level and increased dependence on biomarker technology. In Alzheimer research, Lock (2013) shows how the prioritization of the molecular scale through the intense focus on biomarkers reflects a reductive approach in epigenetic science. Biomarkers enable the molecularization of the environment, and they can obscure an individual’s cultural or personal experience (Abu El-Haj 2007). Overall, the molecularization of the environment enables the individualization of environmental impacts.

In my interpretation, what counts as the environment is also directly related to the biomedical frameworks applied in measuring and tracing environmental factors to changes in genetic expression. Different approaches to epigenetics will focus on distinct areas of the environment. For a neuroscientist who studies epigenetics and brain plasticity, the environment or environmental stimuli can include daily experiences (Szyf 2009; Weaver et al. 2006; Yehuda et al. 2016). For a molecular biologist, environmental stimuli can refer to exogenous material like methyl groups that attach to DNA sequences (Darling et al. 2016). Moreover, for a scientist working at the National Institute of Environmental Health Sciences, environmental agents are defined as “mold, pesticides, air pollution and some foods and medications”

(NIEHS 2017). The various ways that environmental targets are rendered visible and intervenable in the application of epigenetics also creates certain forms of responsibility and not others.

Responsible Environments

From my reading of the STS literature, environments are multiple, porous, scalar and spatio-temporal, but in practice this proves difficult to methodologically study in randomized clinical trials. For instance, as I explain below, the maternal environment is not isolated from home environments or toxic atmospheric environments, yet the maternal environment is studied as if it were a controlled and contained environment. In the pregnancy trials I studied, participants lived within environments of stress, pollution, violence, and racism—social phenomena that are hard to control for in a randomized clinical trial. More significantly, the hyper focus on maternal environments justifies targeting women's bodies and behaviors for interventions as if they were the only environments responsible for adverse health outcomes in future generations (Maher et al. 2010; Richardson et al. 2014; Warin et al. 2012).

Despite the complex role of environmental factors influencing health outcomes, scientific discourses often collapse the whole environment to the maternal environment (Kenney and Müller 2016) and target the maternal environment as the first source of environmental exposure for developing fetuses (Singh 2012). Richardson's work underscores how women's bodies increasingly become what she calls "maternal vectors" or "an intensified space for the introduction of epigenetic perturbations in development" (2015, 221). In South Africa, Manderson shows how the increased focus on women as "foetal containers" reinforces the idea that women are "vehicles of poor intergenerational health" (2016, 154). In my own observations and interviews with scientists, I found that pregnant women were framed as "windows into the health of future generations." Pregnant women with a high body mass index (BMI) are also framed as having "risky wombs or intrauterine environments" (McPhail et al. 2016). Further, within the postgenomic paradigm, the individualized responsibility, blaming, and risk placed on pregnant women and mothers is also shaped by race and class (Mansfield and Guthman 2015; Saldaña-Tejeda Forthcoming).

The framing of women's bodies as responsible and intervenable environments, in the name of disease prevention for future generations, is not new. Feminist historians and anthropologists have traced the emergence of population health and eugenics to state-endorsed projects that targeted pregnant bodies and mothers as ways to maintain the social and biological (re)production of future citizens and soldiers (Davin 1978; Ginsburg and Rapp 1991; Stoler 1995). Feminist scholars of color bring crucial attention to how the practices of targeting women's bodies for health interventions have unequally focused on brown and black bodies (Bridges 2011; Gutiérrez 2008; López 2008; Mullings and Wali 2001; Roberts 1997). In addition, scholars guided by reproductive and environmental justice paradigms expose how concepts of the environment are inextricable from race and reproduction (Sasser 2013).

My intervention into the social and theoretical examination of epigenetics focuses on using ethnographic data to illustrate the effects of epigenetic frameworks on

pregnant women. Whereas much of the literature has focused on discursive analysis of scientific media, my work explores the clinical translation of epigenetics in the design of prenatal interventions, on the ground. Conceptually, my approach attends to how women's bodies (especially brown and black bodies), have a long history of being marginalized while remaining at the center of certain scientific enterprises (Benjamin 2013; Landecker 2000; Tallbear 2013). My aim is to spotlight the ways in which epigenetic knowledge and practice can both reinscribe older prescriptive models of genetics and race (Abu El-Haj 2007; Mansfield and Guthman 2015), *and* provide an entry point for challenging those same models. Epigenetics has the potential to help us reconceptualize what counts as the maternal environment and responsibility. However, as I show below, this opportunity is restricted by the experimental designs of current studies. Finally, my approach to epigenetics is one that foregrounds what happens when multiple and intersecting scales or domains of the environment are in play. I argue that rather than talking about epigenetics as a monolith, we thus might talk about a collection of epigenetic adaptations, which emerge from different disciplinary approaches and interpretations. Like epigenetics itself, *epigenetic adaptations* refers to the adaptations of epigenetic knowledge that emerge from the various translations and experiences of epigenetic theories *in situ*.

This article, based on ethnographic fieldwork and data collection, explores the clinical experience of the environment—specifically, how different scientific approaches and interpretations of epigenetics turn particular domains of the environment into targets for intervention. Here I focus on three domains: pregnant bodies as environment, home as environment, and everyday experiences as environment. The flexible definitions of the environment are significant in the clinical translation of epigenetics because what counts as the environment influences the sites and types of interventions tested in evidence-based medicine. Moreover, the determination of what constitutes a risky environment (requiring intervention) generates material and bodily consequences for pregnant women.

Clinical Trials on the Maternal Environment

Between 2012 and 2014, I collected ethnographic data from two sites: the SmartStart trial, in the United States, and the StandUP trial, in the United Kingdom. The SmartStart trial was funded by a multi-million dollar grant from the National Institutes of Health (NIH) and it is one of seven trials in a larger consortium. In the United Kingdom, the StandUP trial was a multi-million pound project funded by the European Union and the National Institute of Health Research that included five different sites across the United Kingdom. Although both clinical trials are funded by government organizations and are publicly available to research online, I have nonetheless changed the names of all people and clinical trial sites.

There are currently more clinical trials testing behavioral interventions of diet and exercise on pregnant women than ever before (Frew et al. 2014). In the United States alone, the NIH are funding more than 100 trials that test prenatal dietary interventions (NIH reporter 2014). The trials that I examine are framed as behavioral clinical trials that analyze the effectiveness of nutritional interventions. The key difference in the nutritional interventions is that the U.S. trial focused on gestational weight gain, and the U.K. trial did not. Instead, the U.K. intervention focused

on providing educational tools and goals to help women eat a low glycemic diet. The U.K. intervention emphasized that it was not a diet for weight loss and did not focus on weighing or counting calories; whereas, the intervention in the United States focused entirely on calorie consumption and weight control.

The different approaches in the interventions reflect the distinct national health policies on maternal nutrition held in the United States and the United Kingdom. The Institute of Medicine (IOM) in the United States recommends that women who have a high BMI should not gain any weight during their pregnancy, or should limit their weight gain to half a pound per week during gestation (IOM 2009). However, the National Institute for Health and Care Excellence (NICE) in the United Kingdom claims that gestational weight gain is not a reliable indicator for healthy pregnancies, and they do not promote weighing women during pregnancy nor do they provide any weight recommendations for pregnant women (NICE 2010).

As a multi-sited ethnography, my work emphasized the connections across different spaces, networks, and actors (Marcus 1998). Both trials stand as international case studies and reflect the limits of standardization and universalization in scientific knowledge production. The key differences to note here between the U.S. and U.K. sites have to do with the different national health care systems and policies, the different ethnic populations that were targeted, and the different scientific expertise of the principal investigators at each trial. For instance, since the United Kingdom has the national health care system (NHS) that integrates, research, care, and medical training, the clinical trial in the United Kingdom was centrally run from within a NHS teaching hospital, which delivered over 2,000 babies annually. The other participating sites were also connected to local hospitals. In addition, all of the data at each site were sent back to the central location for analysis. In contrast, the structure, organization, and recruitment of the U.S. consortium was dispersed across different hospitals, clinics, and research institutions. At the site I observed, it was challenging to recruit pregnant women because they had to ask permission from individual private and public clinics to recruit their pregnant patients.

For data collection, I focused on examining the perspectives of each trial's designers and participants. To illuminate the trials from the perspective of the research participants, I followed the pregnant subjects through their clinical trial journey. I observed the recruitment process, consent, data collection, and the implementation of each intervention. To understand the design and implementation of the trial from the staff's perspective, I interviewed individual staff members and collaborators for both sites, including research assistants, midwives, consultants, health trainers, and principal investigators. Those responsible for designing and monitoring the trial implementation hailed from a range of scientific disciplines including physiology, psychology, and midwifery.

At the SmartStart trial, I participated as a volunteer staff member. Since I had previous experience working on nutritional interventions and had an advanced degree, I was trained as an interventionist. I recruited participants, collected bio-specimen and survey data, and delivered the intervention to the women in the treatment group. As both ethnographer and staff member on the SmartStart trial, I gained first-hand understanding of the pregnant participants' lives, challenges, and their social conditions. As a feminist ethnographer, my analytical approach and methods are intended to reveal the taken-for-granted and often obscured roles of

race, gender, and power in the production of scientific knowledge (Benjamin 2013; Franklin 1995; Traweek 1988).

Pregnant Bodies as Environments

One of the first ways that environments came into view in the studies I observed could be seen in the animal models used as a basis for the clinical trials. The principal investigator at the StandUP trial, Dr. Smith, a physiologist by training, explained at a conference:

We do a lot of our work in animal models. We go backwards and forwards between animal models and the clinic. And these models are incredibly important to us. For our animal models we give rats and mice absolutely delicious things to eat, and then they get fat and then we make them pregnant, and this is a good model of obstetric obesity. And we've been looking at the *children* when they grow up, or the offspring of these rats and mice, and it's extraordinary. They have very high blood pressure, they become fatter, and they have abnormal glucose control. Therefore, we believe that the fetal development is very susceptible to the *maternal environment* and that it predisposes children to disease as they are exposed to these metabolic conditions in utero (emphasis added).

Dr. Smith cites her published research to argue that animal models provide evidence for the adverse effects of maternal obesity on *human* offspring. The use of animal models to understand environmental epigenetics in humans is common practice. For instance, Michael Meany and Moshe Szyf, geneticists at McGill University, use animal models to make the case that “maternal care” can influence fetal programming (Weaver et al. 2004). By showing how the PI uses animal models at the StandUP trial, I emphasize how her methods and expertise shape her understanding of environmental targets for prenatal interventions in humans. It is through the animal models that certain environmental targets are constructed as important in the design and implementation of the prenatal interventions. As a physiologist interested in metabolic disorders, the PI focused her attention on how the maternal metabolic environments of the rats could affect the development of fetal rats.

In Dr. Smith's rat models, maternal and metabolic environments are prioritized while other aspects of the environment disappear. What about the temperature of the laboratory, or how scientists handle and force-feed the rats? Those aspects are also a part of the maternal rat environment and the rat fetus's environment. The underlying assumptions involved in translating animal models to human interventions, or assuming the standardization of animal environments in laboratory settings is a classic concern in STS (Freccero 2011; Haraway 1997; Thompson 2013; Wolf 2010). For instance, Kenney and Müller point out that the animal model by Meany and Szyf (Weaver et al. 2004), mentioned above, focused entirely on motherly behavior (characterized as licking and grooming), which “black-box[es]” other aspects of the environment like the “cage, food, the other rat pups” (Kenney and Müller 2016, 31).

By ethnographically examining the clinical translation of the rat models to human interventions, I find a similar set of insights. For instance, like the rat models, other aspects of the women's environment such as housing, toxic exposure, and stress did not count as key environmental targets in the clinical trial implementation. In the prenatal intervention, the aim was to change the food entering the pregnant body, which affected the maternal metabolic state and the intrauterine environment. By standardizing and controlling other scales or domains in the lab, the pregnant body emerges as the key environmental target for intervention. In this way, the animal models show us what selected parts of the environment emerge as significant and how those selected factors are then correspondingly applied or assumed in the clinical experimentation on pregnant women.

The point I underscore is that while Meany, Szyf, and Dr. Smith all use animal models to focus primarily on the maternal environment, a key difference across the studies is what emerges as the environmental target for intervention. Meany and Szyf selectively focused on the licking and grooming as key environmental factors that influence genetic expression in the pups (Weaver et al. 2004). Dr. Smith focused on the maternal metabolic state of the rat, which is why the rats are force fed to replicate an "obesogenic" maternal environment. The environmental targets are different in each study because they are approaching, interpreting, and translating the animal models in distinct ways based on their distinct expertise.

The different ways in which environments are conceived directly affect the kind of interventions that are designed. In the Meany and Szyf study, they claim that intervening on "motherly behaviors" can impact (and potentially even reverse) epigenetic modifications in genetic expression (Weaver et al. 2004). In Dr. Smith's animal models, the environment that is targeted for intervention becomes the maternal metabolic environment, which influenced her choice on designing a nutritional intervention for pregnant women.

At the same conference quoted above, Dr. Smith explains that through her animal models she has found "that the *maternal metabolic state* plays a very important role in the future risk of disease in the developing child [. . .]. The suggestion, which is not unusual, is that the *intrauterine environment* has a prolonged effect on the health of the child" (emphasis added). The PI's understanding of environment incorporates a temporal as well as a spatial component. The "future risk of disease" and the "prolonged effect on the health of the child" reflect a temporal understanding of how environmental modification can be carried across time and space. There are two key assumptions that undergird the PI's notion of epigenetic environments; one aspect is that scales of the environments can be controlled and standardized through experimentation, and the other is that the pregnant body can be studied as a temporal environment that extends into the future.

The relationship between parts of the maternal environment to the whole maternal environment is selectively rendered visible through the scientist's expertise and use of animal models. This particular approach emphasizes pregnant bodies as environments for fetal development, rather than bodies situated within multiple scales of the environment. Duden's (1993) and Casper's (1998) work offer ways of understanding how the fetus becomes the main focus while the pregnant person disappears from view by being reduced to an environment or space for fetal development. Overall, the selective prioritization of the maternal metabolic state,

intrauterine environment, or fetal environments individualize responsibility onto mothers and obscures women's (and rats') lived experiences; experiences that are not part of the environment that matters in these particular applications of epigenetic science.

Home as Environment

Dr. Jones, the PI for the SmartStart trial is a health psychologist by training, and she offers a different set of insights on what counts as an environment in her studies. Whereas the StandUP trial focused on the metabolic state or intrauterine environment, the SmartStart trial focused primarily on the idea of the home environment. In addition, Dr. Jones did not use animal models, but instead applied her expertise in nutrition and health counseling to design and implement nutritional interventions on pregnant women, who were classified as overweight and obese. Twenty years ago, when Dr. Jones began her work on behavior, obesity, and pregnancy, the research was situated primarily in the field of health psychology. However, with the resurgence of epigenetics, her work became meaningful for understanding how nutrition during pregnancy changes genetic expression in fetal development, *and* how maternal behavior during early development may also affect gene expression.

The main goal of the SmartStart intervention was to minimize weight gain. Following the recommendations published by the IOM, pregnant participants in the intervention group were counseled to limit their weight gain to only a half a pound per week during their pregnancy. To meet the weight gain goal, the women in the experimental group had to follow a strict meal plan that included meal replacement beverages. In addition, each participant had to meet physical activity goals and meet with a nutritional counselor every other week throughout their pregnancy. The intervention is rigid by anyone's account; however, the participants are not forced to do anything, and compliance varied across participants. The strict meal plan aimed at monitoring weight gain and calorie consumption. Exposure to food, or calories, was associated with weight gain, and different home environments indexed different kinds of food exposure.

In Dr. Jones's words, "the intervention is an intensive environmental manipulation because we are *reducing exposures to food*, and so it is a pretty intensive approach to reduce intake of food—we tell them exactly what to eat, when to eat, and what not to eat, and give them something to use instead of eating" (emphasis added). When I asked what exactly she meant by "reducing exposure to food," Dr. Jones responded by saying that for instance, "having a meal replacement shake [instead of a sandwich], you're not having ham and cheese and bread, and mayonnaise, and mustard, and everything else that you could put on a sandwich, you just have this one thing, and you don't have all the excess food in the house." In Dr. Jones's perspective, she reduced pregnant woman's exposure to food by literally reducing the availability of food in their homes. Food as exposure and exposure to food seem to be two sides of the same coin. If food acts as an environmental factor in the uterine environment, then reducing the exposure to food in the home environment is by extension another way to reduce the food that enters the uterine environment. As such, food as an environmental factor is meaningful at both the scales of the home environment *and* the uterine environment.

Dr. Jones further characterizes the home environment as follows: “I think we can change their home environment and I think that is exciting because of the babies who are going to come and play and develop in that home environment. I think the women in the intervention will have a more healthy home, food, and exercise environment.” For Dr. Jones, the home environment is both a psychological factor in changing behavior, and within the epigenetic literature, it can also act as an environment that influences biological and social development. Following her description of the home environment, I asked her about how the SmartStart trial specifically intervenes in women’s behaviors and environments.

On a basic level, we are giving the women scales, measuring cups, pedometers. We give them some environmental cues. We are asking them to exercise, so that usually leads to more sneakers around the house, or gym clothes around the house and yoga tapes and exercise tapes. We can manipulate it here and there, but I think in this study it is more exercise cues, and some dietary cues, meal plans and shakes. That is manipulating their home environment. We also tell them to do a cabinet clean out; to take out all the junk from their home.

In the excerpt, Dr. Jones explains that tennis shoes, exercise tapes, or the types of food in the kitchen cabinets are all aspects of the home environment that can affect or “cue” behavior, which then affects what kind of exposure to food people have. The woman’s behavior, along with what kinds of things she includes into her home environment can, by extension, affect the food that the developing fetus is exposed to and the home environment in which the future child develops. In this way, the pregnant body is the intermediary environment between the fetal environment and the home environment.

In both trials, the principal investigators render the environment in different ways, which make certain epigenetic environments subject to examination and intervention and not others. At the SmartStart trial, the maternal environment is shaped by calories and it includes the home environment. Whereas in the StandUP trial, the key design aspects of the nutritional intervention were informed by the animal models, which focused on the maternal metabolic environment. The different ways that experts approach, monitor, and categorize environmental targets shape the different kinds of interventions that are designed and implemented. However, regardless of whether scientists refer to home environments or intrauterine environments, the same body is targeted for intervention and held responsible and accountable.

Moreover, the home environment rendered in the scientist’s imaginary indexes a particular socioeconomic status. For instance, some home environments do not have the extra funds to buy exercise equipment or to fill the kitchen cabinets with calorie-controlled snacks or even fresh produce. Some home environments are situated within larger community environments referred to as “food deserts,” which do not have affordable or healthy food (Walker et al. 2010). Assuming that the home or maternal environment is stable, safe, and controllable allows for the intervention to target individual behaviors rather than targeting interventions at the level of community and state policies. Framing the home or maternal environment through food,

behavior, and choices enables the responsibility and accountability to be centered on individual bodies. If scientists were to imagine a different maternal environment, one that takes into consideration the multiple scales of intersecting environments at play—it would no doubt redistribute responsibility beyond the individual alone.

Everyday Experiences as Environment

In the clinical trials I observed, monitoring weight, diet, and the food available in one's home environment were framed as individual and behavioral choices. However, as Mullings and Wali state in their work on reproduction in Harlem: “[W]hat appear to be personal risk factors and individual lifestyle choices are best understood in the context of a larger structure of constraints and social choices conditioned by race, class, and gender” (2001, 2). Moreover, environmental contexts are not only structured by gender, race, and power, they also shape material conditions of *exposure* for individuals (Agard-Jones 2016; Alaimo 2016) and for pregnant bodies (Davis Forthcoming; Menzel Forthcoming). The “larger structures,” which can also shape epigenetic modifications, are illustrated in greater detail through the experiences of the pregnant participants. Drawing from conversations in medical anthropology, feminist STS, and reproductive justice paradigms, my analysis of the pregnant participants' experiences emphasizes how race, gender, and class shape their conditions of exposure.

Based on my work as a nutritional counselor and interviews with other nutritional counselors at the SmartStart trial, the intervention visits were rarely about the food that the pregnant participants ate. That is, the intervention visit itself was entangled with the pregnant women's families, work schedules, transitions, unemployment, evictions, and childcare. The conversations during the nutritional interventions were less about calories or weight and more about managing life in general.

To illustrate the different experiences of the pregnant participants, I turn to an ethnographic case that I call Participant Iris. The ethnographic case illustrates similar issues that different participants experienced, but I have modified identifiable details so that it cannot be traced back to one individual. In so doing, I maintain the anonymity of the different participants I worked with while at the SmartStart trial. Iris's case highlights some of the patterns I observed across different participants but does not intend to stand in for all of the women's experiences.

Iris is a woman of color who is deemed obese and volunteers to participate in a clinical trial. At the SmartStart trial, the majority of women who I worked with and observed were women of color. The study protocol stated that the target enrollment for the trial site that I observed was 50% Hispanic and 50% non-Hispanic women. In conversations with staff members, the term Mexican American was often used to reference the Hispanic group. Since I spent much of my time recruiting and delivering the intervention at the public clinics rather than at the private clinics, Iris's case is representative of the participants I observed who were from a lower economic class.

The trial protocol defined the patient population at the public clinics as more ethnically diverse, lower socioeconomic status (SES), and younger, compared to the private clinic population, which had older patients with high SES. Although the trial aimed to recruit an equal number of participants at different income ranges,

it was difficult to implement it. On a basic level, it was hard to recruit pregnant overweight women for nutritional interventions, and it was harder to recruit them from private clinics for a wide variety of reasons related to insurance systems, access, and attitudes toward clinical experimentation. From my observations, the monetary incentive meant a lot to the women who were unemployed at the time, but it was not the only reason why women participated.

Iris had two kids and was pregnant with a third. She was active in her church and ate at the weekly potluck, which was one of the only cooked meals her family had each week. Money was tight since she and her husband were unemployed. Iris also suffered from depression, insomnia, and at 24-weeks gestation, she was diagnosed with gestational diabetes mellitus (GDM). Throughout the trial I observed symptoms of prenatal depression, and was trained to triage any serious cases to my supervisors, who were clinical psychologists. In addition, the trial identified a significant number of women with gestational diabetes. The higher rates of GDM among the participant population was due in part because the trial used a more conservative measure for diagnosing GDM compared to standard care. After Iris was diagnosed with gestational diabetes, she met with a dietician every other week and a diabetes specialist. Altogether, she had about eight health appointments each month including her participation in the clinical trial. Near the end of the pregnancy, she struggled with eviction and eventually she and her family moved out of their apartment to live with some in-laws.

At the weekly staff meetings, I presented Iris's case and the significance of her mental, emotional, and physical living conditions. However, I was reminded that I had to focus on delivering the intervention as it is delivered to everyone in a standardized manner. This reflects what is measured and what is not, or rather what is considered an environment to target and what is not. For instance, food may be an environment; using the meal replacement shakes may reduce the participants' exposure to food; and the intervention may change their home environment, but the stability of their income and housing are not considered measurable environmental factors in the randomized clinical trial.

Sometimes I felt conflicted asking about Iris's food journal, calorie goals, and steps she walked that week because she had other environmental factors to worry about. She usually preferred to talk about the immediate and material challenges she faced every day, and I would listen. Despite her stressful days, Iris kept coming to meet with me every two weeks. In the end, she delivered what the trial classified as a "healthy baby," and she met the trials weight gain goals. The study followed up with her six months after delivery to weigh her and her baby and take bio-samples for testing. The biomarkers they test will indicate whether or not the nutritional intervention changed any genetic expression by reducing the baby's risk of developing obesity and diabetes. What remains unchanged are the participant's living conditions.

The trial was not designed to target the environmental factors that emerged as significant in Iris's everyday experiences. During our intervention sessions, Iris commented that fast food was often cheaper than fresh produce. When I asked her about her steps and how she could fit in a walk during the day, she told me that she does not feel safe walking around her neighborhood. In addition, meeting the physical activity requirement was difficult because Iris suffered from insomnia and

depression, which made it difficult to feel motivated to exercise. Although it was laborious, Iris still kept attending, and even though she did not completely comply, she made an effort.

As an interventionist on the trial, I felt that multiple understandings of the environment competed for my attention and care. On the one hand, I saw how Iris's environment and living conditions influenced her health and emerged as an active force in the intervention visits; on the other hand, I had a concept of the environment from the SmartStart trial that focused on food or calories and the home environment. Further, what counts as "the maternal environment" from the participants' perspective indexes an entirely different set of concerns that implicate a different set of interventions. More importantly, the view of the maternal environment from the pregnant woman's experiences prompts us to reconsider the bodies that are held responsible and the environments that are targeted for intervention.

The effort to use epigenetics in these trials as a way of predicting genetic expression in the future obscures the multitude of social, political, racial, and gendered environments that influence the expression and possibility of health outcomes in the present. More specifically, focusing on changing what a woman eats during pregnancy in an attempt to reduce the risk of obesity for her child in the future overlooks the quality of life that may already affect marginalized people of color. Racial and gender disparities in health play a role in the development of people's lives and opportunity. Consequently, how we define the environments that come to matter in epigenetics is a political as much as a scientific project that compels us to reconsider the distribution of maternal responsibility.

Conclusion

By exploring the experimental, clinical, and lived experiences of the environment, I show how different ways of knowing are always partial and influenced by perspective, expertise, or approach (Haraway 1988, 1989; Strathern 2004). Here, I have illustrated how bodies, homes, and lived experiences can be rendered as the environment through the clinical translation of epigenetic theories. Whereas the first two examples are situated within genetics, physiology, and psychology, the last example drew from feminist frameworks to underscore the perspective of the pregnant participant. The ethnographic exploration of epigenetics also illuminates how different frameworks shape what counts or does not count as an environment worth targeting for intervention. For instance, in different scientific settings the target for intervention can be food, bodies, and behaviors. The hyper focus on any one of these domains obscures the interconnectedness and multiplicity of epigenetic environments. Furthermore, as I have shown above, what constitutes the maternal environment also shapes the contours of prenatal interventions and responsibility. Framing certain environments as risky, in this case the pregnant bodies with a BMI over 30 results in their subjection to different kinds of prenatal care and treatment.

Finally, I juxtaposed the different animations of the environments to reveal a fracture between the researchers' and the pregnant women's experiences of the environment. Along that fracture lay the political impacts of epigenetic science on pregnant women. For instance, by taking seriously the idea that how studies

conceptualize the maternal environment shapes the interventions that are designed, future trials could employ a different approach, one that starts by asking what pregnant women and their partners/families need and want. What environmental domains require intervention from their perspectives? This approach will require reconceptualizing participants as collaborators in clinical trials. It might also require reconsidering whether the randomized clinical trial method is the best avenue for translating epigenetic science.

The politics that undergird the selective translations and applications of epigenetics are situated within a scientific and economic milieu that prioritizes individual responsibility and risk. These scientific milieus produce a translation of epigenetics that collapses the environment into discrete variables and individualizes responsibility onto pregnant bodies. Moreover, these same milieus enable a dependence on ethnic/racial categories as a way to determine study populations in epigenetic science. An alternative approach to incorporating a focus on race in epigenetics might look at the impacts of racist environment on pregnant women.

In the context of obesity during pregnancy and epigenetics, relying on older tools, methods, and biomedical framings of reproduction risks producing new epigenetic knowledge that reinscribes categories of race and gender. Epigenetics has the potential to reconceptualize what counts as the environments that are targeted for prenatal care—and/or it can be used to justify reproductive surveillance and monitoring in relation to racial and gender hierarchies. By asking ourselves what should count as maternal environment, we can start to reimagine a maternal environment that (re)distributes responsibility across bodies, communities, and government.² At stake in the conceptualization and clinical translation of maternal environments are the treatment and care of diverse people who collectively participate in social and biological (re)production. As many feminist science scholars have stressed, *how questions are framed, who gets to ask those questions, and what counts as legitimate knowledge* have consequences for epigenetics and social theory.

Notes

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1. Richardson and Stevens define postgenomics “both temporally, as the period after the completion of the sequencing of the human genome, and technically, in reference to the advent of whole-genome technologies” (2015, 3).

2. For examinations of distributed reproduction, see Michelle Murphy’s 2011 piece. See also Hannah Landecker’s 2011 article, in which she comments on the lateral distribution of responsibility and states that “the idea of male/female or generational responsibility for the future health of generations is simultaneously in

tension with the very idea that individuals could meaningfully control their environments in such a way as to intentionally direct future phenotype” (p. 22).

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