Racial Inequities in Cardiovascular Disease in New Zealand

Warren Miner-Williams
Research Officer, Person Centred Research, School of Clinical Sciences, Faculty of Health and Environmental Sciences, Auckland University of Technology, 90 Akoranga Drive, Northcote, Auckland, 0627, New Zealand

ABSTRACT

The literature is replete with studies pertaining to ethnic inequities in healthcare. A thorny subject that has been described for decades and yet has few remedial solutions. The pattern of ethnic inequities in healthcare is a global phenomenon that is not confined to any specific race or culture. Worldwide, cardiovascular disease (CVD) is the topmost cause of death and a substantial burden on healthcare resources. In New Zealand CVD is the leading cause of death, accounting for 40% of all deaths annually. Diminished life expectancy is one example of racial inequity in healthcare between Māori and Pākehā (the non-indigenous population). This review attempts to clarify the muddy waters of 175 years of post-colonial healthcare inequity in New Zealand and in particular the causes of inequity in the incidence of CVD and mortality in Māori. Such dialogue will hopefully stimulate discussion among policy makers and clinicians to redress the ethnic inequities in healthcare.

Keywords: Inequity in healthcare; Treaty of Waitangi; Institutional racism; Interpersonal racism; Internalized racism; Indigenous people

Introduction

Racial inequities in cardiovascular disease in New Zealand

Worldwide, cardiovascular disease (CVD) is the topmost cause of death and equally one of the most preventable causes of mortality. Coincident with rapid economic development across the world CVD is a considerable burden on healthcare resources in both the developed and rapidly developing countries [1]. CVD is the leading cause of death in Aotearoa/New Zealand, that annually accounts for 40% of all deaths, with proportionally more deaths per 100 000 occurring in Māori [2]. Diminished life expectancy is one example of racial inequity in healthcare between Māori and Pākehā (the non-indigenous population). Ischaemic heart disease (IHD), a major cause of CVD deaths, accounts for 40.2% of Māori deaths from this condition in those aged less than 65 years, compared to 10.5% of Pākehā deaths [3].

The literature is replete with studies pertaining to ethnic inequities in healthcare. A thorny subject that has been described for decades and yet has few remedial solutions. The pattern of ethnic inequities in healthcare is a global phenomenon that is not confined to any specific race or culture. The socio-economic, cultural and environmental conditions in which people live greatly influence their chances of being healthy.

“Indeed factors such as poverty, social exclusion and discrimination, poor housing, unhealthy early childhood conditions and low occupational status are important determinants of most diseases, deaths and health inequities between and within countries” [4].

The main determinants of health have been conceptualised as a complex, layered spectrum of factors generated by the macro-policy environment such as neo-liberal economic growth strategies that have widened the gap between the rich and the poor [5]. Suggest four interdependent factors that can be grouped together as spheres of influence that impact health and which may form the basis of a health policy intervention (Figure 1). The main influences that affect health can be described as; promoting health; protecting health; and threatening health.

Dahlgren and Whitehead warn that all too often strategies are aimed at correcting a single level of influence, whereas interventions that address several levels may be more effective [5]. Equally evident is that positive actions at one level may be offset by negative actions at other levels; e.g. health education for young people being nullified by insufficient funds for school lunches [5]. Surviving and living a healthy life remain closely related to the socioeconomic background of individuals and families that are reflected in substantial and ever increasing social inequities in health [6]. However, very few countries have developed strategies for the integration of equity-oriented health policies into economic and social legislation. The reduction of inequities in the health status between less privileged socioeconomic groups and their more privileged counterparts, Dahlgren and Whitehead suggest should be key to all international, national and local health policies [6].

For some, inequity in healthcare is tantamount to racism [7-10]. In New Zealand, Harris states that racism is an important determinant of health and a cause of ethnic inequities [11]. Embedded in the socio-economic, cultural and environmental influences of New Zealand health is ethnic identity. Māori are tangata whenua, the indigenous people of Aotearoa/New Zealand, who collectively have a lower level of educational, occupational and income status, and have poorer health than Pākehā, [non-Māori] [12]. Howden-Chapman suggest that such ethnic inequities arise from other, pervasive characteristics of New Zealand society that result in poor health in the Māori and Pacific peoples, e.g. income inequity [13]. Population-based policies, directed to the whole population, rather than targeted social and health measures, are (paradoxically) likely
to provide the greatest benefit for low income groups and those with higher risk factors [13]. However, Howden-Chapman et al. warn that targeting ‘the poor’ will fail to minimise the overall social variations in health, may stigmatise poor people and even accentuate health inequities. They suggest that a range of population wide, mutually reinforcing redistributive policies are required to redress the imbalance in income, education, employment, housing and health services [13]. Since their editorial 16 years ago little has changed in New Zealand; indeed the inequity between the rich and the poor is greater today than in 2000 [14].

Race is a social construct not a biological reality typically used in a mechanical and uncritical manner as a proxy for immeasurable biological, socioeconomic and/or sociocultural factors [7-9]. Jones in an invited commentary uses an example that is true to Māori and Pākehā in New Zealand today; “Black” in the US is over-represented in poverty, but the majority of poor people are “White,” as not all Black people are poor [7]. She continues by suggesting there is no single Black culture just as there is no single White, Hispanic or Asian culture. Genetically, “races” are a heterogeneous mixture of geographic stock from many parts of the world. Race is a social classification in our race-conscious society that conditions many aspects of our lives; which then result in profound differences in life opportunities [7]. Whereas ethnicity is a reflection of our genetic and cultural heritage, race is a measure of identity imposed by society that results in the consequent societal constraints associated with that identity. The race label assigned by an investigator or volunteered by a study participant, Jones suggests, is an “excellent measure of exposure to racism” (p. 300) and that it is this aspect of race that impacts health and results in race-associated inequities that are large, occur throughout a life span and involve many different organ systems [7]. Indeed being classified as “White” by others has been shown to produce statistically significant advantages in health status, irrespective of how a person self-identifies [15]. Associations of observer-ascribed skin colour found a significant gradient in self-rated health by skin colour; those with darker skin colour reported poorer health [16]. Perreira and Telles concluded that darker skin colouration influences self-rated health primarily by an increased exposure to class discrimination and low socio-economic status [16]. In New Zealand reported that skin colour perceived as white was advantageous and health-protective whereas the social-assignment of being Māori was associated with being at risk of differential and discriminatory healthcare [17]. The routine documentation of race-associated inequities in health, without adequate explanation is not benign as it impedes the advancement of scientific knowledge, limits efforts at primary prevention, and contributes to ideas of biologic determinism [7].

In 2000, Jones published a 3 level framework for understanding racism that included: institutionalized, personally mediated, and internalized racism. The concept of the levels that she suggested which form the basis of race-related influences in poor health outcomes [18] (Figure 2). Institutionalised racism manifests itself in differential access to material conditions, (e.g. quality education and housing) and access to political influence. Personally mediated racism is defined as prejudice and discrimination, where prejudice means differential assumptions about the abilities, motives and intentions of others according to

---

**Figure 1:** The main determinants of health.

**Figure 2:** The impacts of racism on health

---

Used with the permission of World Health Organization Regional Office for Europe (2007)
their race and discrimination means differential actions toward others according to their race. Internalized racism is defined as acceptance by members of the stigmatized races of negative messages about their own abilities and intrinsic worth. It is characterized by them not believing in others who look like them, and not believing in themselves [18].

The framework devised by can be superimposed upon the determinants of health stratified by [5,18]. Racism affects health partly because indigenous, minority populations often experience less favourable social and economic circumstances that allows access to healthcare, (institutionalised racism) and partly because of direct psychosocial stress caused by personally mediated racism [12,18-20]. Within New Zealand and internationally, racism is recognised as an important determinant of health [10]. Rata and Zubaran warn that it is low socioeconomic status that is the cause of Māori health disadvantages not ethnicity [21]. They argue that ethnic labelling itself may contribute to negative stereotypes that produce racism, which in turn feed the cycles of discrimination for ethnic minority groups. They suggest that public healthcare policies based on ethnic distinctions should be abandoned in favour of “a more tenable approach that recognises a series of other causal factors in social disadvantage” [21]. As the current strategies are failing Māori, new initiatives addressing social disadvantage and poverty are urgently needed to redress the inequities in healthcare.

The health of indigenous people is significantly less than the non-indigenous in communities where traditional ways of life, environment and livelihoods have been taken over by the worst of Western culture e.g. unemployment, poor housing, alcoholism and drug use [22]. In Aotearoa/New Zealand from the formation of our bicultural society (Māori the indigenous peoples of New Zealand and Pākehā non-indigenous people) with the signing of the Treaty of Waitangi in 1840, Māori were immediately marginalised. The fabric of Māori society; tribal laws, languages, dress, sacred ceremonies, rituals, healers and native remedies were suppressed by colonial laws and regulations [23]. The dispossession of lands and subsequent poverty meant Māori could not compete equally within the imposed and dominant western culture to obtain the benefits of healthcare, education and employment. For a more detailed account of the effect of colonisation of New Zealand Māori [24-26].

However, after 176 years it is unhelpful to ascribe current inequities to intergenerational psychological trauma caused by the impact of a cataclysmic event such as colonisation rhetoric such as: “If Māori are getting less, non-Māori are getting more” [21,26,27]. If as Jackson suggests, Māori health needs arise as a consequence of their indigenous rights being breached, will Māori health improve with the reinstatement of such rights? Likewise it is naïve to argue that genetic differences are not likely to be part of the complex contributory factors for racial inequities in New Zealand when, for example, this is clearly untrue in the light of research that shows melanocortin-3 receptor gene variants in Māori are linked to obesity and the early onset of type 2 diabetes [28-32]. When seeking the reason why health inequity exists in New Zealand today it is not helpful to pound the colonisation drum. Modern health inequity requires an up-to-date reason as to why Māori have increasing rates of CVD when those for Pākehā are decreasing. Rata and Zubaran dismiss post-colonial trauma as the root of Māori inequities in healthcare and suggest that sociocultural factors, with or without an ethnic dimension are more likely the cause of poor outcomes in health [21]. When reviewing risk factors why are 40% of Māori males and 50% of Māori females, aged 15-64 years, continuing to smoke when only 20-21% of Pākehā are? Why 26% of adult Māori are obese compared to only 18% of Pākehā? Using face-to-face interview techniques of 12 500 people. Harris et al. [11] reported an association between experiencing racial discrimination and socioeconomic deprivation with smoking and CVD. Is racial discrimination and socioeconomic deprivation the cause of CVD or is CVD the result of exposure to risk factors such as smoking and obesity? It is dangerous to deflect attention away from the risk factors of CVD and blame post-colonial trauma. Likewise it is inappropriate to blame racism for the voluntary disregard of CVD risk factors such as smoking and obesity; as such factors hold the same proportion of risk for all ethnicities.

Of the many papers that outline health inequities for Māori, very few actually outline what Māori require. An exception to this is the work of who suggests that in New Zealand the availability of care is inversely proportional to the need of the population served, where those who need healthcare the most, receive the least; the inverse care law first suggested by Curtis suggest that three key factors must be addressed in order to invoke changes in health equity; a) acknowledging the role of society, b) understanding the policy imperatives, and c) exploring the clinician’s role. This approach they conclude is consistent with the human rights of Māori as tangata whenua, the rights of Māori as outlined within the Treaty of Waitangi and New Zealand health policy [33,34]. A recurrent theme in the understanding of health inequities is the way society distributes health resources, of material wealth, housing, education and employment [35]. Redressing inequities in power, money and resources should be viewed as restorative actions outlined by the Treaty of Waitangi.

**Racial inequities in New Zealand and the increased risk of CVD**

Although the causes of CVD in minority ethnic groups are not completely understood, researchers agree that socioeconomic factors are important. Although the classic risk factors associated with CVD vary significantly between minority ethnic groups, morbidity and mortality are much higher in these groups than in Caucasian and Asian populations [36]. The prevention of CVD across all ethnic groups relies on; identifying associated risks, establishing a safe threshold for such risks and reducing the risk factors that may precipitate the disease. The Framingham study identified 7 major risk factors; age, sex, blood pressure (BP), glucose intolerance, total and high-density cholesterol, smoking and left ventricular hypertrophy, that may be used to calculate the probability of developing CVD [37]. In addition to these risk factors the study by added; apolipoproteins B:A ratio, history of hypertension, history of diabetes, diet, inactivity, alcohol use, psychosocial stress, and abdominal obesity (which was better than BMI as a predictor of myocardial infarction in all ethnic groups) [38,39]. Population screening using risk factor analysis requires the application of a validated and reliable tool which is currently insensitive and not specific enough to apply to
ethnically heterogeneous populations[36]. Other risk prediction instruments include; PREDICT, FINRISK [40], SCORE [41] and ETHRISK [42]. More controversial risk factors have been proposed and include; metabolic syndrome, micro albuminuria, waist circumference, haemostatic factors, and homo-cysteine levels [36]. In New Zealand the national guidelines for the prevention of CVD contain an age stratification which make screening very cost effective and lists: personal history of CVD; age; sex; smoking status; apolipoprotein imbalance; hypertension and diabetes as the principal risk factors [43]. The guidelines also list other well established determinants of CVD risk: atrial fibrillation; obesity; impaired carbohydrate metabolism; metabolic syndrome; nutrition and dietary patterns; physical inactivity; family history of premature CVD; socioeconomic position; depression, social isolation and social support. When using the Framingham Risk Equation the guidelines list adjustments that include ethnicity. However, evaluating the guidelines and appropriateness for screening in primary care suggests they would be more effective if they incorporated U.S. recommendations on the use of aspirin and initial antihypertensive treatment rather than on achieving blood pressure targets [44].

Higher proportions of Māori live in more deprived areas than non-Māori (Figure 3). In 2013, 23.5% of Māori lived in decile 10 areas compared with 6.8 percent of non-Māori. Only 3.8% lived in decile 1 areas as compared to 11.6% of non-Māori).

In New Zealand CVD and diminished life expectancy is one example of racial inequity in healthcare between Māori and Pākehā (Figure 4). Between 1951 and 2011 the life expectancy of Pākehā at birth had increased steadily from 68 to 80 years for males and from 72 to 83 years for females. However, during the

Note: Crude rates and prioritised ethnicity have been used [45]

**Figure 3:** Neighbourhood deprivation distribution of Māori and non-Māori, 2013.

**Figure 4:** Life expectancy at birth for Māori and non-Māori by gender.
same period the life expectancy of Māori males only increased from <56 to 72 years and from 68 to 76 for Māori females [45].

The standardised mortality rates for CVD in males and females are shown in Table 1. The data show that between 2010 and 2012 the total CVD mortality rate among Māori was more than twice as high as that among non-Māori (RR 2.17, CI 2.08-2.26). In 2012-14, Māori were more than one-and-a-half times as likely as non-Māori to be hospitalised for cardiovascular disease (RR 1.64, CI 1.61-1.67). The mortality rate for heart failure among Māori was more than twice that of non-Māori (RR 2.36, CI 1.76-3.17), and Māori were about four times as likely as non-Māori to be hospitalised for heart failure (RR 4.01, CI 3.83-4.21). The inequity was greater for females where the heart failure hospitalisation rate among Māori females was nearly four-and-a-half times that of non-Māori (RR 4.82, CI 4.47-5.29) and the rheumatic heart disease hospitalisation rate for Māori was nearly five times that of non-Māori (RR 5.23, CI 3.99-6.87) and the rheumatic heart disease hospitalisation rate for Māori females was nearly four-and-a-half times that of non-Māori females (RR 5.30, CI 4.47-6.29) [45].

In New Zealand childhood obesity is believed to be the major contributing factor in adult obesity and is associated with disease conditions such as hypertension, dyslipidaemia, type 2 diabetes and subsequent CVD. Childhood obesity is more prevalent in Māori children (11.8%) compared to those with Caucasian heritage (5.5%) [46]. Cardio metabolic health can be improved by reducing obesity and increasing cardiorespiratory fitness, however, it remains unclear whether fatness or fitness is crucial to improving cardio metabolic health and decreasing CVD risk in children [46]. In adults fitness is key to improving cardio metabolic health.

Cultures apart

A neoliberal perspective of the NZ health system has seen a shift away from a governmental health responsibility towards one of individualised lifestyle accountability. Rather than investment in the prerequisites of good health, that of income, housing and nutrition, healthcare promotion policies are cloaked in the political ideology of neoliberalism, whereby the fundamentals of health, unemployment, poverty, lack of education, all important social determinants of health are largely ignored and seen as poor personal choices made by a population who are able to rectify such social and economic conditions [47]. Such social determinants of health, once seen as a failure of the state are now viewed as a personal failure requiring personal accountability [48]. Health promotion messages of; “Five plus a day,” “Be healthy, be active”; “Eat healthy and lose weight” are widely publicised throughout Western societies, are hailed as panacea to modern day health concerns and chronic disease [47]. However, as social and economic conditions worsen the burden of treating chronic disease on national health expenditure increases exponentially [49]. Unable to afford healthy lifestyle options Māori are frequently subjected to negative stereotypes and deficit explanations that unfairly direct fault at Māori for being obese, unhealthy, and sedentary along with accusations that they make poor lifestyle choices [48,50]. Accusations such as these ignore genetic, contemporary socio-cultural and environmental factors that contribute to their unhealthy lifestyle. The imposition of neoliberal personal responsibility

Table 1: Cardiovascular disease indicators, by gender, Māori and non-Māori.

| Indicator                        | Māori Total | Māori Females | Non-Māori Total | Non-Māori Females |
|----------------------------------|-------------|---------------|-----------------|-------------------|
| Total cardiovascular disease mortality | 346.9       | 232.3         | 286.8           | 168.2             |
|                                  | (328.6–365.9)| (219.2–246.1) | (275.7–298.3)   | (165.3–171.0)     |
| Total cardiovascular disease hospitalization | 3725.6      | 2710.2        | 3186.4          | 2537.2            |
|                                  | (3600.3–3791.8)| (2658.6–2762.6) | (3145.3–3228.0) | (2522.1–2552.3)   |
| Stroke mortality,                | 44.7        | 50.6          | 48.2            | 30.9              |
|                                  | (38.3–51.9) | (44.5–57.3)  | (43.7–53.0)     | (29.8–32.1)       |
| Stroke hospitalization           | 355.1       | 374.2         | 365.7           | 245.1             |
|                                  | (335.3–375.8)| (355.4–393.6) | (352.1–379.8)   | (240.8–249.5)     |
| Heart failure mortality,         | 5.4         | 5.2           | 2.2             | 2.2               |
|                                  | (3.3–8.3)   | (3.3–7.0)    | (3.9–6.9)       | (2.0-2.5)         |
| Heart failure hospitalization    | 676.1       | 434.8         | 547.5           | 179.7             |
|                                  | (648.7–704.3)| (414.3–456.0) | (530.6–564.7)   | (176.4–183.0)     |
| Rheumatic heart disease mortality| 5.0         | 5.7           | 5.4             | 0.9               |
|                                  | (3.6–6.9)   | (4.3–7.5)    | (4.4–6.7)       | (0.7–1.1)         |
| Rheumatic heart disease mortality| 27.6        | 48.6          | 38.7            | 6.8               |
|                                  | (3.6–6.9)   | (43.4–54.1)  | (35.4–42.2)     | (6.2–7.4)         |

Notes:
1. Data from [45]
2. Rates are for individuals of 35+ years, per 100,000, between years 2010-2012
3. Figures are age-standardised to the total Māori population as recorded in the 2001 Census. Prioritised ethnicity has been used – see ‘Ngā tapuae me ngā raraunga: Methods and data sources’ for further information.
4. Sources: Mortality Collection Data Set (MORT), Ministry of Health; National Minimum Data Set (NMDS), Ministry of Health
neglects the complexity inherent in this issue and the barriers that influence choices available to Māori [48].

With the introduction of the “National Science Challenges” (NSC) aimed at restructuring national scientific research funding, Māori health research and researchers were rarely recognised. Such lack of recognition has led to cries of multicultural neoliberalism whereby the New Zealand government were purposefully failing to substantively include Māori input or perspectives forcing Māori to continually claim and defend space for indigenous research in New Zealand [51].

The concept of health may differ between cultural groups [46]. Although the Western idea of health is the absence of disease, the concept for Māori is somewhat different, as it includes spirit, family, mind, land and physical well-being (Figure 5). For Māori issues of Te Whenua (land), Te Reo (language) and Whanaungatanga (extended family) are central to their culture and health [46,10,11]. It is therefore a mistake to educate for a healthy lifestyle unless Māori are aware of the links between poor physical health and how this impacts on their spiritual, mental and family wellbeing.

This expanded model of 5 interconnected aspects of Māori health and wellbeing includes land as part of their worldview. Whenua [land] is seen as being centrally relevant to health and illness by Māori spiritual healers [52]. The holistic view includes land as it is seen as a fundamental part of the person’s existence and an inclusive part of Māori identity. Any strategy for the modification of lifestyle will therefore be limited unless it includes an holistic perspective of health suggests that western medicine often fails to acknowledge a Māori worldview, and as a result the power of self-determination for Māori is reduced [46,53]. However, they caution that Māori health practitioners must not assume that Māori patients or researchers want Māori specific content. This was evident in their study where Māori specific options were declined by most of the participants.

Cameron et al. screened rural and urban Māori (n=252, 243, respectively) with non-Māori (n=256) aged between 20-64 years of age for CVD and associated risk factors. They found that rural Māori were at greatest risk of CVD because of higher levels of; obesity, dyslipidaemia, hypertension, smoking, type 2 diabetes and hyperuricaemia, than urban Māori, and urban non-Māori (Figure 6) [54]. A number of researchers have concluded that the development of an integrated healthcare programme is required to address the increased risks of CVD in rural and urban Māori that is sensitive to their cultural perspective of health [54-56]. While interventions that focus upon such risk factors are important they will fail to fully address the ethnic inequities that exist in New Zealand healthcare unless the socioeconomic inequities such as poverty are addressed first [33]. A weakness of interventions developed to improve Māori cardiovascular health suggest lies in their aim to change Māori behaviour rather than the behaviour of society towards Māori. The fundamental causes of ethnic differences in healthcare are rooted in; culture, biology, racism, economic deprivation and the politics of the dominant culture [9,33].

Low levels of engagement with primary healthcare providers has been observed in elderly patients of ethnic minorities with chronic disease conditions [57]. Such poor engagement reinforces a sense of powerlessness in the very people who require the greatest support. They suggested that clinicians must

Data taken from Cameron et al. [54]. Obesity is age adjusted body mass index kg/m²

Figure 5: The interface of the pathophysiology of CVD and cultural awareness for Māori.

Figure 6: Risk factors of CVD for Pākehā, rural and urban Māori.
be willing to be guided by their patients’ perceptions of need, not by their own assumptions of similarity and beliefs about illness and clinical management. As chronic conditions such as CVD are increasingly managed within the primary healthcare sector, Government policy must advocate greater engagement with vulnerable populations that emphasize health equity and provide incentives to general practitioners to adopt new approaches [58].

Using kaupapa Māori action research ("Māori Utilisation and Experience of Ischaemic Heart Disease Management") to improve the health and well-being of Māori within the northern region of Aotearoa/New Zealand has presented new opportunities to work with Māori communities and utilise a more sensitive approach to raising awareness of Māori to the dangers of CVD [59]. The integration of kaupapa Māori concepts in health research, combining the best of Māori worldview and western medicine is a novel way forward for positive outcomes for Māori cardiovascular health [53].

Although the New Zealand guidelines for the assessment and management of CVD recommend that high risk patients, or those who have previously had a CVD event, should be offered antihypertensive, antplatelet and lipid lowering therapies, (New Zealand Guidelines Group, 2003), only 67% of patients with diagnosed CVD were prescribed medications by their primary care physician [60]. In a 2012 study, only 60% of patients discharged from NZ hospitals with coronary heart disease were prescribed sufficient daily doses of statins. Amongst those at risk of CVD the prescription rates and long term adherence to preventive medications is much lower in Māori and Pacific Island ethnic groups [61]. The reasons for non-adherence have been indicated as: cost, regimen complexity, cultural beliefs regarding medication and; amongst patients with diabetes, depression [62]. One strategy designed to simplify the medication regimen is to combine the preventive medications in a ‘polypill’. In a 2011 randomised control trial (RCT) named Improving Adherence using Combination Therapy (IMPACT) Selak et al. assessed the use of a polypill, aimed at improving adherence amongst Māori and non-Māori patients at high risk of CVD in a New Zealand [61]. This study showed that the administration of a fixed dose combination drug treatment contains aspirin, statin, and two blood pressure lowering agents led to improved adherence and high acceptability to patients at risk of CVD, and their primary healthcare physicians. However, there was no statistical improvement in risk factors with the polypill when compared to the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63]. The mean discontinuation rate for the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63]. The mean discontinuation rate for the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63]. The mean discontinuation rate for the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63]. The mean discontinuation rate for the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63]. The mean discontinuation rate for the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63]. The mean discontinuation rate for the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63]. The mean discontinuation rate for the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63]. The mean discontinuation rate for the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63]. The mean discontinuation rate for the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63]. The mean discontinuation rate for the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63]. The mean discontinuation rate for the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63]. The mean discontinuation rate for the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63]. The mean discontinuation rate for the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63]. The mean discontinuation rate for the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63]. The mean discontinuation rate for the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63]. The mean discontinuation rate for the control group. The reason for this is likely to be that individual treatment modalities were already high in the control group, so improvement was relatively small [63]. The mean discontinuation rate for the fixed dose combination polypill treatment was 20% per year, a relatively small [63].

**Moving forward**

In an effort to redress the imbalance in CVD between Māori and Pākehā Maori leaders in CV health suggested an action plan to improve health sector responsiveness to Māori The plan has six categories that reflect the need for a multi-level, multi-sector approach to improving cardiovascular outcomes [67].

1. Policy development- the need for prioritise Māori health to gain explicit recognition of the Treaty of Waitangi in all health policy directives.
2. Information systems- The need for complete and consistent collection of ethnicity data in order to monitor CVD inequalities.
3. Needs assessment- CVD needs assessments for Māori communities.
4. Quality standards- Strongly recommend Māori-specific and equity-based performance indicators are applied to the cardiac healthcare.
5. Workforce development- To recruit Māori cardiovascular health workers.
6. Research- Both quantitative and qualitative research pertaining to access and equity of healthcare for Māori with CVD is required.

Bramley suggest that any health policy that aims to achieve equity for Māori CVD must be encompass all six points. Sadly the recommendations of the group have had little impact on the inequity between Māori and Pākehā [67]. Indeed suggest that institutional racism inherent in the New Zealand health system may result in perpetuating inequity between Māori and Pākehā. In New Zealand there are few successful CVD prevention and treatment programmes aimed at Māori, however some established schemes include: a) The PREDICT-CVD an electronic medical record decision support system was implemented in the ProCare Primary Health Organisations (PHOs) in Auckland; b) One Heart, Many lives, has been established to increase awareness, assessments, and treatment of Māori, a social networking program targeting CVD in Māori males using methods of risk reduction that include lifestyle changes and cost-effective chronic medication; c) The Indigenous Health Framework (HIF), developed by the University of Otago, is comprised of the Hui process and the Meihana model and used in the medical interview to provide a clinical assessment framework [35]. This scheme aims to translate the principles of cultural competency into an everyday approach for use by health practitioners to improve health service delivery for Māori patients and their Whanau [68]. Themes common to many successful CVD prevention schemes for indigenous peoples include many of the points outlined by including: A dedicated focus on the indigenous population, widespread community involvement, often using of indigenous community health workers, a focus on individuals at a high-risk CVD and regularly contact between the program and participants [67].

Traditionally the medical management of acute conditions is characterised by an initial consultation with a primary health provider followed by patient compliance with a treatment regimen, a strategy that is evidently failing Māori. When managing chronic conditions such as CVD it is imperative that patients take more active role patients in the day-to-day decisions about the management of their illness [69]. To achieve ideal care health providers must interact with informed, empowered patients who are active in managing their own health [70-72]. In order to bridge the cultural divide between minority groups such as Māori and New Zealand’s Pākehā dominated western medicine and better manage chronic conditions such as CVD,
c Culturally appropriate nurses should be employed by DHB's and attached to PHO's as chronic care managers. The role of a care manager is to develop individualised care plans in collaboration with the patient and the care providers that optimize the patient's health status and quality of life [73-76]. A number of studies have demonstrated the effectiveness of such patient-provider collaborations, including the LEONARDO project, with its extension the NARDINO project and the studies of highlighted the necessity to standardise the role chronic care managers within primary care to improve the implementation of chronic disease management strategies [69,73]. In view of the success of schemes such as the LEONARDO project similar provisions should be undertaken to increase the management of CVD in New Zealand.

Conclusion

Biculturalism has challenged New Zealand society to confront racial inequity and its debate has promoted societal recognition of human rights [57]. New Zealand has struggled to put equity principles into practice, indicating will without enactment. Equity they suggest is not systematically addressed below strategic levels and that it does not shape funding decisions, program development, implementation and monitoring. Despite examples of exceptional practice, driven by individuals, there is little incentive to pursue equity in New Zealand. Variation in healthcare provision for minorities between District Health Boards (DHBs) and between Primary Health Organizations (PHOs) implies substantial regional inequity. New Zealand healthcare has a fractured system of individual authorities with little or no governmental policies to address healthcare inequity and develop synchronous policies across the many DHBs. Detailed planning and the implementation of healthcare policy was devolved to DHBs who often further devolved responsibility to PHOs [57]. Despite inequities being recognised in DHB strategic documentation they have not been addressed systematically below this level.

In New Zealand, inequities in health are regarded as avoidable, unnecessary and unjust. The reduction of such inequities is one of the main objectives in the New Zealand Health Strategy. The Health Equity Assessment Tool (HEAT) was devised by the Ministry of Health in 2002 for the assessment of healthcare interventions to reduce inequities and guide the development of DHB strategies to address the potential of policies, programs and service interventions for reducing inequities [77]. However, in practice there has been little evidence that inequity has been addressed for Māori, Pacific peoples or Asian minorities, or for people on low-incomes or by geography [77]. Inequity in healthcare has been defined as a complex "wicked" problem. Wicked problems are difficult to clearly define; have many interdependencies and are often multi-causal; attempts to address them often lead to unforeseen consequences, they are often unstable, usually have no clear solution, are socially complex, hardly ever sit conveniently within the responsibility of a single organization, involve changing behaviour, and are often characterized by chronic policy failure [78]. Addressing such problems such as inequity in healthcare requires a governmental approach to construct and manage a collaboration of the many interested parties to develop policy for funding, monitoring and regulation, education and culture changes [79]. Government must realise that health in the community is also dependent upon other issues such as social welfare and housing. Because health inequities are insufficiently monitored, governments are poorly informed of their failure to address these issues that are a fundamental human right.

If, as reported, social deprivation, abuse, neglect, household dysfunction and poverty remain risk factors for chronic disease in adulthood such as CVD then government policies must address this [80-85]. The right to health, for the state to provide the highest attainable standard of physical and mental health, is not an unreasonable expectation of citizens of the state [85]. The right to health is essential for alleviating poverty [86]. Moving people out of poverty requires much more than money [87]. If the New Zealand government is to address the inequities in healthcare it requires a greater commitment to a more redistributive social and economic agenda and to protect the most vulnerable members of society, our children, from developing chronic diseases in adulthood it is essential to reduce poverty [88]. Child welfare needs to be urgently addressed and children at risk of social deprivation, abuse, neglect and household dysfunction must be identified and supported e.g., with home visits from district health nurses and quality foster care.

Author Contributions

WMW is the sole researcher and author of this work.

References

1. World health organization (2008) The global burden of disease: 2004 update. Geneva: World Health Organization.
2. New Zealand Ministry of Health (2015) Mortality and demographic data, 2012. In: Health, MO (ed.) Wellington, New Zealand: New Zealand Government.
3. Ministry of Health (2015) Mortality and demographic data, 2012. In: HEALTH, MO (ed.).Wellington: New Zealand Government.
4. World Health Organization (2005) Commission on social determinants of health: note by the Secretariat (document number: EB115/35). In: SHARING, IRFI (ed.). Geneva, Switzerland: World Health Organization, Executive Board, p: 115.
5. Dahlgren G, Whitehead M (1991) Policies and strategies to promote social equity in health. Stockholm: Institute for Future Studies.
6. Dahlgren G, Whitehead M (2007) European strategies for tackling social inequalities in health: Levelling up part 2. World Health.
7. Jones CP (2001) Invited commentary: “Race,” racism and the practice of epidemiology. Am J Epidemiol. 154: 299-304.
8. Nazroo JY (2003) The structuring of ethnic inequalities in health: Economic position, racial discrimination, and racism. AJCN. 93: 277-284.
9. Williams DR (1997) Race and health: Basic questions, emerging directions. Ann Epidemiol. 7: 322-333.
10. Robson B, Harris R (2007) Hauora: Maori standards of health IV: A study of the years 2000-2005, Te Ropu Rangahau Hauora a Eru Pomare Wellington, New Zealand.

11. Harris R, Cormack D, Stanley J, Rameka R (2015) Investigating the relationship between ethnic consciousness, racial discrimination and self-rated health in New Zealand. *Plas One*.

12. Ministry of Health (2002b) Reducing Inequalities in Health. In: HEALTH, MO (ed.) Wellington, New Zealand: New Zealand Government.

13. Howden-chapman P, Blakely T, Blaiklock A, Kiro C (2000) Closing the health gap. *NZ Med J*. 113: 301-302.

14. New Zealand Ministry of Health (2006) New Zealand burden of diseases, injuries and risk factors study, 2006–2016 [Online]. New Zealand Government. Available: New Zealand Burden of Diseases, Injuries and Risk Factors Study.

15. Jones CP, Truman BI, Elam-evans LD, Jones CA, Jones CY, et al. (2008) Using “socially assigned race” to probe white advantages in health status. *Ethn Dis*. 18: 496-504.

16. Perreira KM, Telles EE (2014) The color of health: Skin color, ethnoracial classification and discrimination in the health of Latin Americans. *Soc Sci Med*. 116: 241-250.

17. Reid J, Cormack D, Crowe M (2016) The significance of socially-assigned ethnicity for self-identified Maori accessing and engaging with primary healthcare in New Zealand. *Health*. 20: 143-160.

18. Jones CP (2000) Levels of racism: A theoretic framework and a gardener's tale. *AJCN*. 90: 1212.

19. Westbrook E, Baxter J, Hogan J (2001) Are Maori under-served for cardiac interventions? *N Z Med J*. 114: 484-487.

20. Davey-smith G (2000) Learning to live with complexity: Ethnicity, socioeconomic position, and health in Britain and the United States. *AJCN*. 90: 1694.

21. Rata E, Zubaran C (2016) Ethnic classification in the New Zealand health care system. *J Med Philos*. 41: 192-209.

22. Stephens C, Nettleton C, Porter J, Willis R, Clark S (2005) Indigenous peoples' health - Why are they behind everyone, everywhere? *Lancet*. 366: 10-13.

23. Gracey M, King M (2009) Indigenous health part 1: Determinants and disease patterns. *Lancet*. 374: 65-75.

24. Miller C (2016) The shared history of public health and planning in New Zealand: A different colonial experience. *Progress in Planning*. 106: 1-21.

25. Pack S, Tuffin K, Lyons A (2016) Accounting for racism against Maori in Aotearoa/New Zealand: A discourse analytic study of the views of Maori adults. *J Community Appl Soc Psychol*. 26: 95-109.

26. Reid J, Taylor-moore K, Varona G (2014) Towards a social-structural model for understanding current disparities in Maori health and well-being. *J Loss Trauma*. 19: 514-536.

27. Reid P, Robson B (2007) Understanding health inequities. In: Robson B & Harris R (eds.) Hauora: Maori standards of health IV.

28. Jackson M (2002) Perceptions, partnership and progress. Tobacco conference.

29. Rochford TS (2012) Ten reasons why genetics does not explain health disparities between Maori and non-Maori. *New Genet Soc*. 31: 99-110.

30. Wong J, Love DR, Kyle C, Daniels A, White M, et al. (2002) Melanocortin-3 receptor gene variants in a Maori kindred with obesity and early onset type 2 diabetes. *Diabetes Res Clin Pract*. 58: 61-71.

31. Wong J, Nock NL, Xu ZY, Kyle C, Daniels A, et al. (2008) A polymorphism (D20S32e) close to the human melanocortin receptor 3 is associated with insulin resistance but not the metabolic syndrome. *Diabetes Res Clin Pract* 80: 203-207.

32. Gladding P, Mackay J, Webster M, White H, Ellis K, et al. (2010) Longitudinal study of a 9p21.3 SNP using a national electronic healthcare database. *Per Med*. 7: 361-369.

33. Curtis E, Harwood M, Riddell T, Robson B, Harris R, et al. (2010) Access and society as determinants of ischaemic heart disease in indigenous populations. *Heart Lung Circ*. 19: 316-324.

34. Hart JT (1971) The inverse care law. *Lancet*. 297: 405-412.

35. Hill S (2008) Socio-economic inequalities in health. In: Dew K and Matheson A (eds.) Understanding Health Inequalities in Aotearoa New Zealand. Wellington: Otago University Press.

36. Lip GYH, Barnett AH, Bradbury A, Cappuccio FP, Gill PS, et al. (2007) Ethnicity and cardiovascular disease prevention in the United Kingdom: A practical approach to management. *J Hum Hypertens*. 21: 183-211.

37. Gordon T, Castelli WP, Hjortland MC, Kannel WB, Dawber TR (1977) High-density lipoprotein as a protective factor against coronary heart-disease - Framingham study. *Am J Med*. 62: 707-714.

38. Yusuf S, Hawken S, Ounpuu S, Bautista L, FranzosI MG, et al. (2005) Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: A case-control study. *Lancet*. 366: 1640-1649.

39. Wells S, Jackson R (2005) Online management of cardiovascular risk in New Zealand with PREDICT™—getting evidence to the ‘moment of care’. Health Care & Informatics Review Online.

40. Bhopal R, Fischbacher C, Vartiainen E, Unwin N, White M et al. (2005) Predicted and observed cardiovascular disease in South Asians: Application of FINRISK, Framingham and SCORE models to Newcastle Heart Project data. *J Public Health*. 27: 93-100.

41. Conroy RM, Pyorala K, Fitzgerald AP, Sans S, Menotti A, et al. (2003) Estimation of ten year risk of fatal cardiovascular disease in Europe: The SCORE project. *Eur Heart J*. 24: 987-1003.

42. Brindle P, May M, Gill P, Cappuccio F, D'agostino R, et
al. (2006) Primary prevention of cardiovascular disease: A web-based risk score for seven British black and minority ethnic groups. Heart. 92: 1595-1602.

43. New Zealand Guidelines Group (2003) Evidence-based best practice guideline. The assessment and management of cardiovascular risk. In: Health MO (ed.) New Zealand Guidelines Group. New Zealand Government.

44. Marshall T (2005) Evaluating national guidelines for prevention of cardiovascular disease in primary care. J Eval Clin Pract. 11: 452-461.

45. Kahukura T (2015) Māori health chart book.

46. Castro N, Lambrick DM, Faulkner J, Lark S, Williams MA, et al. (2013) Decreasing the cardiovascular disease burden in Māori children: The interface of pathophysiology and cultural awareness. J Atheroscler Thromb. 20: 833-834.

47. Ayo N (2012) Understanding health promotion in a neoliberal climate and the making of health conscious citizens. Crit Public Health. 22: 99-105.

48. Warbrick I, Wilson D, Boulton A (2016) Provider, father and brother: Sedentary Māori men and their thoughts on physical activity. Int J Equity Health.

49. Heidenreich PA, Trogdon JG, Khavjou OA, Butler J, Dracup K, et al. (2011) Forecasting the future of cardiovascular disease in the United States a policy statement from the American Heart Association. Circulation. 123: 933-944.

50. Wright J, Harwood V (2012) Biopolitics and the 'obesity epidemic': Governing bodies, Routledge.

51. Prussing E, Newbury E (2016) Neoliberalism and indigenous knowledge: Māori health research and the cultural politics of New Zealand's "National Science Challenges". Soc Sci Med. 150: 57-66.

52. Mark GT, Lyons AC (2010) Māori healers' views on wellbeing: The importance of mind, body, spirit, family and land. Soc Sci Med. 70: 1756-1764.

53. Rolleston AK, DoughtY R, Poppe K (2016) Integration of Kaupapa Māori concepts in health research: A way forward for Māori cardiovascular health? J. Prime Health Care. 8: 60-66.

54. Cameron VA, Faatoese AF, Gillies MW, Robertson PJ, Huria TM, et al. (2012) A cohort study comparing cardiovascular risk factors in rural Māori, urban Māori and non-Māori communities in New Zealand. BMJ Open.

55. Faatoese A, Gillies M, Robertson P, Doughty R, Whalley G, et al. (2010) Comparison of rates of diagnosed and undiagnosed cardiovascular disease and its risk factors among Māori and non-Māori communities in New Zealand. Eur Heart J. 31: 747-747.

56. FaatoeseAF, Pitama SG, Gillies TW, Robertson PJ, Huria TM, et al. (2011) Community screening for cardiovascular risk factors and levels of treatment in a rural Māori cohort. Aust N Z J Public Health. 35: 517-523.

57. Sheridan NF, Kenealy TW, Connolly MJ, Mahony F, Barber PA, et al. (2011) Health equity in the New Zealand health care system: a national survey. Int J Equity Health.

58. Sheridan NF, Kenealy TW, Kidd JD, Schmidt-Busby Jig, Hand JE, et al. (2015) Patients' engagement in primary care: Powerlessness and compounding jeopardy: A qualitative study. Health Expect. 18: 32-43.

59. Kerr S, Penney L, Barnes HM, Mccreanor T (2010) Kaupapa Māori action research to improve heart disease services in Aotearoa, New Zealand. Ethn Health. 15: 15-31.

60. Mehta S, Wells S, Riddell T, Kerr A, Pylypchuk R, et al. (2011) Under-utilisation of preventative medication in patients with cardiovascular disease is greatest in younger age groups (PREDICT-CVD 15). J Prim Health Care. 3: 93-101.

61. Selak V, Elley CR, Crengle S, Harwood M, Doughty R, et al. (2011) Improving adherence using combination therapy (IMPACT): Design and protocol of a randomised controlled trial in primary care. Contemp Clin Trials. 32: 909-915.

62. Gellad WF, Grenard J, Mcglynn EA (2009) A review of barriers to medication adherence: A framework for driving policy options, RAND.

63. Selak V, Elley CR, Bullen C, Crengle S, Wadhams A, et al. (2014) Effect of fixed dose combination treatment on adherence and risk factor control among patients at high risk of cardiovascular disease: Randomised controlled trial in primary care. BMJ.

64. Patel A, Cass A, Peiris D, Usherwood T, Brown A, et al. (2015) A pragmatic randomized trial of a polypill-based strategy to improve use of indicated preventive treatments in people at high cardiovascular disease risk. Eur J Prev Cardiol. 22: 920-930.

65. Thom S, Poulter N, Field J, Patel A, Prabhakaran D, et al. (2013) Effects of a fixed-dose combination strategy on adherence and risk factors in patients with or at high risk of CVD. The UMPIRE randomized clinical trial. JAMA. 310: 918-929.

66. Castellano JM, Sanz G, Penalvo JL, Bansilal S, Fernandez-ortiz A, et al. (2014) A polypill strategy to improve adherence results from the FOCUS project. J Am Coll Cardiol. 64: 2071-2082.

67. Bramley D, Riddell T, Crengle S, Curtis E, Harwood M, et al. (2004) A call to action on Māori cardiovascular health. N Z Med J. 117: U957.

68. Pitama S, Huria T, Lacey C (2014) Improving Māori health through clinical assessment: Waikare o te Waka o Meihana. BMJ.

69. Ciccone MM, Aquilino A, Cortese F, Scicchitano P, Sassara M, et al. (2010) Feasibility and effectiveness of a disease and care management model in the primary health care system for patients with heart failure and diabetes (Project Leonardo). Vasc Health Risk Manag. 6: 297-305.

70. Cecere A, Scicchitano P, Zito A, Sassara M, Bux F, et al. (2014) Role of care manager in chronic cardiovascular diseases. Ann Gerontol Geriatric Res. 1: 1005.

71. World Health Organization (2002) Innovative care for chronic conditions: Building blocks for action [Online]. Geneva, Switzerland: World Health Organization.
72. Wagner EH (1998) Chronic disease management: What will it take to improve care for chronic illness? *Eff Clin Pract*. 1: 2-4.

73. Brunetti ND, Dellegrottaglie G, Di Giuseppe G, DI Biase M (2014) Remote tele-medicine cardiologist support for care manager nursing of chronic cardiovascular disease: Preliminary report. *Int J Cardiol*. 176: 552-556.

74. Ishani A, Greer N, Taylor BC, Kubes L, Cole P, et al. (2011) Effect of nurse case management compared with usual care on controlling cardiovascular risk factors in patients with diabetes: A randomized controlled trial. *Diabetes Care*. 34: 1689-1694.

75. Deales A, Fratini M, Romano S, Rappelli A, Penco M, et al. (2014) Care manager to control cardiovascular risk factors in primary care: The Raffaello cluster randomized trial. *Nutr Metab Cardiovasc Dis*. 24: 563-571.

76. Lukewich J, Edge DS, Van Denkerkhof E, Tranmer J (2014) Nursing contributions to chronic disease management in primary care. *J Nurs Adm*. 44: 103-110.

77. Ministry of Health (2002a) The health equity assessment tool. In: Department of Public Health (ed.) Wellington, NZ: University of Otago and Ministry of Health.

78. Australian Public Service Commission (2012) Tackling wicked problems: A public policy perspective. Canberra Australia: Australian Government.

79. Whitehead M, Dahlgren G (2006) Concepts and principles for tackling social inequities in health: Levelling up Part 1. In: EUROPE, WHOROF (ed.) Copenhagen, Denmark: World Health Organization.

80. Su S, Jimenez MP, Roberts CTF, Loucks EB (2015) The role of adverse childhood experiences in cardiovascular disease risk: A review with emphasis on plausible mechanisms. *Curr Cardiol Rep*.

81. Nemeroff CB (2016) Paradise lost: The neurobiological and clinical consequences of child abuse and neglect. *Neuron*. 89: 892-909.

82. Danese A, Dove R, Belsky DW, Henchy J, Williams B, et al. (2014) Leptin deficiency in maltreated children. *Transl Psychiatry*. 4.

83. Danese A, McEwen BS (2012) Adverse childhood experiences, allostasis, allostatic load and age-related disease. *Physiol Behav*. 106: 29-39.

84. Gonzalez A (2013) The impact of childhood maltreatment on biological systems: Implications for clinical interventions. *J Paediatr Child Health*. 18: 415-418.

85. United Nations Treaty Series (1996) International covenant on economic, social and cultural rights. United States of America. UN General Assembly.

86. Yusuf S, Woodham J (2014) Human rights agenda for social change. *Jswhr*. 2: 131-160.

87. Bick J, Nelson CA (2016) Early adverse experiences and the developing brain. *Neuropsychopharmacology*. 41: 177-196.

88. Pearce J, Dorling D (2006) Increasing geographical inequalities in health in New Zealand. *Int J Epidemiol.* 35: 597-603.

**ADDRESS FOR CORRESPONDENCE**

Warren Miner-Williams, Research Officer, Person Centred Research. School of Clinical Sciences, Faculty of Health and Environmental Sciences, Auckland University of Technology, 90 Akoranga Drive, Northcote, Auckland, 0627, New Zealand, Tel: 6231-09 921 9999; E-mail: warren.miner-williams@aut.ac.nz

Submitted: December 09, 2016; Accepted: December 16, 2016; Published: December 24, 2016