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Closing the Gap: Protecting Predictive Neuroscience Information from Health Insurance Discrimination

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CLOSING THE GAP: PROTECTING PREDICTIVE NEUROSCIENCE INFORMATION FROM HEALTH INSURANCE DISCRIMINATION

ABSTRACT

Recent neuroscience advances have made it possible to predict illnesses, such as Alzheimer's disease, before the onset of any symptoms. This capability is similar to the use of genetic information to predict illness, which began to emerge roughly twenty-five years ago. While predictive information is incredibly useful, the benefits come with a heavy cost. Predictive information can easily be used as a basis for unfair discrimination by health insurance companies, a problem exacerbated by the fact that predictive information is far from perfect. Congress acknowledged and addressed this concern when it preemptively passed the Genetic Information Nondiscrimination Act to prohibit unfair health insurance discrimination on the basis of genetic information. Congress further reinforced and expanded the protections afforded to predictive genetic information with the passage of the Affordable Care Act. However, no analogous protection has been afforded to predictive neuroscience information, which can provide roughly the same information that genetic information can.

This Comment argues that there is a gap in protection for predictive neuroscience information in the large-group and self-insured health insurance markets. This gap is significant because it allows health insurers to undercut the protections afforded to predictive genetic information by the Genetic Information Nondiscrimination Act and the Affordable Care Act. In order to close the gap, this Comment proposes the addition of a health-status-related factor in the Affordable Care Act for predictive neuroscience information. This solution would bring the health insurance discrimination protections for predictive neuroscience information in line with those that already exist for predictive genetic information.

INTRODUCTION

Technology in the health industry has grown by leaps and bounds, allowing doctors to detect illnesses earlier and intervene in more effective ways. One of the most amazing health-related advances in recent years has been the ability to predict illnesses before any symptoms manifest.[1] Genetic information first opened the door to harnessing this predictive power, and the technology has advanced rapidly, giving medical professionals insight they could only have dreamed of just twenty-five years ago.[2] But with this predictive power also came the potential for abuse and unfair discrimination by health insurance companies.[3] Congress saw this negative potential and did something unprecedented, preemptively shutting the door on unfair genetic discrimination by passing the Genetic Information Nondiscrimination Act (GINA) in 2008.[4]

Just a few years later, another predictive modality has begun to emerge in the form of predictive neuroscience. Neuroscience researchers have realized how useful their findings could be for healthcare and medicine in adding to the predictive power already being harnessed by genetic information.[5] It has become increasingly clear that, in many ways, genetic information and neuroscience information are analogous and tend to reinforce one another, thereby enhancing the strength of the predictive powers of each.[6] However, while GINA and parts of the Patient Protection and Affordable Care Act (ACA) protect predictive genetic information from unfair health insurance

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[1] This idea will be discussed in detail in Part III of this Comment.

[2] *See* U.S. Dep't of Energy Human Genome Project, *About the Human Genome Project*, HUM. GENOME PROJECT INFO. ARCHIVE 1990–2003, http://web.ornl.gov/sci/techresources/Human_Genome/project/index.shtml (last modified Feb. 11, 2015) [hereinafter *About the Human Genome Project*] (describing the achievements of the Human Genome Project).

[3] This comment distinguishes between fair and unfair discrimination in the insurance context.

[4] *See* Jessica L. Roberts, *Preempting Discrimination: Lessons From the Genetic Information Nondiscrimination Act*, 63 VAND. L. REV. 439, 440–41 (2010) (discussing GINA's unique preemptive nature).

[5] *See* Steven K. Erickson, *Blaming the Brain*, 11 MINN. J.L. SCI. & TECH. 27 (2010) (discussing advances in neuroscience relating to criminal culpability); Philip E. Tetlock, Gregory Mitchell & L. Jason Anastasopoulos, *Detecting and Punishing Unconscious Bias*, 42 J. LEGAL STUD. 83 (2013) (discussing neuroscience and bias detection).

[6] For example, the apoE4 gene is a risk gene for developing Alzheimer's disease, but researchers can also predict Alzheimer's disease through neuroimaging of amyloid plaques in the brain. *See* Press Release, Am. Acad. of Neurology, Plaque Build-up in Your Brain May Be More Harmful Than Having Alzheimer's Gene (Oct. 15, 2012), *available at* http://www.sciencedaily.com/releases/2012/10/121015161912.htm; Press Release, Perelman School of Med. at the Univ. of Pa., Path of Plaque Buildup in Brain Shows Promise as Early Biomarker for Alzheimer's Disease (July 15, 2013), *available at* http://www.sciencedaily.com/releases/2013/07/130715105128.htm; *What We Know Today About Alzheimer's Disease*, ALZHEIMER'S ASS'N, http://www.alz.org/research/science/alzheimers_disease_causes.asp (last visited Apr. 27, 2015).

discrimination,[7] predictive neuroscience information has not been afforded analogous protection. Given the strong connection between the predictive information that can be gleaned from both genetic and neuroscience information, it is apparent that one can be used to supplant the other, especially when only predictive genetic information is protected.[8]

Health insurers make money based on their ability to underwrite risk and identify who will be healthy and who will be sick, highly incentivizing them to find any legal means of doing so.[9] Because predictive neuroscience information can give health insurers essentially the same knowledge as predictive genetic information,[10] predictive neuroscience information can be used in place of predictive genetic information to get around the protections that GINA and ACA afford to genetic information.

This Comment addresses the disparity in protections afforded to predictive genetic information and predictive neuroscience information, arriving at the conclusion that predictive neuroscience information should be protected from health insurance discrimination in the same way that genetic information is protected. Part I will begin by briefly introducing GINA and discussing why it was enacted. It will present the backdrop against which GINA was proposed and passed. Part I will also discuss the protections afforded by Title I of GINA and why Title I was so important. It will conclude by exploring arguments supporting and opposing GINA's passage.

Part II will discuss additional existing health insurance discrimination protections currently in place. The Health Insurance Portability and Accountability Act's (HIPAA) relevant protections and limitations will be discussed first, followed by a brief overview of ACA's protections. Part II will

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[7] *See* Patient Protection and Affordable Care Act, Pub. L. No. 111-148, 124 Stat. 119 (2010) (codified in scattered sections of Internal Revenue Code and Titles 29 and 42 of the United States Code); Genetic Information Nondiscrimination Act of 2008, Pub. L. No. 110-233, 122 Stat. 881 (2008) (codified as amended in scattered sections of the Internal Revenue Code and Titles 29 and 42 of the United States Code).

[8] *See supra* notes 5–6.

[9] Health insurers are incentivized to utilize medical underwriting to offset the effects of adverse selection in the health insurance market because they know that (1) "[p]eople are more likely to buy insurance if they have reason to believe they will incur high costs in the near future" and (2) "[a] small proportion of the insured population accounts for a very large share of total claims costs." MARK MERLIS, NAT'L HEALTH POLICY FORUM, FUNDAMENTALS OF UNDERWRITING IN THE NONGROUP HEALTH INSURANCE MARKET: ACCESS TO COVERAGE AND OPTIONS FOR REFORM 5 (2005), http://www.nhpf.org/library/background-papers/bp_underwriting_04-13-05.pdf.

[10] *See supra* note 6.

conclude with a discussion about the trend toward greater health insurance discrimination protections.

Part III will introduce the emergence of predictive neuroscience. It will begin by discussing the increasing relevance of predictive neuroscience information and then move on to give concrete examples of how this information can be used to predict illnesses today, with the aim of demonstrating that this is an increasingly relevant problem and that use of this information for discriminatory purposes is entirely feasible given the current state of the technology.

Part IV will argue that predictive neuroscience information should be protected in the same way that predictive genetic information is protected from health insurance discrimination under GINA and ACA. Part IV will demonstrate that while predictive genetic information is fully protected from health insurance discrimination, there is a gap in the existing protections that allows health insurers in the large-group and self-insured markets to discriminate on the basis of predictive neuroscience information.

Once this gap has been identified, Part IV will then explain why the gap is problematic. It will focus on how the analogous nature of the information that can be gleaned from each modality allows health insurers to get around existing predictive genetic information discrimination protections afforded by GINA and ACA, and why the incentive to do so is so strong. Indeed, predictive neuroscience information may be even more deserving of protection than genetic information due to its greater predictive reliability. A brief evaluation of the number of people who could potentially be affected by this gap in protection will further evidence the extent of the problem.

Finally, Part IV will propose potential ways to close this gap in order to bring health insurance discrimination protections for predictive neuroscience information in line with those of predictive genetic information. The easiest and most practical solution would be to add a health-status-related factor in ACA for predictive neuroscience information. Alternative solutions include amending Title I of GINA to include predictive neuroscience information and crafting new GINA-type legislation specifically for predictive neuroscience information.

I. THE GENETIC INFORMATION NONDISCRIMINATION ACT

A little over two decades ago, researchers were just beginning to understand the human genome and the potential to harness the information it could provide.[11] As the value of this genetic information became evident, the potential to unfairly discriminate based upon this information became equally evident.[12] These rapid genetic advances demonstrated the need for protective legislation that would prohibit discriminatory uses of genetic information.[13] This Part will discuss why GINA was enacted and examine the backdrop against which it was enacted. This Part will then discuss the health insurance antidiscrimination provisions found in Title I of GINA and their importance.

In 1990, the U.S. Department of Energy and the National Institutes of Health founded the Human Genome Project as an international scientific research project with the goal of mapping the entire human genome.[14] The project, which remains the largest collaborative biological project in history,[15] was declared complete in April of 2003,[16] when approximately 3 billion DNA base pairs were sequenced and mapped.[17] It was a staggering accomplishment and revolutionized the fields of science, medicine, and health. This information helped researchers understand diseases, allowed physicians to target their therapies and interventions,[18] contributed to the advance of forensic sciences,[19] and aided the understanding of evolution,[20] among other advances.

However, the implications of the Human Genome Project's successful completion were not all positive. There was widespread concern that this

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[11] *See About the Human Genome Project*, *supra* note 2.

[12] COUNCIL FOR RESPONSIBLE GENETICS, GENETIC DISCRIMINATION: A POSITION PAPER (2001), http://www.councilforresponsiblegenetics.org/pageDocuments/2RSW5M2HJ2.pdf.

[13] *See id.*

[14] *See About the Human Genome Project*, *supra* note 2.

[15] *Human Genome Project*, WELLCOMETRUST, http://genome.wellcome.ac.uk/node30075.html (last visited Apr. 27, 2015).

[16] *All About the Human Genome Project*, NAT'L HUM. GENOME RES. INST., http://www.genome.gov/10001772 (last updated Mar. 18, 2014).

[17] U.S. Dep't of Energy Human Genome Project, *Major Events in the U.S. Human Genome Project and Related Projects*, HUMAN GENOME PROJECT INFO. ARCHIVE 1990–2003, http://web.ornl.gov/sci/techresources/Human_Genome/project/timeline.shtml (last modified Jan. 22, 2015).

[18] *Human Genome Project*, NAT'L INSTS. HEALTH, http://report.nih.gov/nihfactsheets/ViewFactSheet.aspx?csid=45 (last updated Mar. 29, 2013).

[19] Dragan Primorac, *Human Genome Project-based Applications in Forensic Science, Anthropology, and Individualized Medicine*, 50 CROATIAN MED. J. 205, 205–06 (2009), *available at* http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2705009/pdf/CroatMedJ_50_0205.pdf.

[20] Press Release, Univ. of Chi. Med. Ctr., Genome Project Opens the Book on Human Evolution (Feb. 13, 2001), *available at* http://www.sciencedaily.com/releases/2001/02/010213081055.htm.

genetic information would be used to discriminate against people in various contexts, most notably in the employment and health insurance contexts.[21] These concerns arose even before the project was completed, as demonstrated by the introduction of the first version of a genetic antidiscrimination bill in the House of Representatives and Senate in 1995.[22] Over the next thirteen years, many other versions of genetic antidiscrimination bills were subsequently introduced in Congress.[23] On May 21, 2008, President George W. Bush signed GINA into law.[24] GINA passed in the Senate by a vote of 95-to-0 and in the House of Representatives 414-to-1, demonstrating the importance Congress proscribed to protecting this newly acquired genetic information from discriminatory misuse.[25] Senator Ted Kennedy called GINA "the first civil rights bill of the new century of the life sciences."[26]

GINA has two main parts, affording specific protection from genetic discrimination in the health insurance and employment contexts.[27] Title I of GINA pertains specifically to health insurance discrimination.[28] Essentially, it prevents health insurers from denying coverage or charging higher premiums to a healthy person based solely on a genetic predisposition to disease.[29] Title I also prohibits health insurers from requesting or requiring genetic information

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[21] *See Genetic Discrimination in Health Insurance or Employment*, NAT'L HUM. GENOME RES. INST., http://www.genome.gov/11510227 (last reviewed Feb. 28, 2012).

[22] *See Genetic Nondiscrimination Federal Legislation Archive*, NAT'L HUM. GENOME RES. INST., http://www.genome.gov/11510239 (last reviewed Mar. 17, 2014) (listing genetic antidiscrimination bills introduced in the House of Representatives and Senate).

[23] *See id.*

[24] Remarks on Signing the Genetic Information Nondiscrimination Act of 2008, 44 WEEKLY COMP. PRES. DOC. 736 (May 21, 2008).

[25] *See* 154 CONG. REC. 7519–20 (2008) (recording vote of the House of Representatives); 154 CONG. REC. 6841–42 (2008) (recording vote of the Senate).

[26] Press Release, U.S. Senate Comm. on Health, Educ., Labor & Pensions, Kennedy, Enzi, Snowe Celebrate Passage of Genetic Information Nondiscrimination Act (Apr. 24, 2008), *available at* http://www.help.senate.gov/newsroom/press/release/?id=313bfde8-f967-46b4-aa9d-11bc73728813.

[27] Since this Comment will focus on the health insurance context, it will not go into the employment discrimination provisions embodied in Title II, which essentially prevent employers from basing job-related employment decisions on individuals' genetic information. *See* Genetic Information Nondiscrimination Act of 2008, Pub. L. No. 110-233, tit. II, 122 Stat. 881, 905–20 (codified at 42 U.S.C. §§ 2000ff to 2000ff-11 (2012)); OFFICE OF MGMT. & BUDGET, EXEC. OFFICE OF THE PRESIDENT, STATEMENT OF ADMINISTRATION POLICY: H.R. 493 – GENETIC INFORMATION NONDISCRIMINATION ACT OF 2007 [hereinafter GINA STATEMENT OF ADMINISTRATIVE POLICY], *available at* http://www.genome.gov/Pages/PolicyEthics/GeneticDiscrimination/SAPonHR493.pdf.

[28] Genetic Information Nondiscrimination Act of 2008, Pub. L. No. 110-233, tit. I, 122 Stat. 881, 883–905 (codified as amended in scattered sections of the Internal Revenue Code and Titles 29, 42 of the United States Code).

[29] *See* GINA STATEMENT OF ADMINISTRATIVE POLICY, *supra* note 27.

or genetic testing, acquiring or using genetic information for medical underwriting purposes, and treating genetic information as a preexisting condition.[30]

Some individuals who undergo genetic testing may find out that they have a higher probability of developing a disease. GINA is a very important protection because without it, these individuals would not be protected from discrimination by health insurers, who are always incentivized to charge more to those with greater likelihood than the normal healthy population of requiring medical care and services.[31] Given the imperfect nature of predictive information, many with predispositions to diseases would never actually develop those diseases.[32] For example, individuals carrying a BRCA gene mutation have an increased risk for developing breast cancer, but the existence of this mutation does not guarantee that person will ever actually develop cancer or when; it just indicates that they are at greater risk for developing cancer than an individual without the mutation.[33] Additionally, the federal government currently exercises limited oversight over the validity and accuracy of existing genetic tests, making it even more difficult to ascertain the information's meaning and whether it is being interpreted correctly.[34] Allowing discrimination on the basis of this imperfect information would result in many people being penalized for a condition that may never develop.[35]

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[30] *See FAQs on the Genetic Information Nondiscrimination Act*, U.S. DEP'T OF LABOR http://www.dol.gov/ebsa/faqs/faq-GINA.html (last visited Apr. 27, 2015).

[31] *See supra* note 9 and accompanying text.

[32] *See BRCA1 and BRCA2: Cancer Risk and Genetic Testing,* NAT'L CANCER INST., http://www.cancer.gov/cancertopics/factsheet/Risk/BRCA (last reviewed Apr. 1, 2015) (explaining that not all women with a genetic predisposition to breast cancer actually develop the disease).

[33] *See id.*

[34] *See* Gail H. Javitt & Kathy Hudson, *Federal Neglect: Regulation of Genetic Testing*, ISSUES SCI. & TECH., Spring 2006, at 59, *available at* http://www.unz.org/Pub/PolicyArchive-2006jan-00016?View=PDF.

[35] *See BRCA1 and BRCA2: Cancer Risk and Genetic Testing*, *supra* note 32. Furthermore, allowing health insurers to discriminate on the basis of this predictive information, even if it were perfect, would result in unfair discrimination on the basis of an immutable characteristic over which these individuals have no control. Existing antidiscrimination laws and Supreme Court jurisprudence suggest that this type of discrimination is impermissible and that these characteristics are strongly deserving of protection. *See* Sharona Hoffman, *The Importance of Immutability in Employment Discrimination Law*, 52 WM. & MARY L. REV. 1483 (2011); Jeffrey S. Morrow, Note, *Insuring Fairness: The Popular Creation of Genetic Antidiscrimination*, 98 GEO. L.J. 215 (2009); J. Atsu Amegashie, A Positive Theory of Immutable Characteristics and Discrimination (Mar. 4, 2009) (unpublished manuscript), *available at* http://www.uoguelph.ca/~jamegash/tolerable_discrimination.pdf. This Comment assumes that discrimination on the basis of immutable characteristics is improper.

GINA's passage had a huge impact on the health insurance antidiscrimination landscape. It was a unique law in that it sought to preempt discrimination, rather than address it ex post facto, as the majority of antidiscrimination laws typically do.[36] As such, it is difficult to know whether genetic discrimination would have become a reality. Given the strong financial incentives for insurers to discriminate, however, it is reasonable to infer that it would have.[37] In any case, Congress acted nearly unanimously in response to what it perceived as a real threat.[38] Such action provides a glimpse into the environment in which GINA was passed.

A major goal of GINA was to encourage continued biological and genetic research.[39] Given the potential for beneficial applications of genetic information across myriad disciplines, it was generally thought that GINA-type legislation would be necessary for people to feel more comfortable availing themselves of existing genetic tests, both for the advancement of research generally and individual diagnostic purposes.[40] In this same vein, the growth of personalized medicine depends upon individuals feeling comfortable obtaining genetic tests for the purpose of crafting individualized medical approaches and developing personalized therapies.[41] The Coalition for Genetic Fairness also postulated that since all humans inevitably would have some genetic anomalies, it was necessary to protect genetic information from misuse because it would provide an unfounded and unfair basis for discrimination.[42]

However, others argued that there was no demonstrated need for legislation like GINA.[43] They claimed there was no widespread showing of genetic discrimination because it had only recently become possible to interpret and make use of genetic information.[44] These opponents disagreed with GINA's preemptive nature and supported a wait-and-see approach, believing GINA to be overly broad in its protections because it would be impossible to know with any certainty what provisions would be necessary without first seeing what

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[36] *See* Roberts, *supra* note 4, at 441.

[37] *See supra* note 9 (discussing medical underwriting).

[38] *See supra* note 25 and accompanying text.

[39] *See* Roberts, *supra* note 4, at 471–74.

[40] *Id.*

[41] *See* David Resnick, *GINA—A Big Step Toward Personalized Medicine*, BOS. BUS. J. (Aug. 21, 2008, 6:04 PM EDT), http://www.bizjournals.com/boston/blog/mass-high-tech/2008/08/gina--a-big-step-toward-personalized-medicine.html.

[42] *See The History of GINA*, COALITION FOR GENETIC FAIRNESS, http://www.geneticfairness.org/ginaresource_history.html (last updated Nov. 10, 2008) (describing several justifications for passing GINA).

[43] Roberts, *supra* note 4, at 469–70.

[44] *See id.*

issues would arise on the genetic-discrimination front.[45] In contrast, other advocates argued that GINA didn't go far enough in its protections because it did not prevent genetic discrimination by life insurers, disability insurers, and long-term care insurers.[46] Another limitation in GINA's protections is that they cease once a genetic condition is manifest.[47] Despite arguments against its passage, GINA was important because it expanded existing protections to include genetic information in an age where the availability of this information and the role it played was becoming increasingly prevalent.

II. ADDITIONAL EXISTING HEALTH INSURANCE DISCRIMINATION PROTECTIONS

In addition to Title I of GINA, which prevents health insurers from discriminating on the basis of genetic information,[48] there are many other laws that protect against health insurance discrimination. This Part will discuss a few of the major federal laws protecting against health insurance discrimination to paint a fuller picture of the existing landscape of health insurance discrimination protections.

A. Health Insurance Portability and Accountability Act

The Health Insurance Portability and Accountability Act of 1996[49] represents one of the most significant federal laws addressing health insurance discrimination. HIPAA was the first piece of legislation in which the federal government addressed health insurance discrimination and sought to afford protections against it.[50] One of the major rationales behind HIPAA's passage was to eliminate discrimination on the basis of an individual's health status and, more specifically, discrimination against individuals with preexisting conditions.[51] Before HIPAA, health insurers could freely deny coverage, limit

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[45] *See id.*

[46] *See Genetic Information Nondiscrimination Act of 2008*, NAT'L HUM. GENOME RES. INST., http://www.genome.gov/10002328 (last updated Apr. 6, 2015).

[47] Anya E. R. Prince & Benjamin E. Berkman, *When Does an Illness Begin: Genetic Discrimination and Disease Manifestation*, 40 J.L. MED. & ETHICS 655, 655 (2012).

[48] GINA STATEMENT OF ADMINISTRATIVE POLICY, *supra* note 27.

[49] Pub. L. No. 104-191, 110 Stat. 1936 (codified as amended in scattered sections of the Internal Revenue Code, and Titles 29 and 42 of the United States Code).

[50] Jessica L. Roberts, *"Healthism": A Critique of the Antidiscrimination Approach to Health Insurance and Health-Care Reform*, 2012 U. ILL. L. REV. 1159, 1178.

[51] *See FAQs About Portability of Health Coverage and HIPAA*, U.S. DEP'T OF LABOR, http://www.dol.gov/ebsa/faqs/faq_consumer_hipaa.html (last visited Apr. 27, 2015).

coverage, or charge exorbitant premiums for individuals with preexisting medical conditions.[52] HIPAA limited the circumstances in which health insurers were permitted to consider such preexisting conditions in determining eligibility for coverage and the extent and pricing of that coverage.[53]

However, HIPAA's antidiscrimination provisions applied only to group health insurance plans, typically obtained through an employer.[54] This left those without access to group health plans unprotected, which could be problematic for many with preexisting conditions because they were unable to obtain any meaningful coverage in the individual health insurance market.[55] Furthermore, while HIPAA required group health insurance plans to provide coverage for individuals with preexisting conditions, it both failed to provide any provisions specifying what must be included in that coverage and failed to cap the premiums at a reasonable rate.[56]

Although HIPAA was a step in the right direction toward limiting health insurance discrimination, there were still many loopholes through which health insurers might consider an individual's health status and preexisting conditions[57] because HIPAA's antidiscrimination provisions only applied to group health insurance plans, not to individual health insurance plans.[58] And even among the discrimination protections provided in the group health insurance market, protections preventing health insurers from providing weak coverage or charging high premiums on the basis of preexisting conditions were notably absent.[59]

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[52] *See id.*

[53] *See id.*

[54] *See HIPAA*, NAT'L MULTIPLE SCLEROSIS SOC'Y, http://www.nationalmssociety.org/living-with-multiple-sclerosis/insurance-and-money-matters/health-insurance/hipaa/index.aspx (last visited Apr. 27, 2015).

[55] *See* Mary Crossley, *Discrimination Against the Unhealthy in Health Insurance*, 54 U. KAN. L. REV. 73, 84 (2005) ("Although the number of persons who obtain coverage through the individual market is relatively small, the stakes are typically high, for purchasers in the individual market may have no other coverage option.").

[56] *See* 29 U.S.C. § 1182(a)(2) (2012) (explaining that HIPAA should not be construed "(A) to require a group health plan, or group health insurance coverage, to provide particular benefits other than those provided under the terms of such plan or coverage, or (B) to prevent such a plan or coverage from establishing limitations or restrictions on the amount, level, extent, or nature of the benefits or coverage for similarly situated individuals enrolled in the plan or coverage"); *id.* § 1182(b)(2) ("Nothing in paragraph (1) shall be construed—(A) to restrict the amount that an employer may be charged for coverage under a group health plan . . . or (B) to prevent a group health plan, and a health insurance issuer offering group health insurance coverage, from establishing premium discounts or rebates or modifying otherwise applicable copayments or deductibles in return for adherence to programs of health promotion and disease prevention.").

[57] *See supra* notes 54–56 and accompanying text.

[58] *See supra* note 56.

[59] *See supra* note 56.

B. Patient Protection and Affordable Care Act

Another major federal health insurance antidiscrimination law is the Patient Protection and Affordable Care Act, which was signed into law in 2010 by President Barack Obama.[60] ACA has been characterized as a "civil rights bill for the sick"[61] and "represents the most significant regulatory overhaul of the country's healthcare system since the passage of Medicare and Medicaid in 1965."[62]

ACA expands HIPAA's discrimination protections in several key ways. First, ACA includes guaranteed issue and renewal provisions, which foreclose health insurers from discriminating at all when deciding whom to accept for coverage.[63] Health insurers now have no option but to accept everyone who applies and continue that coverage until the insured wishes to terminate it.[64]

Second, ACA expands HIPAA's protections by including both group health plans and individual health plans in its prohibition against the consideration of preexisting conditions[65] when determining eligibility or coverage, thereby imposing preexisting-condition restrictions on everyone.[66]

Third, ACA adopts and builds upon HIPAA's idea of health status by broadly imposing a prohibition on discrimination on the basis of health status under both group health plans and individual health plans.[67] ACA sets forth nine health-status-related factors and prohibits health insurance discrimination on the basis of any of those factors.[68] The factors are (1) health status,

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[60] *See* Remarks on Signing the Patient Protection and Affordable Care Act, 2010 DAILY COMP. PRES. DOC. 196 (March 23, 2010).

[61] Lee-Lee Prina, *Health Reform: What Foundations Are Saying and Funding*, HEALTH AFF. GRANTWATCH BLOG (Apr. 9, 2010), http://healthaffairs.org/blog/2010/04/09/health-reform-what-foundations-are-saying-and-funding/?cat=grantwatch.

[62] *Frequently Asked Questions about FASD*, FETAL ALCOHOL SPECTRUM DISORDERS CTR. FOR EXCELLENCE, http://fasdcenter.samhsa.gov/aboutUs/aboutFASD.aspx (last updated Apr. 17, 2015).

[63] 42 U.S.C. §§ 300gg-1(a) to (b), 300gg-2(a) (2012).

[64] *Id.*

[65] *See* Lisa Smith, *Health Insurance: Paying for Pre-Existing Conditions*, INVESTOPEDIA, http://www.investopedia.com/articles/pf/09/covering-medical-costs.asp (last visited Apr. 27, 2015) ("Most insurance companies use one of two definitions to identify such conditions. Under the 'objective standard' definition, a pre-existing condition is any condition for which the patient has already received medical advice or treatment prior to enrollment in a new medical insurance plan. Under the broader, 'prudent person' definition, a pre-existing condition is anything for which symptoms were present and a prudent person would have sought treatment.").

[66] *See* 42 U.S.C. § 300gg-3(a).

[67] *Id.* § 300gg-4(a).

[68] *Id.*

(2) medical condition, (3) claims experience, (4) receipt of health care, (5) medical history, (6) genetic information, (7) evidence of insurability, (8) disability, and (9) any other health-status-related factor determined appropriate by the Secretary of Health and Human Services.[69]

Finally, with respect to individual health plans and small-group health plans of up to fifty enrollees,[70] ACA allows health insurers to consider only four criteria in determining what premiums to charge: (1) whether the insurance coverage is for an individual or for a family, (2) what geographic location or area the enrollee is in, (3) the enrollee's age, and (4) whether the enrollee uses tobacco products.[71] This provision strongly limits health insurers' discretion in setting premiums and eliminates many bases for discrimination.

The existing federal health insurance discrimination protections have been steadily increasing with the passage of HIPAA in 1996,[72] GINA in 2008,[73] and ACA in 2010.[74] A critical distinction exists between a preexisting condition, which is considered to be "anything for which symptoms [are] present and a prudent person would have sought treatment,"[75] and predictive information, which necessarily presents itself prior to the onset of symptoms. The current federal health insurance discrimination protections fail to address this predictive information outside the genetic realm. This failure presents a critical gap in discrimination protections in the health insurance arena as it pertains to certain kinds of predictive health information, such as predictive neuroscience information.

III. THE EMERGENCE OF PREDICTIVE NEUROSCIENCE

Neuroscience research has advanced rapidly, and the information that scientists have been able to glean from these advances has fostered growth with applications in many fields of study.[76] The emerging ability to predict disease with neuroscience techniques represents one of the most exciting of

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69 *Id.*

70 A small-group health plan is typically sponsored by an employee and is defined to be a health plan covering up to fifty enrollees. *Consumer Guide to Group Health Insurance*, NAT'L ASS'N OF HEALTH UNDERWRITERS, http://www.nahu.org/consumer/GroupInsurance.cfm (last visited Apr. 27, 2015).

71 42 U.S.C. § 300gg(a)(1)(A).

72 *See supra* note 49 and accompanying text.

73 *See supra* note 24 and accompanying text.

74 *See supra* note 60 and accompanying text.

75 Smith, *supra* note 65.

76 *See supra* note 5.

these advances.[77] Similar to genetic advances, which received protection with the passage of GINA in 2008,[78] these neuroscience advances also need to be protected to prevent health insurers from improperly using this information to discriminate. This Part will discuss the emergence of predictive neuroscience information and how health insurers can use this information to unfairly discriminate.

The past twenty years have seen rapid and exciting advances in the field of neuroscience. Neuroscience, the scientific study of the nervous system, has countless applications that have the potential to revolutionize the way we think about everything from disease to criminal culpability to bias detection.[79] President George H. W. Bush "designat[ed] the 1990s as the Decade of the Brain 'to enhance public awareness of the benefits to be derived from brain research.'"[80] As a part of this project, the Library of Congress and the National Institute of Mental Health partnered to sponsor various activities and publications to foster and encourage brain research and increase public awareness of the implications of these emerging neuroscience discoveries and advances.[81]

More recently, this continuing commitment to fostering neuroscience research spawned many interdisciplinary research undertakings. In 2013, the Human Brain Project launched.[82] This project is intended to be a ten-year European research initiative with the goal of better understanding how the brain functions through the simulation of the human brain with supercomputers.[83] The project recognizes the valuable insights that can be gained through neuroscience advances and seeks to harness this information to benefit research in the fields of neuroscience, medicine, and computing.[84]

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[77] For example, Alzheimer's disease can be predicted through neuroimaging of amyloid plaques in the brain. *See* Eric Karran, Marc Mercken & Bart De Strooper, *The Amyloid Cascade Hypothesis for Alzheimer's Disease: An Appraisal for the Development of Therapeutics*, 10 NATURE REVS. DRUG DISCOVERY 698 (2011), *available at* http://www.nature.com/nrd/journal/v10/n9/pdf/nrd3505.pdf; Press Release, Perelman School of Med. at the Univ. of Pa., *supra* note 6.

[78] *See supra* note 24.

[79] *See* Erickson, *supra* note 5; Tetlock et al., *supra* note 5.

[80] *Project on the Decade of the Brain*, LIBR. CONG. (Jan. 3, 2000), http://www.loc.gov/loc/brain/.

[81] *Id.*

[82] *See HBP Summit 2013: Overview*, HUM. BRAIN PROJECT, https://www.humanbrainproject.eu/hbp-summit-2013-overview (last visited Apr. 27, 2015).

[83] *See Overview*, HUM. BRAIN PROJECT, https://www.humanbrainproject.eu/discover/the-project/overview (last visited Apr. 27, 2015).

[84] *See id.*

On April 2, 2013, the Obama administration proposed a similar collaborative research initiative in the United States with the goal of mapping the activity of every neuron in the human brain.[85] The Brain Research through Advancing Innovative Neurotechnologies Initiative (BRAIN Initiative), also known as the Brain Activity Map Project, is based upon the Human Genome Project,[86] seemingly signaling the beginning of an era of neuroscience advances similar to the era of genetic advances that took place in the 1990s and early 2000s.[87]

One of the most practical applications of this growth in neuroscience research lies in the areas of medicine and health. In particular, the emergence of neuroscience as a modality to predict disease onset before any symptoms manifest is a powerful tool for researchers and has the potential to revolutionize how we approach and treat disease.[88] In some cases, outcomes may be significantly improved through early or preventative treatment, favorably altering the course of the disease entirely.[89] Additionally, predictive neuroscience advances may help medical professionals identify how diseases will react to certain treatments and allow for treatments to be tailored to produce the best outcomes.[90] Because health and medicine have the potential to affect everyone, the importance and application of predictive neuroscience advances are widespread.

One of the most promising advances in predictive neuroscience has been in the field of Alzheimer's disease research. Alzheimer's disease is the most common form of dementia, accounting for sixty to eighty percent of all

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[85] *See* OFFICE OF THE PRESS SEC'Y, THE WHITE HOUSE, FACT SHEET: BRAIN INITIATIVE (2013), http://www.whitehouse.gov/the-press-office/2013/04/02/fact-sheet-brain-initiative.

[86] *See supra* Part I.

[87] *See* John Markoff, *Obama Seeking to Boost Study of Human Brain*, N.Y. TIMES, Feb. 18, 2013, at A1, *available at* http://www.nytimes.com/2013/02/18/science/project-seeks-to-build-map-of-human-brain.html.

[88] *See, e.g.*, *Biomarkers Can Predict Risk for Alzheimer's Several Years Before Symptoms Appear*, NAT'L INST. ON AGING (June 15, 2013), http://www.nia.nih.gov/newsroom/announcements/2013/06/biomarkers-can-predict-risk-alzheimers-several-years-before-symptoms.

[89] *See* Press Release, Am. Inst. of Physics, Detecting Alzheimer's Early: Optical Scientists, Psychiatrists Develop Minimally Invasive Eye Test for Alzheimer's (Dec. 1, 2005), *available at* https://retainyourmindfulness.wordpress.com/2011/05/24/sciencedaily-your-source-for-the-latest-research-news-and-science-breakthroughs-updated-daily-science-video-share-blog-print-bookmark-email-detecting-alzheimers-early-optical-scientists-p/ ("If we can get treatments early . . . we can slow the disease to the point where we've effectively cured it.").

[90] *See* James Woolley & Philip McGuire, *Neuroimaging in Schizophrenia: What Does It Tell the Clinician?*, 11 ADVANCES PSYCHIATRIC TREATMENT 195, 197–99 (2005), *available at* http://apt.rcpsych.org/content/11/3/195.full.pdf.

dementia cases.[91] It is a progressive disease that gradually worsens, and it is the sixth leading cause of death in the United States, indicating a high societal disease burden.[92] Although currently there is no cure for Alzheimer's disease, the ability to predict it could have important implications in terms of preparing patients and their families, especially since the affected patient will eventually become dependent on his or her caretakers.[93] One of the prevailing theories behind Alzheimer's disease is the amyloid hypothesis, which proposes that beta-amyloid deposits in the brain fundamentally cause the disease.[94] Neuroimaging has allowed researchers to view the buildup of amyloid plaques in the brain, which may predict the onset of Alzheimer's disease.[95] Additionally, the analysis of cerebrospinal fluid (CSF), found around and inside the brain and spinal cord, may also serve a predictive function since the amyloid fragments that make up brain plaques are also present and can be identified in CSF.[96] Research has shown that these biomarkers are very good predictors for identifying which patients will develop cognitive impairment and how soon the impairment will set in,[97] though it is important to note that these predictions are far from perfect.[98]

Another Alzheimer's disease development on the horizon is the use of a simple, noninvasive eye test to detect changes in the widths of certain blood vessels in the retina, which correspond to the amount of plaque buildup in the brain.[99] An eye test would be far less expensive than a brain scan and could

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[91] *See What Is Alzheimer's?*, ALZHEIMER'S ASS'N, http://www.alz.org/alzheimers_disease_what_is_alzheimers.asp (last visited Apr. 27, 2015).

[92] *See id.*

[93] *See, e.g.*, Paula Spencer Scott, *What is Alzheimer's Disease*, CARING, https://www.caring.com/articles/alzheimers-disease (last visited Apr. 27, 2015).

[94] *See* Karran et al., *supra* note 77.

[95] PET scans are the most common form of neuroimaging used to detect amyloid plaque buildup in the brain. *See* Press Release, Am. Acad. of Neurology, *supra* note 6; Press Release, Perelman School of Med. at the Univ. of Pa., *supra* note 6.

[96] Geert De Meyer et al., *Diagnosis-Independent Alzheimer Disease Biomarker Signature in Cognitively Normal Elderly People*, 67 ARCHIVES NEUROLOGY 949, 951 (2010).

[97] *See supra* notes 95–96.

[98] While predictive biomarkers show great promise, they are not perfect predictors of disease. Some people who exhibit brain scans that would seem to indicate the extensive presence of a disease biomarker function perfectly well and will never develop the disease, while others with scans showing identical biomarker levels are very impaired. *See* Richard Mayeux, *Biomarkers: Potential Uses and Limitations*, 1 NEURORX 182, 186 (2004), *available at* http://www.ncbi.nlm.nih.gov/pmc/articles/PMC534923/pdf/neurorx001000182.pdf.

[99] *See* Press Release, Cognoptix, Cognoptix Drug/Device Test Identifies Alzheimer's Disease via *Beta Amyloid* Signature in the Eyes in a 10-Subject Proof-of-Concept Trial (Jan. 3, 2013), *available at* http://www.businesswire.com/news/home/20130103005076/en/Cognoptix-DrugDevice-Test-Identifies-Alzheimer%E2%80%99s-Disease-Beta#.UwFFxUJdX68; Press Release, *supra* note 89.

serve as a more accessible tool for Alzheimer's disease prediction in the near future.[100] These studies suggest that researchers may now have the ability to detect and predict the onset of Alzheimer's disease years before any symptoms of memory loss or cognitive decline manifest.[101] And there is evidence to support that predictive neuroscience advances in the field of Alzheimer's disease may even have greater predictive value than the corresponding genetic test, which hints at exciting research opportunities.[102]

Neuroscience advances that predict other neurodegenerative diseases, such as Huntington's disease and Parkinson's disease, are following in the footsteps of Alzheimer's disease research, and it may soon be possible to use neuroscience techniques to predict their onset before symptoms manifest as well. Preliminary studies indicate that individuals with Huntington's disease may have measurable characteristics indicating decline before actual diagnosis and symptom onset occur.[103] For example, current predictive research for Huntington's disease indicates that magnetic resonance imaging (MRI) measurement of gray-matter volume in the brain has shown promise in predicting the clinical diagnosis of Huntington's disease before symptom onset.[104]

Similarly, neuroscience advances may soon contribute to the prediction of Parkinson's disease onset and progression. Currently, it is not possible to predict the onset or progression of Parkinson's with any certainty.[105] However, neuroimaging has provided evidence of decreased volume in the cortex of the

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[100] *See* Press Release, *supra* note 99.

[101] However, Alzheimer's disease has no known cure, so early detection would only help prepare the individual and their family for what is to come, but wouldn't be able to prevent it. *See* Michael C. Purdy, *Alzheimer's Markers Predict Start of Mental Decline*, WASH. UNIV. ST. LOUIS NEWSROOM (May 13, 2013), https://news.wustl.edu/news/Pages/25412.aspx.

[102] A brain scan for amyloid plaques is a better predictor of Alzheimer's disease than getting a genetic test. *Brain Plaque vs. Alzheimer's Gene*, CHI. TRIB., Sept. 29, 2013, http://articles.chicagotribune.com/2013-09-29/lifestyle/sns-201302191850--tms--harvhltl69l0313e-20130219_1_apoe4-brain-scan-alzheimer.

[103] Nat'l Insts. of Health, *Neurobiological Predictors of Huntington's Disease (PREDICT-HD)*, CLINICALTRIALS, http://clinicaltrials.gov/show/NCT00051324 (last updated Dec. 4, 2014).

[104] *See* Jan Kassubek et al., *Thalamic Atrophy in Huntington's Disease Co-varies with Cognitive Performance: A Morphometric MRI Analysis*, 15 CEREBRAL CORTEX 846 (2005), *available at* http://cercor.oxfordjournals.org/content/15/6/846.full.pdf+html; *see also* Mario Quarantelli et al., *Default-Mode Network Changes in Huntington's Disease: An Integrated MRI Study of Functional Connectivity and Morphometry*, 8 PLOS ONE, no. 8, art. no. 72159, 2013, at 1, http://www.plosone.org/article/fetchObject.action?uri=info%3Adoi%2F10.1371%2Fjournal.pone.0072159&representation=PDF; D. Stoffers et al., *Contrasting Gray and White Matter Changes in Preclinical Huntington's Disease: An MRI Study*, 74 NEUROLOGY 1208 (2010), *available at* http://www.aronlab.org/Pubs/Stoffers_Neurology.pdf.

[105] *See* Karen Weintraub, *Researchers Take Step Toward Predicting Parkinson's*, USA TODAY, Aug. 26, 2013, http://www.usatoday.com/story/news/nation/2013/08/26/parkinsons-predictor/2701019/.

brain and decreased resting metabolic activity that appear prior to the onset of Parkinson's dementia.[106] Additionally, biomarkers found in the CSF show promise in predicting Parkinson's onset and the severity of motor dysfunction.[107] Though further research is necessary, it may soon be possible to use neuroimaging and CSF biomarkers to predict the onset and progression of cognitive impairment and motor dysfunction in Parkinson's patients.

In addition to predictive implications for neurodegenerative diseases, neuroscience advances also have predictive implications for psychotic mental disorders, such as schizophrenia. There is compelling evidence from neuroimaging to suggest that schizophrenia is a brain disease.[108] Neuroimaging techniques have highlighted structural brain changes that appear to be correlated with the symptoms of schizophrenia, including enlargement of the lateral ventricles in the brain, undersized temporal lobe volume, and prefrontal lobe abnormalities.[109] Additionally, functional magnetic resonance imaging (fMRI) studies, which measure brain activity as opposed to brain structure,[110] suggest that brain activity in response to certain cognitive demands may appear different for at-risk individuals.[111] These advances may make it possible to predict disease course, response to treatment, and potential for relapse.[112] Neuroimaging has the potential to elucidate much in the mental-illness sphere in particular because currently, psychiatric disorders, such as schizophrenia, are diagnosed solely via symptom-based diagnostic criteria.[113] If reliable and identifiable structural or functional brain changes can be correlated with schizophrenia symptoms and onset, it follows that neuroimaging may make it

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106 *See* Lisa C. Silbert & Jeffrey A. Kaye, *Neuroimaging and Cognition in Parkinson's Disease Dementia*, 20 BRAIN PATHOLOGY 646 (2010), *available at* http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3327506/.

107 *See* Jian Wang et al., *Biomarkers of Parkinson's Disease: Current Status and Future*, 18 DRUG DISCOVERY TODAY 155, 159, 160 nn.3–4.

108 *See* Peter F. Buckley, *Neuroimaging of Schizophrenia: Structural Abnormalities and Pathophysiological Implications*, 1 NEUROPSYCHIATRIC DISEASE & TREATMENT 193, 202 (2005), *available at* http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2416751/pdf/ndt-0103-193.pdf.

109 *See* Shawn J. Kile, *Neuropsychiatric Update: Neuroimaging Schizophrenia*, 40 PSYCHOPHARMACOLOGY BULL. 156 (2007).

110 *What Is fMRI?*, CTR. FOR FUNCTIONAL MRI, http://fmri.ucsd.edu/Research/whatisfmri.html (last visited Apr. 27, 2015).

111 *See* Paul C. Fletcher, Editorial, *Functional Neuroimaging of Schizophrenia: From a Genetic Predisposition to the Emergence of Symptoms*, 127 BRAIN 457, 457 (2004), *available at* http://brain.oxfordjournals.org/content/127/3/457.full.pdf+html.

112 *See* Woolley & McGuire, *supra* note 90, at 197–99.

113 *See* AM. PSYCHIATRIC ASS'N, DSM-5: DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS 87 (5th ed. 2013).

possible to predict schizophrenia before any of these diagnostic symptoms manifest.

Another predictive neuroscience application on the horizon relates to multiple sclerosis (MS). MS affects as many as 2.5 million people throughout the world and is the most common cause of progressive disability found in young adults.[114] MS is a disease where the body attacks the central nervous system of the patient.[115] As a result of this attack, myelin, the protective tissue that surrounds the nerve cells of the brain, spinal cord, and optic nerves, is damaged.[116] Neuroimaging technologies are helping researchers make strides toward the prediction of MS through the discovery of visible differences in gray matter in the brain that correlate with the disease, which are detectable in the very earliest stages of MS.[117] Although MS research initially focused on white matter, MRI technology has enabled researchers to better visualize gray matter, and it has become clear that gray matter pathology plays an important role in the development of MS.[118] Studies have shown that gray matter atrophy in the thalamus of the brain has been associated with MS.[119] According to one researcher, "[t]halamic atrophy is an ideal MRI biomarker because it's detectable at a very early stage . . . . It has very good predictive value."[120] Additionally, MRI studies have shown that darker gray matter, suggestive of increased iron deposits, may be another biomarker for MS, which may allow physicians to more accurately identify patients at risk for developing the disease.[121] Given the rapid advances in MS research with the help of MRI technology, it may soon be possible to predict who will develop MS prior to any symptom onset.

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[114] Press Release, Wiley, Multiple Sclerosis Progression Can Be Predicted With MRI (Nov. 5, 2008), *available at* http://www.eurekalert.org/pub_releases/2008-11/w-msp110508.php.

[115] *Definition of MS*, NAT'L MULTIPLE SCLEROSIS SOC'Y, http://www.nationalmssociety.org/about-multiple-sclerosis/what-we-know-about-ms/what-is-ms/index.aspx (last visited Apr. 27, 2015).

[116] *Id.*

[117] *See* Justin Morris Honce, *Gray Matter Pathology in MS: Neuroimaging and Clinical Correlations*, 2013 MULTIPLE SCLEROSIS INT'L, art. no. 627870, at 1, http://www.hindawi.com/journals/msi/2013/627870/; *see also* Elisabeth Andreadou, *Neuroimaging in Multiple Sclerosis*, *in* NEUROIMAGING—CLINICAL APPLICATIONS 317 (Peter Bright ed., 2012), *available at* http://cdn.intechopen.com/pdfs/31418/InTech-Neuroimaging_in_multiple_sclerosis.pdf; Nancy L. Sicotte, *Neuroimaging in Multiple Sclerosis: Neurotherapeutic Implications*, 8 NEUROTHERAPEUTICS 54, 58 (2011), *available at* http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3075734/pdf/13311_2010_Article_8.pdf.

[118] *See* Honce, *supra* note 117, at 1.

[119] Carola Finch, *MRI Can Predict Onset of Multiple Sclerosis*, EXAMINER (Apr. 23, 2013, 8:53 PM MST), http://www.examiner.com/article/mri-can-predict-onset-of-multiple-sclerosis; *see* Honce, *supra* note 117, at 15.

[120] Finch, *supra* note 119 (internal quotation marks omitted).

[121] *See* Press Release, *supra* note 114.

The recent emergence of this predictive neuroscience technology may soon make it possible to predict a variety of diseases before their clinical onset. Although only Alzheimer's disease can currently be predicted with any relative certainty using neuroimaging techniques, a great volume of research is being done on many other diseases, which are following closely in the footsteps of Alzheimer's disease research in the search for predictive correlates that will allow researchers to identify those who may develop diseases years before any symptoms manifest. These health-related benefits raise exciting new possibilities, but they are accompanied by costs because this predictive information also has the potential to be the basis for unfair health-related discrimination.

IV. THE NEED FOR PROTECTION FOR PREDICTIVE NEUROSCIENCE INFORMATION

Predictive neuroscience information should be protected in the same way that genetic information is protected under GINA and ACA from health insurance discrimination. As predictive neuroscience advances become increasingly part of the health and medical landscape, the potential to harness this neuroscience information for good is countered by the potential to misuse the information in order to unfairly discriminate against people based upon its predictive value. This is problematic because while predictive information may serve many useful purposes, it is far from perfect.[122] The potential to misinterpret and misuse predictive information is high, and as such, this information deserves protection to ensure that health insurers do not unfairly discriminate against those who undergo these predictive neuroscience tests.

This Part will discuss existing discrimination protections in greater depth, with each provision narrowing the range of excluded areas from protection. This analysis will show that after all the existing provisions and protections have been applied and considered, there remains a narrow gap in the discrimination protections where predictive neuroscience information in the large-group and self-insured health insurance markets is still unprotected. After identifying this gap in protection, this Part will discuss why this gap is problematic and then conclude by suggesting potential ways to close this gap by extending appropriate protection to predictive neuroscience information.

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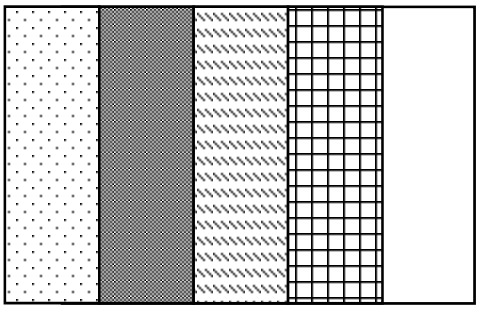
[122] *See* Mayeux, *supra* note 98, at 186–87.

A. A Gap in Existing Health Insurance Antidiscrimination Provisions

This section will demonstrate that there is a gap in existing health insurance discrimination protections by discussing various provisions of ACA. Each provision will narrow the area excluded from protection. Once the applicable protections have been applied, this section will show that there is still a small area where predictive neuroscience information in the large-group and self-insured health insurance markets is excluded. The following diagram is an illustrative overview of the analysis:

FIGURE



Key

Guaranteed Issue and Guaranteed Renewal provisions: protect against all enrollment discrimination but not against rate discrimination.

Preexisting Condition provision: protects against existing condition discrimination but not against discrimination on the basis of any predictive information.

Health Status provision: protects against predictive genetic information discrimination but not against predictive neuroscience information discrimination.

Rating Factors provision: prevents individual and small-group markets from considering predictive neuroscience information in setting rates but doesn't prevent large-group and self-insured markets from considering the same information.

The gap: no protection from rate discrimination for predictive neuroscience information in the large-group and self-insured markets.

1. Guaranteed Issue and Guaranteed Renewal

ACA's protections are expansive and mark a significant step forward in limiting health insurance discrimination. In particular, ACA's guaranteed issue and guaranteed renewal provisions ensure that health insurance will be accessible to all and take away any opportunity for health insurers to

discriminate in deciding who they will cover.[123] These provisions apply to both the individual and group markets, meaning that health insurers are required to accept every employer and individual who applies for health insurance and must renew that coverage at the insured's election.[124] This is important because health insurers previously could discriminate in two main areas. First, they could discriminate in deciding whom to accept for enrollment. Second, they could discriminate in deciding what rates to charge those they had accepted for enrollment and how extensive to make the coverage offered. ACA's guaranteed issue and guaranteed renewal provisions take away the discretion of health insurers to discriminate in the first scenario since the provisions require them to accept everyone who applies for enrollment and require that the enrollment continue as long as the insured wishes.[125] Consequently, the opportunity for discrimination is now limited only to the second scenario, in which health insurers discriminate by setting disparate rates for their enrollees.

2. Preexisting Conditions

Within this second scenario, ACA offers further discrimination protections with its preexisting condition provision, which states that health insurers are not permitted to impose any preexisting condition exclusions with respect to the coverage they offer.[126] A preexisting condition is "anything for which symptoms were present and a prudent person would have sought treatment."[127] This definition makes clear that ACA's preexisting condition protections only extend to conditions after the onset of symptoms. This has the effect of excluding any predictive information from ACA's preexisting condition protections since, by definition, predictive information applies when one does not yet have a condition but, for one reason or another, has a greater likelihood of developing a condition in the future. Accordingly, since health insurers are not permitted to exclude existing conditions from coverage, ACA protects those with existing conditions from discrimination based on their condition.[128] However, the preexisting condition provision fails to address predictive information since predictive information is clearly outside the definition of an existing condition under ACA. So while the preexisting condition provision

123 *See* 42 U.S.C. §§ 300gg-1(a) to (b), 300gg-2(a) (2012).
124 *Id.*
125 *Id.*
126 *See id.* § 300gg-3(a).
127 *Supra* note 65 and accompanying text.
128 42 U.S.C. § 300gg-3.

narrows the opportunity for health insurance discrimination by protecting those with existing conditions, it leaves all predictive information unprotected.[129]

3. Health Status

A further protection afforded by ACA is the health status provision, which prohibits health insurers from discriminating based on the health status of an enrollee.[130] This provision sets forth nine health-status-related factors: (1) health status, (2) medical condition, (3) claims experience, (4) receipt of health care, (5) medical history, (6) genetic information, (7) evidence of insurability, (8) disability, and (9) any other health-status-related factor determined appropriate by the Secretary of Health and Human Services.[131]

Currently, predictive health information can come in the form of genetic or neuroscience information.[132] However, the sixth ACA health-status-related factor explicitly closes the door on health insurance discrimination on the basis of any genetic information,[133] seemingly in acknowledgment of Title I of GINA, which was enacted two years prior to ACA.[134] It appears that Congress intended to embody and reinforce Title I of GINA, which prevents health insurers from denying coverage or charging higher premiums to a healthy person based solely on a genetic predisposition to developing a disease.[135]

Additionally, when read in light of ACA's other provisions and taking into consideration the explicit mention of genetic information as a health-status-related factor,[136] Congress actually expanded the scope of existing genetic information protection markedly in the health insurance realm. An important limitation of GINA prior to ACA was that its protections ceased once a condition had become manifest.[137] GINA defines manifest as any condition that "could reasonably be detected by a health care professional with appropriate training and expertise in the field of medicine involved."[138] This limited GINA's protections to predictive genetic information since once the

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129 *See id.*

130 *See id.* § 300gg-4(a).

131 *Id.*

132 See *supra* Parts I and III for more detail on how genetic and neuroscience information can be used to predict diseases.

133 42 U.S.C. § 300gg-4(a)(6).

134 GINA STATEMENT OF ADMINISTRATIVE POLICY, *supra* note 27.

135 *See* 42 U.S.C. § 300gg-3(a).

136 *See id.* § 300gg-4(a)(6).

137 *See* Prince & Berkman, *supra* note 47, at 655.

138 29 U.S.C. § 1191b(d)(7)(B)(ii) (2012).

condition had become manifest, that individual was no longer protected from health insurance discrimination under GINA.

Interestingly enough, with the passage of the ACA, GINA's once-critical disease-manifestation line has seemingly been rendered irrelevant. The definition of preexisting condition ("anything for which symptoms were present and a prudent person would have sought treatment")[139] and manifest condition (any condition that "could reasonably be detected by a health care professional with appropriate training and expertise in the field of medicine involved")[140] are almost analogous and provide a seamless transition from the predictive genetic protections of GINA to the preexisting condition protections of ACA. For example, if an individual has a genetic predisposition to developing Alzheimer's disease, under GINA, health insurers may not use this information to unfairly discriminate against him or her in making coverage and enrollment decisions or in setting his or her premium rates.[141] Once the Alzheimer's disease "could reasonably be detected by a health care professional with appropriate training and expertise in the field of medicine involved,"[142] GINA's protections would cease. But ACA picks up where GINA's protections leave off, extending its preexisting condition protections to that same individual with Alzheimer's disease once "symptoms [are] present and a prudent person would [seek] treatment."[143] It is difficult to imagine a scenario in which an individual would lose GINA's protections once his or her condition has manifested and ACA's preexisting condition protections wouldn't immediately take effect.

In effect, ACA's previously discussed protections and GINA's genetic information protections demonstrate that there is a narrow gap where predictive neuroscience information is the only information excluded from protection from health insurance discrimination. In analyzing the other health-status-related factors, it becomes clear that none address predictive information. The first factor, health status, is very vague because it appears to have no force of its own, as its definition is simply inclusive of the second through the eighth factors.[144] The second through the fifth factors, in addition

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139 *See supra* note 65 and accompanying text.

140 42 U.S.C. § 300gg-91(d)(17)(B)(ii).

141 29 U.S.C. § 1182(b)–(d); 42 U.S.C. §§ 300gg-1(b) to (c), (d)(19), 300gg-53(a) to (d).

142 42 U.S.C. § 300gg-91(d)(17)(B)(ii).

143 *See supra* note 65 and accompanying text.

144 *See* 42 U.S.C. § 300gg-4(a)(1); *Health Status*, HEALTHCARE.GOV, https://www.healthcare.gov/glossary/health-status/ (last visited Apr. 27, 2015).

to the eighth factor (medical condition, claims experience, receipt of health care, medical history, and disability) do not have anything to do with predictive information.[145] And the sixth factor, genetic information, was previously discussed.[146] This leaves only the seventh factor, evidence of insurability, to consider.[147]

Evidence of insurability has been interpreted under governmental guidance from the U.S. Department of Labor to refer to "[c]onditions arising from acts of domestic violence as well as participation in activities like motorcycling, snowmobiling, all-terrain vehicle riding, horseback riding, and skiing."[148] When read in light of this guidance, it is clear that evidence of insurability does not refer to predictive information. This lends itself to the conclusion that while Title I of GINA and the sixth health-status-related factor protect predictive genetic information,[149] ACA's health status provision does not protect predictive neuroscience information from health insurance discrimination.

4. Rating Factors

While ACA's preexisting condition and health status provisions do not protect predictive neuroscience information from health insurance discrimination, ACA does protect a certain class of the insured through its rating factors.[150] For the individual and small-group markets, ACA permits only four factors to be considered when health insurers set premium rates.[151] These four rating factors are (1) whether the plan covers an individual or a family, (2) the rating area, (3) age, and (4) tobacco use.[152] This limitation means that regardless of the fact that predictive neuroscience information is not protected specifically from health insurance discrimination, ACA's rating factors prohibit health insurers from considering anything other than these four factors when setting premium rates in the individual and small-group markets. However, ACA's rating factors specifically do not apply to the large-group and

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145 *See* 42 U.S.C. § 300gg-4(a)(2) to (5), (8).

146 *See supra* note 133 and accompanying text.

147 *See* 42 U.S.C. § 300gg-4(a)(7).

148 *FAQs About Portability of Health Coverage and HIPAA*, *supra* note 51 (discussing HIPAA's health status factors, which were amended to reflect ACA's protections and are based off of ACA's health status factors).

149 *See supra* notes 133, 141 and accompanying text.

150 42 U.S.C. § 300gg(a)(1)(A).

151 *See id.*

152 *Id.*

self-insured markets, leaving predictive neuroscience information in those groups unprotected.[153]

B. Why this Gap Is Problematic

As there are no further antidiscrimination provisions on point, a careful analysis of the existing health insurance discrimination protections reveals a narrow gap in protection for predictive neuroscience information in the large-group and self-insured markets. This section will discuss why this gap is significant and why it should be closed.

While the gap in protection appears narrow, it is a critical gap that requires attention because a significant part of the overall insured population falls within this gap. Employers who have over fifty employees most commonly utilize large group plans.[154] The ten largest employers in America in 2013 alone employed over 5.6 million employees,[155] so it is not difficult to see that many people could potentially be affected by this gap. In addition, the gap in protection is problematic because, with the increasing availability of predictive neuroscience technologies and information, it gives health insurers a way to undercut the protections provided by GINA and ACA. Furthermore, if health insurers are prohibited from discriminating on the basis of genetic information, they are highly incentivized to turn to any alternative legal means of obtaining the same information to serve the same purpose.[156]

1. Predictive Neuroscience Information Could Be Used to Undercut GINA

On the one hand, predictive neuroscience information is closely analogous to predictive genetic information. Genetic advances and the power to harness the information provided by such advances raised problems that ultimately drove Congress to enact GINA.[157] Many of those same problems are posed by predictive neuroscience information with respect to discrimination. For example, one of the most widespread considerations behind the passage of GINA was the desire to encourage and advance scientific research and ensure that individuals would feel comfortable availing themselves of genetic testing

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153 *See id.*

154 *See Small and Large Business Health Insurance: State & Federal Roles*, NCSL, http://www.ncsl.org/research/health/small-business-health-insurance.aspx (last updated Feb. 1, 2015).

155 Alexander E.M. Hess, *The 10 Largest Employers in America*, USA TODAY, Aug. 22, 2013, http://www.usatoday.com/story/money/business/2013/08/22/ten-largest-employers/2680249/.

156 *See supra* note 9 and accompanying text.

157 *See* Roberts, *supra* note 4, at 471–74; Resnick, *supra* note 41; *The History of GINA*, *supra* note 42.

without fear of that same helpful information being used against them in a discriminatory manner.[158] Identical considerations lend support to the protection of predictive neuroscience information. As predictive neuroscience technologies continue to advance, the use of this information will become more common. But in order for research to take place, and targeted therapies and earlier disease interventions to become a reality, subjects must be willing to participate in studies that may reveal predictions about their disease development in the future. And in order to find willing subjects, there must be adequate protection for those subjects against misuse of the predictive information gleaned from the research studies.

This same logic holds true for the encouragement of early predictive neuroscience testing for early intervention purposes in individual patients. The ability to predict a disease before any symptom onset facilitates research and development of earlier treatments that may delay or minimize the harm imposed by that disease on that individual.[159] For example, Alzheimer's disease prediction before symptom onset could allow for future treatment to target the disease before irreversible brain damage or mental decline occurs.[160]

While genetic information and predictive neuroscience information pose many of the same problems, predictive neuroscience information is not protected from health insurance discrimination. This protection gap is problematic because, since genetic information and predictive neuroscience information are analogous in many ways, it is possible for health insurers to glean much of the same information from predictive neuroscience information that they could get from genetic information.[161] Since Title I of GINA prohibits health insurers from requesting or requiring genetic information or genetic testing, acquiring or using genetic information for medical underwriting purposes, and treating genetic information as a preexisting condition,[162] health insurers may be incentivized to get around the antidiscrimination provisions of GINA and find alternative ways of evaluating their risk in insuring individuals.[163] Health insurers could easily turn to other predictive

158 *See supra* note 157.

159 *See* Cynthia A. Lemere & Eliezer Masliah, *Can Alzheimer Disease Be Prevented by Amyloid-β Immunotherapy?*, 6 NATURE REVS. NEUROLOGY 109, 116 (2010).

160 *See id.*

161 *See supra* note 6 and accompanying text.

162 *See* 29 U.S.C. § 1182(c)(1), (d)(1)–(2) (2012).

163 *See supra* note 9 and accompanying text.

technologies that are not specifically afforded the same protections as genetic information to get roughly the same information.

For example, the apoE4 gene is the strongest risk gene known for predicting Alzheimer's disease and is implicated in about 20%–25% of all Alzheimer's cases.[164] Under GINA, health insurers cannot ask whether a patient has this form of the gene, require them to undergo testing for the gene, or use information about the gene in making enrollment, coverage, or premium decisions, as long as they do not yet exhibit symptoms sufficient for a diagnosis of Alzheimer's disease.[165] However, it has recently become possible to alternatively predict Alzheimer's disease through neuroimaging of amyloid plaques and analysis of amyloid fragments in the CSF.[166] Because this predictive neuroscience information is not afforded any protection against health insurance discrimination in the large-group and self-insured markets, health insurers could get around GINA's protections by turning to predictive neuroscience information that may tell them roughly the same thing. It is crucial that this predictive neuroscience information be protected so health insurers cannot render those genetic protections obsolete by merely circumventing the protection and using roughly analogous predictive neuroscience information as a substitute for the genetic information they are prohibited from using.

2. *Predictive Neuroscience Information Provides a Greater Incentive for Abuse than Predictive Genetic Information*

Although predictive neuroscience information is in many ways analogous to genetic information, it is also unique and raises its own issues that merit consideration. On the one hand, predictive neuroscience information, and neuroimaging in particular, show structural and functional changes occurring in the brain or elsewhere. These changes may be representative of disease in the future if neuroscientists can develop strong enough and reliable enough correlations between the neuroimaging information findings and future disease onset.[167] Once neuroscientists find a good biomarker, which can include

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164 *What We Know Today About Alzheimer's Disease*, *supra* note 6.

165 *See supra* notes 28–30.

166 *See supra* notes 95–96.

167 *See* Christopher J. Honey, Jean-Philippe Thivierge & Olaf Sporns, *Can Structure Predict Function in the Human Brain?*, 52 NEUROIMAGE 766, 774 (2010), *available at* http://chialvo.org/Curso/UBACurso/DIA9/Papers/HoneyStructureFunction.pdf; *Alzheimer's and Dementia Testing for Earlier Diagnosis*, ALZHEIMER'S ASS'N, http://www.alz.org/research/science/earlier_alzheimers_diagnosis.asp (last visited Apr. 27, 2015).

anything from blood or spinal fluid proteins to brain changes visible through imaging, and the biomarker has been validated, which means its predictive value has been confirmed through studies, they can then use this predictive information in a variety of health-related settings.[168] However, it is unclear in many cases what the relationship is between many of these identifiable changes and disease manifestation.[169] Researchers are constantly seeking to elucidate how these visible neuroimaging changes may contribute to disease and to determine whether they contribute to the cause of the disease or whether they are effects of the underlying disease.[170] Furthermore, once the biomarkers are discovered and validated, additional research must be carried out to determine the correlation's reliability and to utilize the relationship between the visible changes and the disease to benefit future research and treatment for patients who may develop, or are currently living with, the disease.[171] As neuroscience research continues to advance and the pathology of diseases are better understood, it is likely that predictive neuroscience information will get swallowed into what is considered to be disease onset, allowing for earlier detection of diseases. But until technology and research advance to that extent, predictive neuroscience information can be very valuable on its own because it shows actual identifiable changes occurring in the brain and elsewhere that are predictive of disease onset in the future.[172]

On the other hand, genetic information is different because, in most instances, it is not actually predictive of disease.[173] It merely indicates that an individual has a higher chance of getting a disease due to the genes he or she possesses.[174] Using genetic information to predict disease onset is very

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168 *Alzheimer's and Dementia Testing for Earlier Diagnosis*, *supra* note 167.

169 *See* Giuseppe Verdile et al., *The Role of Beta Amyloid in Alzheimer's Disease: Still a Cause of Everything or the Only One Who Got Caught?*, 50 PHARMACOLOGICAL RES. 397, 397 (2004), *available at* http://ac.els-cdn.com/S104366180400091X/1-s2.0-S104366180400091X-main.pdf?_tid=98292002-464b-11e3-9a5b-00000aacb35d&acdnat=1383677804_8f3e2d8ba040216ca574e38b192ba85d (explaining the debate between Alzheimer's researchers as to whether the beta-amyloid protein is a cause or an effect of the disease).

170 *See id.*

171 *See Alzheimer's and Dementia Testing for Earlier Diagnosis*, *supra* note 167.

172 *See* Judy Illes & Matthew Kirschen, Editorial, *New Prospects and Ethical Challenges for Neuroimaging Within and Outside the Health Care System*, 24 AM. J. NEURORADIOLOGY 1932 (2003).

173 There are exceptions, though, where genetic information is fully predictive of the disease. For example, Huntington's disease is a prime example of a one hundred percent predictive value, since if you have the Huntington's gene, it is only a matter of time before you develop the disease. *See What Is Huntington's Disease (HD)?*, HUNTINGTON'S DISEASE SOC'Y OF AM., http://hdsa.org/what-is-hd/ (last visited Apr. 27, 2015).

174 U.S. Nat'l Library of Med., *What Does It Mean to Have a Genetic Predisposition to a Disease?*, GENETICS HOME REFERENCE, http://ghr.nlm.nih.gov/handbook/mutationsanddisorders/predisposition (last updated Apr. 20, 2015).

complex. First, having the gene puts an individual at higher risk for the relevant disease, much like having a particular biomarker puts him or her at higher risk.[175] But merely having the gene is, in most cases, not sufficient to manifest the disease.[176] The gene almost always needs to interact with the environment and be switched on or off, so to speak, for disease manifestation to occur.[177] In most cases, genetic testing can only determine whether a particular gene or genes are present, which in turn signals a predisposition for a certain disease or condition associated with that gene or genes.[178] Whether that predisposition results in the disease or condition in the long run ultimately depends on myriad factors that cannot be predicted.[179]

For example, the apoE4 gene implicated in Alzheimer's disease increases an individual's risk for developing the disease.[180] A carrier with one copy of the apoE4 gene is three times more likely than someone who doesn't have any copies of apoE4 to develop Alzheimer's disease.[181] And a carrier with two copies of the apoE4 gene is roughly twelve times more likely than someone who doesn't have any copies of the gene to develop Alzheimer's disease.[182] But even with the increased risk that accompanies being a carrier of the apoE4 gene, it is impossible to know how that gene will interact with that individual's environment and whether that gene–environment interaction will switch on the gene in such a way that results in Alzheimer's disease, as is evident in the fact that the apoE4 gene is far from determinative when it comes to developing Alzheimer's disease.[183] One-third of Alzheimer's disease patients do not carry any copies of the apoE4 gene, and some people who carry two copies of the apoE4 gene never develop the disease.[184]

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175 *See infra* notes 180–84 and accompanying text.

176 For a more in-depth discussion of gene–environment interaction, see Ruth Ottman, *Gene–Environment Interaction: Definitions and Study Designs*, 25 PREVENTATIVE MED. 764 (1996).

177 *See id.*

178 *See* Walter C. Willett, *Balancing Life-Style and Genomics Research for Disease Prevention*, 296 SCIENCE 695 (2002), *available at* http://www.sciencemag.org/content/296/5568/695.full.pdf.

179 *See id.* There are certain genetic conditions that do not need to interact with the environment to be "switched on." *See supra* note 173.

180 *See* Jim Schnabel, *Why Does apoE4 Make Alzheimer's More Likely?*, DANA FOUND. (July 7, 2011), http://www.dana.org/News/Details.aspx?id=43163.

181 *Id.*

182 *Id.*

183 *See* NAT'L INST. ON AGING, ALZHEIMER'S DISEASE GENETICS FACT SHEET 5 (2011), http://www.nia.nih.gov/sites/default/files/alzheimers_disease_genetics_fact_sheet_0.pdf.

184 *See id.* at 4.

If neuroimaging information shows identifiable structural and functional changes that differ from a healthy person's scans,[185] then as long as that information is strongly and fairly reliably correlated with disease manifestation and onset, neuroscience information may be even more useful than genetic information is in a predictive sense. Genes need to be switched on and off by the environment, which is very unpredictable.[186] Brain scans, however, show actual changes in the brain that may be indicative of a cause or effect of some disease that will develop or manifest down the road.[187]

A recent study in the journal *Neurology* found that a PET scan showing high levels of beta-amyloid was a better predictor of future Alzheimer's disease onset than a genetic test for the apoE4 gene.[188] If neuroscience information is more useful than genetic information in predicting disease onset, there is a strong argument that it is more deserving of discrimination protection because it is even more likely to be abused than predictive genetic information.

C. Closing the Gap

Closing the gap in protection could be achieved fairly easily. There are a number of acceptable solutions. This section will briefly suggest a few of the simplest ways that this could be achieved.

The easiest way to close the gap would be to include predictive neuroscience information under health-status-related factors in the same way that genetic information is already listed in ACA.[189] This would quickly and effectively close the gap by covering all predictive information, preventing health insurers from discriminating on the basis of any predictive information instead of just genetic information, as ACA currently reads.[190] There are two ways to include predictive neuroscience information under health-status-related factors in ACA. First, ACA could be amended to add another health-status-related factor. The alternative, and far more likely solution, would be for the Secretary of Health and Human Services to act under the final open health-status-related factor in ACA by determining that predictive neuroscience information should appropriately be included as a

185 *See* Illes & Kirschen, *supra* note 172.
186 *See* Ottman, *supra* note 176.
187 *See* Honey et al., *supra* note 167.
188 *Brain Plaque vs. Alzheimer's Gene*, *supra* note 102.
189 *See* 42 U.S.C. § 300gg-4(a)(6) (2012).
190 *See id.*

factor.[191] This solution would be the quickest and most practical option since the Secretary could act in her agency capacity without congressional action.[192]

Other more complex methods would include extending Title I of GINA to apply not only to genetic information but also to predictive neuroscience information, or drafting a new GINA-like bill specifically for predictive neuroscience information.[193] Though more extensive effort would be necessary to pursue either of these two options, there could be some merit in considering them. However, given the relative ease and practicality of the health-status-related factor option, implementation of these alternative options seems unlikely.

CONCLUSION

The lack of protection for predictive neuroscience information represents a gap in the existing health insurance discrimination protections found in HIPAA, GINA, and ACA. This gap allows health insurers to use predictive neuroscience information to discriminate when determining premium rates and coverage in the large-group and self-insured markets. Despite the expansive protections afforded by ACA and GINA for predictive genetic information, the failure to extend the full measure of this protection to predictive neuroscience information is highly problematic.

Health insurance discrimination has received significant attention in recent years with the passage of GINA and ACA in 2008 and 2010, respectively. While their provisions have expanded protections against unfair health insurance discrimination markedly, there remains a gap in protection for predictive neuroscience information in the large-group and self-insured markets. The reason this gap is significant is because it allows health insurers to undercut existing provisions protecting predictive genetic information by turning to unprotected predictive neuroscience information, which can effectively tell them the same thing, and in some cases is even more reliable from a predictive standpoint than genetic information. It is important that the gap in protection be addressed as soon as possible to prevent this type of unfair discrimination from taking root.

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[191] *See id.* § 300gg-4(a)(9) ("Any other health status-related factor determined appropriate by the Secretary.").

[192] *See id.*

[193] For more on this idea as applied to the employment context, see Stephanie A. Kostiuk, Note, *After GINA, NINA? Neuroscience-Based Discrimination in the Workplace*, 65 VAND. L. REV. 933 (2012).

Closing the gap would likely be a fairly simple and straightforward matter, requiring only the addition of a health-status-related factor in ACA for predictive neuroscience information. This would explicitly close the existing gap in protection and ensure that the millions of people who make up the large-group and self-insured health insurance markets are afforded the same protection as their counterparts in the small-group and individual markets. This simple fix would bring protection for predictive neuroscience information in line with existing health insurance protections for predictive genetic information and foreclose discrimination on the basis of all predictive information. Closing this gap could easily be achieved with minimal effort and would have maximal impact on those who are outside the scope of current protections.

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