Chapter 4
Your DNA is Not Your Destiny: Behavioral Epigenetics and the Role of Emotions in Health
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ABSTRACT
In a series of studies published in 2000 and later, researchers began to demonstrate the importance of epigenetic influences on gene expression. Genes might be silenced through methylation, or their expression facilitated by acetylation. A further step occurred when behaviors and psychological states were noted to regulate the activity of genes. A body of evidence has now been accumulated that assesses the specific genes affected by behavioral influences such as nurturing, by lifestyle interventions such as meditation, by emotions, and by alleviating psychological conditions such as depression, anxiety and post-traumatic stress disorder (PTSD). Comparisons of the relative lengths of telomeres in identical twins, who start life with identical genes, show that emotional stress can result in one twin having a cellular age that is as much as 10 years older by age 40. New studies in the field of energy psychology also indicate that these psychological and emotional stressors may be remediated much more rapidly than previously believed possible, and that behavioral and psychological influences regulate the genes responsible for inflammation, immune function, and cellular regeneration, among others. These advances provide fruitful new avenues for research into the epigenetic properties of simple behavioral and emotional skills such as meditation, the Relaxation Response, and Emotional Freedom Techniques (EFT), and point to the potential of these methods as potent anti-aging and medical interventions.

Keywords: epigenetics, behavior, emotions, meditation, Relaxation Response, EFT (Emotional Freedom Techniques), stress, aging.

BEHAVIORAL EPIGENETICS AND THE ROLE OF EMOTIONS IN HEALTH
The Central Dogma of Molecular Biology was first articulated by Sir Francis Crick in a series of lectures in the 1950s, and formalized in a paper in the journal Nature. It stated that the information required for the processes of life was found in the DNA, and that, via RNA, DNA was the sole source of the blueprints that governed both the structure and function of cells. Crick’s paper argued that information flows from DNA > RNA > protein, but never the other way around, nor does any outside influence determine gene expression.

The Central Dogma was the dominant biological model for half a century. Crick eventually extended this model of genetic determinism to emotional, behavioral, and mental processes, declaring in his final book, “You, your joys and your sorrows, your memories and ambitions, your sense of personal identity and free will, are in fact nothing more than the behavior of a vast assembly of nerve cells and their associated molecules.” Yet while genetic determinism was regarded as an irrefutable paradigm that formed the cornerstone of molecular biology, puzzling pieces of experimental evidence began to appear that contradicted it. These studies showed that genes could be affected by their environment.

By the turn of the twenty-first century, the epigenetic model of gene expression had been worked out, with methylation and acetylation of genes being the primary mechanisms of action. Methyl groups, by adhering to the cytosine molecule of a DNA strand, could silence a gene, while acetyl groups, facilitating the unwrapping of the histone strands around which DNA is coiled, could promote increased gene expression.

A study of the suppression of a gene known as the Agouti gene in mice showed that, when the gene was silenced by feeding mother mice a diet rich in methyls, pervasive physiological effects occurred. Mice in which the gene had been silenced had half the incidence of diabetes and cancer and lived about twice as long as non-methylated Agouti mice. The potential health implications for human subjects of epigenetic interventions were apparent in this and similar studies.
Another landmark study examined the effects of parental nurturing on the promotion of stress-dampening genes. It found that baby rats whose mothers licked and groomed them were better able to cope with stress as adults. The nurtured rats showed acetylation of genes in the brain regions responsible for regulating stress. Not only did behavior alone produce this effect, but the behavior and associated molecular modification was found to be heritable. Nurtured rat pups nurtured their own offspring, producing similar molecular changes without any difference in the genetic sequence. This study was one of the first to point towards the epigenetic effects of behavior, as opposed to biochemical interventions such as injection of methyls or acetyl groups into the hippocampi of rats.

Members of the same research team then extended their inquiry to human subjects. The brains of normal healthy individuals were compared to those of schizophrenics. The team found hypermethylation of the brain regions responsible for regulating stress in the schizophrenic brains, but not in those of normal subjects. The schizophrenic group had all the genetic information required to dampen stress, but it had been epigenetically silenced. This study showed that psychopathology, in this case schizophrenia, was associated with molecular changes in the limbic system of the brain, which regulates the stress response, emotion, and attachment.

Further research demonstrating the epigenetic influence of psychological states followed. Cole et al found that socially isolated and depressed subjects had differential expression of many genes when compared to normal individuals, including those that code for stress hormones such as cortisol and epinephrine. Kawai et al examined the gene expression of medical students: baseline and during a state of heightened anxiety just before their licensing exams. He also found changes in gene expression. Whilst in 2008, Oberlander et al found that psychopathologies may be passed epigenetically from one generation to another with no alteration in the DNA sequence. Alongside the proliferation of research into the function of particular genes; the importance of epigenetics became apparent to the scientific community.

Two landmark studies examined the effects of simple behavioral interventions on gene expression. Dusek et al compared the gene expression between individuals who were experienced in a stress-reduction method called the Relaxation Response and those who did not use this method. The research team found differential gene expression between the experienced relaxers and the non-relaxers. The researchers then taught the latter group the Relaxation Response and performed another gene assay eight weeks later. They found that the expression of 1,561 genes had changed, including those responsible for the scavenging of free radicals, inflammation, and programmed cell death. The anti-aging and regenerative effects of a simple behavioral stress reduction technique were highlighted by this study.

Ornish et al examined a group of 30 men with prostate cancer. The researchers taught the subjects to meditate, engage in moderate exercise, and eat a low fat diet. In three months, a second whole-genome assay was undertaken. The expression of 501 genes had changed. Oncogenes, such as those that promote prostate and breast cancer, had been downregulated. Genes that promoted immunity and cell regeneration had been upregulated. This study showed that a simple lifestyle intervention could directly affect gene expression in a very limited time frame. Positive emotions such as those associated with laughter were also discovered to alter the expression of genes. Other human studies also found molecular changes in the brain associated with emotional stress states.

Telomeres, the telomerase molecule base pairs that serve as terminators of DNA strands, are regarded as the most reliable marker of aging. A comparison of the telomere lengths of identical twins provides further evidence of the epigenetic effects of stress. In one pair of twins studied, one sister developed childhood leukemia at the age of 2 while the other did not. The treatment team of the twin with cancer examined the medical history of both twins, who had been raised together with virtually identical environmental influences. They found only one difference; the twin who developed leukemia had a stressful event (a tonsillectomy) at the age of 6 months, which they hypothesized triggered the expression of the oncogenes present in both sisters.
A longitudinal epidemiological study of 17,421 adults examined the relationship between childhood stress and adult disease. It found that adults who, as children, had experienced adverse events had higher rates of cancer, cardiac disease, hypertension, diabetes, and many other illnesses. The median age of the sample was 57, suggesting that time does not heal emotional wounds laid down in childhood. Other studies have examined the medical effects of adult traumas such as combat experience in Vietnam, Iraq, and Afghanistan. They found that veterans who develop post-traumatic stress disorder (PTSD) as a result of combat emotional trauma have a higher utilization of medical services than those who do not. In veterans with clinical PTSD, differential expression of stress genes in the brain is noted. The association between stress hormones such as epinephrine (adrenaline) and disease was underlined in a study that found epinephrine receptors on the surface membranes of cancer cells. Depression and elevated cortisol correlate with arousal of the limbic system of the brain, which is central to the regulation of the stress response. Reviews have found psychological distress and disease to be strongly correlated.

The cellular age of another set of monozygotic twins in their late 30s was examined by means of telomere assay. When their personal and medical histories were examined, one was relatively stressed and the other relatively unstressed. The stressed sister had a husband with Huntington’s disease, which resulted in his becoming violent and abusive. She was his caregiver for several years, until his eventual death. She was found to have a cellular age, based on telomere length, 10 years older than that of her sister, who had lead a relatively uneventful life. Studies have shown that such substantial differences between siblings who begin life with identical information encoded in their genes are not uncommon.

Contemporaneous with this research, a new set of studies began to challenge the prevailing view of the tractability of psychological conditions through which stress manifests. PTSD, for instance, has been viewed as “a chronic and recurring condition” in review articles. Another review of an aggressive PTSD treatment regime found that “the treatment program’s impact on the course of illness had been negligible”. Anxiety and depression, though treatable, also usually persist for extended durations.
Figure 2. Divergence of gene expression as identical twins age. The yellow areas are common to both. The chromosomes on the left are at the age of 3; those on the right, 50.

Figure 3. Fifty-year-old monozygotic twins show marked external physiological differences.
Table 1: The reduction of psychological symptoms in EFT studies (Italics indicates a randomized controlled trial).

<table>
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<tr>
<th>ANXIETY</th>
<th>Treatment</th>
<th>N</th>
<th>Measure</th>
<th>Improvement</th>
<th>Significance</th>
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<tr>
<td>Benor, Ledger, Toussaint et al., 2009</td>
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<td>15</td>
<td>TAI</td>
<td>31%</td>
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<td>Brattberg, 2008</td>
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<td>HADS</td>
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<td>Church, Hawk, Brooks et al., 2010</td>
<td>EFT 6 sessions</td>
<td>59</td>
<td>SA-45</td>
<td>56%</td>
<td>p &lt; .0002</td>
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<td>Sezgin &amp; Ozcan, 2009</td>
<td>EFT 1 session + self-treat</td>
<td>32</td>
<td>TAI</td>
<td>37%</td>
<td>p &lt; .05</td>
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<td>Church, 2010</td>
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<td>11</td>
<td>SA-45</td>
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<td>216</td>
<td>SA-45</td>
<td>57%</td>
<td>p &lt; .0001</td>
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<td>Church, Geronilla, &amp; Dinter, 2009</td>
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<td>SA-45</td>
<td>50%</td>
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<td>Rowe, 2005</td>
<td>EFT weekend intensive</td>
<td>102</td>
<td>SA-45</td>
<td>54%</td>
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<tr>
<th>DEPRESSION</th>
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<td>HADS</td>
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<td>Rowe, 2005</td>
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<td>102</td>
<td>SA-45</td>
<td>68%</td>
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<th>PTSD</th>
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<td>59</td>
<td>SA-45</td>
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<td>Church, Pina, Reategui et al., 2009</td>
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<td>Church, 2010</td>
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<td>PCL-M</td>
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<td>7</td>
<td>PCL-M</td>
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<th>PAIN</th>
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<td>30</td>
<td>SUD</td>
<td>22%</td>
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<td>SUD</td>
<td>45%</td>
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<tr>
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</tbody>
</table>

Abbreviations: SAS Speaker Anxiety Scale. TAI Test Anxiety Inventory. SA-45 Symptom Assessment-45. HADS Hospital Anxiety & Depression Scale. BDI Beck Depression Inventory. IES Impact of Events Scale. SUD Subjective Units of Distress. CROPS Child Report of PTSD Symptoms. PCL-M PTSD Checklist-Military
Using a novel behavioral intervention called Emotional Freedom Techniques (EFT), researchers have recently shown that chronic clinical PTSD can be remediated in 86% of patients in just six sessions, with gains being reliably maintained on follow-up (p < .0001). Similar studies of EFT and PTSD followed subjects for up to a year and found that the effects held over time.\textsuperscript{30,31} EFT was likewise found to resolve phobias in a single session.\textsuperscript{32,33} It was equally effective in very limited treatment time frames for depression and anxiety.\textsuperscript{34-36} A randomized controlled trial delivering EFT over the internet found a significant improvement in fibromyalgia symptoms.\textsuperscript{37} Other studies showed immediate reductions in pain when emotional memories were treated.\textsuperscript{38} Evidence for a rapid reduction in the primary stress hormone cortisol after EFT administration has been noted,\textsuperscript{39} and reviewing the published research on the epigenetic effects of EFT and similar interventions traces the mechanisms of action by which these techniques affect genetic, hormonal, and neurological processes, including methylation and acetylation of stress genes.\textsuperscript{40} These studies show that stress-related psychological conditions and emotional traumas may be successfully treated in time frames that are small fractions of those required by earlier forms of therapy.

**CONCLUDING REMARKS**

Taken together, these studies indicate that:

- Emotional states are epigenetic – they regulate gene expression.
- Stress can produce large differences in telomere length by age 40.
- When emotional states are improved by the acquisition of stress-reduction skills, beneficial changes in gene expression are found.
- Stressful emotional states may be changed rapidly.
- The genes affected by emotional change are important to health and aging, governing such cellular functions as programmed regeneration, free radical scavenging, inflammation, and immune function.

The next generation of research is expected to identify a subset of genes most reliably associated with changes in emotional state, rather than requiring a whole-genome assay of all 23,688 genes. Such a subset (possibly fewer than 50 genes) has the potential to allow the biological diagnosis of psychological problems such as PTSD, depression, and anxiety. It will also permit the biological assessment of the effects of behavioral interventions such as the Relaxation Response, meditation, yoga, and EFT, permitting the rapid evolution of effective, non-invasive, and patient-oriented treatment plans. As behavioral epigenetics takes root in primary care, patients and healthcare providers will increasingly recognize that DNA is not destiny, and they will utilize emotional stress reduction as a potent medical and anti-aging intervention.

**REFERENCES**


FURTHER READING
EFT website: www.EFTUniverse.com
EFT training: www.EFTPowerTraining.com

DECLARATIONS
The author derives income from presentations and books in the field of energy psychology.

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ABOUT THE AUTHOR
Dawson Church, PhD, CEHP, established the Foundation for Epigenetic Medicine (Santa Rosa, CA USA) to research emerging psychological and medical techniques that may produce radical health transformation. His best-selling book, The Genie in Your Genes, pioneers the field of epigenetics, explaining the remarkable self-healing therapies now emerging from this science. Dr. Church is the editor of Energy Psychology, a peer-reviewed professional journal, and through EFT Power Training he trains groups on how to apply these breakthroughs to health and athletic performance.