Social Epigenetics: Incorporating Epigenetic Effects as Social Cause and Consequence

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Abstract

Epigenetics is a field of study that invites an interdisciplinary interaction of the social and biological sciences. This collaboration has, in fact, led to a blossoming research community over the past two decades, which is using new data, methods, and conceptual frameworks to address a host of old and emergent research questions. A recent (2014) search of PubMed found over a thousand articles on social, behavioral, and cognitive epigenetics. If one includes epidemiological epigenetic studies that incorporate either social causes or consequences in their research, the number expands nearly threefold. Yet, social epigenetics is a still nascent field, marginalized and misunderstood in social science. In this essay, we attempt to review basic epigenetic concepts and the way in which epigenetics has, and can be, of use to social and behavioral scientists in addressing some of the most fundamental sorts of questions their disciplines raise.

INTRODUCTION

Reference to epigenetics, and the use of epigenetic concepts, has grown over the recent past in many social and behavioral science disciplines (Goodman, Heath, & Lindee, 2003; Landecker & Panofsky, 2013) and has an already extensive history within cognitive psychology (Lester *et al*., 2011; Miller, 2010). However, a long history of the abuse of genetic concepts in the social sciences has also been followed by a subsequent disregard by many for the potential role of genetics in phenomena of behavioral and social interest. Much of the abuse of genetic constructs in the social sciences came from simplistic presumptions that complex socially expressed or constructed traits, such as race, intelligence, class, and so on, could be traced to specific fixed underlying genetic expression and destiny, an approach fueled by racism, classism, and sexism. While genetic causation retained a following among more biologically inclined social sciences (e.g., demography,

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neuropsychology, and epidemiology) the relationship of genetics to behavior and broader social constructs has been largely shunned until recent times. Newer generations have been more willing to investigate the social consequences and causes of genetic influences, especially as embodied in the notion of a mutable, even environmentally socially responsive, epigenetic influence on phenotypes, rather than an emphasis on, in the short-run fixed or immutable, DNA genotypes imposed as dominant upon the social landscape. The potential for "social epigenetics," embracing the integration of epigenetic measures and mechanisms into social and behavioral science research, as cause, consequence, or mechanism, is an exciting and potentially very fruitful line of inquiry.

FOUNDATIONAL RESEARCH: WHAT IS EPIGENETICS?

The origins and uses of the term "epigenetic" are varied (Haig, 2004). Usage vaguely familiar to that of modern molecular biologists dates at least to Waddington's (1942) reference to underlying mechanisms by which "genes of the genotype bring about phenotypic effects." For at least the past two decades it has, more or less, had a familiar meaning defined (in the negative) as "the study of mitotically and/or meiotically heritable changes in gene function that cannot be explained by changes in DNA sequence" (Riggs, Martienssen, & Russo, 1996). The notion that epigenetic changes to gene function are heritable dates back to the mid-1960s. However, other definitions remain in use, and the centrality of heritability to a definition of epigenetics is not universally accepted. Bird, for example, defines epigenetics more broadly and as "the structural adaptation of chromosomal regions so as to register, signal or perpetuate altered activity" (2007). And, for much of the work relevant to social scientists, this broad definition is appropriate and appealing. This definition emphasizes, as did Waddington's, epigenetic processes that alter gene expression, including DNA methylation as well as other DNA and chromatin modifications that have not yet been demonstrated to be heritable.

At the risk of over-abstraction, it is perhaps helpful to pause and present things in a simplified form. Consider a gene, or DNA, is largely fixed where each gene is associated with an expression of genetic information or "trait." Here, trait simply means a specific molecular genetic expression, or the way in which information from a gene is used on a molecular level (not trait in the abusive sense of race, intelligence, etc.). The gene's expression can be altered by various molecular processes which are by (negative) definition epigenetic, changes to the gene from outside. These epigenetic influences on gene expression are in contrast to the strictly genetic influences that include mutations or polymorphisms in the DNA sequence. Some of these processes are, in turn,

influenced even by the wider environment and social phenomena such as diet, stress, or environmental exposures. Thus, the social world can influence the expression of genes in a molecular manner. In turn, many believe this epigenetically altered genetic expression can have a broader influence on phenomena of social interest, ranging, for example, from specific genetically related diseases, endocrine function, and bodily reactions to stress, to even antisocial behavior or suicidal tendencies. There is evidence that some epigenetic changes can occur *in utero*, such that the environment of one generation can influence the epigenetic state of the next generation. And, there is evidence of truly heritable epigenetic transmission from one generation to the next through sexual reproduction, implying potentially intergenerational effects from, as a hypothetical example, one generation's childhood dietary behavior to the next generation's childhood health. Lastly, it is worth noting that some discussions often refer to the cumulative epigenetic changes in gene expression as the "epigenome" or a "ghost gene" although this collection of epigenetic changes does not exist, in any cohesive molecular sense, independent of the gene.

At the molecular level, epigenetic processes involve the placement of chemical tags directly on the DNA or on the proteins that help organize the DNA (Rodríguez-Paredes & Esteller, 2011). These chemical tags are critical in controlling the expression of the gene and in maintaining genetic stability and can be passed on to daughter cells at cell division. By far the most studied tag is the methyl group, a carbon atom with three hydrogen atoms (CH_3) that can be attached at specific locations on our DNA. DNA methylation is only one of several epigenetic mechanisms. However, because methylation studies are so prevalent, including among the examples we cite, methylation merits a brief explanation. In humans, methyl groups can be attached to only one of the four DNA bases, cytosine (C), and only when the cytosine is followed by guanine (G) in the DNA sequence. Because the cytosine and guanine on the same strand are joined by a phosphate (p) molecule, the potential DNA methylation sites are referred to as CpGs. CpGs are not evenly distributed across our genome. Regions of our DNA with a high density of CpGs are referred to as CpG islands. Borrowing from oceanography, CpGs occurring a short distance from an island are on the shore, and moving further out, the shelf followed by the deep sea. The density and location of CpGs in our DNA sequence is important. CpG islands occur more frequently within genes, in regions responsible for turning the gene on and off (promoter regions), whereas as low-density CpGs occur more frequently in DNA that is between genes and is responsible for maintaining genetic stability. Biologists are busy cataloging the location and density of methylated CpGs in various tissues from an array of developmental, exposure, and diseased states; however, much remains to be learned about the biological effects of methylation at specific CpGs.

CUTTING-EDGE RESEARCH: USING EPIGENETIC DATA IN THE SOCIAL SCIENCES

Just as one does not have to be an oncologist to study the social correlates of cancer, for example, one does not have to be a molecular biologist to consider the role of epigenetics in social and behavioral models. In most instances, research addressing epigenetic influences is inherently collaborative and increasingly in the model of "big science" involving a team of, perhaps, molecular biologists, epidemiologists, behavioral experts, disease specialists, research physicians, cognitive psychologists, laboratory technicians, and so on, all of whom will be involved in formulating the central research problem on which social and behavioral scientists collaborate, identifying gene regions or targets of epigenetic interest, defining likely epigenetic covariates, supervising appropriate biological assays, and so on. This is true regardless of whether the driving research question is one generated from within the social sciences (e.g., environmental justice research on differential exposure of minority populations to potential epigenetic damage) or one generated by any other discipline that simply relies upon expertise from the social and behavioral sciences for the context in which those disciplines would view the research problem at hand.

To facilitate a somewhat pragmatic discussion of epigenetic research and social science in this big science context, let us start with a word about operationalization and measurement. Again, although methylation is only one form of epigenetic change, it has had attention from molecular biologists and social scientists in particular because of potential epigenetic effects on prominent diseases (e.g., various cancers), behaviorally related body processes (e.g., cortisol response) and evidence of potentially direct cognitive pathways (e.g., suicidal tendencies). Quantitative measures, usually percentage, of methylation in specific gene regions or sites of interest are the most common measurements analyzed. Processing cell samples (e.g., saliva, tissue samples, epithelial cells, etc.) to obtain laboratory measurements of methylation for various regions or sites of the gene is still carried out in many individual laboratories but is increasingly a big business and a matter of contracting out for measurements using biological assays collected by researchers. For this reason, we will not spend time here on various laboratory methods and technical procedures. Instead, the focus here is on the potential to be achieved from integrating such measures into social and behavioral scientific research and the overarching issues of research design and data analysis surrounding epigenetic work of importance to the social and behavioral sciences.

One distinction, of relevance, is between measurements from deductively targeted genetic sites of interest (e.g., pyrosequencing for measurement of methylation in specific promoter regions) and the increasing use of inductive

or exploratory measurements across broad swaths of the genome (e.g., global array analysis and genome-wide sequencing of bisulfite-modified DNA, although these methods can also be used to obtain targeted site information). The challenges these different approaches present to statistical analysis can be very difficult, ranging from small sample size issues for precious biological tissue samples, to the large amount of data generated by global arrays and genome sequencing and complex statistical methods required for analysis. Commercial laboratories providing methylation measurement services, especially array measurements, also tend to provide basic software for analyzing the data and complementary routines are available in public software, largely contributed routines in the R-software suite. Again, the complexity of statistical analysis for much epigenetic data often calls for a big science approach with a knowledgeable statistical analyst.

Thus, without trivializing the complexities of methylation analysis, it is not unreasonable to say the social scientists will usually encounter epigenetics as a database, not unlike many they are familiar with, of quantitative measurements of methylation for either target regions of the gene or for large sections of the genome. Analytical approaches may be deductive and targeted at specific genetic sites using familiar analytical techniques or inductive and requiring high-throughput analysis, often using proprietary laboratory-supplied software. In the remainder of this essay, we do not dwell on laboratory or statistical methodology details. Instead, as suggested, we take an approach in which epigenetic measures are operationalized and available as variables for a specific research problem and focus on the conceptual or functional use of epigenetics as a construct and framework within the social sciences. We do this by focusing, in turn, on recent examples of work, and some speculation as to the future of this work, in which epigenetic measures are used by social scientists as independent causal effects, dependent variable outcome measures, intervening variables representing epigenetic mechanisms, durational effects carrying causation from one point in time to a later manifestation of outcomes and intergenerational variables potentially carrying the effects of gene–environment interactions across generations.

KEY ISSUES FOR FUTURE RESEARCH: THE UTILITY AND ROLES OF EPIGENETIC DATA IN THE SOCIAL SCIENCES

Epigenetic Variation as a Dependent Outcome

The most common existing use of epigenetic variation to date is that of a biological assay, used as a dependent variable, or biomarker, of health outcomes in studies to identify exposures responsible for the epigenetic change.

Epidemiologists, demographers, health and medical researchers, anthropologists, neuropsychologists, and many others, have all shown interest in the potential for incorporating epigenetic assays and measurements as potentially more direct indicators of environmental effects than abstract measures of allostatic health, body condition, or future disease outcomes. Epigenetic variations provide a measure of induced change in genetic fortunes. These may be studied simply as indicators of environmental exposure or be targeted because of known associations with eventual disease outcomes. When studying rare diseases with complex lifelong pathways, such as most cancers, the prospect of studying outcome measures that may be more immediate, but indicative of both environmental causation and thereby induced predisposition toward eventual disease outcomes, is appealing. Although there are many studies that have employed epigenetic variation as a direct outcome measure in this manner, two examples may be of particular interest in an interdisciplinary context: dietary studies and epigenetic toxicology.

An early interest in the effect of social behaviors on epigenetic change emerged in studies of the relationships between diet and methylation (Van den Veyver, 2002) and of diet to other epigenetic mechanisms (Romagnolo, Dashwood, & Ziegler, 2012). These interests have fueled an emerging science of nutrigenomics. Not surprisingly, most evidence that dietary intake affects epigenetics exists for folate, which supplies building blocks of DNA methylation. However, a long list of other dietary components have been studied, including alcohol, flavonoids, polyphenols, lycopene, methionine, B-vitamins, resveratrol, choline, genistein, and sulforaphane, among others, and such common foods containing some of these compounds as green tea, spinach, sunflower seeds, baker's yeast, soy, broccoli sprouts, fiber, or cruciferous vegetables (Hardy & Tollefsbol, 2011). One area of particular attention in dietary studies is in prenatal diets where animal studies suggest folate or choline deficits can cause lifelong hypomethylation. Another great interest in dietary studies concerns the potential treatment aspects of diets which contain dietary compounds that modulate epigenetic programming and can be used to target methylated promoters known to contribute to cancers. Studies of various cancers sometimes consider both overall genome-wide reduction in DNA methylation (global hypomethylation) resulting in chromosomal instability and hypermethylation within the CpG islands of a specific gene promoter, often tumor suppressing genes, as an outcome variable. The prospects of identifying hypermethylated, cancer-related, promoters and targeting them with dietary demethylating agents before disease develops drives a great deal of interest in studies of diet and methylation.

Another interest in the use of epigenetic change as outcome variables comes from communities who are in search of sensitive, widely detectable,

indications of environmental or toxicological exposure. The use of epigenetic variations as direct outcomes in examining toxicological or environmental effects has emerged as a new field of considerable potential (Sahu, 2012; Szyf, 2011). Researchers who examine diffuse environmental hazards and issues of environmental justice, for example, have long struggled with the fact that outcome measures such as disease incidence rates measure only the potentially rare events that exist at the end of a long and complex pathway that may nonetheless be impacted by environmental exposures and bodily predisposition to eventual outcomes. Use of measures such as epigenetic variations that are more immediate mechanistic effects of environmental exposures is of great value in such circumstances. Focusing on epigenetic mechanisms may explicate an actual pathway of ultimate harm, while addressing the research problems posed by disease outcomes that may remain both rare in exposed populations and may not manifest for years hence. This interest has again given rise to a host of studies of specific environmental toxicants such as endocrine disruptors, heavy metals, and other persistent and common environmental pollutants in our air, food, and water supplies. Most environmental epigenetic studies have utilized methylation measures. However, the still emerging and new field of environmental epigenomics has grown to encompass studies across the entire range of epigenetic mechanisms (Godfrey, 2012; Hou, Zhang, & Baccarelli, 2011).

Epigenetic Variation as a Causal Influence

While most studies have emphasized and utilized epigenetic change as a dependent variable or outcome of environmental circumstances, other profitable areas of epigenetic research have focused on using epigenetic changes or biomarkers as an independent variable responsible for direct, or eventual, disease or behavioral outcomes. Some studies have gone even further and explored both causes and consequences of epigenetic variation in a single study, bringing epigenetic variation into the more realistic role of an intervening variable. Again, two examples may suffice to illustrate the uses and potential of this epigenetic approach: neurocognitive/behavioral epigenetics and epigenetic cancer studies.

Within the social and behavioral sciences, neurocognitive and behavioral epigenetics is likely the most familiar application of epigenetics to many and has been the subject of most futuristic speculation. Most, not all, of the research in neurocognitive epigenetics has focused on identifying epigenetic variations associated with cognitive or behavioral outcomes. Epigenetic variation has been considered, for example, as a potential explanation in processes of memory, learning, age-related cognitive changes, depression, aggression, mental illness, and other cognitive outcomes of interest. Studies

have also examined epigenetic effects on behaviorally related endocrine response systems and for specific mental illness outcomes. Epigenetic variations have been implicated in the neurodegenerative disorders such as Alzheimer's and Parkinson's disease, the most common form of inherited mental retardation (fragile X syndrome), Rett syndrome, and more recently, as a potential influence on autism spectrum disorders.

Given the difficulty of brain tissue sampling, one of the most notable studies of this sort is a cadaver study of suicide victims (McGowan *et al.*, 2009). This study replicated earlier animal model work supporting a plausible association between methylation of glucocorticoid receptors and later life abilities to cope with stress. By comparative analysis of those who suffered childhood abuse and those who did not, the authors also suggest abusive stress may be a source of DNA methylation, bringing methylation into the role of an intervening variable affecting later suicide among child abuse victims. This study is only one of many suggesting a link between stress and later life cognition and health. Miller (2010) summarizes this research and several threads of more recent, specifically cognitive, epigenetic research including changes in gene expression through histone modification that are related to subsequent learning and memory, as well as age-related changes in memory, across the lifespan.

Undoubtedly, the greatest strides in studying epigenetic change as a direct cause of socially important outcomes have been in the epidemiological study of methylation and disease outcomes, especially cancer. After nearly two decades of numerous studies seeking to identify specific misregulation in epigenetic mechanisms (including DNA methylation, histone modification, nucleosome remodeling, and RNA-mediated targeting) that can culminate in cancer, there is little doubt that such connections are abundant (see Dawson & Kouzarides, 2012, for one recent summary). However, despite these numerous studies implicating specific epigenetic misregulation with both tumor suppression and cancer outcomes, there are not, as yet, definitive epigenetic biomarkers identified as suitable for routine clinical practice. One forefront of epigenetic cancer research in the coming decades will be to advance research to the point of contributing to clinical diagnosis and treatment of specific cancers. Of course, identification of specific pathways is also potentially critical to preventive measures. While much has been achieved, even in this most prolific area of research, epigenetics is a nascent science.

With a growing number of epidemiological studies, the potential role of methylation, as not only a cause but also an intervening effect between social behaviors and disease or behavioral outcomes is naturally receiving considerable attention. Research in developmental psychology by Frances Champagne and others (see Champagne, 2009 for an overview), for example, provides examples from animal model research suggesting early exposures

to stress could epigenetically modify later life stress response mechanisms; or that juvenile isolation could influence alter epigenetic pathways that contribute in turn to isolation syndrome among adults; and other similar examples of species-specific social behavior with epigenetic intervening effects that subsequently impact social behavior. Discussion of mechanisms of socially mediated epigenetic misregulation in human populations (e.g., excessive alcohol consumption that reduces folates related to epigenetic changes) coupled with studies linking such epigenetic changes to disease outcomes (e.g., liver cancer), suggest a fuller understanding of the relation of social behaviors to disease, or behavior, through specific epigenetic pathways is within reach. For many diseases such as breast cancer, where the major correlates include not only heredity (e.g., BRCA1) but significant influences that are largely social in character (e.g., alcohol, diet, obesity, childbearing, and breastfeeding, etc.), epigenetics may provide a critical missing link in the pathways between social behavior and socially significant disease and behavioral outcomes.

Again, even in the most advanced areas of epigenetic research, our understanding of the complex pathways linking environments to epigenetics to outcomes is only now emerging. The potential of such research excites the imagination. Yet, such speculation is also warranted to the extent that it stimulates new studies and explorations of problems that have long defied the simple bifurcated heredity plus behavior equation. Particularly from a social science or public health perspective, the environmentally interactive and intervening role of epigenetic change may be critically important for understanding both cause and consequence of socially important "epidemic" conditions (e.g., obesity or autism) and ill-defined syndromes (e.g., chronic fatigue, environmental sensitivity, fibromyalgia) that have been largely socially and symptomatically defined, with, as yet, poorly understood biological pathways. With such high-value targets in sight, and a new paradigm with some seeming promise, the occasional speculative, or even wildly speculative, hypothesis is perhaps deserving of some latitude.

Epigenetics and Durational Effects

For social epigenetics, one of the most interesting and useful elements of the conceptual framework afforded is the durational aspects of epigenetic effects. As in much of the research cited earlier regarding social behaviors (e.g., Champagne, 2009), epigenetic influences such as an environmental exposure may occur early in the lifespan, or *in utero*, and those relatively durable epigenetic changes carry consequences forward in time such that they then manifest in an impact on disease or biological and cognitive outcomes in later life. This durational model, carrying the effects

of environmental exposures far removed in time to ultimately observed probabilistic outcomes, provides a mechanism suitable to numerous social and epidemiological inquiries. Animal models demonstrating that maternal crack cocaine exposure alters epigenetic profiles, resulting in dysfunctional social interactions among offspring throughout their life course, have raised awareness of the plausibly groundbreaking potential for such studies in human populations. There is, as yet, more speculation than research in this regard. Kuzawa and Sweet (2009), for example, argue the correlated racial disparities in birth weight differences and cardiovascular diseases across groups offer a rationale for considering epigenetic links between early-life environmental factors, such as maternal stress during pregnancy and adult racial disparities in cardiovascular diseases. Similarly, Thornburg, Shannon, Thuillier, and Turker (2010) argue that such differences, along with research linking rapid growth in the womb to metabolic disease and obesity and also to breast and lung cancers, calls for research to determine the epigenetic processes underlying these linkages.

Demographers and epidemiologists have also long been interested in durational processes in which *in utero*, or early life-course, events and stress have resulted in long-term adult morbidity and mortality in historical populations. Such durational effects are often simply regarded as a "black box," increasing frailty among the exposed populations and the likelihood of later morbidity and mortality. Epigenetics provides a conceptual framework that may well lead us to more specific pathways by which distinctive early life-course exposures and events influence the likelihood of particular morbidity outcomes over the life course. The potential for such research is significant. However, the implementation of longitudinal life-long epigenetic studies of early life-course effects on epigenetic change and probabilistic outcome variables, such as morbidity, is anything but simple compared to a short-term clinical trial, for example, to examine the effects of stress or diet on epigenetic misregulation. An increasing recognition for the big science model needed to address such questions, including the linkage of large longitudinal population studies to genetic and epigenetic data, provides some hope for significant advances in such research despite the many challenges faced in such work.

Transgenerational Epigenetics

Without question, the most controversial and one of the most exciting prospects regarding epigenetics has been some initial evidence for transgenerational transmission of epigenetic effects (Kaiser, 2014). This research goes beyond the durational model, which supports possible mutigenerational (but not transgenerational) effects. In a multigenerational model early-life

or *in utero* conditions may create epigenetic changes that are manifest in later life course outcomes of offspring but exposure of those offspring occurred after sexual reproduction and is not transgenerational or inherited. However, some epigeneticists argue, and some evidence from animal model experiments suggests, that induced epigenetic changes can be transmitted meiotically, or through sexual reproduction, to the next generation or even several generations hence. This research is still in very early stages, with a variety of protocols, some supporting studies, and some failing to find support. Difficulties in conducting such research are vastly greater, yet again, than those faced by life-long studies of epigenetic effects. Research has been exclusively confined to animal models. In addition, a biomolecular mechanism for this intergenerational transmission epigenetic misregulation has also not yet been elaborated. So, as with all truly cutting-edge research, the jury is still deliberating not simply the promise but the reality of transgenerational epigenetics.

Yet, while scholars are still divided and there are skeptics, there are also sufficient converts to transgenerational epigenetics, and enough cumulating supporting evidence, to ensure that this line of research will continue. Transgenerational epigenetic change will ultimately either be refuted or, perhaps more likely, grow into in a new, and paradigm-shifting, field of future study. If transgenerational epigenetic hypotheses are confirmed, the processes to be studied will not be a simple replicate of studies into heritability among future generations. Even among those who strongly support the notion of transgenerational epigenetic change, this claim is often accompanied by the appropriate qualification that continuing methylation and demethylation influences attenuate the force of transmission across generations in ways that will need to be better understood.

The prospect of transgenerational epigenetics for social science is revolutionary to say the least and one which fuels enthusiastic speculation. The idea, for example, that social circumstance of one generation may contribute to epigenetic changes which are then transmitted to, and impact the life-course prospects, of successive generations, could help to explain the intergenerational effects of circumstance on social life that have been found persistent, even in the face of intergenerationally changing social environments and circumstance.

Limits and Further Considerations

There are many limits to the growing enthusiasm for epigenetic research and its expansion into other disciplines including the social sciences. Many current studies, for example, rely ultimately on a selection by the dependent variable and introduce the possibility of reverse causation. If we select tumor

samples, for example, to bioassay, how do we ensure that epigenetic changes found in patients with tumors, and not controls, are not symptomatic rather than causal. One emerging answer to this problem is the statistical use of a Mendelian randomization approach (Relton & Smith, 2010) equivalent to an instrumental variables model with genotype acting as an instrument for the exposure of interest. Another problem in conducting such research is simply the expense and difficulties of obtaining biological assays and the big science funding model required of a truly interdisciplinary epigenetic study. However, there has been an explosion of large data resources combining social and genome-wide array data (e.g., Add Health, the Health and Retirement Study, Wisconsin Longitudinal Study, Framingham Heart Study) and similar resources for genome-wide methylation studies are emerging. The linkage of tissue banks and large-scale longitudinal population surveys provide another potential avenue for social epigenetic research. A growing number of epigenetic databases with linked methylation and survey data are available for public use and efforts by funding agencies to support standardized assays, held in common for future research, have resulted in a growing body of secondary data resources.

Another substantial hurdle lies in the lack of standardized protocols for complex hypotheses and the most challenging studies such as transgenerational epigenetic research. There is no magic shortcut to a mature epigenetic science and only continued study and careful replication will resolve these issues over the near future.

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WIKIPEDIA ANNOTATION AND GLOSSARY

Allostatic Health, http://en.wikipedia.org/wiki/Allostatic_loaD

BRCA1, http://en.wikipedia.org/wiki/BRCA1

DNA Mutations, Polymorphisms, http://en.wikipedia.org/wiki/Mutation; http://en.wikipedia.org/wiki/Polymorphic_DNA

Epigenetic Mechanisms, Methylation, Chromatin Modifications, Histone Modification, Nucleosome Remodeling, RNA-mediated targeting, http://en.wikipedia.org/wiki/Epigenetics#Mechanisms

Epigenomics, Methylation, Hypomethylation, Hypermethylation, Pryosequencing, Global Array Analysis, Genome-wide sequencing, Bisulfite modification, http://en.wikipedia.org/wiki/Epigenomics

Endocrine disruptor, http://en.wikipedia.org/wiki/Endocrine_disruptor

Glucocorticoid Receptor, http://en.wikipedia.org/wiki/Glucocorticoid_receptor

Mitosis, Mitotically, Meiosis, Meiotically, http://simple.wikipedia.org/wiki/Mitosis; http://simple.wikipedia.org/wiki/Meiosis

Mendelian Randomization Approach, Instrumental Variables Model, http://en.wikipedia.org/wiki/Mendelian_randomization

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KATHLEEN F. ARCARO SHORT BIOGRAPHY

Kathleen F. Arcaro is an Associate Professor in the Department of Veterinary Sciences at the University of Massachusetts-Amherst. Her research is focused on discovering epigenetic biomarkers of breast cancer risk and understanding the epigenetic mechanisms underlying drug resistance in the treatment of breast cancer. Her long-term study of breast milk is aimed at determining the extent to which the exfoliated epithelial cells in milk can be used to assess breast cancer risk.

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