Metaphors of Menopause in Medicine

A thesis presented in partial fulfilment of the requirements for the degree of

Master of Arts

in

Psychology

at Massey University, Wellington, New Zealand

Patricia Ruth Niland

2010

Abstract

Medical textbooks have previously represented women's bodies and menopause life transitions by using notions of 'machine productivity' and 'machine breakdown' (Martin, 1987). This study aimed to explore whether these representations have changed, especially given recent HRT clinical trial results. Eight relevant compulsory medical textbooks for first and second year medical students at two New Zealand Universities were identified. A Foucauldian discourse analysis (Parker, 1990) was undertaken on relevant content to identify representations of menopause, HRT, women's bodies, and ageing. Five major discourses were employed in the textbooks in descriptions of menopause and HRT: failure, estrogen deficiency as disease; HRT as saviour; obscurity and the new discovery discourse. Menopause continues to be represented as resulting from a 'failure' of a machine-like body. Although the recent HRT clinical trials were reported as a serious risk factor in half of the textbooks, HRT was also represented as a saviour particularly against postmenopausal osteoporosis. The discovery of 'new' drugs to 'treat' HRT and the 'postmenopausal' patient were heralded with much excitement. Medical textbooks continue to use failure discourses to describe women's bodies at menopause. New risk-based HRT assessments for 'patients' with menopause 'symptoms' are promoted. These portrayals reinforce linear and reductionist ways of thinking about menopause and women at midlife and provide few spaces for resistance or alternative constructions to more accurately reflect women's embodied worlds.

Acknowledgements

I wish to acknowledge my supervisor, Dr Antonia Lyons, who was my inspiration for embarking on this academic journey. Her dedicated support, guidance and friendship throughout this challenging endeavour have enabled me to appreciate how rewarding it is to achieve this goal.

Special thanks to my father, Joe, who has an unwavering belief in my ability to succeed at whatever I do and to my daughters, Geena, Alice and Lauren, who encouraged me every day to be myself but keep writing. I am also grateful to Chris for his generosity in always lending me books from the Hay library and his wise advice, enthusiasm and ongoing support for me to complete this project.

Finally, I dedicate this thesis to Barbara Seaman (1935 – 2008), a principal founder of the women's health feminism movement who persistently and fearlessly challenged medical authority in order to give women more informed and safer choices for their healthcare.

Table of Contents

List of Tables

List of Figures

Chapter 1: Menopause and Hormone Replacement Therapy

Introduction

Menopause exposes all women to a paradox: although they are healthy, they will be rendered sick by the medicalisation of their bodies in this life transition. In New Zealand there are nearly half a million women aged 50 to 70 years (Statistics NZ, 2010) and on a more global scale there are millions of mid-life women who, although they are healthy, must negotiate menopause as an illness that risks their future health. The 'remedy' for this 'illness' is a drug, hormone replacement therapy (HRT), and in its peak year in 2001 there were almost 400,000 publicly funded HRT prescriptions issued to women in New Zealand (Pharmac, 2005). Another paradox that women face is that although HRT is prescribed to preserve their future health, it has been shown to have serious health risks. Clinical trials now suggest that millions of women in the future could be exposed to increased risk from cardiovascular disease and breast cancer due to treatment (HRT) for an illness that can be argued not to exist (menopause). This research seeks to examine the paradoxes surrounding menopause, and in doing so, present new knowledge to women about menopause and HRT.

In this chapter I examine the historical, cultural and socio-economic context of menopause and hormone replacement therapy (HRT). For menopause, this context is underpinned by a physiological phenomenon, so I will firstly outline the current biomedical knowledge base of menopause in terms of menstrual cycles, hormone levels and bodily changes and 'symptoms'. For HRT I also firstly provide a 'beginner's guide to HRT' to show the biomedical rationale for its use in menopause. This is followed by a discussion of HRT in its historical context including the background to the Women's Health Initiative (WHI) clinical trials and its current controversies, which are yet another step in the story of estrogen and its warnings.

What is Menopause?

Menopause can be defined as a woman's last menstrual period and this event "takes place in a gradual process of physiological change, occurring concurrently with age and developmental changes, within a psychosocial and cultural context" (Hunter & O'Dea,

1977, p.199). It is not helpful to locate menopause as a discrete event – the last menstrual period - because this implies it happens quickly and reifies it as a 'thing'. However, it is informative to focus on a period of developmental physiological change, a transition, because this helps women to make sense of their bodies changing over time as they did during puberty. Hunter and O'Dea (1977) propose that the meaning of these bodily changes is "likely to be intimately related to sociolinguistic influences, such as discourses of gender, ageing and reproduction [and] a woman's subjectivity is therefore positioned between her perception of biological changes and the discursive constructions of the menopause, which are influenced by social, political and cultural practices and traditions" (p.199). Before we can examine these 'discursive constructions' of menopause and the meaning of these biological changes, it is important to understand exactly what these bodily changes are. There is much confusion and mis-information about what menopause is in the media (Buchanan et al, 2001) and many competing interests represent this experience to women (Moynihan & Cassels, 2005). There are also differing views of menopause from women themselves. They report that it is an experience to be overcome with healthy habits and a "get on with it" life style (Breheney & Stephens, 2003); and this experience represents images of women as healthy and strong with control over their bodies and themselves (Hvas & Gannik, 2008). In terms of private ageing, women report "the changes in ovarian physiology as altering their reproductive status, and they thus experience the emergence of a new identity" (Ballard et al, 2009, p.269).

Physiology

From a biological perspective, menopause can be viewed as a 'gradual process of physiological change' which is part of women's reproductive development over time. Menopause within this perspective is therefore seen as a *transition phase* rather than a discrete event, providing a clearer idea of what menopause means for the physiology of our bodies. The physiological changes that define this transition phase have been chartered and documented in the Stages of Reproductive Aging Workshop model (STRAW; Soules et al, 2001) which is shown in Figure 1. This workshop was convened with the aim of defining a generic biomedical model of menopause as a dynamic, developmental transition. The goals were to clarify the existing confusing nomenclature of menopause, define reliable research measures and "identify clinical and basic

knowledge gaps for researchers, health practitioners and the public" (Soules et al, 2001, p.845). Although this focus on women's 'reproductive system' is biologically reductionist and essentialist (Kaufert & Lock, 1997; Gannon, 1999; Meyer, 2001; Perz & Ussher, 2008), this workshop attempted to locate only physiological changes that could be reliably measured and there was open debate about this by a number of medical specialists and academic researchers with all affiliations openly stated.

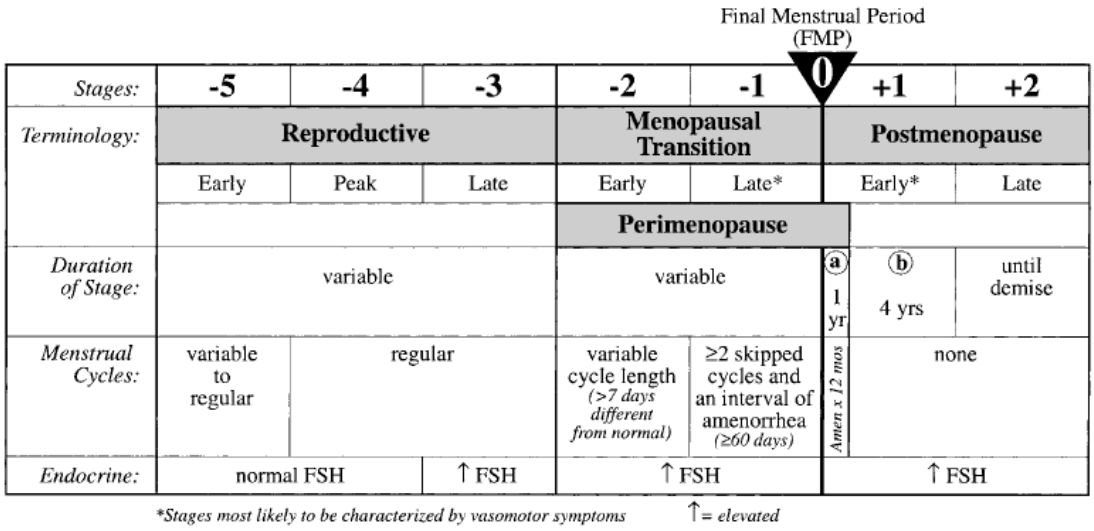

Figure 1: Stages of normal reproductive ageing in women, taken from Soules et al, 2001, p.845

The STRAW workshop identified only two reliable and consistent physiological changes for menopause: changes over time in menstrual cycles and changes over time in the levels of the Follicle Stimulating Hormone (FSH) which stimulates ovulation. Menstrual cyclicity can be determined by a woman's personal recorded menstrual history and FSH levels can be measured by a blood test. These two criteria then form the basis for the STRAW model, a 'general staging model' for healthy women who will progress through these stages but there will always be individuals "who seesaw back and forth between stages or skip a stage altogether" (Soules et al, 2001, p.846). Other biological markers such as 'duration and amount of menstrual flow', estradiol (estrogen), progesterone and luteinizing hormone (another ovulation stimulator) levels were not included due to their high variability in women. Also, fertility was not included because "relative fertility in an individual is nearly impossible to measure and is co-dependent on the fertility of the male partner" (Soules et al, 2001, p.847).

This developmental reproductive system, as defined by menstrual cyclicity and FSH levels, has seven stages that precede and follow the final menstrual period (FMP) which is stage 0, the 'anchor' point in the process. Stages -5 to -3 are the reproductive interval; stages -2 to -1 are the menopause transition or peri-menopause; and stages +1 to +2 are the post menopause phase (see Figure 1). Women's ages in these stages is too variable to be generalised, a guide is that "the menopausal transition usually begins when women are in their mid-to-late 40s, and can last several years, most commonly 4–5 years" (Nelson, 2008, p.760). This model defines menopause as a developmental transition based on menstrual cyclicity and FSH levels only, therefore I now turn to the dynamics of these criteria.

Menstrual cycles

The STRAW workshop described the physiology of menstrual cycles for a normal, healthy woman. After a woman's first period (entry into stage -5) it normally takes several years for her to have regular menstrual cycles. They then occur every 21–35 days for a number of years (stages -4 and -3). In the early menopause transition (stage -2) a woman's menstrual cycles remain regular but the length changes by 7 days or more (e.g., her regular cycles are now every 24 days instead of every 31 days). The late menopause transition (stage +1) is identified when a woman skips two or more menstrual cycles and has at least one inter-menstrual interval of 60 days or more. Stages +1 and +2 are the postmenopause phase and begin (in retrospect) when a woman does not have a period for 12 months.

FSH Levels

The reasoning for using FSH levels is that they are a signal for ovulation occurring or not occurring. On days 1 to 5 of the menstrual cycle (period bleeding begins on day 1) FSH levels increase to stimulate the ovary follicles to begin developing an ova/egg and to produce estradiol to thicken the uterus to receive the egg once it matures. A greater than normal level of FSH (> 10 mIU/ml is the research cutoff value) at this stage signals that there may be a lack of oocytes (or immature eggs) in the ovaries. The FSH level therefore rises to stimulate the ovary follicles more. This is like revving a car to go faster when it is slowing down. However, this increase is not straightforward as its effect is subtle and there is high variability in women which makes it very difficult to set meaningful cutoff levels for different ages Also, the estradiol level in the early cycle

may be elevated (>80 pg/ml) too which can suppress a higher FSH level therefore a second FSH reading should be taken in the next cycle. Together this shows how we can reliably recognise the bodily changes of the menopause transition, accepting the variability involved. Next I will examine the bodily changes as they are experienced by women.

Bodily Changes and Symptoms

The bodily changes of menopause as experienced by women are typically reported as 'symptoms' by the medical community and by women themselves. As with the representation of menopause generally, the reporting and publicizing of these symptoms is influenced by differing research approaches. For example, a biomedical study identified 50 menopause 'symptoms' and then tested whether women reported them before and after menopause (Abraham et al, 1994). On the other hand, a socio- cultural study shifted the focus from vaginal dryness rates and changing sexual activity to gain some cultural understanding of what menopausal changes mean for women's sex lives (Winterich, 2003). A Medline and Cochrane meta-analysis of research on menopause symptoms and therapies from 1966 to 2006 shows that "many symptoms have been attributed to menopause, but only vasomotor dysfunction [50% of women] and vaginal dryness [30% of women] are consistently associated with this time of life in epidemiological studies. Other common symptoms such as mood changes, sleep disturbances, urinary incontinence, cognitive changes, somatic complaints, sexual dysfunction, and reduced quality of life may be secondary to other symptoms, or related to other causes" (Nelson, 2008, p.760). Vasomotor dysfunction refers to a 'hot flash', experienced as a feeling of intense heat in the body with sweating and rapid heartbeat which may typically last from two to thirty minutes. It is worth noting here that the medical science community today admits that the "mechanisms underlying menopausal vasomotor problems are not clearly understood" (Page et al, 2006, p.345). The general premise is that "…VMS occur due to a disruption in pathways modulated by ovarian hormones that maintain temperature homeostasis" (Deecher, 2009, p.77). Nelson (2008) also cites the cultural differences in reporting of symptoms:

> African-American women reported more frequent vasomotor dysfunctions than did White women, who in turn reported more than did Hispanic, Chinese, and Japanese women. White and Hispanic women had sleep difficulties more often

> than did African-American, Chinese, and Japanese women. Hispanic women reported body pain more frequently than did White women. In surveys done in Asia, most Asian women had body or joint pain rather than vasomotor symptoms, although proportions varied with ethnic group. (p.764)

As well as cultural differences in the interpretations of bodily changes in the menopause transition, there is also variation in individual women's interpretations. Hvas & Gannik (2008) found that Danish women's images of their menopause experiences ranged between a feeble and osteoporotic woman, lacking energy and sex-appeal to a healthy and vital woman in control of her body and herself. Other women report feelings of freedom from pregnancy as well as apprehensions about bodily changes and self care (Bertero, 2003). Women have also variously interpreted menopause as a non-event, a continuation of self, the end of reproductive life, a sign of ageing and an unspoken taboo (Hunter & O'Dea, 1997).

Thus the physiological bodily changes and symptoms of the menopause transition as identified by the medical science community are somewhat conservative and women's perceptions of these bodily changes vary across sociolinguistic and cultural locations. This returns us to our definition of the menopause transition, specifically that it is located within women's perceptions of their bodily changes and the discursive constructions of their socio-cultural and political worlds. These discursive constructions will now be explored from an historical and socio-economic perspective.

Historical Perspective and the Influence of Medicine

Menopause is a life transition that has been constructed differently at different times and places. Sybylla (1997) shows this 'history of menopause' in her Foucauldian genealogy of this life stage as it has been represented in the medical literature over time. Sybylla proposes that our knowledge and practices concerning menopause have more to do with historical circumstance than with scientific progress towards greater truths. This historical contingency can be examined by focusing on the power relations inherent in the relationship between "menopause, the feminine, and various social groups and forces at particular points in time in some western cultures" (Sybylla, 1997, p.202). This type of analysis focuses on power relations because it views them as constitutive of

women's subjectivity, and it is therefore vital to focus on forms of power domination which may suppress or alienate them. The focus for menopause is on the medical profession because of the asymmetrical power relationship which may develop between them and the public. The medical profession produces knowledge from its own perspective and the public tends to uncritically accept this knowledge as the 'truth'. This dominance is evidenced in everyday life where only doctors have the authority to diagnose physical and mental illness, prescribe drugs, perform surgery and sign birth and death certificates (Germov, 1998). This dominance has also been allied to a 'take over' of the responsibility of the church to maintain the morality of its flock. The new secular morality is the healthy body and medicine maintains this new virtue by rationally applying its knowledge and practices to produce healthy, dependable and competent bodies (Turner, 1992). To examine the way in which this dominance has influenced women, Sybylla outlines three shifts in the menopause discourse over time.

Firstly, early in the nineteenth century women were seen as victims of their reproductive organs and their uterus was deemed to be the cause of their female weakness. A typical Victorian medical view of this power of the uterus over women's sanity is seen in Mason's (1845) pronouncement that "so remarkable a control does it exercise over the mind and its reasoning powers, that the greater frequency of insanity…has been referred to the mode and regularity with which menstruation is performed" (as cited in Sybylla, 1997, p.205). Later, this cause shifted to a woman's ovaries as espoused by the Victorian physician, Tilt (1870) who declared that "the ovarian nisus [impulse] seems to…so influence the brain that woman, no longer the mistress of her own actions, is literally "fuddled with animal spirits"…" (as cited in Sybylla, 1997, p.205). Thus the medical view was that women of reproductive age were to be managed by the guiding influence of the medical profession so as to avoid the disturbances of their reproductive organs which could induce mental or physical illness. This dominant framing of women's bodies as vulnerable and weak was achieved in part by the medical profession's direct access to women's bodies and its authoritative claim to be the only legitimate source of knowledge about women's health. However, as Sybylla (1997) asserts, the medical profession also existed within a much wider network of power relations through their membership on hospital boards which brought them into close contact with the business and governmental leaders of the middle class. To view the dynamics of these power relations Sybylla uses Foucault's body/power lens which

focuses on what type of body a society needs. In the nineteenth century the industrial economic system required docile bodies for its production processes and therefore it promoted an ordered, decent and healthy lifestyle for men, women and children. A woman's body was shaped for reproduction so as to produce the bodies required for the work force and this confined her to her fragile health and the domestic care of her home and family under the guidance of medical experts. Male bodies on the other hand were shaped as forceful, hard and rational because capitalism needed strong entrepreneurs to implement and control production. What did this mean for the Victorian menopausal woman? Sybylla identifies two types of power that the state and middle classes focused on younger productive women only - bio-power for docile productive bodies and pastoral power for controlling the body through health/pathology. Therefore the menopausal woman was no longer in the game, her 'femininity' no longer required and she was thought "to gain masculine qualities, which were seen as an improvement over her former femininity" (Sybylla, 1997, p.204).

In the mid-twentieth century the medical profession's framing of the menopausal woman was diametrically opposed to that of the nineteenth century. As Sybylla (1997) notes, this era saw a multitude of domestic and human science experts ascend in power to scrutinise and appraise the housewife, home and family. For women this meant a new scientific regime for nutrition, hygiene, sexual relations and childrearing which meant her domestic role as housewife and nurturing mother to her husband and children was now directed by experts beyond her home. At the same time the war brought a new ideal of masculinity for men of industry. The scientific and technological advancements which ensued meant that industry now required a "hardheaded, ultrarational, efficient, scientific technoman" who in turn needed a "modern wife", one who maintained the family home under expert guidance and "who was soft, emotional, "dumb", passive, and sexually appealing" (Sybylla, 1997, p.210). What did this mean for the twentieth century menopausal woman? In contrast to the menopausal Victorian woman who was surplus to production requirements, the twentieth century "modern wife" was still required to support her "technoman", to nurture him and be an attractive sexual partner. A strategic move to maintain women's feminine, domestic servitude beyond their reproductive years came from the medical profession. The gynecologist Robert Wilson's book *Feminine Forever* (Wilson, 1966) portrayed menopause as a loss of femininity and sexual attraction and positioned HRT as the remedy for this crisis.

Therefore, "the mission of HRT was to maintain the well-being of husband, state, economy and herself" (Sybylla, 1997, p.211). In Victorian times the strategy to find fault in female bodies was focused on the fragility of their reproduction, now the strategy was to find fault with the post-reproductive body.

In the late twentieth century women's participation in the workforce has increased and they have realised their independence from female domesticity in pursuing their own careers and lifestyle choices. This suits the capitalist drive for consumerism but what effect does it have on the menopausal woman today? We can answer this by looking at what type of body is required by the late twentieth century capitalist economy. In this era the workforce requirements have changed with more globally competitive industrialised nations using superior scientific and technological innovations to compete for consumer-production dominance. This requires a workforce that is "mobile, active, innovative, resourceful, creative, flexible, risk-taking and highly individualistic" (Sybylla, 1997, p.213). The 'ideal body' here is an active and youthful one and a strategy to achieve it by the state is to promote the capitalist rhetoric of 'individualism' with its virtues of personal health and fitness. For the menopausal women this individualism discourse is reflected in the medical experts now exhorting her to 'make up her own mind' about using HRT for menopause based on the facts as presented by their profession and her own personal assessment of individual risks for her future health. However, this 'choice' is influenced by a strategic fault finding in bodies and now the menopausal woman's body is not surplus to production like her Victorian counterpart, or required to be sexually attractive and nurturing to her husband, rather she needs a fit, youthful body. This positions her in relation to twentieth century man as a "playmate, a fellow consumer, and necessarily a fellow earner of income, a woman who can actively participate in his life style" (Sybylla, 1997, p.215). Older women are persuasively offered this ideal body and life style by the medical profession with the choice of HRT use. As Sybylla (1997) notes, the pathology of the older woman's menopausal body is positioned as the 'Other' to the youthful "ideal body of medicine" and the "ideal body of industry" (p.215).

What is Hormone Replacement Therapy?

HRT is a synthetically produced estrogen hormone. In our bodies, natural estrogen is a group of steroid compounds which function as the primary female sex hormones. The

three types of estrogens are estrone (E1), estradiol (E2, 17β-estradiol) and estriol (E3). They are synthesised primarily in the ovaries, testes, adrenal glands and also our fat tissues. Estrone is a precursor to estradiol; it is a breakdown or storage form of it. Estradiol is the strongest estrogen emitted by the ovaries and the most active in our tissues. Follicle stimulating hormone (FSH) and luteinizing hormone (LH) are chemical messengers from the pituitary gland that stimulate the follicles in the ovary to mature. The follicles, in turn, emit estradiol which thickens the lining of the uterus. Ovulation occurs with a surge of LH that triggers the most mature follicle to burst open and release an egg. If the egg is not fertilised, the lining of the uterus detaches and this is menstruation. It is thought that in the menopause transition there are less follicles therefore less estradiol is emitted, therefore FSH increases to try and 'rev up' the follicles. Over time, as a woman ages, follicular development is no longer needed so there is no need for estradiol to be emitted in such amounts. In the medical field, the symptoms of menopause like 'hot flashes' are thought to be caused by this 'lack of estradiol' and the remedy is to replace the estradiol that is missing with synthetically produced estrogen in order to treat the symptoms, namely HRT. The basic ingredient of HRT is pregnant mare's urine which is sourced by the pharmaceutical companies from 'pee farms' where horses are stalled continuously for years for this purpose only (Equine Advocates, 2009). HRT production is a billion dollar industry for global pharmaceutical companies with sales through public medical authorities and direct to consumer advertising. In New Zealand there were 115,000 publicly funded HRT prescriptions issued in the June 2008-2009 year (J. Arnold, Pharmac, personal communication, February, 3, 2010).

The Story of Estrogen and its Warnings

This section gives a brief historical account of HRT from its discovery in the 1940s through to its position in medical science today. This historical perspective is taken primarily from Barbara Seaman's (2003) account of HRT in her book 'The Greatest Experiment Ever Performed on Women' but also includes the major research findings since the publication of this book. Seaman was a woman's health activist who founded the National Women's Health Network which lobbied the American government to fund the WHI clinical trials. I have located this brief historical account of HRT with a focus on the "truth community" of medical science (Fleck, 1939/1979; Rose, 2007) because this provides an answer to the HRT Debate Symposium question: "…why, for

four decades, since the mid-1960s, were millions of women prescribed powerful pharmacological agents already demonstrated, three decades earlier, to be carcinogenic?" (Krieger et al, 2005, p.740).

The history of HRT is really the history of estrogen and its warnings. It begins in the early 1800s in Germany where the Merck company medicines catalogue offered women who were debilitated by menopause a plethora of products to soothe her days and nights. The choice of drugs included amylnitrate, belladonna, cannabis, and the most popular Merck Opium. Today Merck is a research-driven pharmaceutical company with revenue in the billions from its global manufacture and marketing of medicines. However, the field of endocrinology really took off with the Parisian neurologist Brown-Sequard's hypothesis that substances secreted into the bloodstream – hormones – affected organs in the body. He famously injected himself with testosterone from dogs' testes to show how his body could be re-invigorated with this 'male' hormone.

The female ovary, however, remained a medical mystery until 1922 when Edgar Allen, an American medical student, presented his thesis which detailed the cellular changes in the sex organs and monthly development of ova in a female mouse. In 1939 Allen joined forces with the biochemist Edward Doisy (who discovered vitamin K) and they extracted female follicle fluid which led to the discovery of estrogen and its effects. At this time the pharmaceutical companies learned of this estrogen research, and foreseeing its potential market revenue in body health, they competed to buy up the biochemists. Ayrest (today the multi-national pharmaceutical company Wyeth) 'purchased' James Bertram Collip, an American biochemist who had produced the first insulin for humans in 1922; he was to become the 'father of Premarin'. In Germany the biochemist Adolph Butenandt was 'purchased' by Schering, a dispensing chemist which is today a multi-national pharmaceutical corporation. Both companies supplied the source produce to these biochemists for their research, namely pregnant mare's urine. Butenandt and his colleagues developed synthetic estrogens, however from the outset Butenandt warned that hormones could be carcinogenic because the properties of some hormones in certain situations promoted cell growth. This warning of the risk of cellular growth was to be repeated over the years. In England the biochemist Edward Charles Dodds refused any pharmaceutical company 'buy-up' and headed up his own research institute.

All of these brilliant biochemists were competing in a race to bring estrogen hormone treatments to the world market.

Dodds and his colleagues became concerned about Butenandt's work because Schering had major links to a German cartel that used free labour from the Nazi concentration camps. In the mid-1950s numerous Auschwitz survivors testified that "they were fed daily doses of liquid estrogen in their rutabaga soup. The women stopped menstruating and the men lost their sex drive, just as the Nazis expected" (Seaman, 2003, p.28). They were concerned that the Nazis would use synthetic estrogen as a sterilization weapon in their warfare. Therefore, in 1938 when Schering applied for a patent on estradiol Dodds and his colleagues quickly produced the synthetic and inexpensive estrogen stilbestrol (DES) and published their research. Synthetic estrogen was now free to be manufactured and marketed by the pharmaceutical companies. To gain FDA approval for its use, they formed alliances and lobbied the FDA. At the same time cancer warnings surfaced in estrogen research. Dodds conveyed his concern to Barbara Seaman in 1960 that synthetic estrogens "were good to cure diseases…but were dangerous for long-time use in healthy people" (Seaman, 2003, p.41). Both Dodds and Allen carried out animal research that showed synthetic estrogens produced breast and cervical cancers. In 1938 two major academic journals published research confirming the benefits of estrogens for treating menopause symptoms but warned that long term use may be carcinogenic. Despite these warnings and a lack of rigorous clinical evidence, in 1941 the FDA officially approved the use of DES to treat menopause symptoms and menstrual disorders. The pharmaceutical companies then targeted their HRT products to women through the media and health experts. Women were encouraged to stay on the Wyeth's HRT, Premarin, by Dr Robert Wilson who, funded by the pharmaceutical company Wyeth, published the infamous book *Feminine Forever*. This 'holy bible' exhorted women to save themselves from the 'living decay' of menopause by taking HRT (Perz & Ussher, 2008). However, in 1971 the FDA removed the approval for DES for healthy women due to the risk of them and their daughters developing breast and genital cancers. The next major cancer warning surfaced in 1975 when the FDA acknowledged research findings that estrogen only HRT increased the rate of endometrial cancer. The medical profession then combined progestin with estrogen to reduce the risk of cancer but in 2002, the next major cancer warning was heard, when the WHI study was stopped early due to the risks to women's health.

Women's Health Initiative

The WHI research study was launched in 1991. It was funded by the US National Institute of Health and was initiated by the National Women's Health Network when they actively lobbied the FDA to decline Wyeth's labelling upgrade for Premarin to be approved as cardio-protective for 'menopausal women'. The National Women's Health Network attended the Wyeth-FDA advisory committee meeting and charged that "no drug had been approved for this purpose in men without a quality clinical trial" (Seaman, 2003, p.150). However, the FDA advisory committee voted to approve the Wyeth application based on the impressive epidemiological research presented. This research came from studies such as Stampfer et al (1991), who conducted a 10-year follow-up of 48,470 cardio-healthy postmenopausal women and concluded that: "Current estrogen use is associated with a reduction in the incidence of coronary heart disease as well as in mortality from cardiovascular disease, but it is not associated with any change in the risk of stroke" (Seaman, 2003, p.756). This link between HRT and cardio-protection was also prevalent in the media and popular press and HRT was routinely prescribed by doctors to give postmenopausal women cardio-protection. The key moment for the WHI came when the FDA staff agreed to recommend a large scale clinical trial and this became a prerequisite for Wyeth's labelling upgrade. A year later Senate approved funding for the FDA recommended clinical trial of Premarin and the WHI was underway.

The WHI consisted of a set of controlled-randomized clinical trials and an observational study that included 161,808 generally healthy postmenopausal women. The clinical trials were designed "to test the effects of postmenopausal hormone therapy, diet modification, and calcium and vitamin D supplements on heart disease, fractures, and breast and colorectal cancer" (National Heart Lung and Blood Institute, 2010). The trial had two main research groups: the estrogen-plus-progestin (E+P) HRT group of women with a uterus and an estrogen-alone (E) HRT group of women without a uterus. In both hormone therapy studies, women were randomly assigned to either the hormone medication or to placebo.

The WHI E+P study planned duration was 8.5 years but it was stopped early after 5.6 years, on 7 July 2002, because of the "increased risks of cardiovascular disease and breast cancer in women taking active study pills, compared with those on placebo

(inactive pills). The study showed that the overall risks exceeded the benefits, with women taking E+P at higher risk for heart disease, blood clots, stroke, and breast cancer, but at lower risk for fracture and colon cancer" (Women's Health Initiative Participant Website, 2008). Given the history of estrogen and its earlier warnings, these results should not have been surprising.

In the aftermath of the WHI clinical trials Wyeth's sales of Premarin declined from $2 billion in 2002 to just over $1 billion in 2006 (Lawton et al, 2003). Women all over the world stopped taking HRT; prescriptions in the United States declined by up to 66% in January to June 2003 (Roberts, 2007, p.159). A New Zealand survey of 776 women who were taking HRT at the time the WHI findings were published reported that 58% of these women stopped taking HRT (Lawton et al, 2003). International and local guidelines for HRT use were amended to account for the WHI findings (Roberts & Lethaby, 2003). There is still much controversy over the trial design and the way the risks of its findings were calculated and reported (see Prentice & Hall, 2007, for a review). One of the major controversies that is threatening to 'un-do' the risk warnings of the WHI and potentially endanger women's health is the 'window of opportunity' argument and its research findings. This argument states that HRT should be initiated within the first few years of menopause as this is the time when its cardio-protective and osteoporosis prevention effects will be achieved.

The 'window of opportunity' argument is based on the criticism that the WHI sample was limited because "…the prevalence of stroke is age-dependent and the numbers under age 60 years were small — too small to test the critical window hypothesis for stroke. An increased risk of transient ischemic attacks and strokes must currently be presumed as likely in women initiating HRT many years after menopause" (MacLennan, 2007) This argument advises therefore that: "…the results of the WHI should not have altered current guidelines regarding optimal use of HT for 3 to 5 years in symptomatic women and in women at substantially increased osteoporotic fracture risk" (Burger, 2007). Journals are now reporting that given these recent analyses of the WHI data and other randomised controlled trials and observational and animal studies, the risk-benefit ratio for women has changed and it is "mostly good news" (MacLennan, 2007). However this 'good news' is countered by the National Women's Health Network (2008a) and other researchers (Prentice & Hall, 2007) as myths promoted by

pharmaceutical funded researchers. The 'myth' that the women in the WHI trials were too old that justifies the 'window of opportunity' argument is countered by the fact that 37% of the WHI E+P sample were aged 50-59 (Prentice & Hall, 2007). Another 'myth' is that the WHI women were unhealthy from obesity, hypertension and smoking and they had pre-existing cardiovascular disease (Burger, 2006). However, in the WHI trial every woman was pre-screened for cardiovascular disease and was found to be healthy in this respect (National Women's Health Network, 2008a). The 'window of opportunity' argument for cardiovascular disease prevention is currently undergoing a clinical trial, the Early versus Late Intervention Trial with Estradiol (ELITE), which is sponsored by the US National Institute of Aging. This randomized clinical trial of 504 postmenopausal women aims to test the hypothesis that synthetic estrogen will reduce the progression of early cardiovascular disease if it is initiated soon after menopause versus later when heart tissue has lost its responsiveness to estrogen. It is estimated to complete in February 2012 (US National Institutes of Health, 2010). In the meantime, the National Women's Health Network position on this 'window of opportunity' hypothesis is that HRT does not have regulatory approval for cardio protection and any practitioner who prescribes it to younger women is making clinically irresponsible decisions that lack scientific evidence and this may endanger their patient's health (National Women's Health Network, 2008b).

However, the 'window of opportunity' argument is being used to justify HRT use despite the lack of clinical trial evidence to date. Wren (2009), Director and Gynecologist of the Sydney Menopause Clinic, provides an example of this prescriptive approach in his article in The Medical Journal of Australia (2009) entitled "The benefits of oestrogen following menopause: why hormone replacement therapy should be offered to postmenopausal women" (p.321). Wren (2009) cites animal studies with estrogen as evidence for the 'window of opportunity' argument. For example, in macaque monkeys the cardio-protective effect of estrogen is lost if therapy is delayed by more than 2 years after menopause. Also, dementia is reduced in "castrated animals fed an oestrogenic therapy regime" (p.323) *and* clinical studies show "women who begin [HRT] at or soon after menopause have a reduced risk of dementia" (p.323). Wren states that "extrapolating these results to humans suggest that HRT should be initiated within 6 years of menopause (the "window of opportunity")" (p.323). It is worth noting here that the "clinical studies" that Wren cites is a reference to Bluming

(2004) whose article provides only a critique of the WHI findings. Also, Bluming, Professor of Clinical Medicine at the University of Southern California, refers to science as a "competitive and aggressive activity, a contest of man against man" and the physician who advises on HRT "is much like a sports commentator watching a fast paced football game. To accurately communicate real-time results of this game to a patient, the physician must understand the rules of play so that the patient betting her health or her life on the outcome has the best chance of winning…" (p.30). Women obviously don't get to 'play the game' or even watch it for themselves.

Summary

Menopause as a biological phenomenon has two reliable and overt physiological signs: a change in the length of the menstrual cycle and an increase in the levels of FSH. Many bodily signs or 'symptoms' of menopause have been reported but only 'hot flashes' and 'vaginal dryness' are consistently associated with this time of life. The historical, cultural and socio-economic context of menopause as viewed through a Foucauldian lens shows that women's experiences of menopause are shaped by the power relations inherent in their worlds as seen in the biomedical social constructions of their bodies and their sexual relationships. The fragile and medically managed body of the 'Victorian mother' had no further use once menopause was reached whereas the body of the mid-twentieth century, expertly managed, mother and wife needed to remain nurturing and sexually attractive, hence the medically managed HRT use for menopause. The 'late-twentieth century fit playmate' can choose to not have menopause by using HRT and she is urged to negotiate the health risks to her body with the medical profession. This option to remain youthful belies the framing of her menopause experience as the fixing of an aged, faulty body in an era of individual choice and freedom. The 'elixir of youth' that the medical profession offers in HRT is a synthetic estrogen produced from female horses' urine. It is prescribed to replace the estrogen deficiency at menopause to relieve menopause 'symptoms'. The story of HRT is really the story of synthetic estrogen and its warnings. These warnings have been sounded for years and have recently been repeated in the findings of the WHI, namely that HRT increases the risk of breast cancer and heart disease. However these findings are currently being challenged and HRT for younger women in menopause is being recommended without adequate clinical trial evidence. Thus, HRT use remains controversial and it seems women's health continues to be endangered today.

Chapter 2: Feminist Resistances to a Biomedical Menopause

In this chapter I discuss resistances to the biomedical construction of menopause as a disease, the woman as a 'patient' and HRT as the medical treatment. Firstly, these resistances are shown through the way in which feminism has progressively engaged with the biomedical model in its second-wave, third wave and post- feminist critiques. Secondly, I outline how these critiques have led to a critical engagement with socio-cultural media representations of menopause and with the medical profession's 'scientific' representation of menopause. Finally, I outline the challenge to the biomedical model that Emily Martin (1986) presented in her cultural analysis of the representation of menopause in medical textbooks.

Second-wave Feminist Critiques

For a life transition like menopause the feminist engagement began when second-wave feminists identified the impact of a male dominated society on women's experiences of their bodies in mid-life. This feminist position strongly contested the chauvinist influence of a male-dominated medical science on women's interpretation of their bodily experiences like menopause. This resistance showed the way in which women's essential and universal biology was medicalised and therefore controlled by the medical profession. Simone de Beauvoir (1949/1993) presented menopause as a transition in a woman's life when she begins to lose the physical form and the fertility which justifies her existence and her future. A woman's social worth, in fact her very survival was based on her sexual value: "to hold her husband and to assure herself of his protection, and to keep most of her jobs, it is necessary for her to be attractive, to please; she is allowed no hold on the world save through the mediation of some man" (p.606). However, as a woman begins to experience this 'dangerous age' she "is appalled at the narrow limitations life has imposed on her" (p.607). These 'limitations' were taken up by other second-wave feminists who militantly challenged the subordination of women by a dominant male society. This resistance is seen in the critiques of feminists like Coney (1994) who challenged the medical profession's definition of menopause:

> There is no area that demonstrates the entrenched sexism of medicine more sharply than that of menopause. The defining of a normal bodily state as diseased, the suggestion that women become worthless with the loss of childbearing capacity, the overlay of chauvinistic attitudes about the importance

> of sexual attractiveness – even the language of menopause is inherently misogynous. (p.54)

These criticisms were also extended to the use of HRT by the medical profession. Dr Robert Wilson, who founded a private trust to promote estrogen for menopausal women and treated thousands of women with it, came under sustained attack from feminists. His infamous book 'Feminine Forever' (1968) which promoted HRT for women was a key social marketing ploy for the pharmaceutical industry. Coney (1994, p.59) describes Wilson as the misogynous 'Hugh Hefner of menopause' who was not just driven by profits but had a vision of himself as a 'medical saint' who would save women from their miserable decay and degeneration. These criticisms were further extended to the FDA which approved HRT use for women in menopause. Birnbaum (1990), a spokeswoman for the National Women's Health Network, stated that "adding the concept of "disease" to menopause has compounded an ageist and misogynous attitude of mid-life and older women as unattractive, asexual, dependent and neurotic" (p.250). This 'disease' of women in menopause was also criticised based on the lack of scientific medical evidence. Feminists like Greer (1991) addressed the causal pathways of estrogen deficiency and menopause 'symptoms' and the lack of substantiated empirical evidence and therefore the unfounded bias of women in menopause being ill:

> Unfortunately we do not know enough about the whole woman, whose anatomy is generally studied as if it were simply a man's body with a reproductive system installed in it, to have any clear idea of how the endocrine balance of well women differs from that of ill women, or, indeed, what degree of variability there is in endocrine function in both well women and sick women. (p.165)

Third-wave Feminist Critiques

This second-wave feminist resistance to the biomedical model focused on the collective rights of women to assert their own sexuality and also their rights to not have their bodies defined or controlled by a male dominated medical profession. Third-wave feminism moves beyond this essentialist and universalistic position with its biological/social distinction and 'women as victim' ethos towards a recognition of the diversity, contradictions and individuality of women. In terms of menopause this approach can be seen in cross-cultural research which shows that menopause cannot be defined universally as a 'disease experience' that all women suffer because of their

female physiology (Gannon, 1999). This research has shown that Japanese women associate menopause with genetics and diet (Lock, 1998); African American women report more positive perceptions of menopause than Caucasian women (Brown et al, 2005); Chinese women's menopause symptom reporting is strongly associated with their view of ageing (Shea, 2006); and women in Thailand and Japan viewed HRT as beneficial to their social status because it allows them to continue in the workforce as they age (Richter, 1997). As well as recognising the diversity of women's experience of menopause, third-wave feminism moves from "woman as "victim" of patriarchy, male sexuality etc. towards a more positive image of woman seen to have the capability to choose from a range of lifestyles" (Sim & Van Loon, 2001, p.156). Women are also encouraged to realise their own agency by actively and critically engaging with the influence of the biomedical model in their lives. In terms of menopause and HRT, this 'individual empowerment' is evident with the medical profession advising women to use their autonomy, their individual risk-based assessments, in consultation with their doctors in making their choice about HRT. For example, the revised guidelines from the NZ Guidelines Group for HRT post-WHI, suggest how the findings may be used for "individual decision-making" (Roberts & Lethaby, 2003). However, post-feminism is critical of this third-wave feminist individual discourse in women's health because it can serve to alleviate the responsibility of social, economic and political institutions to provide knowledge of, and access to, healthcare for all women.

Post-feminist Critiques

One of the ways in which post-feminism has moved towards a critical and activist engagement with the biomedical model is in post-structuralist critiques of biological essentialism. As Roberts (2002) asserts: "Cultural, historical and psychical forces are viewed as potent in the making of sexed bodies, while biology is seen to function only as a limit, potentiality or raw material which can never be accessed or known outside of culture" (p.7). This social constructionist approach views life transitions like menopause as constructed within socio-historical times and places and influenced by inherent political power relations embedded in discourses of gender, ageing and reproduction, as discussed in Chapter 1. These discursive constructions in our socio-cultural worlds can be uncovered to show how they influence women's perceptions of our bodies and our health. This in turn problematises and disrupts the dominance of the biomedical model and opens up new interpretations of women's menopause experience. However, this

focus on the discursive framing of menopause and HRT can be criticised for what could be termed a 'discursive essentialism', a privileging of the 'social' over biology. As Lock (1998) states: "…recent anthropological research suggests…..it is more appropriate to think of biology and culture as being a continuous feedback relationship of ongoing exchange in which *both* are subject to variation" (p.410). Lock bases this assertion on cross-cultural research findings that menopause symptoms are reported differently across cultures *and* there is considerable cultural diversity in the physical incidence of the diseases (osteoporosis, breast cancer and heart disease) associated with menopause.

Roberts (2007) extends this premise that there is a dynamic relationship between biology and culture to argue that menopause is an embodiment that is culturally shared. The many feminist approaches to embodiment theorise it as a complex interplay between biology and culture whereby bodies materialise within social interactions that are situated within knowledge and power structures in particular times and places (Foucault, 1969/1989; Butler, 1990; Grosz 2005). As noted, Lock (1998) found that in menopause women's physical symptoms are culturally diverse. For example, Japanese women identify shoulder stiffness whereas American women report hot flashes. According to Roberts, this suggests that women share culturally located forms of embodiment in menopause which in turn disrupts the idea of a boundary between the body and culture. To further engage such views on embodiment with the materiality of the body, Roberts draws on actor network theory to invoke the body as an *active* object as well as a cultural one in the world of science. Hormones therefore are viewed not as essential bodily messengers that *produce* sexual difference, but as *active* agents, material and semiotic entities that message across the bio-social boundary. The implication of this for HRT is that it can be seen as an active (material and semiotic) messenger that moves into culturally situated bodies in highly cultural and technical forms within intricate socio-economic and political discourses. Within these discourses HRT as a messenger cannot be removed from the social as it is involved in complex interactions between entities in the body, biochemists, pharmaceutical companies, general practitioners and their patients, pregnant mares producing urine, scientists and their medical journals. Thus, although the biomedical model seeks to maintain the 'biological facts' of hormones like HRT, it is not possible to separate the bio-social, and given the discursive complexity of the bio-social, post-feminists are now advocating a

collective and critical position towards menopause and HRT. This position begins with a critical stance towards the representations of menopause in our society.

Representations of Menopause

As discussed, women's interpretations of the bodily changes of menopause and their use of HRT are located within the discursive complexity of the bio-social. A way to examine these interpretations of menopause within the bio-social is to critically focus on media representations of health and illness. Lyons (2000) argues for a critical analysis of our socially and culturally produced representations of health and illness because they generate and reproduce meanings that can influence our attitudes towards others with diseases and our own interpretations of bodily sensations, hence our subjectivities. In terms of menopause, this critical analysis of media representations can also uncover the dominant political power relations underpinning the discursive constructions that influence women's interpretations and subjectivities for their health in this life transition.

Gannon and Stevens (1998) examined socio-cultural attitudes to menopause as represented in popular print over a 15 year period and concluded that although the frequency of articles increased, almost all of them "focused on menopause as a negative experience or disease and in need of medical treatment" (p.1). Although women resist these negativities by viewing menopause as a time for increased personal freedom, self-awareness and self-worth (Perz & Ussher, 2008), they are impacted by this negative social attitude. They approach this life transition by having negative "apprehensions" about self-care (Bertero, 2003) and "getting through the event" and "soldiering on" (Shore, 1999) and only avoiding it when life stress makes them too sick to worry about it (Winterich & Umberson, 1999).

The research that focuses on the media visibility of mid-life and 'menopausal' women further shows this impact of negative social attitudes. Kaufert and Lock (1997) examined the visual images of menopausal women in pharmaceutical literature and the mass media. They found that in the 1970s this woman was depressed and unwell and in the 1990s she glows with fitness and embraces health as a virtue. This new virtue for a healthy lifestyle is also embedded in the decisions that women make about taking HRT (Stephens & Breheny, 2008). It is also present in the medical literature where there are

now two models of the menopausal women – one on the light side who takes her hormones and one on the dark side who does not (Kaufert & Lock, 1997).

In relation to the visible age-related changes in body appearance Whittaker (1998) critically evaluated the visual representations of menopausal women and HRT in medical and pharmaceutical advertisements and demonstrates they portray hegemonic male definitions of the 'feminine' rather than women's own lived experience of menopause. Whittaker (1998) argues that throughout history visual portrayals of women have privileged the 'male spectator' whereby the female is represented as frail and capricious yet naturally, sexually tempting. An example of this visual depiction for menopause is seen in an advertisement for HRT that shows a 'before' Picasso picture of a disjointed, ugly older woman and an 'after' Renoir picture of a beautiful and tempting burlesque woman. The impact of this imagery for women in menopause is that since it is constructed from a male idealised 'feminine' they are separated from their own bodily experiences of it. The dominance of this male idealised femininity in the media industry is underpinned by the creative force and ultimate decision makers being male and when the advertising representatives of pharmaceutical companies tried to deviate from the traditional forms of femininity the doctors did not prescribe the treatments advertised (Whittaker, 1998). However, it must be noted that men too are victims of a cultural consumerism that commodifies their bodies with an idealised masculinity that is youthful, strong and sexually capable. Our social attitudes towards women and men as represented in the media are also influenced by systemic capitalist socio-economic factors.

As well as the visibility of women in media representations, their invisibility also reflects social attitudes. Casper and Moore (2009) have explored how particular places, spaces, policies, and practices in today's society "exhibit and celebrate some bodies while erasing and denying others" (p.3). They have demonstrated the social power dynamics that marginalise and suppress victims of war, natural disasters and poverty so that they "are socially present but are culturally missing in action" (p.10). It can be argued that in society mid-life women are also "missing in action" due to a society which is culturally obsessed with youth and beauty. The scarcity of alternative visibilities for them is evidenced by their invisibility in socio-cultural mediated locations such as advertising. Rosewarne (2006) analysed 177 outdoor advertisements

and found that less than four percent of women were shown as older than 30 years and when older women were portrayed they were cast in negative roles. The invisibility of these women infers not only their lack of attractiveness for media advertising but their social unattractiveness which signals their omission from society (Rosewarne, 2006). An impact of this invisibility for women in menopause is that they experience 'public', visible age-related alterations in the appearance of their bodies and 'private', invisible age-related physiological changes in their bodies that they ascribe to the menopause (Ballard et al, 2009). In relation to the invisible biological changes of menopause, younger women who experienced early menopause were diagnosed by their doctors with psychiatric rather than menopause symptoms because of their age so they felt they were going mad and it was all in their heads (Broughton, 2008). As we have seen, women and their menopause transition have been invisible or when visibly represented, this has been in a predominantly negative way in their socio-cultural worlds. This discursive constructions of menopause that underpin these media representations are worthy of attention.

The critical social science discursive analyses of menopause have focused on many texts such as pharmaceutical pamphlets, medical journals and feminist, popular media, science and self-help texts. These analyses have shown that there is a multiplicity of menopause constructions available for women's interpretations of their menopause experience. Coupland and Williams (2002) found that menopause and HRT use is situated within conflicting 'biomedical', 'alternative therapy' and 'emancipatory feminist' discourses, each with different health and lifespan ideologies that further different sets of economic and/or political agendas. For example, the 'alternative therapy' discourse exhorts women in menopause to be agentic and active and create their own 'natural' and healthy lifestyles whereas the 'emancipatory feminist' discourse situates menopause as a life transition that brings a positive personal freedom. Both of these discourses reject the biomedical medicalisation of menopause but the 'alternative therapy' discourse is just as ageist as the biomedical discourse, because it positions women to defend themselves against their ageing bodies. Lyons and Griffin (2003), in their analysis of four medical, alternative and feminist self-health books, found that the 'natural' discourse of 'menopause as a natural bodily transition' was counter-posed with the biomedical discourse of 'menopause as illness' and this polarisation leads to the 'menopause as confusing' discourse whereby menopause and women's bodies are

framed as complex and confusing. Further, although medical expertise still dominates women's bodies and menopause, the "responsibility for the 'management' of menopause as a chronic condition lay solely with individual women" (p.1629). Hvas and Gannik (2008) analysed popular Danish medical booklets, articles, magazines and books and found that the biomedical model is challenged by multiple discourses, including 'eternal youth' and 'consumer'. The 'eternal youth' discourse idealises youthfulness in society "while ageing and old people are belittled and made invisible" (p.164). In this discourse menopause is another negative sign of ageing and HRT is one of the many medical life elixirs that counters bodily decay. In this sense this discourse is aligned with the biomedical model but its objective "is not just avoidance of weakening and diseases, but conservation of sexuality and physical attractiveness" (p.165). In the 'consumer' discourse, patients are consumers of health services and medical professionals are service providers. Women in menopause are therefore health consumers with the right to quality information and health options and free to make their own informed health choices. However, the biomedical discourse remains influential here as the 'management of menopause' is guided by the doctor's expertise with women being encouraged to seek their advice. Despite there being a multiplicity of menopause constructions available for women's interpretations of their menopause experience, the dominance of the biomedical model continues to permeate these discourses. This dominance is achieved in part by the power and prestige that the medical profession obtains from its location in our enlightened, rational and objective scientific world. Yet, as I argue next, this is the location from which this discourse can be challenged.

The medical profession certifies its knowledge as scientific 'truth' in part through its published academic literature. When this literature portrays menopause as disease and deficiency, this view of women may therefore become the sanctioned wisdom for women's lives (Rotosky & Travis, 1996). However, scientific practice is not an objective and value-free enterprise because scientists themselves are embedded in an historical, cultural, socio-economic and political world (Kuhn, 1962). Therefore science as a discursive entity can be analysed for its strategies to construct its objects and to investigate them (Komesaroff et al, 1997). The way that medicine represents its 'object of study' - our bodies, illness and disease - can therefore be analysed in terms of the cultural influences underpinning its knowledge 'truths'. In terms of menopause, Emily

Martin (1987) has contested the biomedical model of menopause in this way by analysing female corporeality in medical textbooks from the eighteenth century through to the late twentieth century. She traces the changes in the metaphors representing women's bodies and shows how they reflect the socio-cultural contexts of the times. Tomlinson (1999) has identified Martin's analysis of metaphors as a rhetorical strategy of intensification. This strategy challenges inherent presumptions in scientific academia by questioning the taken-for-granted metaphors used in their seemingly neutral theorising and debates.

Martin's Marxist thesis is that scientific medicine's representation of women's bodies reflects the alienation and fragmentation in the modern consciousness of capitalist society. Early industrial capitalism alienated men and women from their life-worlds – their families, their materiality and their communities. Men were located into the public sphere of work where they were also separated from their own labour, and middle-class women were located into the private and isolating sphere of the nuclear home where they were separated from their production – their children - by experts in childrearing. At the personal level there is fragmentation of the embodied self by medical science which treats the person as a machine whose body parts could be isolated, removed, transplanted or repaired. Martin (1987) also asserts that as well as fragmenting women into body parts, scientific medicine subjects them to a hegemonic male-dominated "model of human nature and social reality" (p.21). The aim of her analysis was to examine women's responses to this dominant cultural ideology for their bodily experiences, although she began with a cultural analysis of the scientific representations of women's bodies.

In terms of menopause, Martin (1987, p.42) asked "what is the language in which menopause is described?" She found that in the twentieth century the dominant metaphor for the body was a hierarchical information processing system and for female reproduction this is reflected in the "female-brain-hormone-ovary system" which is controlled by a 'central processor', the hypothalamus. Martin (1987) quotes an example of this metaphor from medical literature:

> From the first menstrual cycle to menopause, the hypothalamus acts as the conductor of the orchestra. Once its baton signals the downbeat to the pituitary,

> the hypothalamus-pituitary-ovarian axis is united in purpose and begins to play its symphonic message, preparing a woman's body for conception and child-bearing. (p.41)

The implication of this metaphor is that menopause is then seen as a "breakdown of a system of authority". This breakdown is caused by "ovarian failure" which is described as the "decreasing ability of the aging ovaries to respond to pituitary gonadotropins" (p.42). At every point in this system, functions "fail" and "falter" and the members of the system decline: "breasts and genital organs atrophy", "wither", and become "senile" (p.42). This imagery of the body as a hierarchically organised system leaves women in menopause associated with breakdown, decay and atrophy. There is another failure for women in the factory machine metaphor, "…the system is seen as organized for a single pre-eminent purpose" "transport" of the egg along its journey from the ovary to the uterus and preparation of an appropriate place for the egg to grow if it is fertilised" (p.44). Menstruation is therefore failed production as a plethora of negative descriptions portray: "The fall in blood progesterone and estrogen, which results from regression of the corpus luteum, deprives the highly developed endometrial lining of its hormonal support; the immediate result is profound constriction…" (p.49). In contrast male reproduction is described as: "Perhaps the most *amazing* characteristic of spermatogenesis is its *sheer magnitude*: the normal human male may manufacture several hundred million sperm per day (p.48, emphasis added)". Martin (1987) states that for menopause this failure in production refers to a failure of the ovaries to produce estrogen: "Estrogens are produced in sub critical quantities for a short time after the menopause, but over a few years, as the final remaining follicles become atretic, the production of estrogens by the ovaries falls almost to zero" (p.51). This failure to produce estrogen is then linked to disease and pathological states where "a woman must readjust her life from one that has been physiologically stimulated by estrogen and progesterone production to one devoid of these hormones" (p.51). Martin's analysis of the way in which medical science has represented women's bodies and menopause demonstrates the culturally embedded metaphor of the 'body as machine', and menopause as a failure in the breakdown of hierarchical control of the female brain-hormone-ovary-system as well as a failure to produce estrogen which results in disease and pathology. The implications of these 'medical facts' for women is that they are

subjected to negative socio-cultural attitudes within a discipline that purports to be 'objective', 'scientific' and 'factual'.

Since Martin's analysis in 1987, research examining medical textbooks has been limited. Vainionpaa and Topo (2005) studied representations of male menopause in two Finnish introductory medical textbooks (gynecology 2001 and urology 2002) and a physician's handbook. They found that the male menopause is constructed by a 'gerontological' discourse where it is presented as a natural ageing transition in contrast to an 'andological' discourse, which views it as a decline in gonad functioning with associated risks for cardio-vascular disease and osteoporosis. From a socio-cultural perspective, it is significant that these researchers state that the existence of the male menopause "has been widely debated" and there is dispute about whether "it really exists" and whether "it only applies to women" (p.842). Lawrence and Bendixen (1992) studied 31 United States medical anatomy textbooks between 1890 and 1989 and found that in illustrations, vocabulary and syntax these 'scientific' texts represented male anatomy as the norm for the human body and it is this "overall female-depends-on-male directionality that discloses what it means for these ostensibly objective, scientific texts to be culturally gendered" (p.933). Anatomy is a powerful and authoritative science that provides scientific medicine with the structure of its subject – the human body – so to construct the 'human' body as male centric sets the scene in medicine for women's bodies to be the 'Other'. More recent research suggests little has changed. Dijkstra, Verdonk and Lagro-Janssen (2008) examined eleven medical textbooks used by two medical schools in the Netherlands in the academic years 2004-05 and 2005-06. They identified bias in these textbooks in that gender-specific information was scarce or absent for cardio-vascular disease, alcohol abuse and pharmacology. and there was a general inference that women's health problems were aberrations of the male norm.

Summary

In summary, this chapter has provided an overview of the different forms of feminist resistances to the biomedical model of menopause. Second-wave feminists strongly contested the chauvinist influence of a male-dominated medical science on women's interpretation of their bodily experiences like menopause whereby they were seen as decayed and sexually unattractive. Third-wave feminism asserted the cultural diversity, contradictions and individuality of women's interpretations of menopause to challenge

the universalistic view that it is a disease needing treatment with HRT. Post-feminism has moved towards a post-structuralist critique of the biomedical model by refuting the essentialist biological 'facts' of menopause. This feminist position engages with menopause as a cultural embodiment that is theorised as a fluidity between the bio-social that is located within a complex set of discourses. This means that HRT is also embedded in the bio-social as a material and semiotic messenger; it cannot be separated out as a scientific biological 'fact' of menopause and must be critiqued from its location within the discursive realm. This focus on the bio-social leads us to examine the media representations of menopause in our society.

Menopause has been predominantly represented negatively as a disease that needs treatment. This representation reflects the negative social attitudes toward women's ageing bodies in society as evidenced in their media visibility as 'old crones' or their invisibility in consumer advertisements that commodify the body. For menopause this cultural obsession with youth and beauty translates into it being represented as a threat to the sexually attractive 'feminine' and HRT is presented as the way to restore and maintain this socially idealised 'femininity'. This culturally projected imagery of menopause conflicts with women's interpretations of their bodily experiences so that menopause becomes a site of contradictions for them. These contradictions are evidenced in the multiplicity of discursive constructions available for women's interpretations of their menopause experience. Further, most of these discourses which include the 'emancipatory feminist', 'naturalist', 'consumerism' and 'eternal youth' discourses, are permeated with the influence of the biomedical model. Despite this dominance, it is argued that the power and prestige of the medical profession can be challenged because scientific 'truths' are culturally embedded. Emily Martin (1986) presented this challenge in her cultural analysis of the representation of menopause in medical textbooks.

Research Aims

This research will firstly replicate Emily Martin's (1987) analysis by focusing on how menopause is represented in the major medical textbooks used by New Zealand medical students in 2009. Secondly it will examine whether these representations have changed since Martin's analysis showed us the 'body as machine' over twenty years ago. Thirdly, the textbooks will be examined to see whether and how HRT is represented,

and particularly whether this representation reflects the recent large scale HRT clinical trials. How HRT is linked to representations of menopause will also be explored.

Chapter 3: Methodology

This chapter describes the research process. Firstly I identify the epistemology that informs this analysis of menopause and HRT. Secondly, I outline the way in which I obtained the full sample of medical textbooks and then identified the key sample. Thirdly I focus on ethical considerations that I encountered while doing this research and discuss my personal interest in this area. Fourthly I describe the analytical approach and outline the procedure I undertook to analyse the key sample of medical textbooks.

Research Approach

This research approaches the analysis of menopause and HRT from the position of social constructionism. This epistemological position has a critical stance towards taken-for-granted knowledge - it assumes that our "objectification of our social world can lead us to assume that the way things are is the way things should naturally be" (Moghaddam, 2005, p.323). Since 'the way things are' for menopause and HRT is dominated by the powerful biomedical model a critical stance is required to explore whether this is "the way things should naturally be."

The way in which social constructionism guides this critical process is seen in its basic assumptions (Burr, 1995). It assumes that the world is not presented objectively to an observer, but is known through human experience which is largely influenced by language. The categories in language emerge from social interaction within a group of people at a particular time and in a particular place. This means that categories of understanding are situational. This knowledge is sustained by social process such that reality is determined by the conventions of communication in force at that time. Reality is therefore socially constructed by interconnected patterns of communication behaviour. This research critically focuses on the language of the medical science community and its implications for the way in which it influences the reality of women's interpretations of menopause in their lives.

Moghaddam (2005) has outlined some of the main implications for social constructionism for theory and methods, such as the notions that language is not a transparent lens, it is constructive, it shapes our thoughts and perceptions and the focus

on meaning making through language must address relativism and dominant ideologies through a focus on power relationships. The present research focuses on the power relations involved in the ideological dominance of the biomedical model for menopause and HRT.

One of the most important criticisms of social constructionism is that it leads us to a relativism where 'anything goes', leading to questions regarding how our knowledge can have a secure foundation and how can we defend our 'truths'? This concern is seen in Feyerabend's (1993) claim that there are no objective criteria to rationally justify our theories, rather our theoretical truths depend on our own world views. Gergen (1999) answers this criticism by asserting that we must firstly abandon the true/false binary and view truth as emerging from our communicative interactions in the public sphere. Within this local realm the value of ideological critique remains paramount: "to engage in critique is to defend a mode of life…it is not left in the position of claiming the only 'truly true'" (p.38). This reply asserts that since there is not one single truth, our choices have to be based on values and we have to take responsibility for the statements we make and the actions we take. As stated, this research seeks to disrupt the taken-for-granted knowledge of menopause and HRT use, and this is required because the 'truly true' ideology of the biomedical view of menopause and HRT may in fact be a dangerous truth to live with. This danger is evidenced by the controversy over the use of HRT for menopause symptoms. The HRT Debate Symposium asked "why decades of repeated warnings about dangers of manipulating and prescribing hormones to 'treat' menopause symptoms were ignored and not translated into health policies" (Krieger et al, 2005, p.741).

This epistemological position also addresses the outstanding lesson identified by the HRT Debate Symposium which was "…the need to frame contemporary research in historical, social, economic, and political context, so as to illuminate the oft concealed technical, administrative, economic and political decisions and values that shape scientific inquiry and its impact on population health" (Krieger et al, 2005, p.741). As noted, given that our objectification of our social world can lead us to believe it is naturally that way, it is important to address the way that dominant ideologies may influence our interpretations since this dominance may marginalise, silence or harm people within their social world.

Theoretical Framework

The theoretical lens used in this research is derived from three conceptual bases. Firstly, Foucault's (1969/1989) archaeology of knowledge and genealogy of knowledge/power, secondly Fleck's (1939/1979) treatise on the genesis of a scientific fact, and thirdly Rose's (2007) discursive analysis of the biopolitics of medical science. Foucault's project is to critique our contemporary claims to knowledge and he initiates this by disrupting our historical continuities, our notions of tradition, causation, evolution and our collective conscious. He proposes that we suspend these pre-existing forms of continuity to show how they were formed and legitimised as the natural order of life. In suspending our belief in the continuity of unities in space and time, Foucault frees them up to be viewed as discursive formations which can then be analysed. A discursive formation "divides up the general plane of things said at the specific level of statements" and there are four directions by which it can be analysed: (1) the formation of objects; (2) the formation of the subjective positions; (3) the formation of concepts; and (4) the formation of strategic choices (Foucault, 1969/1989, p.130). However, for Foucault discourses are more than just ways of thinking and producing meaning, rather they constitute our subjectivities, our ways of being in the world. This move towards a more dynamic account of our 'experience of ourselves' retains the archaeology of knowledge where a given system of thought is uncovered but now describes the way it enunciates our subjectivities through mechanisms of power. This was the theoretical lens that initially informed the analytic approach of this research.

My analysis required a way of negotiating the scientific medicine discourse of medical textbooks, a way of understanding its discursive construction and a way of questioning the authority of its statements. To do this, I used the vital biopolitics approach of Nikolas Rose (2007). This approach focuses on our 'vitality', our 'form of life', not in the terms of a traditional critical analysis of the politics of health as illness and wellness, but in terms of "a politics of "life itself"" (p.3). This vital biopolitics derives from techno-medical innovation and is concerned with our emerging "capacities to control, manage, engineer, reshape, and modulate the very vital capacities of human beings as living creatures" (p.3). This approach was useful for two reasons. Firstly, it is a contemporary genealogy that captures the present continuities and futures of our subjectivities in the rapidly changing world of medical science. Roses's aim in doing this is that it may "fortify our abilities, in

part through thought itself, to intervene in that present, and so to shape something of the future that we might inhabit" (Rose, 2007, p.5). This approach helped me to locate menopause and HRT within a contemporary genealogy of the biomedical discourse and therefore to gain a focus of their possible futures. This is important given that research is now showing that HRT has come "full circle" (Roberts, 2007) and new generation HRT treatments are being developed (Komm, 2008).

Secondly, Rose makes use of Ludwig Fleck's 'style of thought' (1939/1979) - a theory of how medical science develops its 'facts' through time and culture. Ludwik Fleck's treatise on the "Genesis and Development of a Scientific Fact" was published in 1939 and translated into English in 1979 at the request of Thomas Kuhn. Fleck (1939/1979) views a scientific 'fact' as "*a thought-stylized conceptual relation which can be investigated from the point of view of history and from that of psychology…*" (p.83, italics in original). His study of the genesis of syphilis from its astrological 'planetary alignment' and religious 'sinful lust' causations through to its definition as a bacterial disease showed that medical knowledge was subject to historical social and cultural influences and this knowledge was developed within communities of research scientists – 'thought collectives'. Fleck (1939/1979) defines a 'thought collective' as "*a community of persons mutually exchanging ideas or maintaining intellectual interaction…it also provides the special "carrier" for the historical development of any field of thought, as well as for the given stock of knowledge and level of culture*" (p.39, italics in original). Rose uses Fleck's 'style of thought' to show how over the last fifty years psychiatry has "gradually mapped out what it considered to be the neuronal and neurochemical bases of human mental life" (p.188). This is seen in a shift from theories to experiments, the development of drugs and imaging equipment and the development of a truth community with specialist researchers funded by public and private bodies. Rose aims to show how within this truth community "the new truths of ourselves arise, not from philosophy, it seems, but from research" (p.190). I used this discursive approach in my analysis of the medical textbooks to focus on the truth community of medical specialists and the discursive entities inherent in their menopause and HRT research: bodies (female), chemicals (estrogen), body functions (female reproduction), experimental model systems (women's bodies), investigative techniques (biochemical assays, drug trials) diagnoses (menopause), human subjects (women) and truth technologies (gold standard drug trials, FDA approvals). This gave me a way of seeing how the medical 'facts' were constructed.

Medical Textbooks

To identify relevant and appropriate medical textbooks, all of the course textbook reading lists for second year New Zealand medical students were collected from the two medical training schools for doctors in New Zealand, namely the Otago Medical School in Dunedin and the Auckland Medical School. Both of these institutions were contacted and supplied their 2009 compulsory and recommended textbook reading lists for their second and third year undergraduate medical students. The first year textbooks were not included because all medical students undertake a general health science course in their first year and do not begin their medical tuition until their second year. The two textbook lists were combined and duplicates eliminated. There were 48 textbooks in this combined list; all of them were then obtained from the Massey University Library (extramural loan), the Otago Medical School Library in Wellington (on site photocopying) or the Dunedin Medical School and Auckland Medical School libraries (inter-loan). Once obtained, all of the 48 textbooks were sighted and perused for references to 'menopause' (including pre-menopause, peri-menopause and post-menopause) and 'hormone replacement therapy' (including HRT, hormone therapy, estrogen therapy and exogenous estrogen). Twenty-eight textbooks had no references to these topic areas so were discarded from the sample. These were textbooks on: Anatomy, Medical Genetics, Biochemistry, Communication Skills, Ethics, Medical Microbiology, Embrology, Neural Science, Haematology, and Immunology. There was also one Psychology textbook that was discarded from the sample because it represented menopause and HRT from a critical health psychology framework whereas the focus of this research is on the biomedical representation of menopause and HRT. The remaining 20 textbooks did reference menopause and/or HRT and were included in the preliminary sample.

Preliminary Sample

The preliminary sample of 20 textbooks comprised approximately 380 pages of text related to menopause/HRT. These were photocopied. Of these pages, 12 textbooks (approximately 30 pages of text) had one or less sentence, paragraph or table entry. This was not considered to be sufficient coverage of the topic areas for analysis so these textbooks were discarded from the sample. They were textbooks on: Physiology (Lecture Notes and Respiratory Physiology), Clinical Chemistry, Histology, Pharmacology (a summary version) and Clinical Skills. The remaining 8 textbooks in this preliminary sample then became the key textbook sample.

Key Sample

The key sample of 8 textbooks comprised approximately 60 pages of text with one or more paragraphs that referenced menopause and/or HRT. However, a further 350 pages of text were examined as not all the references to the topic areas were listed in the textbook indeces. This sample included two physiology textbooks, four pathology textbooks and two pharmacology textbooks, as shown in Table 1. Martin also had Guyton and Hall in her research, the 1986 edition.

Table 1: Key Sample of Medical Textbooks

Reference	Textbook
Text 1	Koeppen, B.M., & Stanton, B.A. (Eds.). (2008). *Berne & Levy physiology* (6th ed.). Philadelphia: Mosby Elsevier.
Text 2	Guyton, A.C., & Hall, J.E. (2006). *Textbook of medical physiology* (11th ed.). Philadelphia: Elsevier Saunders.
Text 3	Kumar, V., Abbas, A.K., Fausto, N., & Mitchell, R.N. (2007). *Robbins basic pathology* (8th ed.). Philadelphia: Saunders Elsevier.
Text 4	Underwood, J.C.E. (Ed.) (2004). *General and systematic pathology* (4th ed.). Philadelphia: Churchill Livingstone.
Text 5	Rubin, R., & Strayer, D.S. (Eds.) (2008). *Rubin's pathology: Clinicopathologic foundations of medicine* (5th ed.). Philadelphia: Lippincott Williams & Wilkins.
Text 6	Kumar, V., Abbas, A.K., & Fausto, N. (2005). *Robbins and Cotran pathologic basis of disease* (7th ed.). Philadelphia: Saunders Elsevier.
Text 7	Katzung, B.G. (2007). *Basic & clinical pharmacology* (10th ed.). USA: McGraw-Hill.
Text 8	Page, C.P., Curtis, M.J., Walker, M.J.A., & Hoffman, B.B. (2006). *Integrated pharmacology* (3rd ed.).. Philadelphia: Mosby Elsevier.

Ethical Considerations

The ethical considerations for this research related not to the data, as the medical textbooks are a public media and free to be cited, but to my responsibility as a researcher to maintain a balanced approach to the topic. In undertaking this research it became apparent that this topic is complex, controversial, emotive and very political with many competing interests

vying for their views to be accepted. This meant that for every research article and book that I read, I learned to check the author's professional associations. It became apparent that researchers proving the efficacy of HRT are predominantly associated with pharmaceutical companies and the professional medical bodies that manage menopause, while the researchers resisting HRT are mainly associated with feminist academia and women's health activists. This meant that in negotiating the scientific medical research to access the biological descriptions of women's bodies and estrogen, I had to maintain an awareness of 'who was talking' and 'listen' to all the voices. This personal ethical issue I encountered is reflected in the research policy implications that the HRT Debate Symposium (2005) stated must be implemented in the future. The Symposium stated that as well as engaging with scientific debates over the harm and benefit of HRT we must engage with its societal context and this means "engaging with core issues of accountability, complexity, fear of mortality, and the conduct of socially responsible science" (p.745).

Personal Reflexivity

My interest in this research topic comes from my personal experience as an older woman approaching menopause and wanting to know what it means for me. I grew up in a 1960s neighbourhood with the spectre of 'the change of life' being alluded to in hushed tones, usually in reference to some un-natural, ill or anti-social behaviour of women in the neighbourhood. With no knowledge or visibility of what this actually meant for women I could only assume that one day I would somehow shrivel up and become a man. This defensive humour and ignorance lasted for years until I entered the pre-menopausal age. Now it mattered and I was on the alert for help from older women who were surprisingly silent or hushed and distressed about their symptoms. They also seemed to be confused and un-informed about HRT or the alternative therapies they were taking. I approached the 'expert', my doctor, and she dismissed me as 'too young for that and come back in a year and I'll measure your hormones'. The popular texts on menopause read like religious tracts for saving your femininity or how to be a sexy healthy goddess in spite of it all. So, I was left without a way of knowing what it means for me, which both frustrated and intrigued me, and has motivated me to help other women to view this experience more intelligently, positively and confidently.

Procedure and Analytic Strategy

The analytic strategy for this research was a Foucauldian discourse analysis. This type of analysis focuses on "*what kind* of objects and subjects are constructed through discourses and *what kinds* of ways-of-being these objects and subjects make available to people" (Willig, 2001, p.91). The medical textbooks are, on the surface, 'easy to read' because the information is at the introductory level, is presented concisely and the subject matter – physiology, pathology and pharmacology – was a generally familiar topic compared to a specialist topic like physics or chemistry. Therefore it was relatively easy to 'get to know' the texts in the key sample before approaching the discursive analysis.

In the first step in this analysis I identified 'menopause' (including pre-menopause, peri-menopause and post-menopause) as the discursive object in the texts. I did this by searching the indices for explicit references. However as I became more familiar with the textbooks I discovered that menopause was linked to specific diseases so I then searched all of these areas for references to menopause and highlighted all explicit and implicit references. I then identified the different ways in which menopause was described through the metaphors used and the themes that re-occurred throughout each text and across texts, to document the way in which menopause is constructed by the biomedical discourse in these medical textbooks. These metaphors and themes were then compared with Emily Martin's findings, particularly differences in how menopause was presented as a discursive object. By identifying how different discourses construct menopause, I was able to explore the subject positions and potential for action they offered. Finally, I examined the consequences for women's subjective experience of menopause that these subject positions offer.

To identify 'hormone replacement therapy' (including hormone therapy, estrogen therapy and exogenous estrogen) as the discursive object in the texts, .I searched the indices for explicit references, however as I became more familiar with the textbooks I again discovered that HRT was linked to specific diseases *and* menopause, so I then searched all of these areas for references to HRT and highlighted all explicit references. I identified the different ways in which HRT was described through the themes that re-occurred throughout each text and across texts. My main focus was whether the WHI clinical trial evidence was cited and the way in which it was represented. I also noted where the WHI

clinical trial evidence was not mentioned where it would reasonably be expected to be, e.g. as a risk for breast cancer. Although Martin (1987) recognised the move towards defining menopause (estrogen deficiency) as a risk to cardiovascular health, her research did not reference HRT use.

Chapter 4: Results and Discussion

The analytic process enabled the identification of five major discourses, namely the 'failure' discourse, the 'estrogen deficiency as disease' discourse, the 'HRT saviour discourse', the 'obscurity' discourse and the 'new discovery' discourse. Each of these discourses is described in detail below and is considered in terms of comparisons with Martin's findings, other menopause and HRT research, and the ways in which women are positioned within these discourses. The current textbooks will be referenced by their text numbers as shown in Table 1.

Failure Discourse

Consistent with Martin's findings on the 'woman in the body' over 20 years ago, nothing has changed in the current textbooks, which all represented women's bodies as reproductive 'machines' or 'systems' with the purpose of producing. Martin found that menopause therefore is represented as failure, a 'breakdown in hierarchical control' and a 'lack of production', as did the textbooks in the present sample. The current textbooks all constructed menopause using this failure discourse and alternative positive representations were absent.

There are two main kinds of failure, that due to a failure in the 'breakdown of hierarchical control' and that due to a failure in production. In the first, the metaphor is grounded in the descriptions of female reproduction. As Martin states: "In overall descriptions of female reproduction, the dominant image is that of a signalling system [and] the female brain-hormone-ovary system is usually described not as a feedback loop like a thermostat system, but as a hierarchy, in which the "directions" or "orders" of one element dominate…" (pp.40-41). This is the dominant image in the current textbooks where the female hormonal system consists of "three hierarchies of hormones": a hypothalamic releasing hormone (GnRH); a pituitary follicle stimulating hormone (FSH) and luteinizing hormone (LSH) "which are secreted in response to the release of GnRH from the hypothalamus"; and estrogen and progesterone which are "secreted by the ovaries in response to" FSH and LSH (Text 2, p.1011). Within these hierarchies, the hypothalamus is the "major control headquarters" (Text 2, p.772). It sends its "hormonal control factors" (GnRH) to the hormone-producing cells" of the pituitary gland (Text 4, p.436). The pituitary gland is 'second-in charge' and "exerts

many essential control functions over the rest of the endocrine system, earning it the title "conductor of the endocrine orchestra'" (Text 4, p.435). This metaphoric 'orchestra' is seen in another description of female reproduction:

> During active reproductive life, the endometrium is constantly engaged in the dynamics of shedding and regrowth. It is controlled by the rise and fall of pituitary and ovarian hormones and this control is executed by proper timing of hormonal release in both absolute and relative amounts. Alterations in this fine-tuning mechanism may result in a spectrum of disturbances, including atrophy, abnormal proliferative or secretory patterns, and hyperplasia. (Text 6, p.1081)

Here "alternations" in the mechanism lead to a "spectrum of disturbances" which positions menopause negatively, as something that has gone wrong with this "fine-tuning mechanism". Firstly, the cycles become "irregular" (Text 1, p.796; Text 2, p.1022) and then the woman "ceases to cycle altogether" (Text 1, p.796; Text 7, p.653; Text 2, p.796). The reasons given for this end of "normal ovulatory function" (Text 7, p.658; Text 8, p.460) are that "follicles do not develop in response to LH and FSH secretion" (Text 1, p.796), the ovary "*fails to* respond to the gonadotropins secreted by the anterior pituitary gland" (Text 7, p.653, italics added), there is a "*loss of* the negative-feedback inhibitor" (Text 1, p.796, italics added) and the "central mechanisms controlling GnRH release" lose control (Text 1, p.796). This 'breakdown' in the brain-hormone-ovary system then leads to what is inferred as the 'end of production'.

The overall purpose of the female-brain-ovary system is the reproduction of life. Guyton and Hall (2006, p.1011) declare that "female reproductive functions can be divided into two major phases: (1) preparation of the female body for conception and pregnancy, and (2) the period of pregnancy itself." This leaves no space for the female who does not reproduce. This same textbook then divides the male reproductive functions "into three major subsystems: (1) spermatogenesis, which means simply the formation of sperm; (2) performance of the male sexual act; and (3) regulation of male reproductive functions by the various hormones" (Text 2, p.996). Here male reproduction is discussed as *active* whereas the female body merely *prepares* for conception. Martin (1987) also identified this contrast between female and male reproduction and notes that the high speed, high volume sperm production machine does not mention the equally high volume of wastage that results from unfertilised

sperm whereas for female reproduction menstruation is described as ""ceasing," "dying," "losing,"" (Martin, 1987, p.48). Another comparison between male and female reproduction in the current textbooks relates to the 'fixed ovarian reserve'. Koeppen and Stanton (2008) state that "because the ovarian follicular reserve represents a fixed, finite number, the rate of which resting primordial follicles die or begin to develop (or both) will determine the reproductive life span of a woman" (Text 1, p.770). In the same textbook, male reproduction "has evolved for **continuous, life-long gametogenesis,** coupled with occasional **internal insemination** with a **high density of sperm…**There is no overall cyclicity of this activity in men**"** (Text 1, p.758, bold text in original). Thus male reproduction is continuous and ongoing, whereas female reproduction is fixed and finite.

The implication of this view of female reproductive life for menopause is that the body has failed its overall purpose. As in Martin's (1987) work, the current textbooks all represent this failure in production. Menopause is seen as the "end of reproductive life" (Text 2, p.1011; Text 4, p.480; Text 3, p.723) due to "ovarian failure" (Text 7, p.658; (Text 8, p.346; Text 6, p.1082; Text 2, p.1022; Text 4, p.473). This is caused by "loss", "withdrawal", "depletion" and "decline" of estrogen secretion (Text 1, p.784-794; Text 7, p.658; Text 2, p.1022; Text 4, p.708; Text 3, p.806;). This lack of estrogen production is in turn caused by the ovaries "burning out" (Text 2, p.1022) or "an exhaustion of the ovarian reserve" (Text 1, p.797) where production falls "below a critical value" and "virtually to zero" (Text 2, p.1022). This "burnout" metaphor implies that menopause is similar to the negative burnout an employee may experience when he or she is no longer motivated to function in their job. Typically this is due to a person being exposed to excessive and prolonged stress in their employment which leads to emotional, mental and physical exhaustion. This metaphor is a modern day version of the 'factory machine breakdown' metaphor whereby people are now the 'resources' in workplaces and they are required to be healthy, fit and effective to produce their creative or informational outputs. The workforce is protected from the threat of burnout 'symptoms' by prevention, coping and recovery strategies (Cordes & Dougherty, 1993).

As the female reproductive system malfunctions, "so do the members of the system decline" (Martin, 1987, p.42). In the textbooks these 'members' or body parts all suffered "atrophy". There is "atrophy of the endometrium" (Text 6, p.1082); "genital

atrophy" (Text 7, p.658); "ovarian atrophy" (Text 6, p.1083); "urogenital atrophy" (Text 8, p.345); "vaginal epithelium atrophies" (Text 1, p.796); and "atrophy of the urethal mucosa" (Text 8, p.345). Guyton and Hall (2006) provide a graphic description of a woman's 'system parts' in 'decline' at menopause:

> When the ovaries of a *fully developed* woman are removed, the sexual organs *regress* to some extent so that the uterus becomes almost *infantile* in size, the vagina becomes *smaller*, and the vaginal epithelium becomes *thin* and easily *damaged*. The breasts *atrophy* and become *pendulous* and the pubic hair becomes *thinner*. The same changes occur in women after menopause. (p.1023, italics added)

The proliferation of negative terms here portrays a woman who is defined essentially as a 'female' biological entity whose body parts have become "infantile", "smaller" and "damaged" on the interior and atrophic, "pendulous" and "thinner" on the exterior. This woman has lost claim to being a 'woman' because her body parts are no longer "fully developed", she has become the 'Other' to the privileged youthful, productive woman. The comparison with younger women's bodies ("fully developed") is repeated in another textbook where the "drop" in estrogen levels is compared to the mean levels of "younger cycling women" (Text 1, p.796). Lock (1998) recognises the bias in this comparison when she states: "…the functioning of bodies of younger women is set up implicitly as the standard for all women, and any possible advantages to the biological changes associated with aging, especially to lowered estrogen levels, are ignored" (p.418). In the current textbooks there was no positive view of the changes in estrogen production for older women. These changes were consistently and clearly situated as negative and exhausting in notions of loss, failure, decline and a body that is no longer "fully developed".

What does this failure discourse mean for women? It frames menopause as a failure on all counts – a failure in the hierarchical control of a finely tuned system, a failure in the production of estrogen and a failure to produce life. How does this 'failure' position women in menopause? Are ageing women unproductive, no longer developed and therefore unnecessary? The negativity in this framing is unnecessary. There are other ways to frame such changes. The following example is a positive view of this change. It is quoted by Emily Martin (1987) from a gynecology textbook:

> It would seem that although menopausal women do have an estrogen milieu which is lower than that necessary for *reproductive* function, it is not negligible or absent but is perhaps satisfactory for *maintenance of support tissues*. The menopause could then be regarded as a physiologic phenomenon which is protective in nature – protective from undesirable reproduction and the associated growth stimuli. (Jones & Jones, 1981, p.799, italics in original)

The known physiological changes of the menopause transition are not disputed but the way in which this failure discourse constructs these changes should be challenged so that women are aware of these influences when they interpret their experience of menopause.

Estrogen Deficiency as Disease Discourse

Throughout the current textbooks menopause is framed as a disease in itself and also as leading to other diseases, specifically those related to the breast, genitals and bone. These two constructions will be discussed and then their implications for the woman in menopause will be examined. Firstly, menopause is defined as an "estrogen deficiency" (Text 7, p.719; Text 8, p.345) such that the "symptoms associated with menopause result from estrogen deficiency" (Text 1, p.796). These symptoms are both physical and psychological. The physical symptoms attributable to menopause (estrogen deficiency) across the current textbooks are listed in Table 2 below.

The psychological symptoms of menopause occur where "the loss of estrogen production by the ovaries has a clear clinical impact" (Text 1, p.784). The psychological symptoms reported by the textbooks are shown in Table 2. Guyton and Hall (2006) go so far as to declare that "a woman must readjust her life from one that has been physiologically stimulated by estrogen and progesterone production to one devoid of these hormones" (Text 2, p.1022) which highlights how a woman's entire "life" is seen as driven by hormones. Guyton also made this declaration in the 1986 edition, so twenty years later this textbook has not changed its exaggerated view of the pervasive impact of hormonal control not just over women's bodies but over their lives as well. However, these physical and psychological symptoms of estrogen deficiency are relatively minor compared to the diseases of the breast, genitals and bone that are due to estrogen deficiency.

Table 2: Symptoms of Menopause in the Medical Textbooks

Physical Symptoms	Text # (page)	Psychological Symptoms	Text # (page)
"hot flashes"	1(796), 3(292)	"psychic sensations of dyspnea"	2(1022)
"hot flushes"	2(1022), 7(659), 8 (345)	"anxiety"	2(1022)
"vasomotor symptoms"	7(659)	"psychopathological states"	7(659)
"dyspareunia (painful intercourse)"	8(345)	"irritability"	2(1022)
"insomnia"	7(659)	"neuropsychiatric effects"	8(346)
"recurrent urinary tract infections"	8(345)	"occasionally various psychotic states"	2(1022)
"fatigue"	2(1022)	"climacteric depression and other psychopathologic states"	7(659)
"palpitations"	8(345)		
"vaginal dryness"	8(345)		
"sweating"	7(659), 8(345)		
"urinary incontinence"	8(345)		
"coronary artery disease increases markedly"	1(796)		
"decreased strength and calcification of bones"	2(1022)		
"acceleration of atherosclerotic cardiovascular disease	1(658)		

The current textbooks do not relate estrogen deficiency per se to benign breast proliferation changes, rather there is a correlation between pre- and postmenopausal women and these changes occurring. Underwood (2004) states that fibrocystic change and cystic hyperplasia of the breast "increase in frequency towards menopause" and the incidence of these lesions "reaches a maximum in the years just before menopause" (p.473). These changes are not related to disease, rather they are structural changes in body tissue which may or may not lead to disease. Yet the implication is that menopause causes these structural tissue changes, inferred by observation only: "in women nearer the menopause cysts are frequently seen" (Text 4, p.474). This observation could equally be framed as a frequency related to a woman's age rather than to menopause.

In terms of breast cancer, the postmenopausal woman is clearly positioned as at risk, as the following quotes from the current textbooks demonstrate:

> Fifty to 85% of carcinomas express estrogen receptors, and such tumors are more common in postmenopausal women. (Text 6, p.1147)
>
> Functioning ovarian tumors that elaborate estrogens are associated with breast cancer in postmenopausal women. (Text 3, p.745)
>
> Women whose natural menopause occurs before 45 years have only half the breast cancer risk of those whose menopause occurs after 55 years. (Text 4, p.480)
>
> Breast cancer is uncommon in women younger than age 30. Thereafter, the risk steadily increases throughout life, but after menopause the upward slope of the curve almost plateaus. (Text 3, p.744)

Where a reason is given for this risk for postmenopausal women, it is the link between breast cancer and hormonal status which is "strongly suggested by the conspicuous association between the incidence of this tumor and the age of menarche, menopause, and first pregnancy" (Text 5, p.848). This "*hormone exposure*" at menarche and menopause association is seen as a "*major risk factor for sporadic breast cancer*" (Text 6, p.1120, italics in original). Here, menopause is associated with other life transitions and female hormones are deemed to be somehow causative by their "conspicuous" presence. Further, this causation is framed as a "risk factor" which reflects the way that scientific medicine now blurs the concept of illness and risk. The meaning of medical

risk is the potential for illness "but it is increasingly being treated as if it were an illness in and of itself" (Conrad, 2007, p.163).

Change in the genital structure of an older woman's body is also treated as a disease in and of itself. These 'genital changes as disease' are presented by Kumar, Abbas & Fausto (2005, p.1082) in a section entitled "Disease of Organ Systems" with a sub-title of "Menopause and Postmenopausal Changes". They state that since menopause "is characterized by anovulatory cycles, architectural alterations in the endometrial glands may be present transiently, followed by ovarian failure and atrophy of the endometrium" (Text 6, p.1082). This is followed by the warning that these "architectural alterations" or "cystic changes" should not be "confused with more active hyperplasia, which exhibits evidence of glandular and stromal proliferation" (Text 6, p.1083). It is confusing as to why this structural change in the endometrium of older women is located in a *disease* section given that all women will be affected by its purported cause – ovarian failure.

There is more confusion and contradiction across textbooks in their description of lichen sclerosus. This is the growth of lesions and atrophy and stiffening of the vulva and vaginal orifice (Text 3, p.713). This 'disease' "occurs in all age groups, but is most common in postmenopausal women" (Text 3, p.713) or "after menopause" (Text 6, p.1066). However, Reuben and Strayer (2008) do not associate this disease with postmenopausal women at all (p.787). Further, Kumar, Abbas, Fausto & Mitchell (2007) state that benign tumors "occur at any age [but] they develop more commonly at the time of menopause" (Text 3, p.724) whereas Kumar, Abbas & Fausto (2005) state that they "are equally common before and after menopause, with a peak incidence at 40 to 60 years of age" (Text 6, p.1090). The only cause given is that "estrogens and possibly oral contraceptives stimulate their growth; conversely they shrink postmenopausally" (Text 3, p.724). As well as being confusing and contradictory, the menopause marker is meaningless as these tumors occur before, during and after menopause. This theme of confusion and contradiction continues with the descriptions of the more serious cancerous tumors of the endometrium and menopause.

Endometrial carcinomas are reported to "occur in postmenopausal women" (Text 6, p.1088), "in the uterus of peri- and post-menopausal women" (Text 4, p.508), they affect "elderly post-menopausal women" (Text 4, p.507) and appear "most frequently

between the ages of 55 and 65 years" (Text 3, p.725). Again the menopause marker appears meaningless as these carcinomas occur in peri-menopause, menopause and post-menopause. Women's hormonal status is again presented as the causal agent. Kumar et al (2007) state that the "clinical setting" for these carcinomas is "estrogen excess" in perimenopausal women and endometrial atrophy in older women (Text 3, p.725). Kumar et al (2005) link endometrial hyperplasia (which may lead to carcinomas) to "prolonged estrogen stimulation of the endometrium by anovulation or increased estrogen production" (p.1085). This textbook also cites "estrogen replacement therapy" as a condition promoting endometrial hyperplasia (Text 6, p.1085). Therefore, it is either too much estrogen in peri-menopause and menopause and from HRT use or too little estrogen in older postmenopausal women with endometrial atrophy. For endometrial carcinoma, even the 'too little estrogen' explanation is modified for postmenopausal women: "In postmenopausal women, there is a greater synthesis of estrogens in body fat from adrenal and ovarian androgen precursors, a finding that may partly explain why there is increased risk of endometrial cancer with age and obesity" (Text 6, p.1087). This speculation reinforces the meaningless of menopause as a marker for the potential cancerous changes in women's bodies over time. The significance of this menopause marker is the "conspicuous presence" of estrogen, although it is confusing whether it is its excess or deficiency that is the problem.

There is no confusion in the current textbooks for the link between estrogen deficiency and bone health, and postmenopausal osteoporosis is named as a disease in itself. The disease is one of the two most common forms of osteoporosis, the other is senile osteoporosis (Text 6, p.1281; Text 3, p.804). Rubin and Strayer (2007) state that "postmenopausal osteoporosis is usually recognizable within 10 years after the onset of menopause, whereas senile osteoporosis generally becomes symptomatic after age 70 years" (p.1110). The symptom of postmenopausal osteoporosis is "an acceleration of bone loss" (Text 7, p.658) which for "susceptible women" is "often particularly severe" (Text 3, p.804) with a "more rapid decline in women in the early postmenopausal years" (Text 8, p.457). This accelerated bone loss leads to osteoporosis. The overt sign of postmenopausal osteoporosis is evidenced in "vertebral, hip and wrist fractures" (Text 7, p.658) which are "rare in middle-aged men" (Text 5, p.1107). Osteoporosis used to be called the 'silent disease' because it was only when fractures occurred that it was diagnosed, however sensitive screening techniques now show up the reduced bone

density. This screening for women in menopause began with a classic study in 1982 by a radiographer, Genant, who meticulously chartered the retention of bone density of 30 women before and after their oophorectomies (ovary removal) over a 24 month period. It was a random double-blind study with women either receiving a placebo, a 0.625 dose of Premarin or a lower dose of Premarin. Genant found that the women on the 0.625 dose of Premarin retained their bone density far more than the other groups. All of the women in Genant's experiment had their ovaries removed, therefore the findings could not be generalised to women with ovaries. However, the truth got lost in translation and "the word went out that rapid bone loss at menopause could be held at bay with 0.625 of Premarin" (Seaman, 2003, p.77).

The scanning of women's bodies continues to show up the insides of their bones as porous and decaying. The textbooks also display pictures of bone scans with osteoclasts resorbing bone (Text 3, p.805) and x-rays of patient's bones with osteoporosis (Text 6, p.1282). The rapidity of this decay is described in an alarmist way by Kumar et al (2007):

> The decline in estrogen levels associated with menopause correlates with an annual decline of as much as 2% of cortical bone and 9% of cancellous bone. This can add up to 35% of cortical bone and 50% of trabecular bone within 30-40 years! Therefore it is not surprising that roughly half of post-menopausal women will suffer an osteoporotic fracture (compared to 2-3% of men of comparable age). (Text 3, p.806)

This observational correlation between the decline in estrogen levels and the decline in bone density for women is deliberately extrapolated so that the contrast between women and men's risk of fracture is magnified. This frames women as diseased and weakened by their own physiology, their very skeletal structure is eroded by their own hormones, whereas men overall remain sturdy by comparison. Correlation does not provide evidence for causation, and there are many hypothesised causes for osteoporosis. However, the current textbooks do converge on the cause of this bone disease in women as being due to estrogen deficiency "because estrogens decrease the number and activity of osteoclasts" (Text 2, pp.991-992). Medical knowledge explains that our bones are not fixed; rather they are continually forming by osteoclast cells absorbing the bone that is there and osteoblast cells growing new bone. As it is thought that estrogens suppress the

osteoclast cells, when there is a decrease in estrogen, these cells are free to absorb more bone. The current textbooks therefore are in agreement that "estrogen deficiency plays the major role in this phenomenon" (Text 6, p.1284). Rubin and Strayer (2008) state that "the increased number of osteoclasts that appears in the early postmenopausal skeleton is the direct result of estrogen withdrawal" (Text 5, p.1109). Although osteoporosis occurs in both men and women (Text 5, p.1107; Text 7, p.719) and ageing is a major determinant, the other major determinant is "estrogen deficiency in postmenopausal women" (Text 5, p.1110). Therefore, although ageing is acknowledged as a cause, "by far the most common such cause is post-menopausal oestrogen withdrawal" (Text 4, p.708). In 1981 Guyton (p.1011) did not link estrogen deficiency with osteoporosis, but from the next edition onwards, and after Genant's results in 1982, it is stated that: "In some women, this effect is extremely severe, and the resulting condition is *osteoporosis*" (1986, p.974, italics in original).

There are many causes for osteoporosis and all of the current textbooks describe them. It is related to alcoholism, diabetes, liver disease and smoking (Text 4, p.709); "lack of physical activity, lack of vitamin C, old age, Cushings syndrome, and malnutrition (Text 2, pp.991-992); and calcium intake, calcium absorption and vitamin D, exercise and smoking (Text 5, p.1109). It is stated that "even young healthy males confined to bed after a limb fracture show substantial bone loss" (Text 4, p.708). The most interesting admission comes from Underwood (2004) which clearly un-does the construction of postmenopausal osteoporosis:

> Many postmenopausal women and elderly men have some degree of osteopenia or bone loss. In most cases this is due to osteoporosis…this results in fractures…commonly of the vertebral bodies leading to a stooped posture (so-called 'Dowager's hump'). This would appear to be a clear indication that spontaneous deterioration in hormonal function (ovarian function in the case of post-menopausal women) leads to a classic ageing phenomenon; this is often treated with hormone replacement therapy (HRT). However, it is now becoming clear that the development of osteoporosis in old age is much more common in those who were inactive or who had diets low in calcium or vitamin D in youth and epidemiological evidence is accumulating that many classic features of old age are controlled in this way by things that happen during the youth of the individual. (Text 4, p.268)

What does the 'estrogen deficiency as disease' discourse mean for women? In this discourse women in menopause are framed as ill due to their own hormone deficiency or excess; their own femaleness renders them sick. This illness is no longer transitory as with menopause symptoms, rather it can lead to life threatening diseases of the breast and genitals and the most pervasive of these diseases is the accelerated decay of their bones which threatens their lives in the long term. This framing positions women as 'damsels in distress', they are vulnerable, they are under siege by their own hormones, at risk of cancer, and their bones are dissolving. They need saving. This salvation is offered to them in their positioning as the 'patient' who can be treated. This 'patient' is discussed across the current textbooks. For example: "These "peri- and post-menopausal symptoms" (Text 4, p.505) are of "sufficient magnitude in about 15 percent of women to warrant treatment." (Text 2, p.1022) and they are the basis for the "management of the postmenopausal patient" (Text 7, p.659). In terms of psychological symptoms, Guyton and Hall (2006) state that "if counselling fails, daily administration of estrogen in small quantities usually reverses the symptoms…" (Text 2, p.1022). Interestingly the 1986 edition of this textbook uses the same sentence but with the word 'psychotherapy' instead of 'counselling' (Guyton, 1986, p.658). Perz and Ussher (2008) query whether women internalise this idea of a weakened, decayed and atrophied body and therefore submissively subject their bodies to medical control by accepting this 'patient' position. It can also be queried here whether women will internalise the idea of an 'at risk postmenopausal' body that although it appears healthy, calls for a greater compliance with medical management. If women accept this passive 'patient' position they are offered salvation in the form of drugs. These drugs include "hormone replacement therapy, calcitonin, bisphosphonates, selective estrogen receptor modulators (SERMs), calcium and vitamin D" (Text 8, p.460). The prescribed drug of choice is oral estrogens which are the "most widely prescribed drug to treat postmenopausal osteoporosis" (Text 8, p.461). This form of salvation will now be examined.

HRT Saviour Discourse

Significantly, six of the eight current textbooks reference the WHI results and interpret them as serious risk factors in HRT treatment. The warnings from the WHI trials have made HRT a very conditional saviour for the 'postmenopause patient' and patient

management is now framed as a consultative interaction between the physician and a woman's own individual risk assessment.

The two physiology textbooks do not report any WHI warnings. Guyton and Hall (2006) state only that symptoms of menopause are usually reversed with "daily administration of estrogen in small quantities" (Text 2, p.1022). Koeppen and Stanton (2008) do not discuss HRT as a treatment although they do outline the molecular basics of the next generation selective estrogen receptor modulators (SERMs). This is HRT that aims to selectively inhibit or stimulate estrogen in various parts of the body. The other six textbooks all report the established warnings about estrogens alone presenting a known risk of endometrial cancer such that progestin (a synthetic form of the hormone progesterone) must be added to the HRT treatment. For example, "HRT increases the risk of ovarian cancer…but the risk is drastically reduced or eliminated when progestins are added to the therapeutic regimen…most commonly in use today for postmenopausal women" (Text 3, p.293).

These six textbooks all cite the WHI findings although the varying ways these results are reported ranges from brief 'risk' sentences that indirectly reference the WHI findings through to reports of and responses to the findings. Brief 'risk' sentences are seen in the assertion in Underwood (2004) that HRT has been "linked with an increased risk of endometrial cancer and, possibly breast cancer" (Text 4, p.481). Rubin and Strayer (2007) take this warning further and report that recent research on estrogen products has "cast doubt on their effectiveness in preventing myocardial infarction and osteoporosis [and shown] increased risk for cancers of the breast and endometrium [and] increased risk of deep venous thrombotic events" (Text 5, p.263). A more definitive 'risk' statement that directly references the WHI findings is seen in Page et al (2006) who state that "a recent large-scale clinical trial showed that HRT was associated with increased mortality. This resulted in HRT falling into disrepute and alternative therapies being emphasized" (Text 8, p.346). This warning is also issued by Kumar et al (2007) who state that since the WHI findings "suggested greater harm than benefit...there has been a precipitous decline in estrogen plus progestin use and a serious re-evaluation of menopausal hormone therapy" (Text 3, p.744). The most detailed reporting of the WHI findings for HRT risks for breast cancer, venous thrombosis, pulmonary embolism, cardiovascular disease and cholecystitis is provided by Kumar et

al (2005). Katzung (2007) directly acknowledges the WHI findings and the risks for cardiovascular disease and breast cancer with HRT but disputes their validity for younger menopausal women by stating that "young women with premature menopause should definitely receive HRT" (Text 7, pp.658-659). The reason for this recommendation is not given in this textbook, however the 11th edition of this textbook (Katzung et al, 2009) does report that "there is no increased risk for breast cancer if [HRT] therapy is given immediately after menopause and for the first 7 years, while the cardiovascular risk depends on the degree of atherosclerosis at the onset of therapy" (p.704). This recommendation reflects the 'window of opportunity' argument discussed previously in Chapter 1.

The conditional HRT saviour discourse constructs HRT as the treatment for menopause symptoms like hot flashes but it is predominantly aimed at saving women from accelerated bone loss due to estrogen deficiency that is reported to occur at postmenopause. However, in this context the six textbooks all recognise the WHI findings and the risks of using HRT that were evidenced. The common theme in the current textbooks is that they present menopause symptoms and the more serious bone loss disease caused by menopause, then the taken-for-granted remedy of HRT is shown to now have 'risks' because of the WHI findings. Kumar et al (2007) assert that "…hormone replacement therapy, prevents or at least delays the onset of osteoporosis. However, according to recent studies, relatively short-term use of combined estrogen and progestin hormone therapy is associated with an *increased risk* of breast cancer, diagnosis at a more advanced stage of breast cancer, and more abnormal mammograms." (Text 3, p.744, italics added). Rubin and Strayer (2007) state that "estrogen therapy is an effective if *controversial* means of preventing postmenopausal osteoporosis. Because hormone treatment carries with it slightly *increased risks* of breast and endometrial cancers other bone-specific antiosteoporotic drugs have been developed" (Text 5, p.1110, italics added). Kumar et al (2005) report the long term benefits of HRT as "maintenance of bone mineral density and prevention of osteoporotic fractures" but now the WHI findings "have provided *new information about the risks* and benefits of HRT" (Text 6, p.427, italics added). Katzung (2007) states that there is reduced "enthusiasm" for HRT therapy for postmenopausal osteoporosis due to "concern that estrogen *increases the risk* of breast cancer and fails to reduce the development of heart disease" (Text 7, p.719, italics added). This makes

HRT a very conditional saviour for women whose bones are dissolving at an accelerated rate.

This HRT Saviour discourse constructs women as postmenopausal patients who require treatment with HRT in order to save them from accelerated bone loss. However, the conditional nature of this treatment given the WHI results is recognised and this concern transfers into the way that the patient is managed. Page et al (2006) state that "the decision to start estrogen replacement therapy and patient adherence to therapy depend not only on its effect on bone but also on its other clinical effects" (Text 8, p.461). These other 'clinical effects' mean that the patient is now at risk and these risks and benefits "must be evaluated for each individual patient in the context of her overall health, individual risk factors, and family history" (Text 6, p.428). Therefore the risks of treatment mean that "optimal management of the postmenopausal patient" requires a "careful assessment of her symptoms" and a discussion with her about the "presence of (or risks for) cardiovascular disease, osteoporosis, breast cancer, and endometrial cancer" (Text 7, p.659).

Despite this risk-based management of the postmenopausal patient, the current textbooks continue to promote HRT with 'slight risk' statements for breast cancer and the 'smallest dose' prescriptions for HRT. Katzung (2007) states that although "no adverse effect of short-term estrogen therapy on the incidence of breast cancer has been demonstrated, a small increase in the incidence of this tumor may occur with prolonged therapy" (Text 7, p.660). Katzung (2007) also warned previously that "concern that estrogen increases the risk of breast cancer and fails to reduce the development of heart disease has reduced enthusiasm for this form of therapy" (Text 7, p.719). After issuing a WHI warning, Rubin and Strayer (2007) then make a similar 'slight risk' statement by stating that "the use of oral contraceptive agents has not been associated with an increased risk of breast cancer, although postmenopausal hormone supplementation slightly augments the risk" (Text 5, p.848). Further, Page et al (2006) issue the WHI warning of "increased cardiovascular events" then continue to recommend that "transdermal estrogen usually controls postmenopausal symptoms and osteoporosis, but has less effect on lipoproteins" (Text 8, p.461). This trend does not stop with breast cancer. Katzung (2007) acknowledges that HRT "is a major cause of postmenopausal uterine bleeding" but "unfortunately" since this bleeding may also be due to endometrial

carcinoma it is recommended that to "avoid confusion, patients should be treated with the smallest amount of estrogen possible" (Text 7, p.660).

Perhaps the best example of this promotion of 'HRT despite the risks' is seen in the therapeutic strategy advocated by Katzung (2007) for the management of the 'postmenopausal patient'. This therapeutic rationale begins by prescribing HRT for physical and psychopathological menopause symptoms in the "lowest dose, for the shortest time" for menopause symptoms where there is a low risk of osteoporosis (Text 7, p.659). However, if there is a risk of osteoporosis (and the current textbooks infer that all women are at risk) then HRT should be given in the "smallest dose monthly as soon as possible after menopause" (Text 7, p.659). There is now no indication for how long HRT use should continue and the only pre-WHI warning provided is that estrogen plus progestin must be combined to avoid endometrial cancer. The benefits of daily HRT use are stated, including the maintenance of bone density and cardiovascular health, but there is no mention of the WHI findings for the risks associated with HRT use previously reported by this textbook. Instead Katzung (2007) acknowledges that the "main disadvantage of continuous therapy is the need for uterine biopsy when bleeding occurs after the first few months" (p.659). This technique of checking that HRT is not causing or exacerbating disease is a further invasive intrusion of a woman's body.

In the context of discussing the assessment risk of HRT with the 'postmenopausal patient' the textbooks do not explicitly state that HRT only defers ongoing bone density changes or that "the same rapid loss of bone (up to 15% over 5 years) will occur when you stop taking ET or HT, no matter how long you have been on it" (New York State Department of Health, 2004). Katzung (2007) states that estrogen blocks bone resorption which leads to a "*transient* increase in bone mineral density because bone formation is not initially decreased" (Text 7, p.719, italics added). This "transience" of the conditional HRT saviour is inferred in the wording used in the other textbooks for the efficacy of HRT on bone density whereby it "slows bone loss" (Text 8, p.461); "or at least delays the onset of osteoporosis" (Text 3, p.744); is used for the "maintenance of bone mineral density and prevention of osteoporotic fractures" (Text 6, p.427); and "can ameliorate some of the bone loss" (Text 3, p.806). However, these representations of the conditional HRT saviour are not explicit that it is only a temporary measure.

What does this conditional HRT saviour discourse mean for women? Since the WHI findings, research has shown that women's most frequently cited information source for menopause and HRT continued to be their doctor, who was ranked significantly higher than pharmacists and other health care professionals for trustworthiness, helpfulness and knowledgeability (Huston, Jackowski & Kirking, 2009). However, since the WHI findings, the conditional HRT saviour discourse now exhorts women to use their autonomy, their individual risk-based assessments, in consultation with their doctors in making their choice about HRT. Therefore women are now invited into the 'objective realm' of medical science to be responsible for calculating the statistical risks for diseases they may or may not acquire, with or without HRT, based on their past, present and future health assessments. Also, as Roberts (2007) asserts, this liberal model of 'choice' "is cleansed of any engagement with issues relating to power differences" (p.146). These power differences are reflected in medical consultations where physicians retain their expertise on the management of women's bodies with the authority of their 'factual' knowledge. This in turn allows them to offer women the impossible 'choice' of which disease they would prefer to have. However, there are contingencies or gaps that exist in this authoritative 'factual' knowledge. It is worth remembering that medicine originated with priests or holy men who maintained the secret and magical knowledge of herbs and potions and who were respected and feared because intentional poisoning was not unheard of (Page et al, 2006).

Obscurity Discourse

During analysis it became apparent that there was a sub-text in the scientific medical information that was represented which was subtle but easily identifiable in the 'estrogen deficiency as disease' discourse and HRT efficacy descriptions. The predominant tone of the textbooks is factual and authoritative, but a closer look at the language, words and meanings, particularly about the contingencies in knowledge, demonstrates what could be called an obscurity discourse. Here the actual causation of diseases from estrogen deficiency and the benefits of HRT are obscure, not fully known and an ongoing project. Furthermore the processes involved in menopause itself are also not well understood.

For example, the current textbooks give an authoritative account of what menopause is in terms of the failure discourse, yet the "causes and processes of menopause are poorly

understood" (Text 1, p.794). The failure discourse constructs menopause as a breakdown in the hierarchy of control of the hypothalamus-pituitary-ovarian system yet this system is the subject of speculation. Guyton and Hall (2006), authorities on menopause, state that if "counselling fails, daily administration of estrogen in small quantities" (Text 2, p.1022) should be given to the 'postmenopausal patient'. They state that in menopause the "estrogens can no longer inhibit the production of gonadotropins" (Text 2, p.1022). Yet this textbook speculates about this process: "Now, after discussing much of the known information about the interrelations of the different components of the female hormonal system, we can attempt to explain the feedback oscillation that controls the rhythm of the female sexual cycle. It seems to operate in approximately the following sequence of three events" (Text 2, p.1021).[1] This speculation is then extended into the specifics of the 'hierarchical control system' where "the cause of this abrupt surge in LH secretion is *not known*. However, *several possible explanations* are as follows…" (Text 2, p.1021, italics added). One of these speculations for the way in which this system develops is that it is initiated in puberty "by some maturation process that occurs elsewhere in the brain, perhaps somewhere in the limbic system" (Text 2, p.1022). Another possible explanation is that "age-related changes in the CNS…may play an important role" (Text 1, p.796). These "speculations" and "possibilities" lead us to question the validity of the medical 'facts' of women's body functioning as represented in the failure discourse. If medicine is not really sure about how female reproduction functions, then by what authority does it claim to intervene in a body transition like menopause? Charles Dodds, one of the first biochemists to synthesise estrogen, warned that our lack of understanding of how even one Graafian follicle in the ovaries develops should make us humble about knowledge of the female reproductive cycle (Seaman, 2003).

Furthermore, the current textbooks all cite the predominant symptom of menopause as hot flashes yet "the mechanisms underlying menopausal vasomotor symptoms are not clearly understood" (Text 8, p.345). The cause of these 'menopausal vasomotor symptoms' is "thought to be linked to increases in LH release and are probably associated not with the pulsile rise in LH secretion but rather with central mechanisms

[1] A previous edition of this textbook is more open about its speculation: "we can *digress from the area of proven fact into the realm of speculation* and attempt to explain the feedback oscillation that controls the rhythm of the female sexual cycle" (Guyton, 1986, p.978, italics added).

controlling GnRH release" (Text 1, p.796). In this example, something has gone wrong with the "major control headquarters" (Text 2, p.772) but just what is still not clear. Similarly, the current textbooks cite a link between estrogen deficiency and proliferative breast disease yet "the aetiology is poorly understood" and although there "is no doubt that ovarian hormones participate in its causation…the means by which the changes are produced are still *obscure*" (Text 4, p.473, italics added). A reason why ovarian hormones are implicated is inferred from a correlation: "The fact that the incidence of benign proliferative changes increases as menopause gets nearer and that failure of ovulation also increases in this time period, *suggests* that the relative imbalance of oestrogen and progesterone in each menstrual cycle *could be* an important aetiological factor" (Text 4, p.473, italics added). As correlation cannot show causality, the cause of breast disease remains unknown and open to further investigation.

The current textbooks do cite the link between estrogen deficiency and breast cancer but "as is the case with all cancers, the cause of breast cancer remains unknown" (Text 3, p.744) and the "pathogenesis of breast cancer is poorly understood" (Text 5, p.847). As with breast disease, a reason why hormonal influences are implicated is inferred from a correlation: "A link between breast cancer and the hormonal status of women is strongly suggested by the *conspicuous association* between the incidence of this tumor and the age of menarche, menopause, and first pregnancy" (Text 5, p.848, italics added). More specifically, it is "hypothesised that the estrogen and progesterone receptors… often present in breast cancer cells, may interact with growth promoters…" (Text 3, p.745). And, as with the research on what causes hot flashes, the "relation of estrogen therapy to cancer continues to be the subject of active investigation" (Text 7, p.660). This "active investigation" reflects that medical science is an ongoing project. This search for answers is also seen with the genital disease, lichen sclerosus. It is cited as a disease associated with postmenopause yet the "pathogenesis is unclear…it has many features of an autoimmune disorder" (Text 6, p.1066). These hypotheses set the direction for future research where scientists and industries compete to discover the causation of diseases and their potential treatments.

The current textbooks are unanimous in their authoritative assertion that estrogen deficiency causes postmenopausal osteoporosis yet this disease "is of uncertain origin" (Text 5, p.1109). Rubin and Strayer (2007) set out the two major determinants of

primary osteoporosis as "estrogen deficiency in postmenopausal women and the ageing process in both sexes" and summarise the "possible mechanisms for these effects" in a diagram (Text 5, Table 26-27, p.1110). This diagram describes the process for postmenopausal osteoporosis by simply naming the "osteoclast" and "osteoblast" cytokines with "estrogen" interfering with them – this is the molecular explanation of bone remodelling. This molecular explanation is a part of the 'cutting edge' of medical science and it is here that the textbooks step onto new ground: "Although *much remains unknown,* recent advances in elucidating the molecular biology of bone have provided *intriguing new hypotheses*…" (Text 6, p.1283, italics added). Kumar et al (2007) have a similar view: "Although a complete understanding of the underlying control mechanisms of bone remodelling is not yet known, there are a number of *exciting new insights*" (Text pp.804-805, italics added). These "exciting new insights" are not substantiated. This textbook also states that "…females are more vulnerable to osteoporosis and its complications. Regardless of the underlying cause…" (p.804). In terms of HRT, the current textbooks are also unanimous in their assertion that it reduces menopause symptoms and slows accelerated bone loss in women and if the risks identified in the WHI research can be negotiated then it should be a treatment for the 'postmenopausal patient'. One of the historical risks that these textbooks openly and clearly warn about is that it "is now recognized that there is no indication for the use of diethylstilbestrol during pregnancy, and it should be avoided" (Text 7, p.660). The 'DES daughters' story gives us a way of exploring the efficacy of HRT. As mentioned earlier, in 1971 the FDA removed approval for the use of this synthetic estrogen to prevent miscarriages and treat menopause symptoms because research showed that it increased the risk of breast cancer and women's daughters developed genital carcinomas, structural deformities and infertility. Katzung (2007) issues the DES warning but then states that "it is not known whether other estrogens have a similar effect or whether the observed phenomena are peculiar to diethylstilbestrol" (p.660). Rather than asking whether HRT works, surely the current textbooks and medical researchers should ask if it is safe for women?

What does this obscurity discourse mean for women? This discourse is similar to the 'menopause as confusing' discourse identified by Lyons and Griffin (2003) in their discursive analysis of four medical, alternative and feminist self-help books. It was represented in the medical profession in debates around whether menopause was a

natural life transition or a medical disease such that "the menopause is so confusing that even the doctors, who are constructed clearly as 'experts', do not have a straightforward view of menopause" (Lyons & Griffin, 2003, p.1636). Lyons and Griffin (2003) also report that in this discourse women's bodies are also framed as confusing, mysterious and relatively invisible in all except the medical books. In the present study the obscurity discourse extends this framing of women's bodies as confusing and complicated to the medical profession which leads us to question their implicit authority as experts on women's bodies and their bodily life transitions such as menopause. If the medical profession is confused in its construction, then can women trust that they are not endangering their health? Germaine Greer (1991) has 'expertly' identified the consequences of this obscurity discourse for the 'postmenopausal patient':

> We do not know by what biological pathway that active agent reaches the target organs. It is at least possible, if not likely, that treatment with replacement oestrogen to ablate the fluctuations in menopausal levels may affect the switch-over to a new pattern of secretion relying upon the stromal cells and the adrenals. The battering of this delicate mechanism with large amounts of exogenous steroids may prevent its establishment, so that the long-term outcome of this kind of haphazard dosing of women … is to compromise their health and accelerate their ageing. (p.167)

New Discovery Discourse

As seen in the obscurity discourse, there is a sub-text in the textbooks that represents the causation of diseases from estrogen deficiency and the benefits of HRT as not known, obscure, and an ongoing project. The treatment of the 'postmenopausal patient' is also an ongoing project. The focus of the HRT saviour discourse is to protect the 'postmenopausal patient' from accelerated bone loss but this saviour has become very conditional since the WHI clinical trials reported their risk findings. The new discovery discourse seeks to overcome these risks and continue to treat the 'postmenopausal patient'. It seeks to eliminate the health risks of HRT by using the newer drugs, the next generation HRTs, which include selective estrogen receptor modulators (SERMs). These drugs selectively inhibit or stimulate estrogen-like action in target tissues. In the current textbooks the new discovery discourse constructs an 'exciting' era of 'new discovery' with these new drugs selectively targeted at reducing bone loss for

postmenopausal women. These new drugs are still under research investigation, though some are FDA approved for use, and they all have benefits and adverse side-effects. They are seen to be a prospective new paradigm for maintaining women's health (Pandar, 2002). This discourse represents the causation of bone loss and the effects of these drugs with the 'molecular story' which reflects the latest technological advances in neurochemical research. In this discourse there remains a sub-text of uncertainty and obscurity. The treatment and management of the 'postmenopausal patient' continues with the goal to achieve the ultimate menopause therapy through these drugs. Komm (2008) sets out the hope that these drugs will:

> Reduce the number and severity of hot flashes, effectively treat vulvar-vaginal atrophy and its symptoms, prevent and treat menopausal osteoporosis, and have favorable effects on lipoprotein profiles, while at the same time would not stimulate the endometrium, not cause uterine bleeding, not increase the risk of vascular events, not be associated with breast pain or tenderness, and potentially reduce breast cancer incidence. (p.984)

Komm is affiliated to the pharmaceutical company Wyeth and this highlights the imperative of drug therapy for menopause which is part of the general shift in medicalisation toward re-setting the social norms of bodies, health and behaviour. Conrad (2007) notes this shift in medicalisation whereby the pharmaceutical biotechnology industries are now major players investing in human enhancement products such as Viagra and HRT and direct marketing them to consumers.

This ongoing project of scientific medicine can be identified in the way that new discoveries, particularly molecular science, are represented in the textbooks. Kumar et al (2005) capture this tone in their introduction where there is "new and exciting information in stem cell biology", "spectacular advances, including completion of the human genome project", "the newest information available – morphologic and molecular" and advances "at the cutting edge of science" which show "the excitement of the topic" (Text 6, p.ix). One of these exciting new discoveries is the next generation HRTs. Wyeth in the United States is conducting drug trials of its next generation HRT drug Aprela. This drug is a new SERM (bazedoxifene) combined with the old conjugated estrogens and if FDA approval can be achieved then this "potential

blockbuster…will help further maintain Wyeth's position as a leading women's health player" (Datamonitor, 2009). However, the trademark details for Aprela are: "pharmaceutical preparations to treat menopausal and post-menopausal female conditions and for the prevention and treatment of osteoporosis" (Trademarkia, 2010). This move beyond "female conditions" to the "prevention and treatment of osteoporosis" suggests a move beyond women's health markets to the treatment of men as well which would make Wyeth a much more lucrative and powerful player in world health markets.

The benefit sought in the Next Generation HRTs is that bone health is maintained while the health risks identified in the WHI clinical trials are eliminated. These drugs "act by increasing bone mass (similar to endogenous estrogen) but without the side effects associated with estrogen use" (Text 3, p.806). They "avoid the increased risk of breast and uterine cancer associated with estrogen while maintaining the benefit to the bone" (Text 7, p.719). One of these FDA approved SERMs for the 'postmenopausal patient' is Raloxifene. The textbooks report its benefits and risks: It "protects against spine fractures…does not prevent hot flushes and imposes the same increased risk of thrombophlebitis as estrogen" (Text 7, p.719). There is an "improvement of bone density of the hip and lumbar spine [with] no evidence of increased endometrial cancer" but the "adverse effects include an increased risk of venous thrombolic events similar to estrogen [and] a slight increase in hot flushes …" (Text 8, p.462). The implications for the 'postmenopausal patient' are that her bone density is improved in her hip and spine and she is protected from spinal fractures. However, the main menopausal symptom is not prevented and may be increased. This adverse effect has been reported elsewhere for Raloxifene: "The most common general side effects were hot flashes (25%) and leg cramps (6%). Hot flashes or flushes tended to occur during the first six months of therapy" (Drugs.com, 2009). Other side-effects reported elsewhere are:

> Severe allergic reactions (rash; hives; itching; difficulty breathing; tightness in the chest; swelling of the mouth, face, lips, or tongue); abnormal vaginal bleeding; back or side pain; blurred vision, loss of vision, or other vision changes; breast pain, tenderness, swelling, lump or discharge; chest pain; coughing up blood; confusion; flu-like symptoms; leg or calf pain, warmth, or swelling; one-sided weakness; shortness of breath. (Drugs.com, 2009)

Further, the textbooks state that there is 'increased risk of venous thrombolic events similar to estrogen' (Text 8, p.462). These 'events' are deep vein thrombosis, pulmonary embolism, and retinal vein thrombosis, all of which can be life threatening. The risks of breast cancer or cardiovascular disease for Raloxifene are not reported in the textbooks suggesting research in this area is ongoing.

This search for knowledge is represented in the current textbooks as a 'story'. This story begins with uncertainty about how the system works: "although a complete understanding of the underlying control mechanisms of bone remodelling is not yet known, there are a number of exciting new insights" (Text 3, p.805). These insights come from molecular science where "although much remains unknown, recent advances in elucidating the molecular biology of bone have provided intriguing new hypotheses in the pathogenesis of osteoporosis" (Text 6, p.1283). This molecular level is seen in the appearance of 'cytokines' (cell molecules) in explanations: "The effects of estrogen on bone mass are mediated by cytokines" (Text 6, p.1284); and "*The hypoestrogenic effects are attributable in part to augmented cytokine production"* (Text 3, p.806, italics in original). Kumar et al (2007) explain that the "story begins with" a receptor activator in the cell called RANK and it involves "novel members of the tumor necrosis factor (TNF) receptor family" that influence bone resorption (Text 3, p.805). The "story" that follows in this textbook is an example of what Rose (2007) has identified as the 'new entities' of molecular neuroscience in the neurochemical discourse: "receptor sites…ion channels…docking and discharge, receptor regulation, receptor blockade, receptor binding" (p.200). Kumar et al (2007) include "cell surface receptors", "ligands" "colony-stimulating factors", "bone-crunching osteoclasts" and "decoy-receptors" in their molecular representation of osteoporosis (Text 3, p.805). These space-age war-like metaphors are perhaps an update to those 'body at war' body metaphors shown by Emily Martin (1994) in her analysis of immunity discourses. In this research Martin also showed the genderised male dominant metaphors of molecular descriptions in medical texts. This is similarly seen here where the SERMs "are believed to act competitively on the [alpha] estrogen receptor and may function as competitive inhibitors in tissues with [alpha] receptors. SERMs can also bind to [beta] estrogen receptor complexes…" (Text 8, p.462). As noted earlier, Lawrence and Bendixen (1992) found that male anatomy was represented as the norm for the human body in medical anatomy textbooks. This cultural gendering of ostensibly objective, scientific

texts is continued here with socio-cultural 'alpha male' masculinity metaphors used in the representation of osteoporosis.

This story is also an illustrated one. Rose (2007) cites the "conventionalised visual simulations" of "molecular specificity" as a way in which the neurochemical discourse makes visible the realities of its 'truths' (p.200). These "simulations" include the typical lock and key symbols that Fleck (1939/1979) identified for the theoretical specificity of immunology. The current textbooks use this "vividness" for bone remodelling by showing a range of typical illustrations. A typical diagram, as shown in Figure 2, displays drawings of the spine, breast, heart and uterus with descriptions of the SERMs acting on them (Text 8, p.462). A typical illustration shown in Figure 3 portrays osteoblast bone cells with protruding ligand (binding) molecules as round shapes fitting into the receptor molecules (cut-out-half circles) of the osteoclast bone molecules (Text 6, p.1276).

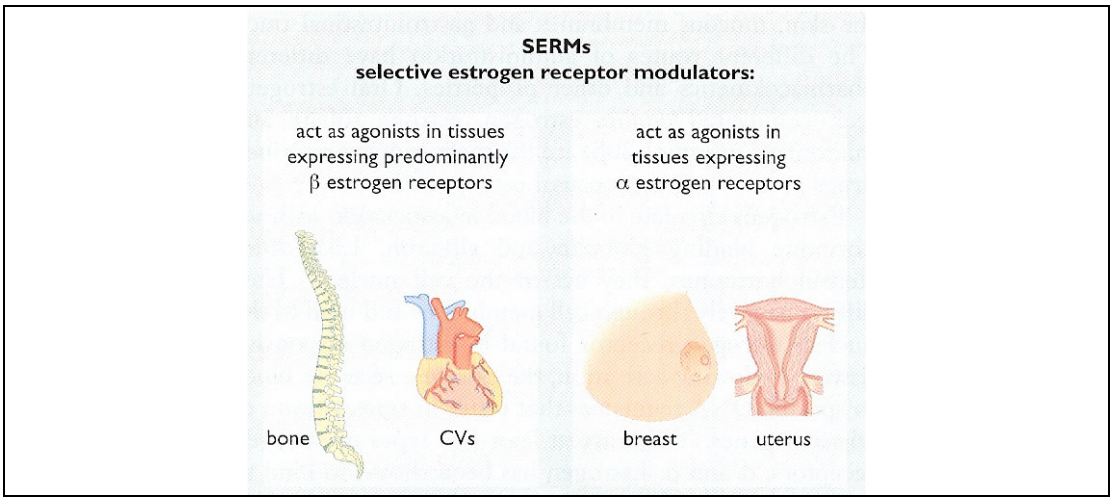

Figure 2: Effects of SERMs on estrogen tissues, taken from Page, Curtis, Walker and Hoffman, 2006, p.462

Figure 4 shows a typical basic illustration with estrogen receptor modulators portrayed as jigsaw pieces that fit into the estrogen receptor cut-out jigsaw pieces (Text 1, p.781). Another typical diagram is shown in Figure 5. This portrays the pathophysiology of osteoporosis by labelling ageing and menopause as separate entities. The ageing entity lists decreased osteoblast cells, osteoprogenitor cells, growth factors and reduced physical activity as key determinants whereas the menopause entity lists members of the neurochemical family: "serum-estrogen" and "IL-1, IL-6, TNF levels, expression of

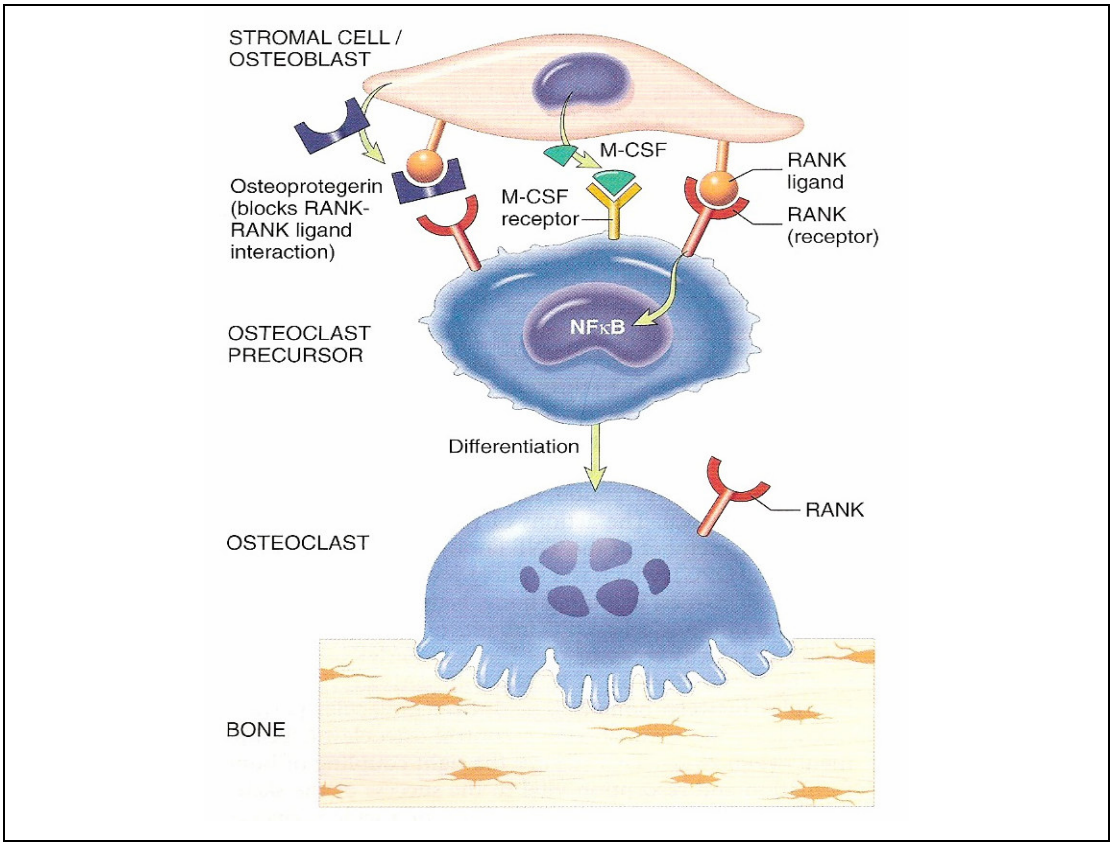

Figure 3: Paracrine molecular mechanisms that regulate osteoclasts, taken from Kumar, Abbas and Fausto, 2005, p.1276

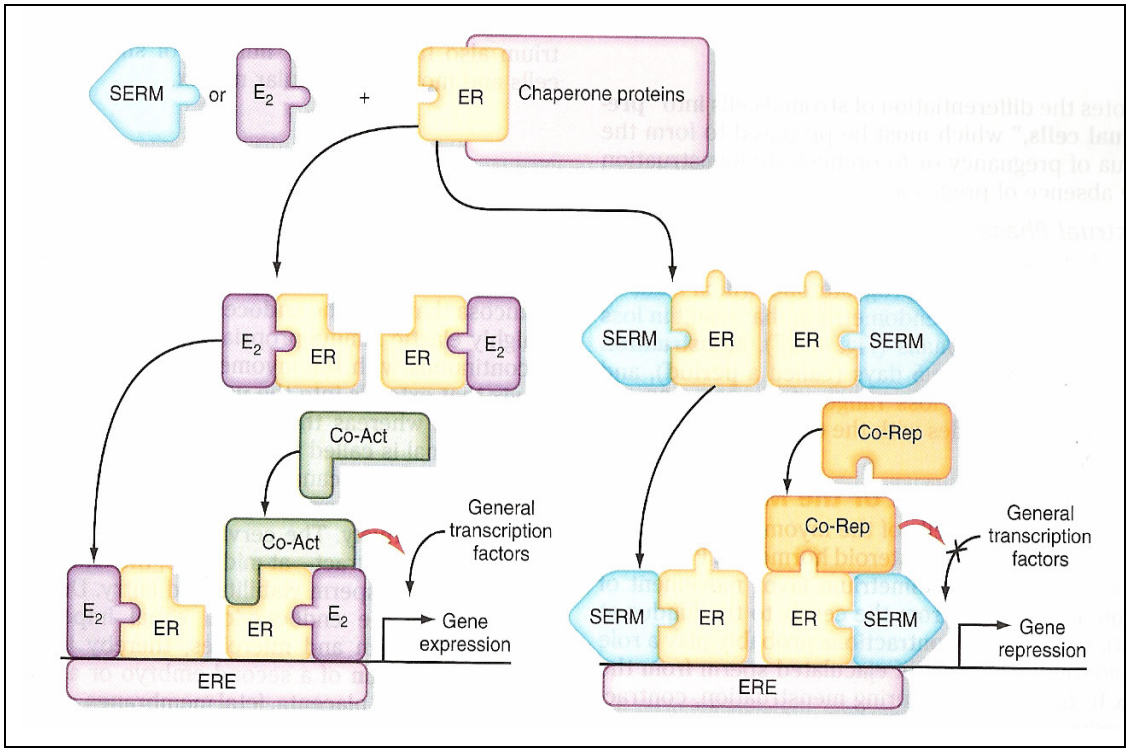

Figure 4: Molecular mechanisms of estrogen receptors, taken from Koeppen and Stanton, 2008, p.781

RANK, RANKL" (Text 3, p.806). Fleck (1939/1979) also highlights this technique of "vividness" or "pictorial quality" as used by "an expert who wants to render an idea intelligible to others" but he warns that "what was initially a means to an end acquires the significance of a cognitive end" (p.117). This means that a visual aid such as a diagram used to explain a concept can become reified as an explanation. Pictures of binding and locking molecules and the naming of them as members of the neurochemical family portrays a neurochemical causation for bone remodelling that is really only one of many theories for this process.

What does this new discovery discourse mean for women? This discourse frames its new knowledge as 'exciting discoveries' and women in menopause therefore are positioned as the recipients of these advancements. However, there is an imperative for drug therapy for menopause that is fuelled by the pharmaceutical companies mandate to colonise new frontiers of what constitutes our bodies, health and behaviour (Conrad, 2007). In terms of Fleck's truth community, the relationship of the pharmaceutical industry and the medical practitioners is seen in this tribute to the pharmaceutical companies by Katzung (2007): "These entities deserve great credit for making possible many of the therapeutic advances that we enjoy today…..these companies are uniquely skilled in exploiting discoveries from academic and governmental laboratories and translating these basic findings into commercially successful therapeutic breakthroughs" (Text 7, p.3). Given that companies like Wyeth earn billions of dollars per annum from their sales of Premarin to women all over the world, it is vital that women are aware of their positioning as the 'postmenopausal consumer'.

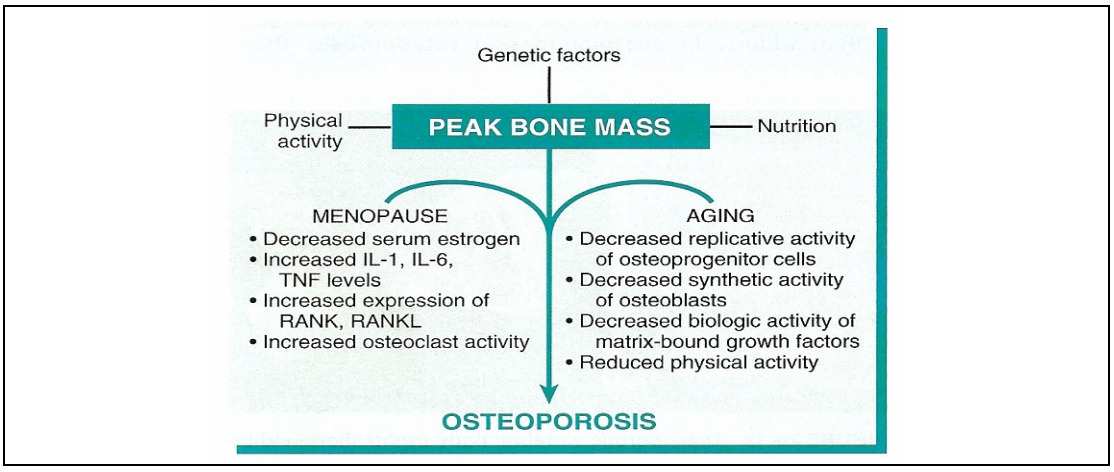

Figure 5: Pathophysiology of postmenopausal and senile osteoporosis, taken from Kumar, Abbas, Fausto and Mitchell, 2007, p.806

Chapter 5: Summary and Conclusions

In terms of Martin's (1987) findings, the current textbooks continue, over 20 years later, to represent menopause as a failure - a failure in the 'breakdown of control in a hierarchical system' and a failure in the purpose of production (to give birth) and more specifically, a failure in the production of estrogen. The consequence of this failure to produce estrogen is that it leads to both psychological and physical symptoms which frame the woman as a 'patient' in need of treatment. Framing of the patient as needing treatment for specific symptoms of menopause such as hot flashes and psychopathological problems goes beyond menopause, and it is at this point that the current research results step beyond Martin's (1987) findings. They demonstrate that in the current textbooks, the failure to produce estrogen leads to diseases of the breast and genitals, and particularly of the bones. This 'estrogen deficiency as disease' discourse continues to frame women as patients in need of treatment, but with the inclusion of bones the stakes are now much higher. The patient now has a major disease – postmenopausal osteoporosis. This construction of the 'postmenopausal patient' implies that *all* women will have this disease, and should therefore be treated with HRT. The 'HRT saviour' discourse constructs two patients, the 'menopause patient' (women with menopause symptoms) who needs 'short term' treatment and the 'postmenopausal patient' (all women) who needs to be saved from having her bones dissolve rapidly due to estrogen deficiency. However, HRT is now a very conditional saviour given the results of the WHI clinical trials which identify the health risks associated with its use.

The 'HRT saviour' discourse is complex and includes serious risks to the health of women from HRT which makes it a very conditional saviour. The 'estrogen as deficiency' discourse provides the rationale that these risks must be negotiated because HRT is promoted as the protection from diseases of the breast, bone and genitals. However, results also demonstrated an 'obscurity' discourse where the causation of diseases from estrogen deficiency and the benefits of HRT are not known, obscure, and an ongoing project. The ongoing search for a 'cure' for postmenopausal osteoporosis now overshadows treatment of menopausal symptoms. This is evidenced in the 'new discovery' discourse where next generation HRTs are now being prescribed and used for the treatment of the 'postmenopausal patient'. These new cures, which seek to overcome the WHI health warnings, are generated through molecular medical science.

This discourse represents the more advanced molecular based HRT similar to a 'neurochemical selves' discourse (Rose, 2007), in which advanced medical technology enables us to scan our bodies, and therefore explain our 'selves', at a finer molecular level. However, there is a drug imperative for using HRT for menopause and this underpins the 'new discovery' discourse. For women in menopause, we must question who benefits from exposing the interior of their bodies – their bones - and framing the picture as one of frightening decay. Despite the focus on patient risk for diseases potentially caused by HRT, the end goal of the rationale for HRT therapy appears to be to give HRT to the 'postmenopausal patient' at all costs to save her bones. We must also question why the therapeutic rationale for women in menopause involves an individual risk assessment of diseases to be managed and avoided? Roberts (2007) also questions why "contemporary medical science is unable to provide coherent or uncontested evidence regarding the risks and benefits of HRT for menopausal women In acknowledging this problem, medical scientists gesture towards hopeful futures and perfect drugs but also, 'in the meantime' (as if there will be a time in which 'the facts' are indisputable), place the burden of responsibility on individual women to make decisions about which medications they wish for to take" (p.146).

These research findings demonstrate that we need to shift the focus from menopause as biological with reproductive and sexual changes to menopause as a socio-cultural phenomenon. Rather than focus on physiological changes, women have emphasised concerns such as their social status, quality of relationships, health and sexual history when they explore their interpretations of menopause (Winterich, 2003). This shift in focus would locate menopause within a women's health paradigm, much like the New View Campaign which challenges the medical and pharmaceutical medicalisation of women's sexuality (Kaschak & Tiefer, 2002). The New View Manifesto of women's sexual problems defines a 'sexual problem' as discontent related to emotional, physical or relational experiences which may emerge within the context of medical, psychological, partnership and socio-cultural, political or economic factors (New View Campaign, 2009). Menopause can be located within this framework, not as a sexual problem, but as a women's bodily transition that is shifted from the biomedical arena and contextualised within the complexity of a shared women's health discourse. This in turn moves social constructions like menopause and its consequent health-threatening HRT use into the political realm where it can be openly and collectively debated.

These research findings also demonstrate that a shift is needed is in the way we represent menopause and HRT in medical literature. Martin regards her work as a "modest force in the world" but it has changed the way millions of women read about themselves in popular books like *Our Bodies, Ourselves* (Boston Women's Health Collective, 2005) and the few college biology textbooks where the metaphors of women's bodies have been re-written (Kirschner, 1999). This research suggests that these medical textbooks should also re-write their metaphors of menopause. This is not just a matter of changing the words, but enabling the medical profession to recognise the negativity inherent in their taken-for-granted knowledge of menopause and HRT and the harmful impact this has for women's interpretations of their bodily experiences. An example of a change in the way menopause can be represented is seen in the way it is framed as a 'restoration':

> After approximately 30 years of menstrual cycles in order to provide transitory fertility, ovarian serenity is restored, estrogen once again becomes stable and levels return to normal, menstruation ceases. The women experiences release from reproductive pressures and is able to participate fully in her career, social and family activities as she need no longer be concerned about the problems associated with menstruation, birth control and pregnancy and is no longer at a heightened risk for endometriosis, uterine fibroids, and breast cancer. (Gannon, 1999, p.68)

Limitations & Future Research

Martin has admitted the limitation of her focus on metaphors in the *Women in the Body* to the exclusion of broader political economic forces (Kirschner, 1999) and this research also does not go beyond the medical textbooks to engage directly with the medical 'truth community', the doctors, gynecologists, medical students, research scientists, government health bodies, health agencies and pharmaceutical companies that are active agents in biomedical discourses. A critical area of engagement at this level is the communication of information about women's health. There is a wealth of information in the community about women's sexual and reproductive lives – puberty, pregnancy, childbirth – but scant attention is given to menopause (Buchanan et al, 2001). Therefore, research that focuses on the health promotion of menopause as a biological

and a social phenomenon and how it is linked to HRT is needed. There are many audiences that this information can be communicated to in the community including colleges, health clinics, medical centres, medical schools, and future research could address the impact of this communication in changing perceptions of what menopause means in women's *and* men's lives. Men's interpretations of the meanings of menopause for the women in their lives, and for women in general, requires further exploration. Dillaway (2008) found that the biomedical model of menopause was strengthened in intimate family interactions because men encouraged their female partners to seek help for their 'symptoms'. Future research in this area could provide further information such as how men are complicit with or resist the biomedical model of menopause for women and how other discursive constructions of menopause influence the relationships between mid-life men and women. This research argues for a shift in focus to a socio-cultural menopause, therefore it is important that men's relationships with women in the context of menopause are explored further.

This research also does not give a 'voice' to the medical students themselves, the recipients of the medical knowledge that is represented in the textbooks. Medical students are exposed to not only overt formal medical training but also to the implicit normative influences embedded in the culture of their profession. A study of the first and second year Otago Medical School students, one of the medical schools used in this research, showed that the students acknowledged a "slipping into the culture" of the medical profession through "subconscious and very powerful" influences (Jaye et al, 2006, p.149). Therefore, it would be informative for future research to explore the ways in which menopause and HRT are represented, or absent, within the context of medical students' informal socialisation. It would also be informative to explore whether medical students are resistant to any dominant biomedical framing, not just menopause.

A further limitation of this research is that women's own articulations of their interpretations of menopause and HRT are not included. Some women have been vocal in their resistance to the biomedical model of menopause and they resist the use of HRT as an immoral weakness or an unnatural and unhealthy remedy (Stephens et al, 2004). However, future research should explore to what extent the WHI warnings have changed women's interpretations of and resistances to HRT. This research is critical because the HRT warnings have been set within controversy and women's knowledge

of them is conflicted, as is medical knowledge. A recent retrospective study of the WHI trial found no significance difference in the incidence of diagnosed lung cancer for women in the combined HRT group and women in the placebo group but significantly more women died from lung cancer in the combined HRT group (Chlebowski et al, 2009). This finding returns us to the warning of Butenandt, a biochemist who first synthesised estrogen in the 1940s, that hormones under certain conditions are carcinogenic because they make cells divide (Seaman, 2003, p.23). This suggests that the HRT warnings will never cease.

Personal Reflexivity

Although I approached this research as an academic with a critical social science lens I also approached it as a woman and as a woman the personal impact of this experience has been immense. I am a woman in the menopause transition and I began to experience hot flashes as I was reading about them in the textbooks so in a sense I was fully embodied in my research. Medical students too report experiencing the diseases they are learning about with such prevalence that 'medical student's syndrome' is a common diagnosis for them (Baars, 2001). At first, I was shocked at the negativity of the metaphors used for menopause, then I became confused as to why a normal physiological bodily process was being associated with severe diseases, and finally I was outraged that women were being prescribed a drug that was shown to cause cancer. As I worked towards uncovering the structure and dynamics of this knowledge I felt *my* body was decayed and decrepit despite looking in the mirror and seeing the same 'me' and despite knowing the discursive complexity of menopause. I am familiar with the representation of older women in my society and am intelligently and emotionally resilient towards it but the decay and disease in the text and images of the textbooks has seeped into my personal consciousness. In this respect these representations are powerful because it is about my mortality. At the other extreme, way out beyond the material body, I have lost a sense of fixity, of concreteness about the world; it seems like my female identity is 'made up', it gazes back at me not only from the modern media but from artworks throughout history and it is a male construction. From paintings of voluptuous renaissance nudes through to the thinnest modern haute couture women I now see images that are passive and about sexual availability and know why I have always felt uncomfortable looking at them. Therefore, as a woman I am disrupted,

conflicted and challenged by this research, yet as a critical social science researcher I now know how important it is to challenge the established continuities.

Conclusion

In conclusion, this research contributes to the field of women's health by critically examining the paradoxes surrounding menopause, and in doing so, presents new knowledge to women about menopause and HRT. These paradoxes emerge from the dominance of the medical profession which medicalises women's healthy bodies and renders them unwell during menopause and then offers to save them from this illness with a drug that may endanger their health. This research is located within the critical discursive resistances to biomedical representations of menopause and HRT and provides a further challenge by directly engaging with the 'truths' of scientific medicine in its medical textbooks. This social constructionist engagement has extended the cultural analysis of the biomedical model of menopause from the 'failed body of production' twenty years ago to the present day 'failed and diseased body' still needing treatment. In its critical examination of this treatment, HRT, this research has extended the knowledge of how the biomedical model has included recent clinical trial evidence and warnings into its 'truth community' and the implications of this for women's interpretations of menopause and their HRT use. This critical examination of the biomedical model of menopause has also extended knowledge of its discursive complexity by demonstrating that scientific representations of menopause and HRT are imbued with obscurity and ongoing discoveries that disrupt its authority over women's bodies. This research has also contributed to the ongoing post-feminist critique of the biomedical model of menopause by attempting to shift menopause and HRT from the biomedical arena into the bio-social realm where there is space to collectively debate their meanings within a shared women's health discourse.

References

Abraham, S., Llewellyn-Jones, D., & Perz, J. (1994). Changes in Australian women's perception of the menopause and menopausal symptoms before and after the climacteric. *Maturitas, 20,* 121-128.

Baars, B.J. (2001). *In the Theater of Consciousness: The Workspace of the Mind*. US: Oxford University Press.

Ballard, K. D., Elston, M.A., & Gabe, J. (2009). Private and public ageing in the UK: The transition through the menopause. *Current Sociology, 57(2),* 269-290.

Beauvoir, Simone de. (1993). *The second sex*. London: David Campbell Publishers.

Bertero, C. (2003). What do women think about menopause? A qualitative study of women's expectations, apprehensions and knowledge about the climacteric period. *International Nursing Review, 50,* 109-118.

Birnbaum, D. (1990). Self-Help for Menopause: A Feminist Approach. *Annals of the New York Academy of Sciences, 592,* 250-255.

Bluming, A.Z. (2004). Hormone replacement therapy: the debate should continue. *Geriatrics, 59,* 30-37.

Boughton, M., & Halliday, L. (2008). A challenge to the menopause stereotype: young Australian women's reflections of 'being diagnosed' as menopausal. *Health and Social Care in the Community, 16*(6), 565-572.

Brown, C., Matthews, K. A., & Bromberger, J. (2005). How do African American and Caucasian women view themselves at midlife? *Journal of Applied Social Psychology, 35(10),* 2057-2075.

Buchanan, M.C., Villagran, M.M., & Ragan, S.L. (2001). Women, Menopause, and (Ms.)Information: Communication about the Climacteric. *Health Communication, 14*(1)*,* 99-119.

Burger, H.G. (2006). Hormone therapy in the WHI era. *Australian and New Zealand Journal of Obstetrics and Gynaecology, 46,* 84-91.

Burr, V. (1995). *An introduction to social constructionism.* London: Routledge.

Butler, J. (1990). *Gender Trouble.* NY: Routledge.

Casper, M.J., & Moore, L.J. (2009). *Missing bodies - The politics of visibility*. NY: New York University Press.

Chlebowski, R.T., Schwartz, A.G., Wakelee, H., Anderson, G., Stefanick, M., et al. (2009). Oestrogen plus progestin and lung cancer in postmenopausal women

(Women's Health Initiative trial): a post-hoc analysis of a randomised controlled trial. *The Lancet, 374,*1243-1251.

Coney, S. (1994). *The menopause industry: A guide to medicine's 'discovery' of the mid-life woman.* New Zealand: Penguin Books.

Conrad, P. (2007). *The medicalization of society – On the transformation of human conditions into treatable disorders.* Baltimore: John Hopkins University Press.

Cordes, C.L., & Dougherty, T.W. (1993). A review and an integration of research on job burnout. *Academy of Management Review, 18*(4), 621-656.

Coupland, J., & Williams, A. (2002). Conflicting discourses, shifting ideologies: Pharmaceutical, 'alternative' and feminist emancipatory texts on the menopause. *Discourse & Society, 13,* 419-445.

Datamonitor (2009). Datamonitor Research Store (*Wyeth: positive Aprela results bode well for planned portfolio strategy*). Retrieved February 15, 2010, from http://www.datamonitor.com/store/News/wyeth_positive_aprela_results_bode_well_for_planned_portfolio_strategy?productid=E551E990-69AB-444B-877B-30023DB06579.

Deecher, D.C. (2009). Thermoregulation and menopause: Understanding the basic pathophysiology of vasomotor symptoms. *The menopausal transition: Interface between gynecology and psychiatry, 175,* 77-87.

Dijkstra, A.F., Verdonk, P., & Lagro-Janssen, A.L.M. (2008). Gender bias in medical textbooks: examples from coronary heart disease, depression, alcohol abuse and pharmacology. *Medical Education, 42, 10,* 1021-1028.

Dillaway, H. (2008). "Why Can't You Control This?" How women's interactions with intimate partners define menopause and family. *Journal of Women and Aging, 20* (1/2), 47-64.

Drugs.com.(2010). Drugs A to Z (*Raloxifene side effects*). Retrieved February 15, 2010, from http://www.drugs.com/sfx/raloxifene-side-effects.html.

Equine Advocates (2009). The Issues (*Premarin*). Retrieved February, 16, 2010, from http://www.equineadvocates.org/issues/premarin/premarin.html.

Feyerabend, P. (1993). *Against method* [3rd ed.]. London: Verso.

Fleck, L. (1979). *Genesis and development of a scientific fact.* London: University of Chicago Press.

Formanek, R. (1990). *The meanings of menopause: historical, medical and clinical perspectives.* Hillsdale, NJ: The Analytic Press Inc.

Foucault, M. (1989). *The archaeology of knowledge.* UK: Routledge.

Gannon, L., & Stevens, J. (1998). Portraits of menopause in the mass media. *Women and Health,27,* 1–15.

Gannon, L.R. (1999). *Women and ageing: Transcending the myths.* London: Routledge.

Gergen, K. (1999). *An invitation to social constructionism.* London:Sage.

Germov, J. (Ed.). (1999). *Second opinion: An introduction to health sociology.* NY: Oxford University Press.

Greer, G. (1991). *The change: women, ageing and the menopause.* London: Penguin Books.

Grosz, E. (2005). Refiguring bodies. In M. Fraser & M. Greco (Eds.). *The Body: A Reader* (pp.47-51). NY: Routledge.

Guyton, A.C. (1981). *Textbook of medical physiology* (6thth ed.). Philadelphia: Elsevier Saunders.

Guyton, A.C. (1986). *Textbook of medical physiology* (7thth ed.). Philadelphia: Elsevier Saunders.

Guyton, A.C., & Hall, J.E. (2006). *Textbook of medical physiology* (11th ed.). Philadelphia: Elsevier Saunders.

Harris, M.C. (2008). Aging women's journey toward wholeness: New visions and directions. *Health Care for Women International*, *29,* 962–979.

Hunter, M.S., & O'Dea, I. (1997). Menopause: Bodily changes and multiple meanings. In J. Ussher (Ed.). *Body Talk: The material and discursive regulation of sexuality, madness and reproduction* (pp.199-222). London: Routledge.

Huston, S.A., Jackowski, R.M., Kirking D.M. (2009). Women's trust in and use of information sources in the treatment of menopausal symptoms. *Women's Health Issues, 19,* 144-153.

Hvas, L. & Gannik, D.E. (2008). Discourses on menopause - Part I: Menopause described in texts addressed to Danish women 1996-2004. *Health, 12,* 157-175.

Hvas, L. & Gannik, D.E. (2008). Discourses on menopause - Part II: How do women talk about menopause? *Health, 12,* 177-192.

Jaye, C., Egan, T., & Parker, S. (2006). 'Do as I say, not as I do': Medical education and Foucault's normalizing technologies of self. *Anthropology & Medicine, 13*(2), 141-155.

Jones, H. W., & Jones, G. S. (1981). *Novak's textbook of gynecology* (10th ed.). Baltimore: Williams and Wilkins.

Kaschak, E., & Tiefer, L. (2002). *A new view of women's sexual problems.* NY: Routledge.

Katzung, B.G. (2007). *Basic & clinical pharmacology* (10th ed.*)*. USA: McGraw-Hill.

Katzung, B.G., Masters, S., & Trevor, A. (2009). *Basic & clinical pharmacology* (11th ed.*)*. USA: McGraw-Hill.

Kaufert, P.A., & Lock, M. (1997). Medicalization of women's third age. *Journal of Psychosomatic, Obstetrics & Gynecology, 18*(2), 81-86.

Kirschner, S.R. (1999). From flexible bodies to fluid minds: An interview with Emily Martin. *Ethos, 27*(3), 247-282.

Koeppen, B.M., & Stanton, B.A. (Eds.). (2008). *Berne & Levy physiology* (6th ed.). Philadelphia: Mosby Elsevier.

Komesaroff, P., Rothfield, P., & Daly, J. (Eds.). (1997). *Reinterpreting meno pause: Cultural and philosophical issues.* NY: Routledge.

Komm, B. S. (2008). Review article: A new approach to menopausal therapy: The tissue selective estrogen complex. *Reproductive Sciences, 15*(10), 984-992 .

Krieger N., Lowy I., Aronowitz R., Bigby, J., Dickersin, K., et al. (2005). Hormone replacement therapy, cancer, controversies, and women's health: historical, epidemiological, biological, clinical, and advocacy perspectives. *Journal of Epidemiological Community Health, 59,* 740–748.

Kuhn, T. (1962). *The structure of scientific revolutions*. US: University of Chicago Press.

Kumar, V., Abbas, A.K., & Fausto, N. (2005). *Robbins and Cotran pathologic basis of disease* (7th ed.). Philadelphia: Saunders Elsevier.

Kumar, V., Abbas, A.K., Fausto, N., & Mitchell, R.N. (2007). *Robbins basic pathology* (8th ed.). Philadelphia: Saunders Elsevier.

Lawrence, S.C., & Bendixen, K. (1992). His and hers: Male and female anatomy in anatomy texts for United States medical students, 1890-1989. *Social Science and Medicine, 35, 7,* 925-934.

Lawton, B., Rose, S., McLeod, D., & Dowell, A. (2003). Changes in use of hormone replacement therapy after the report from the Woman's Health Initiative: cross sectional survey of users. *British Medical Journal, 327,* 845-846.

Lock, M. (1998). Menopause: Lessons from Anthropology. *Psychosomatic Medicine, 60,* 410-419.

Lyons, A. (2000). Examining Media Representations: Benefits for Health Psychology. *Journal of Health Psychology, 5*(3), 349-358.

Lyons, A.C., & Griffin, C. (2003). Managing menopause: a qualitative analysis of self-help literature for women at midlife. *Social Science & Medicine, 56,* 1629-1642.

MacLennan (2007). Viewpoint: HRT: a reappraisal of the risks and benefits. *The Medical Journal of Australia, 186*(12), 643-646.

Martin, E. (1987). *The woman in the body.* Buckingham: Open University Press.

Martin, E. (1994). *Flexible bodies: The role of immunity in American culture from the days of polio to the age of AIDS*. Boston: Beacon Press.

Meyer, V. F. (2001). The medicalisation of menopause: Critique and consequences. *International Journal of Health Services, 31*(4), 769-792.

Moghaddam, F. (2005). *Great ideas in psychology: A cultural and historical introduction.* Oxford: Oneworld Publications.

Moynihan, R., & Cassels, A. (2005). *Selling sickness: How drug companies are turning us all into patients.* NSW, Australia: Allen & Unwin.

National Health Lung and Blood Institute (2010). Women's Health Initiative (*WHI in dbGAP*). Retrieved February, 15, 2010, from http://www.nhlbi.nih.gov/whi.

National Women's Health Network (2008a). Women's Health Activist July/August 2003: Feature Article (*The backlash against the WHI: Myths and facts about menopause hormone therapy*). Retrieved February 15, 2010, from http://www.nwhn.org/newsletter/options/print.cfm?info_id=323.

National Women's Health Network (2008b). Menopause Hormone Therapy and Cardiovascular Protection (*Does age matter? What we know so far about age of initiation of hormone therapy and heart disease in women)***.** Retrieved February 15, 2010, from http://www.nwhn.org/healthinfo/detail.cfm?info_id=10&topic=Fact%20Sheets.

Nelson, H.D. (2008). Seminar: Menopause. *Lancet, 371,* 760-770.

New View Campaign (2009). Challenging the medicalisation of sex (*The new view manifesto*). Retrieved February 15, 2010, from http://www.fsd-alert.org/manifesto3.asp.

New York State Department of Health (2003). Osteoporosis *(FDA-Approved medications for osteoporosis prevention and/or treatment*). Retrieved February 15, 2010, from http://www.health.state.ny.us/diseases/conditions/osteoporosis/fda.htm.

Page, C.P., Curtis, M.J., Walker, M.J.A., & Hoffman, B.B. (2006). *Integrated pharmacology* (3rd ed.). Philadelphia: Mosby Elsevier.

Panda, S.N. (2002, January). *Selective estrogen receptor modulators.* Paper presented at the 45th All India Congress of Obstetrics and Gynaecology. Retrieved February

15, 2010, from http://www.obgyn.net/educational-tutorials/educational-tutorials.asp?page=/english/pubs/features/presentations/panda6/panda6-ss

Parker, I. (1992). *Discourse dynamics: Critical analysis for social and individual psychology.* London: Routledge.

Perz, J., & Ussher, J.M. (2008). "The horror of this living decay": Women's negotiation and resistance of medical discourses around menopause and midlife. *Women's Studies International Forum, 31,* 293–299.

Pharmac (2005). *Annual Review.* Retrieved February 15, 2010, from http://www.pharmac.govt.nz/2005/11/10/ARev05.pdf

Prentice, R. L., & Anderson, G. L. (2007). The woman's health initiative: lessons learned. *Annual Review of Public Health, 29,* 131-150.

Reeves, G., Beral, V., Green J., Gathani, T., & Bull D. (2006). Hormonal therapy for menopause and breast-cancer risk by histological type: a cohort study and meta-analysis. *Lancet Oncology,* 7, 910-918

Richter, J.M. (1997). Menopause in different cultures. *Journal of Psychosomatic, Obstetrics & Gynecology, 18,* 73-80.

Roberts, C. (2002). A matter of embodied fact: Sex hormones and the history of bodies. *Feminist Theory, 3*(1), 7-26.

Roberts, C. (2007). *Messengers of sex: Hormones, biomedicine and feminism.* NY: Cambridge University Press.

Roberts, H. (2007). Hormone replacement therapy comes full circle: Further evidence on the early benefits and late risks does not change advice to menopausal women. *British Medical Journal, 335,* 219-220.

Roberts, H., & Lethaby, A. (2003). Revised key messages for hormone replacement therapy from the New Zealand Guidelines Group. *Australian and New Zealand Journal of Obstetrics and Gynecology, 43,* 273–279

Rose, N. (2007). *The politics of life itself - Biomedicine, power, and subjectivity in the twenty-first century.* New Jersey: Princeton University Press.

Rosewarne, L. (2006, July). *Advertising and ageism: The politics of the invisible older woman. Equality and diversity in the third age.* Paper presented at the British Psychological Society National PSIGE Conference. Retrieved February, 15, 2010, from http://www.bps.org.uk/media-centre/press-releases/releases$/psige-section/invisible.cfm

Rotosky, S.S., & Travis, S.B. (1996). Menopause research and the dominance of the biomedical model 1984-1994. *Psychology of Women Quarterly, 20,* 285-312.

Rubin, R., & Strayer, D.S. (2008). *Rubin's pathology: Clinicopathologic foundations of medicine* (5th ed.). Philadelphia: Lippincott Williams & Wilkins.

Seaman, B. (2003). *The greatest experiment ever performed on women.* NY: Hyperion.

Shea, J. L. (2006). Chinese women's symptoms: relation to menopause, age and related attitudes. *Climacteric, 9,* 30-39.

Shore, G. (1999). Soldiering on: An exploration into women's perceptions and experience of menopause, *Feminism & Psychology, 9*(2), 168-178.

Sim, S., & Van Loon, B. (2001). *Introducing critical theory.* US: Totem Books.

Soules, M.R., Sherman, S., Parrott, E., Rebar, R., Santoro, N., et al. (2001). Executive summary: Stages of reproductive aging workshop. *Journal of Women's Health and Gender-based Medicine, 10*(9), 843-848.

Stampfer, M.J., Colditz, G.A., Willett, W.C., et al. (1991). Postmenopausal estrogen therapy and cardiovascular disease. Ten-year follow-up from the nurses' health study. *New England Journal of Medicine*, *325,* 756–762.

Statistics New Zealand Tatauranga Aoteoroa (2010). Population (*Age and Sex*). Retrieved February 15, 2010, from http://www.stats.govt.nz/methods_and_services/access-data/TableBuilder/2006-census-pop-dwellings-tables/population/age-and-sex.aspx.

Stephens, C., & Breheny, M. (2008). Menopause and the virtuous woman: the importance of the moral order in health. *Health, 12*(1), 7-24.

Stephens, C., Carryer, J., & Budge, C. (2004). To have or to take: Discourse, positioning, and narrative identity in women's accounts of HRT. *Health*, *8*, 329-350.

Sybylla (1997). Situating menopause within the strategies of power. In P. Komesaroff, P. Rothfield & J. Daly (Eds.). *Reinterpreting meno pause: Cultural and philosophical issues* (pp.200-221). NY: Routledge.

The Boston Women's Health Collective (2005). *Our bodies, ourselves.* NY: Touchstone.

Tomlinson, B. (1999). A rhetoric of medical anthropology. *Medical Anthropology Quarterly, 13(1),* 7-31.

Topo, P. (1997). Climacteric hormone therapy in medical and lay texts in Finland from 1955 to 1992. *Social Science and Medicine, 45*(5)*,* 751-760.

Trademarkia (2010). Pharmaceutical Products *(Aprela)*. Retrieved February, 15, 2010, from http://www.trademarkia.com/aprela-77324812.html.

Turner, B.S. (1992). *Regulating bodies: Essays in medical sociology.* London: Routledge.

Underwood, J.C.E. (Ed.) (2004). General and Systematic Pathology (4th ed.). Philadelphia: Churchill Livingstone.

US National Institutes of Health (2010). ClinicalTrials.gov (*ELITE: Early versus late intervention trial with estradiol*). Retrieved February, 15, 2010, from http://clinicaltrials.gov/ct2/show/results/NCT00114517.

Vainionpaa, K.J., & Topo, P. (2005).The making of an ageing disease: the representation of male menopause in Finnish medical literature. *Ageing and Society, 25*(6), 841-861.

Whittaker, R. (1998). Re-framing the representation of women in advertisements for hormone replacement therapy. *Nursing Inquiry, 5,* 77-86.

Willig, C. (2001). *Introducing qualitative research in psychology – Adventures in theory and method.* Buckingham: Open University Press.

Wilson, R.A. (1966). *Feminine forever.* London: W.H. Allen.

Winterich, J. (2003). Sex, menopause and culture: Sexual orientation and the meaning of menopause for women's sex lives. *Gender and Society, 17*(4), 627-642.

Winterich, J. A., & Umberson, D. (1999). How women experience menopause. The importance of social context. *Journal of Women & Ageing, 11*(4), 57-73.

Women's Health Initiative Participant Website (2008). Findings (*Health risks and benefits 3 years after stopping randomized treatment with estrogen and progestin).* Retrieved February 15, 2010, from http://www.whi.org/findings/ht/eplusp_3yr.php.

Wren, B.G. (2009). The benefits of oestrogen following menopause: Why hormone replacement therapy should be offered to postmenopausal women. *The Medical Journal of Australia, 190,* 321-325.