"Warrior genes" and the disease of being Māori

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Abstract: The propensity of Māori towards violence and aggression has been assigned to the expression of a unique monoamine oxidase gene (*MAO*) popularly known as the 'Warrior Gene'. This assignment suggests that the violence supposedly exhibited by Māori is due to the very nature of Māori himself. Although this assignment and assessment by Pākehā falls into the usual pattern of stereotypes offered about Māori over the last 200 years, it is instructive to examine the truth of the matter, since it was based on the results of a scientific investigation. Behavioural problems have been reported in people who have exhibited abnormalities in the expression of unusual forms of *MAO* genes and their conditions have been variously described as diseases. Could those Māori who express the 'warrior' gene be diagnosed as having a medical condition similar to those with diseases such as Brunner syndrome or Norrie disease, two diseases involved in the expression of abnormal *MAO* genes? As a consequence is being Māori just another disease?

Keywords: gene expression; Māori domestic violence; monoamine oxidases; warrior genes

Introduction

Māori, who account for only 14.7% of the New Zealand population, commit more acts of violence than any other ethnic group in the nation, and the reasons for this apparent fact are not obvious (Statistics New Zealand, 2008). Some blame poverty and deprivation while others believe it stems from a century and a half of colonization. There are others who would blame this propensity of Māori towards violence on his very nature resurrecting racial stereotypes of the past that have been discredited. If not nature, then what else might be the cause for such a disparity in criminal convictions between Māori and Pākehā for acts of violence?

Recently, a new idea was presented that raised howls of protest from both scientific and political commentators. It was proposed that the high criminality of Māori was due to the expression of a 'warrior' gene that rendered Māori "more prone to violence, criminal acts, and risky behaviour." (Anonymous, 2006; Lea & Chambers, 2007). This neo-Darwinian approach to human behaviour claims that because Māori evolved in a high-risk environment, survival favoured those mutations that contributed to his survival and hence the frequency of the 'warrior' gene in the Māori population became enhanced over those found in other races.

The idea is that Māoridom is a warrior society that evolved under the stresses of war and ocean exploration resulting in a natural character that embraces aggression; hence the proclivity of Māori towards violence lies within his own nature. All of which presupposes that survival in the natural world is simply a function of aggression and violence and that Māori experienced more of this than most.

In this essay I have tried to gauge the 'truth' of the 'warrior' gene hypothesis and understand what that 'truth' might mean for Māori. That the apparent violence surrounding Māori arises from his nature is a very dangerous idea, and one that needs to be taken seriously especially by Māori. The invention of social theories underpinning racial stereotypes is inevitable, but the coupling of those theories with eugenic dogma regarding genetic suitability was the foundation of the Nazi death camps (Kevles, 1999) and Serbian ethnic cleansing (Naimark,

2001) of the last century. Unlikely to happen in New Zealand perhaps, but ethnic minorities should always be aware and vigilant of how they are perceived by Eurocentric majorities.

The 'warrior' gene

At the 2004 Annual Meeting of the American Association of Physical Anthropologists in Tampa, Florida, Ann Gibbons a scientific journalist (Gibbons, 2004), called the monoamine oxidase A gene a 'warrior' gene. She was speaking not so much as a scientist, but as a populariser of dry-as-dust science for the masses. As I will show, the term 'warrior' gene was an inappropriate label for a gene whose cellular and metabolic functions have not been clearly defined.

Monoamine oxidase genes (*MAO*) code for enzymes called monoamine oxidases. Monoamine oxidases (MAOs) are enzymes that are involved in the breakdown of neurotransmitters such as serotonin and dopamine and are, therefore, capable of influencing feelings, mood, and behaviour of individuals. The levels of these MAOs in brain and other tissues are important because the levels of MAOs determine just how quickly metabolism of these neurotransmitters occurs or whether metabolism occurs at all. It appears that the levels of these MAO enzymes in brain tissue can have marked effects on behaviours ranging from anxiety and panic disorder to aggression and violence.

MAOs come in two major forms called A and B. The A-form is an item of particular scientific interest because its gene contains a 30bp repeat polymorphism (*MAO-A*30bp-rpt) that is associated with its transcriptional regulation (Sabol, Hu, & Hamer, 1998). Studies have also detected a 3-repeat allele of the *MAO-A*30bp-rpt that corresponds to a lower MAO-A activity and higher dopamine levels. Lea & Chambers (2007) measured the presence of an additional polymorphism which they used for scoring the most common haplotype (AGCCG) and determined that the AGCCG haplotype frequency was elevated in Māori subjects who also carried the 3-repeat allele form of the *MAO-A*30bp-rpt as compared with non-Māori (Lea & Chambers, 2007). From this estimation of population frequency Lea and Chambers (2007) then went on to ask a number of questions concerning the distribution of *MAO* genes in Polynesia and their contributions to alcohol and tobacco use and whether or not this gene could prove of value in the development of methods for the treatment of the smoking and drinking habits of Māori people.

Based on their observations regarding population frequency of this particular form of the *MAO* gene the authors then went on to state:

> It suggests to us that Polynesian males who embark on long, dangerous canoe voyages and engaged in (and survived) war with other island tribes carried the AGCCG haplotype, coupled with the 3-repeat allele of *MAO-A*30bp-rpt, to Aotearoa (New Zealand) where they both increased in frequency due to rapid population growth (Lea & Chambers, 2007).

The paper by Lea and Chambers (2007) was reviewed by outside experts in the field of population genetics. Merriman & Cameron (2007) pointed out several serious flaws in the scientific reasoning. For example, there are both high and low activity forms of MAO but the relationship between these forms and aggressive behaviours is not clear.

> We believe that this conclusion is based on science with insufficient investigative rigor, both in interpreting and applying the relevant literature, and in generating new data. Central to the argument of Dr Lea and colleagues is the assumption that the low-activity *MAO-A* allele is associated with aggression in Māori males. This assumption cannot be made without the appropriate genetic epidemiological experiments being

> done to test for an association between *MAO-A* and aggression. However no such study has ever been reported (Merriman & Cameron, 2007).

And further stated:

> There is no direct evidence to support the claim that the *MAO-A* gene confers 'warrior' qualities on Māori males, either modern or ancestral. Furthermore, the assumption that a genetic association in Caucasian applies in Māori; the use of the "warrior gene" label in the context of human *MAO-A* aggression studies; generalising from a sample of 17 individuals not representative of the general Māori population; and the lack of scientific investigative journalism have combined to do science and Māori a disservice (Merriman & Cameron, 2007).

Not only has the science reported by Lea and Chambers been criticized for its lack of rigour, but the ethics of claiming "genetic explanation for negative social and health statistics for Māori" has been questioned by Crampton & Parkin, (2007).

> To make the causal claim on the evidence of association alone is naïve. In this case, the naïvety is masked by the positive public stereotype of the cutting edge scientist reporting a breakthrough. Of special concern is the fact that Dr Lea has made extravagant claims concerning the "warrior gene" in Māori despite having himself at times cautioned against risking the naive leap to a simplistic causal connection.

The assignment of behavioural functions to expression of specific genes based on such thin evidence and in the absence of epidemiological studies designed specifically for the investigation and identification of cause and effect is scientifically unsound.

Behavioural deficiencies associated with *MAO-A*.

Accepting perhaps that the relationship between the 'warrior' gene and Māori behavioural traits is weak, what is the evidence that *MAO* genes in general have been found to underlie behavioural peculiarities in other disease states? Was the 'warrior' gene hypothesis simply a step too far, or was it a reasonable expectation based on what is known about MAO and behavioural abnormalities? While "reasonable expectation" is not a substitute for hard evidence if speculations are made then what are the foundations of those speculations regarding *MAOs*?

A deficiency in monoamine oxidase A (MAO-A) has been shown to be associated with aggressive behaviour in men of a Dutch family (Cases et al., 1995). Selective deficiency of MAO-A in several males resulted in a syndrome of "borderline mental retardation and abnormal behaviour including impulsive aggression, arson, attempted rape, and exhibitionism" (Brunner, Nelen, Breakefield, Ropers, & van Oost, 1993, p.578). Low platelet MAO activity is associated with characteristics related to hypomanic behaviour and sensation seeking (Siever & Coursey, 1985). High levels of MAO-A appear to protect against the impact of childhood maltreatment and adversity on the development of antisocial behaviour and conduct disorder in whites (Widom and Brzustowicz, 2006). However, the protective effect was not found for non-white abused and neglected individuals. They concluded that the differences between white and non-whites might arise from "contextual" factors (e.g. environmental stressors) and a question concerning the suitability of using the *MAO-A* promoter VNTR polymorphism as a proxy for *MAO-A* levels in non-white populations.

The list of behavioural disorders implicating MAO-A is long (Table 1). Low levels of MAO activity and mutations on the *MAO-A* gene have been associated with enhanced aggression in animals (Cases et al., 1995; Brunner et al., 1993). All of those listed in Table 1 point to

negative behaviours many of which are either mental handicaps or borderline disorders that in today's world are considered undesirable. None listed are traits that, anyone would consider, confers advantages in terms of survival and in fact probably serve the exact opposite. That is, possession of these traits would pose severe disadvantages especially in the ancient world of the voyaging and war-like Polynesians.

Table1. Behavioural disorders implicating MAOs

Disorder	Reference
Anxiety	Chen et al. (2004)
Personality disorders	Samochowieca et al. (2004)
Antisocial behaviour	Caspi et al. (2002); Samochowiec et al. (2004)
Violence and risk taking	Lea and Chambers (2007); Widom and Brzustowicz (2006)
Risk taking	Lea and Chambers (2007)
Antisocial behaviour	Widom and Brzustowicz (2006)
Aggressive behaviour	Newman et al. (2005); Buckholtz & Meyer-Lindenberg (2008); Cases et al. (1995); Rosenberg (2006)
Impulsive aggression	Buckholtz & Meyer-Lindenberg (2008)
Mental disorders	Pinsonneault et al. (2006)
Obesity	Need et al. (2006)
Impulsivity	Manuck et al. (2000)
Depression and suicidality	Bernet al. (2007)
Impaired impulse control	Rosenberg et al. (2006)
Mental retardation (Brunner syndrome)	Rosenberg et al. (2006)
Mental retardation, autism, seizures, sleep disturbances (Norrie disease)	de la Chapelle et al. (1985); Levy et al. (1989)
Panic disorder	Deckert et al. (1999)

Thus, the association of *MAOs* with numerous antisocial and borderline psychotic conditions might lead some to speculate that every aspect of negative behaviours might be connected to *MAOs*; it seems that Māori violence has been placed into this category of a mental health concern without recognizing that aggression and violent behaviours also have important environmental factors that may contribute to the overall pattern. Behavioural abnormalities cannot be isolated from environmental conditions excepting in those who are indeed mentally disturbed. One is tempted to ask if the nature of Māori is now being forced into a health arena that borders on a category called 'mental defect'.

Implications of the 'warrior' gene

To say that the 'warrior' gene exists in the company of psychopaths and borderline psychotics is not too great a stretch of the imagination. Certainly, the behavioural abnormalities associated with *MAO* genes are many, but for most of humanity the *MAO* system functions quite well and quite consistently. While conviction rates for domestic violence of Māori exceed those of any other group there is no indication that the *MAO* system carried by Māori functions any differently from that of any other ethnic group and certainly no evidence to indicate that it has anything to do with violent behaviour in Māori (Hook, 2009).

The danger of the warrior gene hypothesis is that if it were true then any violent behaviours exhibited by Māori could be ascribed by the uninformed to this gene in the same way that the

abnormal behaviours of patients with say Brunner syndrome or Norrie disease have been blamed on abnormities in their *MAO* genes. Ascribing Māori domestic violence to genomic expression of a *MAO* gene raises the possibility that being Māori could then be placed in the abnormal category of syndrome and disease and subject to therapeutic interventions. Being Māori might then be controllable with drugs and all Māori children subject to interventions similar to those today who have been diagnosed with hyperactive syndrome.

It is one thing for newspapers to promote their fetishes but it is another for scientists to be the source of speculation and fantasy about the nature of Māori ('Warrior gene' blamed for Maori violence, 2006; Maoris attack 'warrior gene' claim., 2006). The nature of Māori and its relationship to violence is not the issue any more than the nature of Pākehā is the issue that underlies their apparent propensity towards colonization, war and genocide. Another danger comes from insurance companies who could, based on the 'warrior' gene hypothesis, refuse to insure Māori people or charge them higher premiums simply because they are Māori and therefore at risk. The implications that follow from the 'warrior' gene hypothesis should it become fact in the minds of the general public are horrendous.

The new eugenics

> "…the strongest and the best are selected for the task of propagating the likeness of God and carrying on his work of improving the race."

Thus said George Huntington Donaldson in his published sermon "Eugenics: a Lay Sermon" as published in 1929 in the Methodist Review and as quoted by Osgoode (2008).

It is probably agreeable that certain genetic traits are favourable while others are not, however, the ethical conundrum is how do we decide as to which ones are desirable and which ones are not? During the early part of the last century and in the name of eliminating "undesirable" traits from the gene pool many nations around the world set about sterilizing the "undesirables." Nazi Germany, however, went a step further and decided to kill them. The application of eugenics as a means of improving the "Master" race involved the elimination of all undesirables which included criminals, degenerates, dissidents, the feeble-minded, homosexuals, the idle, the insane, the religious, and the weak. It went on to include the Jews, Gypsies, and just about everyone else that Hitler didn't like.

Germany was not the only nation to embrace the "science" of eugenics. Others included the United States, Australia, New Zealand, Canada, Western Europe, and the United Kingdom. However, most of the world stopped short of killing, being satisfied with just sterilizing their undesirables. Only Germany decided that the most efficient way of dealing with the undesirables was by killing them, although Russia killed many of their undesirables too. Even the church supported the belief and practice of eugenics. In fact the United Methodist church recently issued an apology for their support of eugenics during the early part of the last century (Osgoode, 2008). Eugenics has been discredited, but it is not dead having simply changed its form.

The new eugenics is not only alive and well it is making giant strides in moving humanity to a place where improvement of the stock has now become simply a parental decision. Eugenics is no longer something imposed by government policy and enforced by goon squads; it is now a choice offered to most parents or prospective parents throughout the western world. The new eugenics is made possible by a process called Preimplantation Genetic Diagnosis (PGD) where the potential for the delivery of a genetically defective child is tested and choices offered (Preimplantation Genetic Diagnosis, 2008, 2009). PGD is the new method for detecting and removing the weak and the undesirable.

Embryo screening for genetic disposition that could lead to diseases such as Huntingdon's, aneuploidy, cystic fibrosis, sickle cell diseases, myotonic dystrophy, fragile X syndrome, haemophilia A, Duchenne muscular dystrophy, acute myeloid leukemia, Turner's syndrome, Down's syndrome, Niemann-Pick disease, pancreatic cancer, polycystic kidney disease, breast and ovarian cancers, deafness, Wilson's disease, is currently available (Preimplantation Genetic Diagnosis, 2009), and no doubt there are new diseases being added to the list even as we speak. The use of PGD to minimize the delivery of babies with these genetic disorders is probably a good thing and undoubtedly here to stay, but let us hope that the 'warrior' gene will never find its way onto that list of genetic undesirables.

Germany, Austria, Ireland, and Switzerland have banned PGD outright and UK, France, the Netherlands, Belgium, Italy and Greece have limited its use (Bell, 2007). Most PGD procedures occur in the United States (Covington & Burns, 2006). PGD is not generally permitted in New Zealand but guidelines for its use are being developed (Bioethics Council, 2008). Under the current government the Bioethics Council may be disbanded (Trevett, 2009); however, PGD can be used under special circumstances and without Ethics Committee oversight provided the disorder could cause the child to be 'seriously impaired.' The deciding factor is whether or not a single-gene disorder has been identified in the family and the risk of an affected pregnancy is 25 percent or greater. Thus, screening for the 'warrior' gene by using PGD could be permissible under the terms and conditions of the Human Assisted Reproductive Technology Act 2004 (the Hart Act) if the gene were determined to be detrimental to a child's long-term survival. Unlikely but one must remain mindful of the past.

Conclusions

Intergroup bias is recognized as an important influence on social behaviour (Blair, 2001). The development of racial stereotypes has been part of the colonization process throughout the settlement of New Zealand. In responding to news of the 'warrior' gene hypothesis Māori MP Hone Harawira said:

> I remember 30 or 40 years ago when I was a kid people said Maori had a natural inclination to play the guitar, that Maori had a natural inclination to play rugby, Maori were good on bulldozers etc. I've stopped listening to all that sort of carry on. (Poverty behind violence, says Maori MP., 2006).

In the case of Māori domestic violence blaming nature as opposed to nurture simplifies the problem because it implies that Māori has no one to blame but himself for his own condition. Contributions to racial stereotyping by trained scientists are unethical and scandalous. It will be interesting to see where this hypothesis of the 'warrior' gene takes us over these next few years and to see if it has any influence in the arena of public policy and public opinion. Newspaper columnist Brian Rudman (2006) said it quite nicely, "With 30,000 genes to investigate, I wonder how long it will be before someone discovers the "naive" gene that a high proportion of scientists seem to carry."

Part of the "evidence" used in the construction of this 'warrior' gene hypothesis is the rather romantic picture that Europeans have regarding the world of the pre-contact Polynesian. The picture offered is one of constant war, struggle, and voyaging over vast distances of the ocean. The images conjured by such suppositions are those of an aggressive, potentially violent, courageous, risk taking, adventurer, and while war and risk taking was no doubt part of the world of the Polynesian, there is no evidence to suppose that Polynesians experienced greater survival pressures than those experienced by any other people around the world. Frequencies of 'warrior' genes in specific populations are unlikely to arise simply from the struggle to survive because survival is an experience common to all people.

While it is true that *MAO-A* genes have been found in disease states associated with behavioural abnormalities in some non-Māori subjects, there is no evidence to indicate that the behavioural characteristics of Māori as a people are in anyway unusual. Māori are not borderline psychotics, retarded, hyper aggressive, depressive, antisocial, impulsive, suicidal risk takers and to suggest otherwise is irresponsible and not supported by the facts. An explanation for the high conviction rates of Māori for violent crimes is to be found not in his nature but elsewhere perhaps such as in his victimhood arising out of 160 years of colonization or in how the justice system deals with people whom most of its Eurocentric white administrators perceive as being excessively violent.

Māori are not the only indigenous people to find themselves accused of violence by their mainstream colonizers. The Inuits, Metis, and Indians of Canada all exhibit a propensity for violence above that of their colonizers (Chartrand & Mckay, 2006; St-Jean, 2000). Even the Australian Aborigines, have high conviction rates for violent crimes (Johnston, 2007). The only common factor amongst all these indigenes appears to lie in the perceptions of their colonizers, their dispossession from their lands, their impoverishment, deprivation, and assimilation. Somewhere in there might lie the real reasons for indigenous violence.

References

Anonymous (2006). 'Warrior gene' blamed for Maori violence. National Nine News. August 8, 2006. Retrieved May 29, 2008 from http://news.ninemsn.com.au/article.aspx?id=120718

Bell, R. (2007) Comparative European approaches to Pre-Implantation Genetic Diagnosis. Symposium Report. Retrieved July 24, 2009 from http://www.bioethics.ac.uk/cmsfiles/files/resources/a4_symposium_report_european_approaches_to_pgd_web_final.pdf

Bernet, W., Vnencak-Jones, C.L., Farahany, N., & Montgomery, S.A., (2007). Bad nature, bad nurture, and testimony regarding MAOA and SLC6A4 genotyping at murder trials. *Journal of Forensic Sciences, 52*(6) 1362-1371. Retrieved June 11, 2008, from http://www.ingentaconnect.com/search/article?title=MAOA&title_type=tka&year_from=1998&year_to=2008&database=1&pageSize=20&index=2

Bioethics Council (2008). *Who gets born? A report on the cultural, ethical and spiritual aspects of pre-birth testing by Toi te Taiao: the Bioethics Council.* Retrieved June 19, 2008, from http://www.bioethics.org.nz/publications/who-gets-born-jun08/html/page1.html

Blair, I.V. (2001). Implicit Stereotypes and Prejudice. In *Cognitive Social Psychology. The Princeton Symposium on the Legacy and Future of Social Cognition(* Chap. 22). Retrieved June 11, 2008, from http://books.google.com/books?id=mYswBBWX5uIC&pg=PP7&lr=&source=gbs_selected_pages&cad=5

Brunner H. G., Nelen M., Breakefield X.O., Ropers H. H., & van Oost B. A. (1993). Abnormal behavior associated with a point mutation in the structural gene for monoamine oxidase A. *Science*, 262(5133) 578-80. Retrieved June 6, 2008, from http://www.ncbi.nlm.nih.gov/pubmed/8211186?dopt=Abstract

Buckholtz, J.W., & Meyer-Lindenberg, A. (March, 2008) MAOA and the neurogenetic architecture of human aggression. *Trends in Neurosciences, 31*(3), 120-129. Retrieved June 11, 2008, from

http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6T0V-4RS9SHM-2&_user=10&_origUdi=B6T4S-4K9C5J2-6&_fmt=high&_coverDate=03%2F31%2F2008&_rdoc=1&_orig=article&_acct=C000050221&_version=1&_urlVersion=0&_userid=10&md5=ffcdff01be3b9a2a89b277fec609dd40

Cases, O., Seif, I., Grimsby, J., Gaspar, P., Chen, K., Pournin, S., et al. (1995). Aggressive behavior and altered amounts of brain serotonin and norepinephrine in mice lacking MAOA. *Comment in Science, 270*(5235) 362-4. Retrieved June 6, 2008, from http://www.ncbi.nlm.nih.gov/pubmed/7792602?dopt=Abstract

Caspi, A., McClay, J., Moffitt, T.E., Mill, J., Martin, J., Craig, I.W., et al. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, 297(5582) 752. Retrieved June 12, 2008, from http://www.ncbi.nlm.nih.gov/pubmed/12161658?dopt=AbstractPlus

Chartrand, L. & McKay, C. (2006). *A review of research on criminal victimization and First Nations, Métis and Inuit Peoples 1990 to 2001.Report prepared for the Department of Justice Canada. January 2006.* Retrieved June 3, 2008, from http://www.justice.gc.ca/eng/pi/rs/rep-rap/2006/rr06_vic1/index.html

Chen, K.; Holschneider, D. P.; Wu, W.; Rebrin, I.; & Shih, J. C. (2004). A spontaneous point mutation produces monoamine oxidase A/B knock-out mice with greatly elevated monoamines and anxiety-like behavior. *Journal Biological Chemistry, 279* 39645-39652. Retrieved June 12, 2008, from http://www.jbc.org/cgi/reprint/279/38/39645

Covington, S.N., & Burns, L.H. (2006). *Infertility counseling. A comprehensive handbook for clinicians, 2nd Edition*. Cambridge University Press.

Crampton, P., & Parkin, C. (2007). Warrior genes and risk-taking science. *Journal of the New Zealand Medical Association, 120*(1250). Retrieved June 13, 2008, from http://www.nzma.org.nz/journal/120-1250/2439/

de la Chapelle, A., Sankila, E. M., Lindlöf, M., Aula, P., & Norio, R. (1985).Norrie disease caused by a gene deletion allowing carrier detection and prenatal diagnosis. *Clin Genet, 28*(4) 317-20. Retrieved June 13, 2008, from http://www.ncbi.nlm.nih.gov/pubmed/2998655?ordinalpos=209&itool=EntrezSystem2.PEntrez.Pubmed.Pubmed_ResultsPanel.Pubmed_RVDocSum

Deckert, J.; Catalano, M.; Syagailo, Y. V.; Bosi, M.; Okladnova, O.; Di Bella, D. et al. (1999). Excess of high activity monoamine oxidase A gene promoter alleles in female patients with panic disorder. *Hum. Molec. Genet. 8* 621-624, [PubMed ID : 10072430]. Retrieved June 19, 2008, from http://hmg.oxfordjournals.org/cgi/content/abstract/8/4/621

Gibbons, A. (2004). American Association of Physical Anthropologists meeting: tracking the evolutionary history of a "warrior" gene. *Science, 304* 818–9. Retrieved June 15, 2008, from http://www.sciencemag.org/cgi/content/summary/304/5672/818a

Hook, G.R. (2009). Does the Domestic Violence Act discriminate against Māori? *MAI Review, 1*. Article 7, 10 pages.

Johnston, T. (August 24, 2007). Far-reaching policy for Aborigines draws their fury. *The New York Times*. Retrieved June 18, 2008, from http://www.nytimes.com/2007/08/24/world/asia/24outback.html?scp=1&sq=indigenous%20domestic%20violence&st=cse

Kevles, D.J. (1999). Eugenics and human rights. *British Medical Journal, 319(72027):* 435-438. Retrieved July 28, 2009 from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1127045

Lea, R., & Chambers, G. (2007). Monoamine oxidase, addiction, and the "warrior" gene hypothesis. *Journal of the New Zealand Medical Association*, *120*(1250). Retrieved June 12, 2008, from http://www.nzma.org.nz/journal/120-1250/2441/.

Levy, E.R., Powell, J.F., Buckle, V.J., Hsu, Y.P., Breakefield, X.O., & Craig I.W. (1989). Localization of human monoamine oxidase-A gene to Xp11.23-11.4 by in situ hybridization: implications for Norrie disease. *Genomics, 2* 368-70. Retrieved June 13, 2008, from http://www.ncbi.nlm.nih.gov/pubmed/2793188?ordinalpos=190&itool=EntrezSystem2.PEntrez.Pubmed.Pubmed_ResultsPanel.Pubmed_RVDocSum

Manuck, S.B., Flory, J.D., Ferrell, R.E., Mann, J.J., & Muldoon, M.F. (2000). A regulatory polymorphism of the monoamine oxidase-A gene may be associated with variability in aggression, impulsivity, and central nervous system serotonergic responsivity. *Psychiatry Research, 95*(1) 9-23. Retrieved June 11, 2008, from http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6TBV-40RTMB4-2&_user=10&_rdoc=1&_fmt=&_orig=search&_sort=d&view=c&_acct=C000050221&_version=1&_urlVersion=0&_userid=10&md5=72bbcd624950f9916e1ebd06f4db765a

Maoris attack 'warrior gene' claim. (August 10, 2006).*Times OnLine*. Retrieved June 12, 2008, from http://www.timesonline.co.uk/tol/news/world/article604593.ece

Merriman, T., & Cameron, V. (2007). Risk-taking: behind the warrior gene story. *Journal of the New Zealand Medical Association*, *120*(1250). Retrieved June 12, 2008, from http://www.nzma.org.nz/journal/120-1250/2440/

Naimark, N.M. (2001). Fires of hatred: Ethnic cleansing in twentieth-century Europe, Cambridge: Harvard University Press.

Need, A.C., Ahmadi, K.R., Spector, T.D., & Goldstein, D.B. (2006). Obesity is associated with genetic variants that alter dopamine availability. *Annals of Human Genetics 70*(3) 293-303. Retrieved June 11, 2008, from http://www.ingentaconnect.com/search/article?title=MAOA&title_type=tka&year_from=1998&year_to=2008&database=1&pageSize=20&index=12

Newman, T.K., Syagailo, Y.V., Barr, C.S., Wendland, J.R., Champoux, M., Graessle, M., et al. (2005). Monoamine oxidase A gene promoter variation and rearing experience influences aggressive behavior in rhesus monkeys. *Biological Psychiatry 57*(2) 167-172. Retrieved June 11, 2008, from http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6T4S-4F37M8J-5&_user=10&_coverDate=01%2F15%2F2005&_alid=752685980&_rdoc=3&_fmt=high&_orig=mlkt&_cdi=4982&_sort=v&_st=17&_docanchor=&view=c&_ct=171&_acct=C000050221&_version=1&_urlVersion=0&_userid=10&md5=a0a72b8f8d3b6d7ed6b1db931781d662

Osgoode, E.C. (April, 2008). *An apology for the support of eugenics by the United Methodist Church.* Retrieved June 15, from http://www.freerepublic.com/focus/f-news/2012353/posts

Pinsonneault, J.K., Papp, A.C., & Sadée, W. (2006). Allelic mRNA expression of X-linked monoamine oxidase a (MAOA) in human brain: dissection of epigenetic and genetic

factors. *Human Molecular Genetics 15*(17) 2636-2649. Retrieved June 11, 2008, from http://www.ingentaconnect.com/search/article?title=MAOA&title_type=tka&year_from=1998&year_to=2008&database=1&pageSize=20&index=7

Poverty behind violence, says Maori MP. (August 8, 2006). *National Nine News*. Retrieved May 29, 2008, from http://povertynewsblog.blogspot.com/2006/08/australia-poverty-behind-violence-says.html

Preimplantation Genetic Diagnosis. (2008). Retrieved June 16, 2008, from the BioCentre website, http://www.bioethics.ac.uk/index.php?do=topic&sid=14

Preimplantation Genetic Diagnosis. (2009) Retrieved July 22, 2009, from, http://en.wikipedia.org/wiki/Preimplantation_genetic_diagnosis#Biopsy_procedures

Rosenberg, S., Templeton, A., Feigin, P., Lancet, D., Beckmann, J., Selig, S., et al. (2006). *Human genetics 120*(4) 447-459. Retrieved July 22, 2009, from http://www.ingentaconnect.com/search/article?title=MAOA&title_type=tka&year_from=1998&year_to=2008&database=1&pageSize=20&index=6

Rudman, B. (August 11, 2006). Gene-pool scientist misses mark. *The New Zealand Herald*, Retrieved June 25, 2008, from http://www.nzherald.co.nz/section/1/story.cfm?c_id=1&objectid=10395645&pnum=0

Sabol, S. Z., Hu, S., & Hamer, D. (1998) A functional polymorphism in the monoamine oxidase A gene promoter. *Hum Genet.* 103 273–9.

Samochowieca, J., Syreka, S., Michaa, P., Ryewska-Wódeckaa, A., Samochowieca, A., Horodnickia, J., et al. (2004). Polymorphisms in the Serotonin Transporter and Monoamine Oxidase A Genes and their relationship to personality traits measured by the temperament and character inventory and NEO Five-Factor inventory in healthy volunteers. *Neuropsychobiology 50* 174-181. Retrieved June 12, 2008, from http://content.karger.com/produktedb/produkte.asp?typ=fulltext&file=NPS20040500021 74

Siever L.J., & Coursey R.D. (1985). Biological markers for schizophrenia and the biological high-risk approach. *Journal of Nervous and Mental Disorder 173*(1) 4-16. Retrieved, June 6, 2008, from http://www.ncbi.nlm.nih.gov/pubmed/3917487?ordinalpos=4&itool=EntrezSystem2.PEntrez.Pubmed.Pubmed_ResultsPanel.Pubmed_RVDocSum

Statistics New Zealand (2008). Retrieved from Statistics New Zealand website, http://www.stats.govt.nz/default.htm

St-Jean, G. (May 15, 2000). Aboriginal peoples and the criminal justice system. A special issue of the *Bulletin, Ottawa*. Retrieved June 25, 2008, from http://www.ccja-acjp.ca/en/aborit.html

Trevett, C. (March 3, 2009). Environment jobs go in shake-up. *The New Zealand Herald* Retrieved April 9, 2009, from http://www.nzherald.co.nz/nz/news/article.cfm?c_id=1&objectid=10561001

'Warrior gene' blamed for Maori violence. (August 8, 2006). *National Nine News*. Retrieved May 29, 2008, from http://news.ninemsn.com.au/article.aspx?id=120718

Widom C.S., Brzustowicz, L.M. (2006). MAOA and the "cycle of violence:" childhood abuse and neglect, MAOA genotype, and risk for violent and antisocial behavior. *Biological Psychiatry 60*(7) 684-9. [Epub 2006 Jun 30]. Retrieved June 6, 2008, from http://www.ncbi.nlm.nih.gov/pubmed/16814261?ordinalpos=5&itool=EntrezSystem2.PEntrez.Pubmed.Pubmed_ResultsPanel.Pubmed_RVDocSum

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