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A rhetorical tale: neurochemistry and the efficacies of antidepressants in Canada

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ABSTRACT

[English]

Recent work in anthropology has speculated on how developments in molecular biology and medicine might bring about new bodies, selves, and forms of sociality. This thesis explores how herbal and pharmaceutical antidepressants differently affect experiences of one's neurochemistry. It does so in two ways. First, it outlines the historical 'social life' of pharmaceutical antidepressants, including their co-production with depression, and the neurochemical body in a particular style of reasoning in biological psychiatry. Second, it presents and analyzes claims made for the efficacy of antidepressants made in vernacular North American books, advertisements, and pamphlets. Although the claims for both herbal and pharmaceutical antidepressants allude to the same realms of value – those of science, nature/history, and personal experience – their different social lives enable different access to the neurochemical body.

[Français]

Des études anthropologiques récentes imaginent l'impact – sur le corps, le soi, et la social – des dévelopments de la biologie moléculaire. Ce mémoire présente les études portant sur la question des antidépresseurs et leurs effets sur les expériences «neurochimiques» du corps en Amérique du Nord. Le mémoire se penche, en utilisant un genre de raisonment de la psychiatrie biologique, sur comment la «vie sociale» des antidépresseurs, dans le contexte de leur co-production avec la dépression et l'efficacité, pourrait faciliter ce corps «neurochimique». Comment des antidépresseurs non-pharmaceutiques, tel le millepertuis commun, conditionnent également les expériences des corps neurochimiques? Ce mémoire contraste la légitimisation – a travers les «régimes de valeur» de la science, de la nature/historie, ou de l'expérience personelle – des antidépresseurs pharmaceutiques et phytothérapeutiques dans les livres et annonces populaires. Les différentes «vies sociales» offrent des entrées distinctes au corps neurochimique.

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CHAPTER 1
INTRODUCTION

`But I'm *not* a serpent, I tell you!' said Alice. `I'm a--I'm a--'

`Well! *what* are you?' said the Pigeon. `I can see you're trying to invent something!'

`I--I'm a little girl,' said Alice, rather doubtfully, as she remembered the number of changes she had gone through that day.

`A likely story indeed!' said the Pigeon in a tone of the deepest contempt. `I've seen a good many little girls in my time, but never *one* with such a neck as that! No, no! You're a serpent; and there's no use denying it. I suppose you'll be telling me next that you never tasted an egg!'

`I *have* tasted eggs, certainly,' said Alice, who was a very truthful child; `but little girls eat eggs quite as much as serpents do, you know.'

`I don't believe it,' said the Pigeon; `but if they do, why then they're a kind of serpent, that's all I can say.'

This was such a new idea to Alice, that she was quite silent for a minute or two, which gave the Pigeon the opportunity of adding, `You're looking for eggs, I know *that* well enough; and what does it matter to me whether you're a little girl or a serpent? '

–from *Alice's Adventures in Wonderland* by Lewis Carroll [1988]

A tale of antidepressants, biology, and values

This tale will explore the claims made in vernacular[1] North American books, advertisements, and pamphlets about how antidepressants 'work'. I will focus on the claims made for an herbal antidepressant, St. John's Wort, to provide a useful foil for the claims made for pharmaceutical antidepressants. For this project I have relied mostly on unobtrusive observation, namely archival research. This is by no means a simple story – there are many characters, settings, and plots – and the claims are often as incomparable as those that Alice and the Pigeon make about little girls and serpents in *Alice's Adventures in Wonderland.*

This tale was prompted by a friend's explanation of how he knows that he has forgotten to take his antidepressant medication. While gesturing around his head, he explained that "my neurons just stop firing … everything slows and there is just no connection. I know it sounds weird, but I can actually feel it."

This experience of one's neurochemistry also appears in published narratives of depression. Elizabeth Wurtzel concludes in her book *Prozac Nation* that "after an accumulation of life events made my head such an ugly thing to be stuck in, my brain chemistry started to agree" (1995: 345 *in* Fee 2000: 85). This is mirrored in a 1997 issue of *TIME*, for which the cover features the image of a terrified man, lacking the top of his head (implying direct access to the brain) with a pipette dropping a liquid, which the caption tells us is a 'mood drug.'

> One week later [after beginning antidepressants] I felt fully restored and resigned myself to a humbling new self-image: *neurochemical robot.* I felt like one of those cutaway human heads used in TV commercials for decongestants. In the process I became highly sensitive to the play of my mental chemicals.... I see a grumpy or irritable person and estimate his or her serotonin level. It's an odd way

[1] I am using historian Katherine Pandora's term 'vernacular' instead of the terms 'popular' or 'lay' culture to circumvent the (belittling) associations of these latter words. Pandora uses the term "to refer to the common, everyday forms of communication and activities that mark a culture;" this might include commercial culture (2001: 492).

> to view the world, or oneself, and I wonder sometimes if I'll ever really get used to it" (Kirn 1997: 62).

I cannot feel my neurons, nor do I experience my social world in terms of neurochemicals. But I wonder – would this be different if I had experienced a crippling depression and used antidepressant medications to help overcome the depression? This paper is thus an exploration of antidepressants and the experiences of the body – especially the brain – they might facilitate. To be more precise, it is an exploration of antidepressants and the *expression of* the experiences of the body. Instead of focussing on individual narratives, however, I will present the claims made for the efficacy of antidepressants in vernacular documents, and contemplate how these claims might facilitate particular embodiments. Antidepressants may be effective both in helping people overcome depression, but also in effecting new experiences of body.

A tale of anthropological interest

This tale might be of anthropological interest for several reasons. One reason is the recent presentation of depression as a worldwide major health burden, and as such, the study of depression and its treatment is culturally relevant. For example, the World Health Organization (WHO) recently published a book entitled *Depression: Social and economic timebomb* (Dawson and Tylee 2001), and estimated that by 2020 depression will be the second leading cause of disability (Ustun and Chatterji 2001). In Canada, a study found that stress, depression and tiredness were among the most frequently reported health problems by women (Walters and Denton 1997). In a Quebec newspaper it was stated that in Quebec the prescriptions for antidepressants in 2001 numbered approximately three million (Leduc 2002); according to the Nonprescription Drug Manufacturers Association of Canada, St. John's Wort was used by about 7.4% of the Canadian population during the height of its popularity in 1999 (Vallis 2002). All of these statements lead me to believe that depression is perceived as relevant, and thus is an interesting site of anthropological investigation.

Another reason why this tale might be of interest to medical anthropology is that it might add to the anthropology of pharmaceuticals (Van der Geest and Whyte 1989). Although it is not uncommon to show how the efficacy of a particular treatment is rationalized in the context of a particular medical tradition (Van der Geest and Whyte 1989; also see chapter two), vernacular explanations of the efficacy of antidepressants have not been a focus of anthropological attention. A final reason that this study is relevant to medical anthropology is that it is a study of medical pluralism[2]. Just as Alice and the Pigeon have different ways of knowing and making claims about serpents, the ways of knowing about (and making claims about) herbal and pharmaceutical antidepressants are not always commensurable.

The role of a medical anthropologist might include seeking "the forms and sources of knowledge in medical cultures as well as their historical roots and development, distribution, legitimacy, validation and accountability" (Gaines 1991: 241). This thesis is indeed about 'the forms and sources of claims of efficacy made about antidepressants' that is structured around the 'historical roots' of antidepressants as well as their 'development, continued legitimacy, and validation. It is divided into four chapters.

Signposts for the journey: Chapter Overviews

In this thesis, I have chosen to experiment with the format of a story of a journey – it is my attempt to liven up an analysis. The scene unfolds in North America, specifically Canada. The two characters are pharmaceutical and herbal antidepressants. This tale will follow them, and their rather busy 'social lives' (Appadurai 1986; 'social history' in Kopytoff 1986); in other words, for the purposes of analysis, this tale traces the history and interactions of antidepressants as if they were people with social lives (Appadurai 1986). This permits me to present possible reasons why these antidepressants enable particular experiences of human biology. The first part explores the social life of pharmaceutical antidepressants as a means of placing in context how these

[2] As Judith Farquhar points out, the idea of 'medical pluralism' is one that prioritizes the particular epistemological standpoint of biomedicine and does not reflect the dynamism and adaptation of traditions. However, in the claims that are presented in chapter three, I argue that this 'medical pluralism' is often the

antidepressants might facilitate an experience of the body in terms of neurobiology. Specifically, I examine the early social life of pharmaceutical antidepressants and the co-production of particular notions of depression, biology and efficacy. The second part is an exploration of the social life of another type of antidepressant: an herbal antidepressant. I will comment on how the social life of this herbal antidepressant enables it to reproduce *and* circumvent these particular notions of depression, biology, and efficacy.

Setting: The co-production of depression and antidepressants

While Alice's journey leaves her more than a little disoriented on a couple of occasions, I hope that the journey on which I guide you has a clear itinerary. Our first section of the journey is similar to the first experiences of Alice in Wonderland. At first there is no question of who Alice is – she is a girl lying by the side of a river. However, after crawling through the rabbit tunnel, Alice falls and floats downwards, ending in a hallway – in which the only door is not suitable for her current size. She finds that she can change her size with different substances, such as the contents of the bottle labelled 'drink me' and the cake saying 'eat me'. Eventually she succeeds in leaving the hallway. However, during these changes, she despairs – "Who am I then?" and decides that if she doesn't like who she is she'll "stay down here until I'm somebody else". Similarly, at first there is little question of what depression is, at least according to its 'official' definition in Canada. This chapter presents this definition, as one of many possible ones lying about, but then explores other entities and definitions that may involve alternate bodies, experiences, and identities, thus bringing the definition into question. This chapter will take the form of a literature review of recent constructionist studies of depression in four different disciplines: history, anthropology, sociology, and psychology.

The chapter will conclude with speculation on the extent to which this definition, with its neurobiological associations, facilitates experiences of neurobiology in depression. I will mention current theories about the implications of the biology of selves in this 'official'

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way that claims are made about different types of antidepressants in Canada, and is one that is currently being institutionalized in Canadian governmental regulations.

definition of mental disorders such as depression (including Rose in press, 2001; Rabinow 1996), however, I will also present the findings from various studies in North America, Britain, and Australia to explore to what extent these neurobiological theories influence the experience of the body (Jadhav m.s.; Gammell and Stoppard 1999; Karp 1992, 1993; Scattolon 1999). As the landscape seamlessly but drastically changes as the ever-changing body of Alice moves through Wonderland, additions to the landscape may drastically alter the picture we have of depression at any point in time.

A note on definitions of depression

In this chapter, I am dealing with a rather specific definition of depression, namely the 'official' definition in the current edition of the *Diagnostic and Statistical Manual of Mental Disorders*. However, depression is an incredibly complex misfortune and has many complex definitions[3]. Indeed, with the plethora of associations in the historical development of depression, melancholia, and acedia, it is no wonder that a single definition of depression has not been unequivocally established in Britain and North America. However, because my interest is in antidepressants, and depression only to the extent that this depression is involved with antidepressants and particular biologies, I focus on the depression that is co-produced with antidepressants in English-language psychiatry in the second half of the 20th century.

Characters: Antidepressants, St. John's Wort, and the trusty armchair

While chapter two explores antidepressants in terms of their production *in* biological psychiatry, chapter three explores claims made for the efficacy of antidepressants *outside* biological psychiatry. I will present and analyse the claims made for the efficacy of antidepressants, which at times seems as logical as the conversations at the table of the March Hare, since it is a tangle of *if, how,* and *why* antidepressants manage depression. After exploring the concept of 'efficacy,' I will then compare and contrast the claims made for the efficacy of pharmaceutical and herbal antidepressants in vernacular documents that aim to invoke 'science.' Differences between the types of claims made

about pharmaceuticals and this herbal antidepressant will be framed in terms of their differential access, because of their different social lives, to different 'regimes of value' (Appadurai 1986:4). Thirdly, I explore other types of value that are invoked in these claims, which I argue are mostly related to either tradition/nature or personal experience. Finally, I will explore how these three regimes of value, namely science, tradition/nature, and personal experience, are juxtaposed. While these regimes of value might have internal consistency (Schutz 1973 [1945])[4], passing from one to the other entails a sort of 'shock' – there is no algorithm to translate experiences from one regime to the other, as each regime has specific forms of self-experience, sociality, and time perspective among other things (Schutz 1973 [1945]: 230). The different regimes of value with their own organization of meaning are incommensurable in Kuhn's sense of the term. Given that a particular biology is associated with depression and pharmaceutical antidepressants, what types of understanding of the body might be associated with other regimes of value?

A brief example of the claims made about St. John's wort

Take for instance an 'informational pamphlet' such as this one for St. John's Wort shown in Figure 1, which can be found at health food stores, bulk herb stores, and regular pharmacies. Because the pamphlet is essentially a marketing tool, I believe that I can safely assume that everything in this pamphlet is intended to imbue the product with value. These pamphlets (and the packaging on the products they extol) almost invariably have some of the printing in green, or pictures of plants or other 'natural' landscape features. There may also be references to the sun, such as "this product is made with sunshine to lift your spirits!" In the pamphlets, there are usually several types of claims. For instance, there is usually some claim to ancient knowledge, a history of the herb, a reference to pertinent medical studies, a reference to a neurotransmitter, and a quote or reference to some biomedical authority. I will argue that there are three themes that recur in these claims for herbal antidepressants: that of science, nature/tradition, and personal experience.

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[3] One division of these different referents that is commonly referred to in anthropology is Kleinman and Good's statement that depression may refer to a common emotion or mood, a symptom, and also an illness (1985: 2).

Taking Flora's St John's Wort Oil Extract

Suggested Use

- Take three to five capsules throughout the day with liquid, or as recommended by a qualified health care practitioner.
- Positive results are often noticed within two to eight weeks. Results vary from person to person.
- For therapeutic and occasional use only.

Considerations

- Traditionally extracted St John's Wort Oil has an excellent safety profile, and no significant side effects have been reported.
- As St John's Wort can cause drowsiness, avoid intake of alcoholic beverages, and take extra care when driving or engaging in activities requiring alertness. It may be better to take the larger part of the dose in the evening so as not to interfere with these activities.
- When exposing yourself to the sun for long periods of time wear sunscreen and a hat, and know your personal limit. Photosensitivity through taking St John's Wort extracts has been reported only at many times above our recommended dosage.
- St John's Wort Oil extract should not be used when pregnant or breast-feeding.
- People under medical supervision, or already taking a prescription antidepressant drug should check with a qualified health care practitioner before starting any new treatment, including St John's Wort.

Call toll-free 1-888-436-6697 to find out more about Flora's St John's Wort Oil Extract

You Can't Argue With Results!

At Flora we are extremely proud of our traditional St John's Wort Oil Extract. We continue to hear amazing success stories from people who use our healing oil, and the results really do speak for themselves.

I...have become a total convert to [Flora's] St John's Wort Oil. I have had tremendous success taking it internally for periods of stress and overwork and resulting stomach upsets, and externally for rough skin and sunburn...

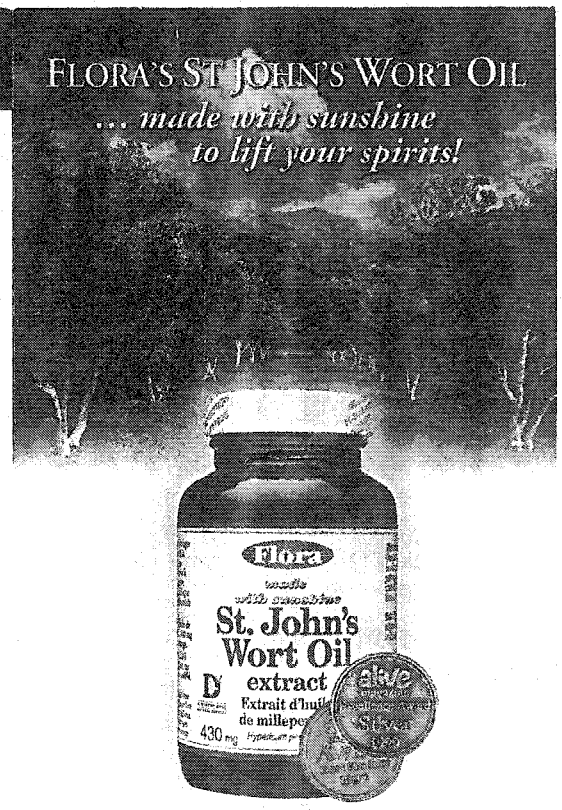
– M.H., Hornby Island, BC

Dear Flora, I was ready to put my husband into a nursing home until my daughter brought me some of your St John's Wort Oil Extract to give him. It was a miracle! But then I switched to a cheaper brand and within days I saw what a difference it made in the results. I marched straight to our local health store and bought your brand again, and since then my husband is better all the time. I don't think I'll have to resort to a nursing home now. His health is 50% better and we hope it will be 100% better before long. Thank you for your company's excellent grade of St John's Wort Oil. It's a miracle.

– V.M., BC

"I'm a professional massage therapist, and I've been using [Flora's] St John's Wort Oil for two years now in my practice... J.F. [is] an...example – a mountain biker with chronic knee pain – nothing else had ever worked before, but St John's Wort Oil Extract loosened it up, de-swelled (sic) it and relieved the discomfort. I myself use the oil for the tendonitis and occasional frozen shoulder that are occupational hazards of my trade, and it really does heal quickly. I also recommend it for any kind of physical over-exertion and as a wonderful calming backrub before bed!"

– S.J.A., Oxford, ON



"Flora's St John's Wort Oil Extract is of the highest quality, wild-crafted and extracted in an optimal manner. I find it to be the most consistently effective treatment for my patients who are suffering from depression."

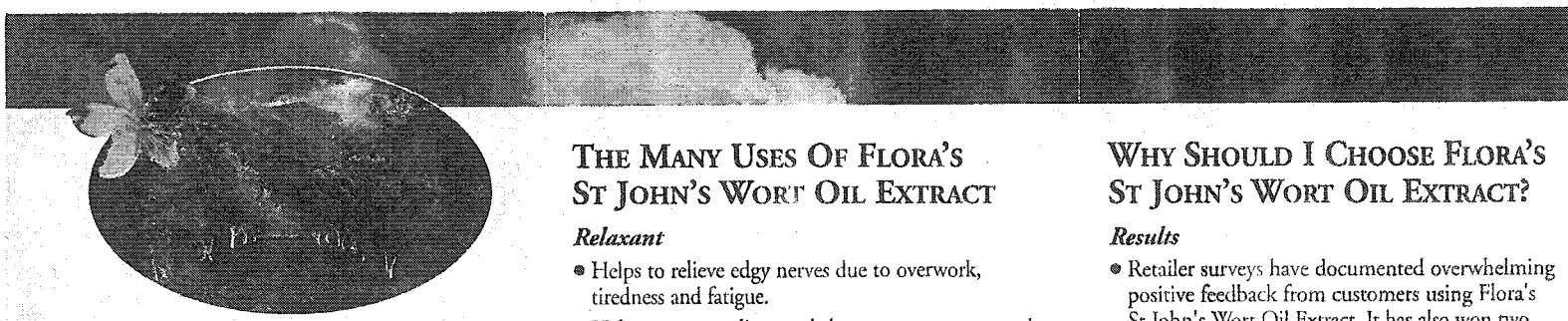
– Carolyn DeMarco, MD

Flora

Quality Health From God's Pharmacy
www.florahealth.com • 1-888-436-6697

12/99

Figure 1: 'Informational' pamphlet for Flora's St. John's Wort Oil



The Intriguing Story Of Flora's St John's Wort Oil Extract ...

In the sun-drenched fields nestled among the Peleponnese mountains of Greece, there is a traditional way of making St John's Wort Oil extract. It's a way that uses time-honoured natural methods, uncompromising standards and the healing energy of the sun.

The mature golden flowers of wild grown St John's Wort plants are harvested manually in the mornings, and are submersed that day in extra virgin olive oil, cold-pressed from freshly picked olives of certified organic origin. This mixture, sealed in glass jars, is then potentized for several weeks in the intense Greek sun of midsummer.

It is this slow, painstaking process alone which maximizes the extraction of the plant's full spectrum of active ingredients into the oil – not just one! This traditional mode of preparing St John's Wort Oil extract is recorded as early as 1618 in the first publication of the British Pharmacopœia.

The extraordinary healing properties of this kind of St John's Wort Oil extract have been valued and praised since antiquity. It is the oil Flora is proud to offer you, and which satisfied customers and doctors recommend.

The Many Uses Of Flora's St John's Wort Oil Extract

Relaxant

- Helps to relieve edgy nerves due to overwork, tiredness and fatigue.
- Helps to return disrupted sleep patterns to normal[1].

Nervine Tonic & Restorative

- St John's Wort extract is now recognized by the medical community as being effective in treating mild to moderate depression[2, 3, 4, 5].

Immune Function

- Current studies indicate antiviral, antibacterial and antimicrobial properties of the compounds that are oil-extracted from St John's Wort[5,6].
- For the relief of a sore throat swallow a teaspoon of oil every three to four hours.

Digestive Anti-inflammatory

- Can be used internally to soothe ulcerated membranes, nervous bowels and gastritis.

Topical Applications

- Use as a massage oil for relief from neuralgia, sciatica, muscle and joint pain and inflammation.
- Soothes irritated skin, bruises, mild burns and especially sunburn.
- Traditionally used for dry skin, diaper rash and chafing.
- Antibacterial properties may reduce healing time.

References:

1 Reuter, H.D., (pers comm)

2 British Medical Journal (August 3, 1996)

3 Linde et al. 1996

4 Wheatley et al. 1997; Vorbach et al. 1997

5 Reuter, H.D. 1997

6 American Herbal Pharmacopœia – St John's Wort Monograph

Why Should I Choose Flora's St John's Wort Oil Extract?

Results

- Retailer surveys have documented overwhelming positive feedback from customers using Flora's St John's Wort Oil Extract. It has also won two Alive awards for Best Herbal Supplement.

Potency

- St John's Wort plants grown in hot, dry climates such as Greece are known to contain higher levels of important active constituents.
- Traditional oil extracts contain important flavonoids that increase over time in the sun extraction process, and which are the key to the action of the total extract.
- Flora's St John's Wort Oil Extract is made with flowers gathered in the mornings of midsummer, when sunlight is the most intense and their active principles are highest.

Doctor Recommended

- Dr. Carolyn DeMarco is a leading Canadian medical doctor committed to natural medicine. Dr. DeMarco states that Flora's St John's Wort Oil Extract is the most consistently effective treatment for her patients who are suffering from depression.

Availability

- Flora's St John's Wort Oil Extract is available in soft gel caps for internal use, and in liquid for both internal and external use. It is also available as a Body Oil for massaging, fragrantly scented with ylang-ylang.

A note on method

One obvious limitation to this archival research is that, although the sources listed above have wide and varied distribution and prominence, I have little way of knowing if the content of these sources are representative of or even influential in the illness narratives of any group of people with depression. I might assume that these are representative of middle-upper class North Americans; however this would simply be speculation. So rather than frame this research in terms of a specific group, I will simply position the author(s) of the document, and talk about the vernacular portrayal of the efficacy of one herbal antidepressant – St. John's Wort – and pharmaceutical antidepressants. Please consider the picture that I will paint as the work in progress that it is.

Plots and subplots: webs and placebos

This story is meant to prepare me for future work in the area of the anthropology of the body and science in the context of depression and its treatment, so many of the conclusions in this last chapter are tentative, and ones that I might follow in future research. This section draws many conclusions about the efficacies of antidepressants.

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[4] These different regimes of value might be usefully interpreted in terms of Schutz's notion of provinces of meaning (1973 [1945]).

CHAPTER 2

THE CO-PRODUCTION OF DEPRESSION AND ANTIDEPRESSANTS

> Can you imagine a world in which, effectively, there was no such thing as depression? (Healy 1997: 4)

Outline of this chapter's journey

In this chapter, I set the scene for the analysis in chapter three by presenting the current 'official' construction of depression in Canada. My two purposes for writing this chapter are interwoven throughout. One is to present the co-production of a particular depression, antidepressants, efficacy, in the context of a style of reasoning in biological psychiatry. The second is to undermine the idea that depression, as constructed in this way, is inevitable by telling the tales of times and lands with other variously-named distress associated with alternate bodies, experiences, and identities. These two purposes use some terminology that requires some clarification: 'style of reasoning' and 'construction'.

A note on 'style of reasoning'

I will use the term 'style of reasoning' to present the co-production of depression and antidepressants. This term is drawn from Fleck's 'thought-style' (1979 [1935]), which has subsequently been reshaped into Hacking's 'style of reasoning' (1992b), Young's 'style of reasoning' (1995), and Rose's 'style of thought' (in press). There are several things that should be mentioned about a style of reasoning. It is an attribute of a collective, not of the psyche of an individual. The collective, or 'thought collective' to use Fleck's terminology, in this particular case is that of biological psychiatry. Also, a 'style of reasoning' is not cognitive in the everyday sense of 'thought' or 'reasoning' but rather includes the materials, narratives, and institutions that are involved with the production of 'facts', as well as the affective domain of the individuals involved (Fleck 1979: 49; Daston 1995: 5; Young 1995). In other words, a style of reasoning "is a way of seeing, a way of explaining, a way in which reasoning [that] is embedded within certain practical and intellectual techniques, conventions about experimentation, about instruments and inscriptions, measurements and model systems" (Rose *in press*). A style

of reasoning is self-authenticating because "it develops a body of types of theory and types of apparatus and types of analysis that are mutually adjusted to one another" (Hacking 1992a: 30). A style of reasoning is also self-vindicating because once this "structurally complete and closed system of opinions has been formed, it offers enduring resistance to anything that contradicts it" (Fleck 1979: 27); failed hypotheses might be vindicated by the development of an auxiliary hypothesis, or by a change in instrumentation or analysis (Hacking 1992a: 30, Young 1995).

Indeed, Hacking argues that objects are inseparable from the style of reasoning in which they are produced; furthermore, in this production, these objects reshape the style of reasoning (Hacking 2002). Thus, we can argue that depression and antidepressants are co-produced (Healy 1997) by a particular style of reasoning in biological psychiatry, which is one that is increasingly operating on a molecular level (Rose *in press*); in turn, depression and antidepressants change the bodies and selves recognized by and produced in biological psychiatry's style of reasoning:

> the neurochemical brain becomes known in the very same process that invents interventions to manipulate its functioning – that is to say, therapeutic drugs (Rose *in press*).

A note on construction

The organization of this chapter is loosely shaped by Ian Hacking's model of different levels of commitment to constructionism (1999). Hacking argues that a work can be considered 'constructionist' if it argues against the inevitability of the status quo – usually, but not always, concomitant with an argument that the status quo is harmful:

> Social Construction work is critical of the status quo. Social constructionists about *X* tend to hold that:
>
> (0) In the present state of affairs, *X* is taken for granted; *X* appears to be inevitable
>
> (1) *X* need not have existed, or need not be at all as it is. *X*, or *X*, as it is at present, is not determined by the nature of things; it is not inevitable.
>
> Very often they go further, and urge that:
>
> (2) *X* is quite bad as it is.

(3) We would be much better off if *X* were done away with, or at least radically transformed (Hacking 1999: *amalgamation of* 6 and 12)

I will use this typology, replacing Hacking's *X* with 'depression,' to structure a literature review of the construction of depression[5]. I first present the 'official' definition of depression in North American psychiatry, as Hacking's precondition, i.e. statement 0 above. In one study of Canadian women's depressive experiences, the women used depression in a way that was similar to that given in the DSM-IV (Stoppard 2000). As mentioned in the introduction, there are many different definitions for depression, and this one is no more taken for granted than are others. However, in many of the documents analyzed in chapter 3, this definition of depression is used more than any other (see Figure 2), which probably has to do with my chosen actors, antidepressants. I present this definition first, because as Fleck claims of the definition of syphilis, "[i]t is only after the choice [of definition] has been made that the associations produced by it are seen as necessary" (1979: 8).

I will then review some historical studies that document possible precursors, also called proto-ideas (Fleck 1979 [1935]) or traces (Rheinberger 1997), of depression. I will also review some possible analogies for depression in other cultures, drawn from anthropological case studies. Sometimes these histories and anthropological case studies were written to illustrate the universalism of depression while other times they were written in order to undermine this universalism. Although there are many purposes for historical and anthropological studies related to depression, I will focus on those that help us imagine other possibilities and question the inevitability of depression.

Some studies however, go on to state that 'depression is quite bad as it is.' This is where I have ambiguous feelings, because although I think in some respects depression is not the best way of dealing with complex misfortune and suffering, it is one way that may be

[5] I argue depression is culturally constructed – or co-produced with antidepressants in a particular style of reasoning – but I am clearly not arguing against the reality and the suffering of depression. Likewise, using an example relevant to our imaginary journey, arguing that my boots have been constructed in no way diminishes their reality and their impact on my experiences. I am instead trying to follow the reasoning of Allan Young in his work on Post-Traumatic Stress Disorder, and attempting to show how depression has been made real (1995: 6) and also how it is sustained as real.

Figure 2: 'Informational' leaflet from Montreal family doctor's office

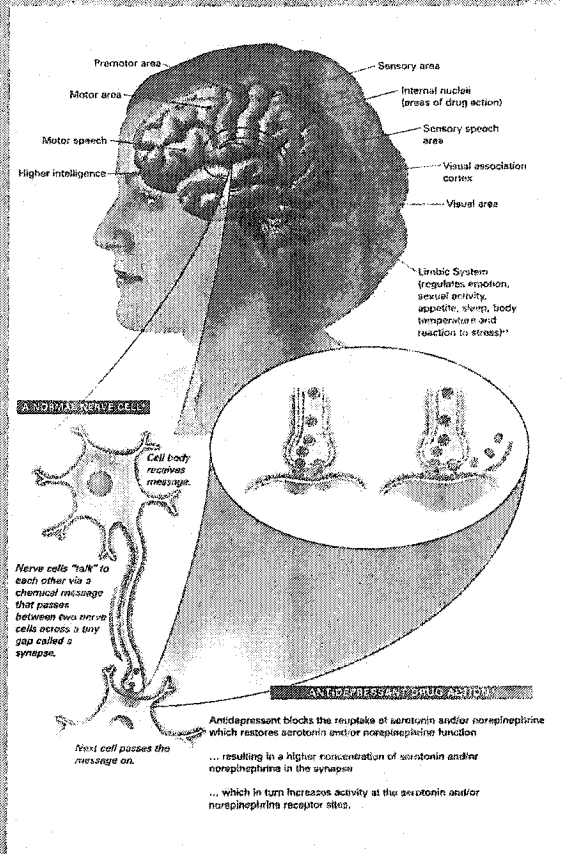
Understanding depression

Depression is very common, affecting 5 to 20 per cent of the population.[1]

The average age of onset for major depression falls between the ages of 20 and 40 years.[2]

What is depression?

- Depression is not about "fault." Depression is a treatable medical illness. It is a neurochemical disorder that can be triggered by external events.[1]
- If you have a depressive illness, you're not alone. Between 5% and 20% of people will suffer from depression at some time in their lives.[1]
- Depression is usually the result of an interaction of biological, psychological and social factors[1], which may include:
  - family history
  - changes in the levels of brain chemicals called neurotransmitters, specifically serotonin and norepinephrine
  - physical illnesses and the side effects of medications used to treat them
  - stressful or traumatic life events (such as the death of a loved one or the breakup of a marriage)



Are there different types of depression?

There are several different types of depression-related mood disorders[1]:

- ***Organic mood disorders*** are often associated with medical illness or drug reactions
- ***Bipolar disorders*** are marked by episodes of both depression and mania (which causes elevated mood, irritability, overactivity, poor judgement and other symptoms)
- ***Major depression*** causes a depressed mood and/or a loss of interest in usual activities, plus other symptoms
- ***Dysthymia*** is similar to major depression, but causes fewer and less severe symptoms
- ***Adjustment disorder*** with depressed mood involves depressive symptoms following crucial life events

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Do you have depression? [2,7]

S	*Sleep*	Have you been sleeping a lot more or less than usual?
A	*Appetite*	Has your appetite changed or have you gained or lost a lot of weight?
D	*Depressed Mood*	Do you feel sad or hopeless?
I	*Interest*	Have you lost interest or pleasure in all or most of your daily activities?
F	*Fatigue*	Do you feel tired and have no energy nearly every day?
A	*Agitation/Retardation*	Do you feel anxious, restless or "slowed down" nearly every day?
C	*Concentration*	Do you have trouble concentrating or making decisions?
E	*Esteem*	Do you feel worthless or guilty and have a feeling that life is not worth living?
S	*Suicidal Thoughts*	Do you have dark thoughts or think about death or suicide attempts?

If you have 5 out of 9 of these symptoms for more than 2 weeks (1 symptom must be depressed mood or loss of interest), you may be suffering from depression.

What is the treatment?

Various kinds of psychotherapy may be used in the treatment of a depressed person. Some common types are:

- **cognitive-behavioural therapy, in which you try to recognize and correct inaccurate and negative impressions of yourself and your circumstances, and try to change behaviours linked to depression[8]**
- **interpersonal therapy, in which you work to develop new, more effective ways of handling interpersonal relationships linked to your depressive symptoms[4]**

There are a number of different types of medications now available for the treatment of depression, all of which correct the level of various neurotransmitters in the brain. These drugs include:

- **tricyclic antidepressants (TCAs): drugs that work on serotonin and/or norepinephrine and other receptor sites[5]**
- **selective serotonin reuptake inhibitors (SSRI's): drugs that work on serotonin only[7]**
- **serotonin norepinephrine reuptake inhibitors (SNRI's): are newer drugs that work on both serotonin and norepinephrine[6]**

Notes

For more unbiased information and support contact:

- **DIRECT (Depression Information Resource & Education Centre) Toll-free 1-888-567-5051 ext. 3000 or www-fhs.mcmaster.ca/direct**
- **Your Doctor**
- **CANMAT (Canadian Network for Mood and Anxiety Treatments) www.canmat.org**

Please consult prescribing information for important safety information before prescribing. PAAB

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useful for some people. According to Hacking's terminology, I have an 'ironic' level of commitment to the construction of depression, in that I'm not sure what can be done about depression right now. I suppose in some ways I am also an 'unmasker,' because to the extent that I think depression is a bad thing, I am attempting to undermine it by presenting some of the functions that it might serve. However, many of the studies critiquing depression go beyond this level of commitment. Because of the precondition with which I am working, that of the American Psychiatric Association's (APA's) definition of depression in the fourth edition of their *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) (1994), most of the studies and critiques presented are dealing with the current biopsychiatric associations of depression, as opposed to psychological or social associations. These studies are critical for many reasons, whether it be for issues of neo-colonialism (Jadhav 1996), cultural inappropriateness (Kleinman and Good 1985), lack of concern for gender (Stoppard 2000), or (over)medicalization (Healy 1997).

0. Introduction to the DSM-IV

The most recent version of the DSM is the fourth edition published in 1995, although there was a text revision in 2000. The definitions of various mental disorders in this manual are the 'official' working definition for most mental health workers in North America, used for research, clinical, forensic, statistical, and insurance purposes (Kutchins and Kirk 1997).

Major depressive disorder

What is commonly referred to as 'depression'? In bestselling books, such as the New York Times best seller *The Noonday Demon: An Atlas of Depression* (Solomon 2001) that will be analysed in chapter 3, 'depression' tends to be indexed to the 'major depressive disorder' in the 'mood disorders chapter' of the DSM-IV[6]. This chapter begins with an overview of the 'building blocks' of the mood disorders, the depressive, manic,

[6] Although he begins his book with the statement "Depression is the flaw in love" (Solomon 2000: 15), and berates the DSM-IV for "ineptly defin[ing] depression as the presence of five or more on a list of nine symptoms" (20), he makes use of the DSM-IV's definition explicitly in other parts of the book (25), in his own narrative of depression (Chapter 2), and refers to these categories throughout the book.

mixed and hypomanic *episodes*. The depressive *episode* does not constitute a diagnosis of depression, but is a precondition for the diagnosis of major depressive *disorder*:

> The essential feature of a Major Depressive Episode is a period of at least 2 weeks during which there is either depressed mood or the loss of interest or pleasure in nearly all activities.... The individual must also experience at least four additional symptoms drawn from a list that includes changes in appetite or weight, sleep, and psychomotor activity; decreased energy; feelings of worthlessness or guilt; difficulty thinking, concentrating, or making decisions, or recurrent thoughts of death or suicidal ideation, plans, or attempts. To count toward a Major Depressive Episode, a symptom must either be newly present or must have clearly worsened compared with the person's pre-episode status. The symptoms must persist for most of the day, nearly every day, for at least 2 consecutive weeks. The episode must be accompanied by clinically significant distress or impairment in social, occupational, or other important areas of functioning. For some individuals with milder episodes, functioning may appear normal but requires markedly increased effort (APA 2000b: 349)

Seven pages reiterating and expanding upon each individual symptom, outlining associated features and disorders, laboratory findings, course, as well as 'specific culture, age, and gender features', follow this paragraph. Later in the chapter, the diagnosis of Major Depressive *Disorder* is described in relation to the Major Depressive *Episode*.

The spell of science and the definition of depression

This 'official' definition of depression involves more than the explicit statements of the DSM-IV; it also involves implicit elements. One of these implicit elements is that of being under the 'spell' of science, by which I mean that depression is marketed as a scientific term. I do not use 'spell' in a perjorative way, but use it in the sense of Walter Benjamin who contrasts the 'aura' of authenticity of the original work of art with the 'spell' of the generic. This 'spell' is related to the commodification of the work and its promotion in terms of advertising rhetoric (Benjamin 1968 [1936]: 231). Again, Figure 2 is an excellent example of the spell of science that takes the explicit definition of depression one step farther. As I will show later in this section, depression's past social

life, namely its being co-produced in the style of reasoning of biological psychiatry, enables this 'spell'. The scientific spell is developed through the positioning of depression in the style of reasoning of biological psychiatry: in laboratories, in research grant proposals, in clinical practice, and in other institutions (Kirk and Kutchins 1992).

There are also several rhetorical devices[7] in the DSM-IV that enable this spell of science. This 'spell' of science is related to American psychiatry's need, in the 1970s, to become more 'scientific' and research oriented in order to gain respectability, prestige and funding to survive as a profession in the United States (Kutchins and Kirk 1997). The first rhetorical device is that of referring to depression as a discrete disorder[8]. In the description of the Major Depressive *Disorder*, is it clear how the DSM-IV reinforces the idea of discrete disorders in at least three ways: the use of diagnosis, numbers, and statistical differentiation. First of all, the diagnosis itself might reify the idea of a discrete disorder (Mirowsky and Ross 1989: 11). In psychiatry, the infectious disease model means that mental disorder is conceptualized as a series of distinct disorders with clear and necessary criteria for inclusion (Mirowsky and Ross 1989). Finally, this discreteness is also reinforced in practice; an anthropologist remarks that

> [d]oing a lot of diagnoses ... and writing those admission notes tends to give one the sense that there *are* underlying essences that can be seen, named, and possibly controlled, even when the actual problem seems elusive and perplexing (Luhrman 2000: 44-45).

The second strategy is the use of a numbering system. Coding distress as a major depressive disorder, mild, single occurrence, and as 296.21 reinforces the separateness and concreteness of the disorders. Also, numbers are an indicator of the virtue 'objectivity' (Daston 1995), as they are frequently used as an indicator of such in scientific journals and popular discourse (Rose 1996). The third strategy does not lie in

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[7] I use the word rhetoric, not to make a negative judgement on the DSM-IV, but to highlight that this presentation of information, like every such presentation, has a particular way of communicating information that appeals to specific realms of value.

[8] The DSM-IV, in the introduction, has a statement to the effect that the definitions purposely do not specify any particular model of mental disorder: "there is no assumption that each category of mental disorder is a completely discrete entity with absolute boundaries dividing it from other mental disorders or from no mental disorder" (APA 1994: xxxi). The organization of the manual, however, implicitly reinforces the disease model (Kutchins and Kirk 1997; Mirowsky and Ross 1989).

the DSM-IV itself, but in the documentation that justifies the choices in the creation of the DSM-IV: the *DSM-IV Sourcebook*. In the sourcebook, many chapters are devoted to proving the distinctiveness of depression (APA 1996, 1997, 1998).

The infectious disease model also lends itself to a particular version of biology[9]. The spell of science, for major depressive disorder at least, is associated with little tangible entities that are blamed for the disorder. The laboratory findings section in the section on Major Depressive Disorder is full of fancy-sounding chemicals, hormones and protocols, although their involvement is still under dispute. In the final overview of mood disorders in the *DSM-IV Sourcebook* it is stated that "although we [the mood disorders work group] briefly considered incorporating laboratory tests into diagnostic criteria for one or more mood disorders," a policy adopted for the DSM-IV as a whole was that laboratory tests would not be used (Rush 1998: 1032). This statement implies that there *are* perhaps laboratory tests for depression. The overview explicitly concludes that "over the next decade, specific laboratory tests may well have a greater role in the diagnosis of selected mood or other psychiatric conditions" (Rush 1998: 1032). This neurochemical aetiology is also associated with depression by the makers of pharmaceutical and herbal antidepressants. For example, on the Zoloft.com website [accessed August 13th, 2002], under the heading 'How ZOLOFT Works,' there is the following explanation:

> Everyone has a normal substance in the brain called serotonin. It is thought that not having enough serotonin may contribute to depression, panic disorder, OCD, and PTSD. How ZOLOFT works for all of these conditions is not known. What is known is that ZOLOFT may help correct the chemical imbalance of serotonin in the brain. This helps relieve your symptoms. It may take several weeks for your symptoms to get better.

Likewise, in the *Physicians' Desk Reference for Nonprescription Drugs and Dietary Supplements*, about half of the entries for St. John's Wort products implicate some neurotransmitter in the description of the product, for example "St John's Wort has been

[9] This particular biology is only one that is presented; much biological research now may or may not use the categorical model, may or may not invoke neurochemistry or regulation of various brain processes, and generally favours an approach in which there is no one determining cause of depression.

found to help alleviate depression through its influence on brain serotonin levels" for Brite-Life by Advocare (Medical Economics 2001: 795).

In the DSM-IV, the spell of science is reinforced in other ways as well, including in the manual's style of presentation. The spell is imbued by the rhetoric of both mechanical objectivity (priority of instruments over people) and aperspectival objectivity (in the impersonal phrasing of the DSM-IV [Stoppard 2000: 26]) to use two terms of historian Lorraine Daston (1995). The spell is also invoked via repetition and redundancy, a sure way of indoctrinating the reader. For instance, the diagnostic criteria are reiterated three times: they are outlined once in the introductory paragraph, expanded upon in the ensuing paragraphs, and then written again in the diagnostic criteria 'cookbook' section. The expansions, however, rarely seem to introduce new material or restrict the criteria in a way that is not listed in the 'cookbook' section. Take, for instance, the paragraph explaining the criterion of sleep disturbance:

> The most common sleep disturbance associate with a Major Depressive Episode is insomnia (Criterion A4). Individuals typically have middle insomnia (i.e., waking up during the night and having difficulty returning to sleep) or terminal insomnia (i.e., waking too early and being unable to return to sleep). Initial insomnia (i.e., difficulty falling asleep) may also occur. Less frequently, individuals present with oversleeping (hypersomnia) in the form of prolonged sleep episodes at night or increased daytime sleep. Sometimes the reason that the individual seeks treatment is for the disturbed sleep (APA 2000b: 350).

The new information in this paragraph could really have been reduced to "Insomnia occurs more frequently than hypersomnia in a Major Depressive episode, and either sleep disturbance may be the reason that the individual seeks treatment". This example is not brought up to suggest that the writing in the manual needs improvement, but rather to suggest that the long descriptive sections serve a purpose other than a simply descriptive one. The long, repetitive re-iteration of information rather gives a scientific authority to the facts, and reduces the chance that they appearing whimsical.

Other diagnoses related to depression

> The diagnosis of major depression represents the severe end of a spectrum that has "normal" sadness as a reaction to life events at the other. Thus, major depression must be differentiated from such other conditions as bereavement and adjustment disorders with depressed mood. The presence of these normal reactions or milder conditions does not preclude the development of MDD and/or the need to provide treatment" – from the *Clinical Guidelines for the Treatment of Depressive Disorders* published by the Canadian Psychiatric Association (Parikh 2001).

Although *The Noonday Demon* indexes depression to the DSM-IV's Major Depressive Disorder, there are four other closely related diagnoses in the 'mood disorders' chapter. First, there is the diagnosis of dysthymic disorder – this diagnosis is also mentioned in *The Noonday Demon*, although to a lesser extent than major depressive disorder. Dysthymic disorder is differentiated from major depressive disorder in the sense that it is characterized by "chronic, less severe depressive symptoms that have been present for many years" (APA 2000b: 379). The sourcebook points out that dysthymic disorder and major depressive disorder, as discrete disorders, may coexist in different ways (see Figure 3, from Keller *et al.* 1998: 724).

There are also many other disorders and "other conditions that may be a focus of clinical attention" (see Table 1) that are related to depression that do not involve psychosis or elements of schizophrenia:

Table 1: DSM-IV diagnoses related to depression

1A: Disorders listed in the 'mood disorders' chapter	
296.xx	Major Depressive Disorder
300.4	Dysthymic Disorder
311	Depressive Disorder Not Otherwise Specified
293.83	Mood Disorder Due to [indicate the general medical condition]
29x.xx	Substance-Induced Mood Disorder

Figure 3: Dysthymia and Major Depression, taken from Asnis, Gregory, Lata McGinn, and William Sanderson (1998)

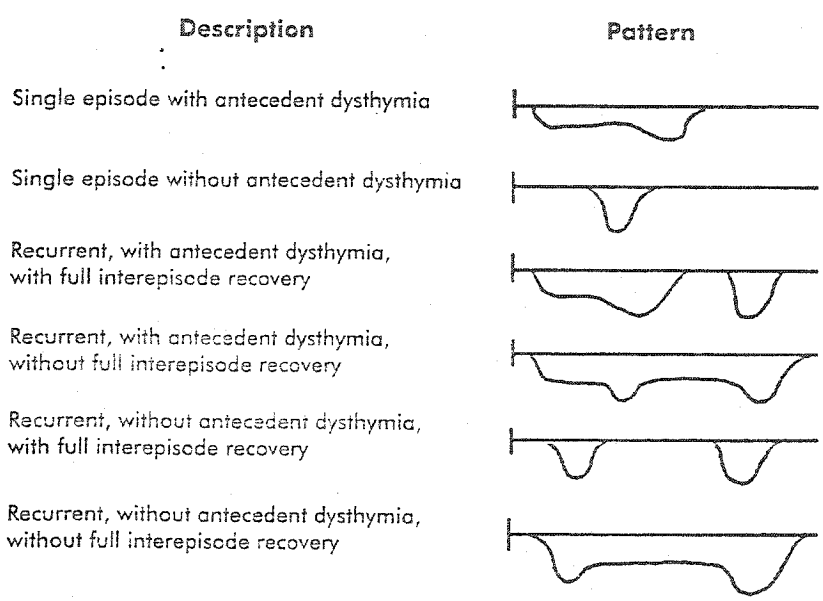


Figure 43–1. Proposed classification and patterns of longitudinal course specifiers for major depression.

1B: Disorders listed in other chapters in the main body of the DSM-IV[10]
309.0 Adjustment Disorder with Depressed Mood
v62.82 Bereavement [not a disorder per se, but a 'condition that may be a focus of clinical attention']

1C: Disorders listed in the appendix of the DSM-IV, provided for further study[11]	
•	Premenstrual dysphoric disorder
•	Minor depressive disorder
•	Recurrent brief depressive disorder
•	Mixed anxiety-depressive disorder
•	Depressive personality disorder

The other side of the spell of science and the definition of depression

While I argued that the diagnosis of major depressive disorder involved the spell of science, including that of discrete disorders, there is clearly a continuum of disorders related to depression. I mention this continuum because I want to highlight the fact that it is not only in vernacular documents that depression is used to connote more than one of the many meanings of depression mentioned in the introduction.

If we look at Table 2, we can see that many of the diagnoses are in fact very closely related to one another. In fact, major depressive disorder, minor depressive disorder, and recurrent brief depression are all based on the major depressive episode with its nine symptoms, although the number of symptoms required and the time over which they must be experienced differ for each diagnosis. I have listed dysthymia on the chart as well as its diagnosis is based on variations of seven of the nine symptoms listed for the major

[10] There are also a few other relevant diagnoses in the manual but I will focus on those that involve some variant of 'depression' in the name and that do not necessarily involve psychotic episodes. Also, I speak of the disorders as applicable to adults only; some may not apply to children or adolescents who sometimes have modified or different criteria.

[11] These disorders are listed in the appendix because Allen Frances, the editor of the manual, did not want to make any changes to the manual in its fourth version without there being enough validating evidence (Frances *et al.* 1989). Thus, while not codable disorders in and of themselves, they are relegated to the appendix in the hope that further studies using these categories will be conducted to assess the validity of the diagnoses (APA 2000).

Table 2 Spectrum of depressive disorders in DSM-IV: number of symptoms of major depressive episode vs duration

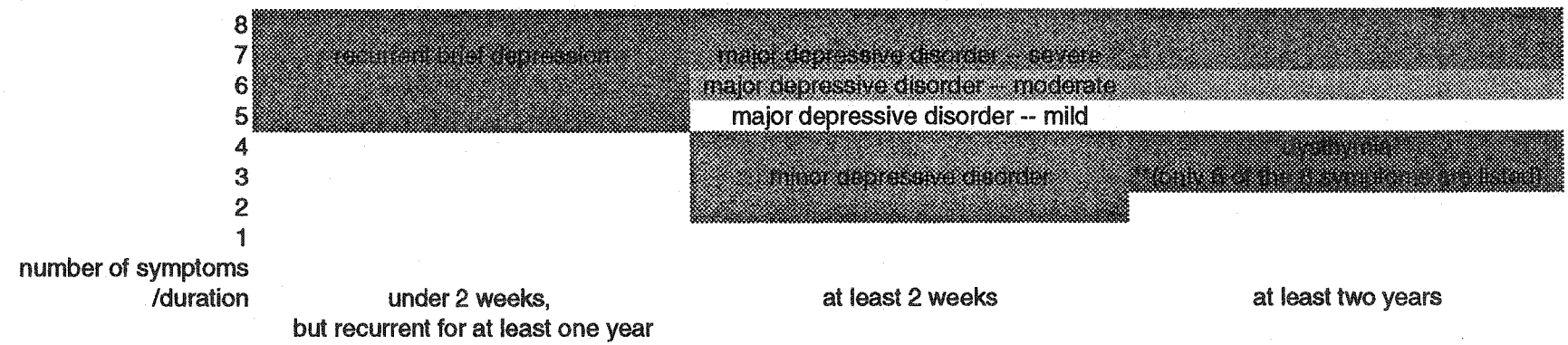
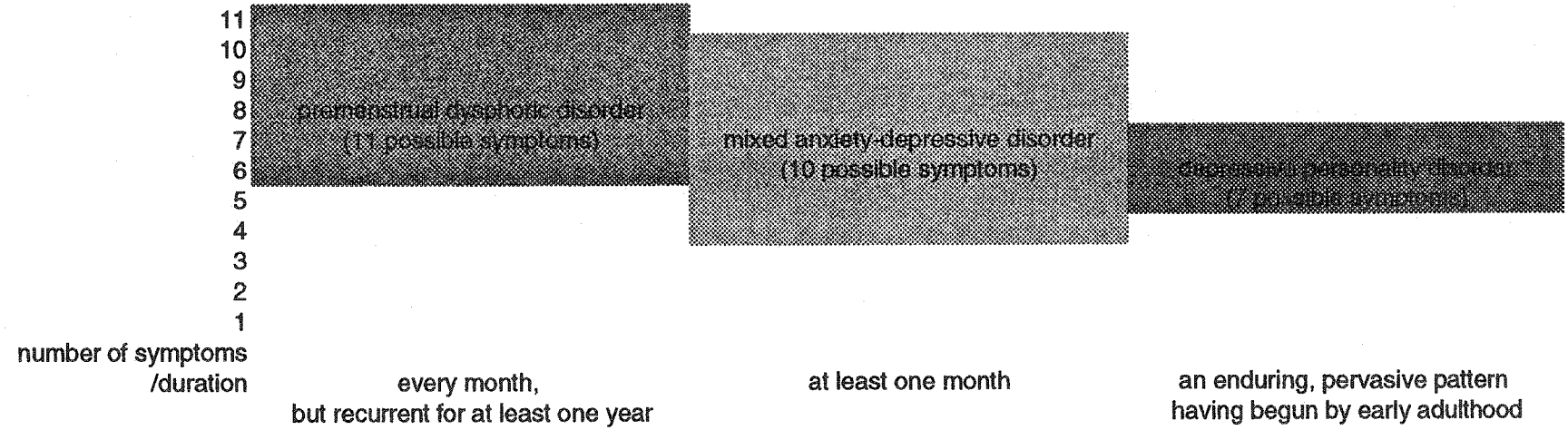


Table 3 Disorders similar to those in Table 2 but each with a somewhat modified symptom base



depressive episode. Although I did not place them on the chart, if a depressive episode does not qualify for a major depressive disorder but there is an event that is related to the distress, then either of the adjustment disorders or the bereavement disorder are suggested possible diagnoses.

There are several closely related diagnoses as shown on Table 3. Premenstrual dysphoric disorder, a disorder listed in the appendix of the DSM-IV, has a related but significantly different symptom base: this base has 6 of the 9 symptoms listed for a major depressive episode, but does not require depressed mood to be present and also has 5 additional symptoms related to anxiety and physical complaints. The criteria for mixed anxiety-depressive disorder have 5 of the 9 symptoms listed for the major depressive episode, with an additional 5 anxiety-related symptoms. Depressive personality disorder entails the pervasive presence of at least 5 out of 7 quite different symptoms since early adulthood.

The authentication and vindication of major depressive disorder

> Indeed the idea that there might be a depression that drugs could treat had in one sense to be invented as had the idea of an antidepressant – and part of the problem to this day is that neither of these inventions has fully worked, at least not with the public at large (Healy 1997: 4)

David Healy presents a compelling and nuanced case for the co-production of depression and antidepressants. In addition to his historical analysis that I will present in a later section, Healy outlines some of the protocols involved with the production of depression and antidepressants. For instance, one of the rating scales for depression that later "became the gold standard for the assessment of antidepressant effects," "is generally conceded to fit almost hand-in-glove with the profile of imipramine" (Healy 1997: 76). Indeed, this Hamilton Rating Scale for depression lists many items for sleep and anxiety in such a way that antidepressants with sedative effects – such as imipramine, one of the first antidepressant – are favoured in trials using this particular scale.

The *DSM-IV Sourcebook* also provides examples of the interaction of laboratory results, protocols, biology and antidepressants in the style of reasoning of biological psychiatry: in other words the self-authentication of facts produced and their subsequent self-vindication when challenged. I will focus on how antidepressants and two specifiers of major depressive disorder, 'with melancholic features' and 'with atypical features,' are co-produced. These two features figure in multiple chapters of the *DSM-IV Sourcebook* that contains the post-hoc justification for the choices made in the making of the manual.

Three articles justify the qualifier 'with atypical features.' The first, "Should Atypical depression be included in the DSM-IV" (Rabkin *et al.* 1996) traces a history of atypical depression that is explicitly linked to antidepressants. Atypical depression was first conceptualized in the 1950s as a way to classify the depression of those patients who responded poorly to imipramine (a tricyclic antidepressant), but well to a monoamine oxidase inhibitor (MAOI), contradicting the (at the time) generally held belief that MAOIs were less effective than tricyclics for depression. It was noted that patients who responded in this atypical manner characteristically tended to have mood reactivity and anxiety. Thus the constellation 'atypical depression' was born, in this self-authenticating style of reasoning.

In the 1970s and 1980s, however, it was demonstrated that phenelzine has a wide range of efficacy among outpatients with major depression and so there was no longer an atypical group of MAOI responders. This, of course, did not result in the disappearance of atypical depression. As Young points out, one of the key features of a style of reasoning is its self-vindication – failures are not seen as failures but rather as a call for the production of auxiliary hypothesis or a change in instrumentation (Young 1995: 10, 279; from Hacking 1992a). And indeed, auxiliary hypotheses were found. It was 'discovered' that the atypically depressed group did not respond especially well to MAOIs but rather responded less well than expected to imipramine. This new definition was buttressed by studies involving the biology behind this tempered response to imipramine and these studies are summarized in a second article entitled "Clinical and Biological Characteristics of Atypical Depression" (Asnis *et al.* 1996).

The protocol for identifying the clinical syndrome also morphed throughout these decades. The reviewers of the first study explain that this morph is due to different studies "tapping into different patient populations" (Rabkin *et al.* 1996: 250). The process comes full circle in the third article: "Pharmacotherapy Response in Atypical Depression: Findings From the NIMH Treatment of Depression Collaborative Research Program" (Sotsky and Simmens 1998). In this article, Sotsky and Simmens build a symptom list for atypical depression based on the maximization of the difference of response to imipramine. They demonstrate that, using a definition with reactive mood with at least one associated feature (one of six definitions that they tested), the atypical patients fared about the same on placebo and imipramine (the placebo fared slightly better) and the nonatypical patients fared significantly better on the imipramine than on the placebo. This vindicates atypical depression in the DSM-IV.

The co-production of atypical depression and antidepressants is still embedded in the social lives of antidepressants. The *Practice Guideline for the Treatment of Patients with Major Depressive Disorder*, put out by the APA, states that MAOIs may be particularly effective with patients with atypical depression, however "in clinical practice, many psychiatrists start with SSRIs in such patients because of the more favourable adverse effect profile" (APA 2000a: 3). The Canadian Clinical Guidelines recommend SSRIs and another agent, moclobemide, over the use of a MAOI because of increased tolerability (Kennedy *et al.* 2001: 39S).

Atypical depression is often compared to melancholic depression, on the terms that atypical depression is 'generally believed' to not be as severe (Rabkin *et al.* 1996: 253). And indeed, the 'melancholic feature' in the DSM-IV is linked to positive response to biological treatment in the DSM-IIIR, and was originally conceptualized as a predictor of ECT response (Rush and Weissenberger 1996: 204). The clinical practice guidelines published by the APA state that the "melancholic subtype is a severe form of major depressive disorder with characteristic somatic symptoms, and it is believed to be particularly responsive to pharmacotherapy and ECT" (APA 2000a: 38).

In addition to the two features already mentioned, other features of Major Depressive Disorder were also co-produced with medications. The catatonic features specifier is believed to be occasionally caused by medication, and was included for its clinical utility because the features respond to particular treatments. Psychotic symptoms were maintained as a modifier in the DSM-IV partially on the basis that "the finding that the presence of psychotic symptoms suggests a more likely need for neuroleptic medications combined with antidepressants or electroconvulsive therapy (Rush 1998: 1026). The two other specifiers for major depressive disorder, 'chronic' and 'with post-partum onset' are clearly not directly co-produced with medication. It is interesting that four out of the six associated features are explicitly co-produced with antidepressants.

Summary for Introduction to the DSM-IV

I have shown how the definition of depression in the DSM-IV is both explicitly and implicitly under the spell of science. This definition is inseparable from antidepressants and is conducive to leading to a biology that involves small and tangible culprits.

1. Depression, time, and space

> `I could tell you my adventures--beginning from this morning,' said Alice a little timidly: `but it's no use going back to yesterday, because I was a different person then.'

In this section we are tracing the adventures of 'depression' over a much longer period of time than 'beginning from this morning'. I will concurrently present two types of histories of depression in this section: one type that supports the idea that depression has been around since the beginning of time, and another type that argues that the proto-ideas or traces of depression in the past are windows into totally different realms of existence.

The rote history

In the vernacular literature analysed in chapter three, any history presented seems to emphasize that depression has been around since the beginning of time. These histories,

presented with varying degrees of depth and/or sophistication, are not histories in a broad sense, but rather a specific, processed, and almost pre-packaged narrative; in other words, "a consensual account that tells the history of the discourse and its object" (Young 1995: 141). These narratives are part of a particular style of reasoning, and as such these histories are part of the current concept of depression. I will refer to this narrative account of depression as the rote history[12]. One instantiation of depression's current rote history comes from the first paragraph found in the "History" chapter of Andrew Solomon's *The Noonday Demon: An Atlas of Depression* (2001). This is a quite thorough account of the rote history – often there is simply a one-line reference to depression being "as old as the Greeks." I use the passage in full to give a flavour of how depression might be presented as being around since the dawn of time:

> The history of depression in the West is closely tied to the history of Western thought and may be divided into five principal stages. The ancient world's view of depression was startlingly similar to our own. Hippocrates declared that depression was essentially an illness of the brain that should be treated with oral remedies, and the primary question among the doctors who followed him was about the humoral nature of the brain and the correct formulation of these oral remedies. In the Dark and Middle Ages, depression was seen as a manifestation of God's disfavor, an indication that the sufferer was excluded from the blissful knowledge of divine salvation. It was at this time that the illness was stigmatized; in extreme episodes, those who suffered from it were treated as infidels. The Renaissance romanticized depression and gave us the melancholic genius, born under the sign of Saturn, whose dejection was insight and whose fragility was the price of artistic vision and complexity of soul. The seventeenth to nineteenth centuries were the era of science, when experiment sought to determine the composition and function of the brain and to elaborate biological and social strategies for reining in the mind gone out of control. The modern age began in the early twentieth century with Sigmund Freud and Karl Abraham, whose

[12] I would rather not use a new term, but the current terms used – 'history' and 'genealogy' are used very differently in related works. For instance, Allan Young refers to the rote history of PTSD as a genealogy, and his different version of this history simply as 'history'; conversely Michel Foucault uses the term

> psychoanalytic ideas of the mind and self gave us much of the vocabulary still in use to describe depression and its sources; and with the publications of Emil Kraepelin, who proposed a modern biology of mental illnesses as an affliction separable from or superadded to a normal mind.
>
> .... The shape and detail of depression have gone through a thousand cartwheels, and the treatment of depression has alternated between the ridiculous and the sublime, but the excessive sleeping, inadequate eating, suicidality, withdrawal from social interaction, and relentless despair are all as old as the hill tribes, if not as old as the hills (Solomon 2001: 286-287).

Following Allan Young's assessment of a typical rote history of PTSD, we can argue that the rote history outlined by Solomon buttresses the following statements: (1) depression can be found in various guises/ manifestations throughout the ages, and that (2) the change in representations are for the most part representative of a trend towards a more accurate and timeless model of depression (Young 1995: 141). This section attempts to provide a more detailed account of this rote history invoked both in articles published in scientific journals and more generalist accounts in popular magazines, but to a very different end.

Traces and trances

> History cannot be logically constructed any more than a scientific event, if only because it involves the progress of vague and indefinable concepts which are about to crystallize.... [The concepts] become a tangle impossible to unravel logically, and organic structure produced by mutual development and with interacting components. At the end of the process, the beginning cannot be understood any longer or even properly expressed in words. If at all, it will be understood and expressed differently than it was originally (Fleck 1979 [1935]: 53).

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genealogy to mean his different version of this history, with the term discourse encompassing the notion of rote history (among other things).

In addition to presenting this rote history in more detail because it is important in the current definition of depression, I hope to present the traces of depression as a way of visiting exotic lands, and imagining other possible distresses with different embodiments, and experiences.

To facilitate this journey I have decided to present the rote history of depression and its treatment under several headers[13], the relationships among which will be explored later on: melancholia, acedia, nerves, psyche, and biological depression. These headings clearly do not 'carve nature (or history) at the joints,' but seem to this time traveller to be culturally salient 'proto-ideas' (Fleck 1979 [1935]: 25-7) or 'traces' (Rheinberger 1997) that are related to depression in its current rote history. These traces could be woven into very different rote histories for other DSM-IV disorders – for instance Obsessive Compulsive Disorder (Healy 1997: 30), somatoform disorder, and various anxiety disorders.

Melancholia[14]

Melancholia was not a discrete disease in the sense of depression in the DSM-IV; rather, melancholia was an imbalance of the naturally occurring humours in the body, and is therefore not separable from the body. Melancholia, the literal translation of the Greek *melaine chole*, was associated with an excess of black bile (Jadhav 1996: 273), which in turn was one of the four humours of which the body was made. Therapy did not involve dealing with a small tangible culprit, but rather involved the mimicry of the body's methods of dealing with illness, involving (among other things) purging and blood letting. Also, in therapy, attention was given to the 'six non-naturals,' namely: air, exercise/rest, sleep/wakefulness, food/drink, extraction and retention of superfluities, as well as passions/perturbations of the soul (Jackson 1986).

[13] I got the idea of tracing various threads from Sushrut Jadhav's excellent review of the history of depression (1996).

[14] For the first two sections – melancholia and acedia – I draw mainly from Stanley Jackson's *Melancholia and Depression: From Hippocratic Times to Modern Times* (1986).

Melancholia also did not necessarily connote the same degree of discreteness. In fact, Babb stated: "As I read the literary evidence, there is no clear distinction in the layman's mind between the melancholy temperament and the melancholic disease" (in *Elizabethan Malady* p. 71 *quoted in* Jackson 1986: 101). Melancholia also had more positive associations such as the image of the gifted and creative melancholic, harkening back to Plato's presentation of the 'divine fury' of writers and Aristotle's presentation of the melancholy genius (Porter 1995).

The humoral aetiology of melancholia was influential for a very long period of time. Many developments in the nascent sciences of the 16th and 17th centuries were merged with humoral concepts. In description and explanation, humoral theory remained strong in this pluralistic atmosphere although melancholia was also attributed to vapours, demons, spirits, chemicals, and/or nerve juice imbalance (Jadhav 1996). Nonetheless, there was still much emphasis on treatment, especially on the practical aspects of humoral theory as presented in two major works written on melancholia. In the 60 manuscript volumes recording the medical practice of Richard Napier, a physician in rural England in the early 17th century, there are few uses of humoral theory as aetiology however many uses in treatment. Napier almost always used purgatives to treat melancholy (in those of higher status) and mopishness (in ordinary people); he also used traditional remedies, bloodletting, religious counselling and prayer, amulets if the astrological positions indicated, as well as pragmatic remedies pulled from Paracelsus' repertoire to care for these disorders.

Acedia

Acedia, first used to describe the state of some Egyptian desert monks near Alexandria in 4AD, was in part a physical disorder although in a very different way from depression. Acedia is the Latin transliteration of a Greek word connoting sluggishness, torpor, i.e. an uncaring state (Jackson 1986). Sometimes acedia was attributed to a demon, such as 'the noonday demon,' coming to tempt the monks. Acedia was initially understood on physical terms, illustrated by Cassian's suggestion of manual labour as therapy. However, by the 12th century, acedia became more of an affective rather than physical

problem, and in the middles ages was included with cardinal sins in the middle ages as "a temptation" against which monks needed to guard themselves (Jadhav 1996: 273).

Furthermore, acedia was not always a 'disease', although it did become so in the thirteenth century as it became linked with humoral theory (Jackson 1985: 50). As a disease, the moral implications were lessened. With the weakening of the church in the fifteenth and sixteenth centuries, acedia lost importance as a concept. Its associated sadness and dejection became associated with terms such as melancholia; its associated sin was referred to as sloth, gaining cultural relevance in the era of the Protestant work ethic (Jackson 1985: 56).

An interlude

> The least demanding grade of constructionism about *X* is historical. Someone presents a history of *X* and argues that *X* has been constructed in the course of social processes. Far from being inevitable, *X* is the contingent upshot of historical events. A historical constructionist could be quite noncommittal about whether *X* is good or bad. How does historical "social" constructionism differ from history? Not much, a matter of attitude, perhaps (Hacking 1999: 19).

The rote history presented in Solomon, I argue, is not constructionist. Although he states that the "shape and detail of depression have gone through a thousand cartwheels" (Solomon 2001: 287), he does not argue that these cartwheels imply a fundamental change in depression. Rather, he claims the constellation of symptoms "are all as old as the hill tribes, if not as old as the hills" (Solomon 2001: 287). I also argue that Jackson, to some degree, uses the history of melancholia to illustrate a fundamental continuity. He emphasizes continuity and similarities with the depression of the late 20th century, rather than the discontinuity and dissimilarities. I do not mean to paint a black and white picture, but rather one in shades of grey – Jackson himself remarks that "[t]o search for the origins of the term and the concept of 'depression' the historian does not need to go beyond the middle of the nineteenth century" (Jackson, 1986). Roy Porter, in 'Mood Disorders – Social Section' (1995), also exemplifies this approach to some extent:

> It should be noted that with melancholy and mania – unlike, say, schizophrenia – disease categories are encountered that were 'framed and named' … at the dawn of learned medicine, concepts destined to root and blossom over the centuries (Porter 1995: 409).

The emphasis of Solomon, Jackson, and Porter, while perhaps "a matter of attitude," changes the experience I have as a time-traveller because I take for granted the language, bodies, and meanings of acedia and melancholia and am thus, in a sense, lulled into a false sense of security. If one is looking for similarities, i.e. traces of a clinical syndrome, then the result is that the history will emphasize the similarities and continuous threads, rather than the differences that might not make much sense – i.e. the threads that led off in other directions or that simply ended before the current day.

In contrast, David Healy (1997) and GE Berrios (1995) emphasize the differences and both argue that depression as it exists today could not possibly have existed before the 20th century. Melancholia and acedia referred to some aspects of what is now termed depression, however the differences are more important than the similarities. I argue that they have the "matter of attitude" that changes their accounts of the history of depression to more of a social constructionist one that denaturalizes the idea of the timelessness of depression. This interlude will use the ideas of Healy (1997), Berrios (1995), and Jadhav (1996) on depression, and the ideas of other historians more generally (Duden 1991; Rosenberg 2002), to look critically at the notion that depression has been present in various guises since the beginning of time. The interlude presents three major dissimilarities that each raise an issue in the 'translation' or commensurability, in a Kuhnian sense, of the experience of melancholy in ancient Greece to something comparable to the experience of depression in present-day North America. Like Alice who only realizes that something is distinctly different about the current state of things when she notices that the waist-coat-wearing Rabbit "actually TOOK A WATCH OUT OF ITS WAISTCOAT POCKET," hopefully some of these translation issues will instigate us, burning with curiosity, to follow some non-familiar embodiments and worlds.

Translation: language

Let us examine the first issue, that of the translation of the language of distress. While a rose might be "indifferent" to being called by another name, people are "interactive kinds": they change and are changed by the language used to name their experiences (Hacking 1999). In his historical account, Solomon treats language and choice of terms as a non-issue: "I use *depression* here to describe states for which we would now use that term" (2001: 286). While Jackson does briefly mention issues with translation and the use of medical texts he does not examine these issues as serious problems. In other words, in treating these issues as surface issues instead of issues affecting the very constitution and reality of the terms, he stays squarely on the history side, rather than the historical constructionism of the Hacking quote.

Even when the language is familiar, for instance in the 16th and 17th century notes of the rural English physician Richard Napier, it is difficult to imagine that the words mean the same things to the authors writing the works as they do to 21st century readers. Indeed,

> The group of conditions nowadays called 'affective disorders' has resulted from the convergence of certain *words* (e.g. 'affective' and its cognates), *concepts* (theoretical notions accounting for 'mood' related experiences), and *behaviours* (observable changes in action and speech associated with neurobiological changes yet to be fully elucidated). The evolution of these elements has been asynchronous and each has a different history; in fact, their convergence only took place during the early part of the twentieth century. Since there is no reason to expect that such convergence is necessarily 'written in the nature of things', it is postulated here that its explanation belongs more to history than to science. If the 'convergence hypothesis' is correct, then those who believe that the history of clinical conditions such as 'mania' and 'melancholia' (as *currently* defined) starts with the Greeks are mistaken. Indeed, such anachronistic approach would only chronicle the *history of the pertinent words* (Berrios 1995: 384, emphasis in original).

Indeed, rather than simply presenting different names for a common underlying phenomenon, the different conditions called 'melancholia' through the ages have fundamental differences and are not necessarily commensurable. Berrios uses the example of Esquirol, who was part of the nineteenth century classification frenzy. Esquirol, influenced by faculty psychology, wanted to more clearly define what he saw as an over inclusive term: 'the word melancholia, consecrated in popular language to describe the habitual state of sadness affecting some individuals should be left to poets and moralists whose loose expression is not subject to the strictures of medical terminology' (1820: 148, quoted in Berrios 1995: 389). He devised the term 'lypemania' to refer specifically to a disease where there are delusions in only one or two domains with much sadness. Berrios says that this term is not really ever taken up, but that lypemania can be considered a 'bridge' category: "it only served to catalyse the transition between the old notion of melancholia (as a primary disorder of intellect) to the new one (as a primary disorder of affect)" (Berrios 1995: 389), one that was concomitant with the change from insanity-as-total to a notion of partial insanity. Thus, even when the wording is consistent, the meaning may not be[15].

> Alice felt dreadfully puzzled. The Hatter's remark seemed to have no sort of meaning in it, and yet it was certainly English.

This problem of the 'translation' of meaning from one era of English to another is also relevant when tracing the word 'depression' through history. Using the Oxford English Dictionary to do a lexical analysis of depression, Jadhav notes that the term depression, linked to the Latin term *De Primere* meaning to 'press down', was in 1391 used in regards to the apparent sinking of phenomena in astrological observations (Jadhav 2000). The term 'depression' was first used medically in the context of cardiovascular medicine of the 1850's, where the term indicated a "reduction in function" (Berrios 1995). It was first used in the phrase 'mental depression', but by 1860 'depression' by itself appeared in medical dictionaries "applied to the lowness of spirits of persons suffering under disease" (Mayne 1860 *in* Berrios 1995: 386). Indeed, over the 19th century, the use of

[15] While Jackson also traces changes, again, his focus is more on the overall similarities – 'a matter of attitude.'

'depression' was becoming more common in contexts related to mood but it was not in and of itself a formal diagnostic category (Jackson 1986). By 1901, 'depression' was defined as:

> a condition characterized by a sinking of the spirits, lack of courage or initiative, and a tendency to gloomy thoughts. The symptom occurs in weakened conditions of the nervous system, such as neurasthenia and is specially characteristic of melancholia (Jastrow 1901: 270 *in* Berrios 1995: 386).

Many of these 1901 associations of depression are understandable in terms of the depression in late 20th century North America, but some are a stretch. Terms such as 'lack of courage,' 'sinking of the spirits,' and 'occurring in weakened conditions of the nervous system' can be rationalized in terms of the current definition(s) of depression, but I doubt they would often spontaneously be mentioned in those ways. And as noted in the section on the DSM-IV definition of depression, melancholia has now become a subfeature of depression instead of depression being 'specially characteristic of melancholia'.

Translation: body

The second major issue of 'translation' dissimilarity is that of the body – Solomon takes for granted that the body in ancient Greek times is the same body that we have in North America at the turn of the 21st century. Indeed, this is perfectly logical if we take the perspective that, through history, culture may change but nature (including the physical body) is ahistorical. But let us take a trip back to the women patients of Dr. Storch, a doctor in the German town of Eisenach in the 1730s. As historian Barbara Duden examined his recorded case histories, she concluded that the bodies of these women patients were completely different than her own in the late 20th century (1991). It was not that Duden could not recognize and relate to some of the comments that these women were making – she could do that – but when she immersed herself in their accounts, especially the parts that did not make sense to a 20th century woman, she could piece together an embodiment that was completely foreign (1991). Simply put, as travellers, our bodies are fundamentally not comparable to the bodies we read about in Jackson's work in ancient Greek times. There are so many things that make my body quite different

from the bodies of the women patients of Richard Napier. For one, no spirits inhabit my body, no demons tempt me, and I simply do not have trouble with black bile. As Duden puts it, the first major difference is that I "have" a body in a (possessive) way that Richard Napier's patients likely did not. The bodies and selves of the women patients are not just similar to ours but with different explanations attached; they *are* different bodies and selves.

Translation: social context

So you, my fellow traveller, might argue 'I don't care about these finer points of interpretation – Jackson clearly shows how the clinical syndrome, whether experienced by similar bodies, stays remarkably consistent.' The third issue, the translation of social functions and contexts of the terms involved, should hopefully convince you that there may be some striking dissimilarities that cause a fundamental problem with the comparison of ancient Greek melancholia and present North American depression. Currently, one might suffer from depression for a period of time, but after a while, whether treated or not, the depression might remit. Even for those suffering from chronic depression, it is the depression that is seen as abnormal whereas the remittance of it is a state that does not necessarily need to be explained. Berrios argues that from this vantage point,

> pre- 1800 concepts of insanity are only apparently intelligible. Up to the eighteenth century, insanity (lunacy, madness, vesania) was opaque in various ways; the hardest to understand being the fact that it predicated of the insane a state of existence (rather than of mind) which was *sub specie aeternitatis* [something that lay outside of time and that would stay once acquired]" (Berrios 1995: 388).

Thus, Berrios continues, what needed explanation before the 1800s was the *remission* of the disorder, rather than its existence per se.

In addition to changes in the overarching concepts of madness and pathology, there are changes in the relations of diagnoses to diseases and symptoms throughout time. First of all, the idea that diseases could be discrete, non-overlapping alternatives is one that was without much credibility until the second half of the 19th century. Even then, Rosenberg

argues that the "narrative that constituted and described each disease became tight, more procedure oriented and rule defined" only in the 20th century (Rosenberg 2002: 248). From the evidence in Jackson's book, it does seem that naming a disease 'melancholia' in ancient Greek times did have the same implications of the naming one 'depression' today, with the ensuing "conflation of diagnosis, prognosis, and treatment protocols (Rosenberg 2002: 240). To examine the pre-conflation situation we can travel back to Jackson's accounts of treatments for acedia and melancholia. Although I briefly mentioned the most emphasized treatment options I did so only out of a desire for consistency. Their listing is very different than the treatments listed for those conditions that are experienced *after* the bacteriological revolution in medicine, because if disorder is a matter of imbalance in the individual, then the treatments used are general ones used to restore balance in certain ways. Thus, statements such as the following excerpt from Solomon might need to be reinterpreted:

> The ancient world's view of depression was startlingly similar to our own. Hippocrates declared that depression was essentially an illness of the brain that should be treated with oral remedies, and the primary question among the doctors who followed him was about the humoral nature of the brain and the correct formulation of these oral remedies (Solomon 2001: 286).

Thus, as we continue our travels back in time, it is important that we keep in mind the different relationships of the treatment to the disease.

This is especially true when it comes to classification. As with any history, the history of depression is entwined in problems of historical interpretation: namely, the reliance upon the description of a clinical syndrome in written texts as a link to the experience of sickness by the people at the time, or the clinical practice at the time.

> "Alice tried to fancy to herself what such extraordinary ways of living would be like, but it puzzled her too much, so she moved on."

<u>Nerves</u>

Another proto-idea that might be relevant to neurobiology, but that is not mentioned in Solomon's rote history, is idea of disorders of the nerves. We might trace the rote history

of nerves beginning with the nerve power or force in Galen's concept of animal spirits. However, by the 17th century, these spirits had materialized into nervous fluid or solids, and by the 19th century, they had transformed into electrical entities (Oppenheim 1991). Until the beginning of the 19th century, the term 'nervous' "had connotations of vigour, force, and freedom of debility," in other words connotations of strength and overabundance rather than disorder (Lutz 1995: 534).

Nerves were related to many disorders though the ages. Oppenheim argues that those suffering from nervous disorders were often heralded as the product of their time: the melancholic in the 16th to 17th century; the hypochondriac in the 18th century; the neurasthenic in the19th century (Oppenheim 1991); and some might argue the depressive in the 20th century (Ehrenberg 1998). In the work *Historical Origins of the concept of Neurosis* (1983), Jose Maria Lopez Pinero traces disorders linked to the nerves, beginning with Renaissance concept of nervous disease in the work of Thomas Willis and Thomas Sydenham. Sydenham, for instance, argues that hysteria and hypochondria were not due to Galenic vapours issued from the womb or spleen, but rather were related to the nervous system. But Lopez Pinero cautions us from assuming that this 'nervous system' is similar to the nervous system that we embody today; it was not until the 19th century that 'nervous disease' connoted something 'neurological' (Lopez Pinero 1983).

Styles of reasoning and neuroses

These nervous disorders, as has been argued for depression, were authenticated within particular styles of reasoning that changed over time, and within these styles were vindicated. I will illustrate this with the term neurosis. The term neurosis was first used by the Scottish physician William Cullen in 1769 as a convenient way to refer to functional nervous disease. When Cullen first coined the term neurosis, there was an expectation that a physical or anatomical basis for the functional nervous disorder would be found; there was not the psychological connotation to neurosis that there might be today. Because of Cullen's reputation, his work was translated into many languages and he is credited with having brought this term into practice (Oppenheim 1991). In practice, the term was translated and used in different contexts, and thus the spread of this concept

also involved various drastic permutations and combinations. Pinel was another famous alienist who took up the term neurosis from Cullen, but in his hands neurosis, "related since its inception to *general* diseases and interpreted *physiologically*," was drastically reconfigured as the anatamoclinical approach, "based on *localization* and a *reduction to the anatomical level*," took hold in the 19th century (Lopez Pinero 1983: 44, emphasis in original). As an organic basis for a disorder was found, the disorder was removed from the umbrella of neuroses, with the effect that over time the neuroses umbrella shrank. By the time neurosis made it from Pinel to his follower Jean-Etienne Esquirol (who we visited earlier with his word 'lypemania'), to E. E. Georget in mid-19th century France, neurosis connoted any disease *without* organic basis. Lopez Pinero states that only followers of Georget, mostly in Germany and France, continued to use the term neurosis, and that the term virtually disappeared in Britain (1983).

Janet Oppenheim, in her study of nervous breakdown in Victorian England, takes a similar stance on the progression of the concept of nervous disorders in the 18th and 19th centuries. She argues that at the beginning, nearly everyone had the expectation that a physical or anatomical cause would be found for all neuroses and functional disorders; towards the end of the century, these umbrella terms were more than a little ratty. Once the umbrella of nerves and neurosis had shrunk, and it was clear that the anatamoclinical approach had failed, other concepts and methods developed to fill the void. Some of these other concepts developed to fill the void emphasized localization without anatomical lesions, i.e. 'functional localizations' (Lopez Pinero 1983). The notion of 'nerves' was not totally discarded as this notion helped to create some prestige for the mad doctors, alienists, and neurologists (whose standing by WWI was much better than that of the psychiatrists[16]). In the middle of the 19th century, for instance, there was spinal irritation, where tender spots on the spinal cord caused various neuroses. There were also the reflex functional nervous diseases, in which specific disorders were caused by another part of the body linked to the area of the disorder via a 'reflex'. Charcot's

[16]This is only one approach taken by the alienists of the 19th century: "Throughout the Victorian and Edwardian periods, they posed as scientists, single-mindedly pursuing their physiological inquiries, or as moral guides, firmly but sympathetically redirecting their patients' thoughts away from morbid into healthy

approach to hysteria, for instance, also left the anatamoclinical approach and instead approached hysteria as a *maladie generale* because it affects the whole person, not just one part (Lopez Pinero 1983). It is clear that throughout the 19th century, nervous disorder underwent some rather radical transformations as it continued to develop with various methods, from the anatomoclinical approach to the functional localizations. It is interesting that the fact of nerves (and later, neurotransmitters) underlying mental disorder has been vindicated in medicine for over a century, with failures in providing 'proof' causing either the development of auxiliary hypotheses or changes in instrumentation (Young 1995: 10, 279; Hacking 1999).

> Biological psychiatry has had 200 years to develop a progressive research programme. It has not done so, yet paradoxically it seems to be very successful and increasing its sphere of influence (Russel 1995: 154).

The co-production of a proto-idea and its treatment

The co-production of a proto-idea of depression and its treatment is evident to some extent with 'neurasthenia', a term introduced in 1869 by Beard. He defined neurasthenia as a distinct disease, although it had an almost infinitely expandable symptom list. The term quickly became popular in the 1880s. He blamed the 19th century, with its urban life, the education of women, and rapid transportation and communication as the cause – and as such it was an American disease (Oppenheim 1991). Rather than this blame critiquing American society, however, it celebrated a productive country's production of a 'distinguished malady' (Lutz 1995: 535). As many other things of distinction in the productive country, neurasthenia was very common in the upper classes of North America at the end of the 19th century, but it was not until the early 20th century that farmers and labourers might be affected by neurasthenia (Lutz 1995). In the 19th century these labourers might have been affected with differently named nervous disorders (recall the melancholia vs. mopishness of Richard Napier's diagnoses), or they might not have been diagnosed with nervous disorders at all. It is important to note that the only therapeutic options for the working classes, at least with respect to nerves, were massive

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channels, or, yet again, as social disciplinarians, resolutely advancing against deviancy on all fronts" (Oppenheim 1991: 35).

drugging or the public asylum – neither of which make a nervous disorder a particularly appealing one for these classes. For the more leisurely classes, other treatments were available, usually involving a combination of physical treatments and moral guidance (Oppenheim 1991). Getting out in the breezes and sunshine, as well as water cures and hydropathic spas were big, especially as an alternative when massive drugging (with iron, quinine, strychnine, arsenic, mercury, and opiates as nerve tonics) and bloodletting were still in favour. Just like a battery needs recharging with electric current, the neurasthenic might be recharged with electric treatments (Oppenheim 1991). Exercise cures (for men) and rest cures (for women) were also ways of regaining 'nervous energy/power' (Lutz 1995). It is also important to note that some of the treatments became milder, with the idea that the nerve juices should be strengthened, rather than keeping harsh treatments to put the body back in line. This differential of treatment options available might be key in understanding the different existences of nervous disorder among different groups in the 19th century.

However, all of the above disorders eventually fell outside the ambit of a biological model. Oppenheim concludes that during the last quarter of the 19th century, the search for proof of the more physical attributes of the disorders lessened and there was a move towards psychological attributes (1991).

Psyche

The most famous proponent of these psychological attributes, at least from the standpoint of the late 20th century, is Freud. In his later years, Freud considered the aetiology of the neuroses to be largely psychogenic, and he emphasized the unique psychodynamics of each mind and self (Porter 1995: 419). This aetiology led to less drastic treatments – neither massive drugging nor bloodletting nor asylum stays were necessarily involved – and as such it is possible that people with less severe troubles ventured to see psychiatrists (Berrios 1995). This led to the development of the private practice, in addition to the ever-present asylums (Berrios 1995).

Once depression became a disorder primarily of psyche, rather than a disorder such as 17th century hypochondria that integrally involved disorders of the internal organs, including 'gastroenteritis', many physical symptoms were no longer integral to the illness concept in North America. It is at this point that the concept of somatization in the DSM-IV begins to make sense, and it is indeed not until the 20th century that the term 'somatization' was coined (Berrios and Mumford: 1995).

Traces of the DSM-IV

This division of psyche and body was so entrenched that the first comprehensive American listing of psychiatric disorders, the *DSM-1*, was organized around the division between disorders of organic and psychodynamic origin:

> The basic division in this nomenclature is into those mental disorders associated with organic brain disturbances, and those occurring without such primary disturbances of brain function, and *not* into psychoses, psychoneuroses, and personality disorders (APA 1952: 12).

The editor of this volume, Adolf Meyer, emphasized environmental influences, and placed psychiatric disorders along a continuum with normality (Young 1995: 98). The influence of Freud and psychodynamic aetiology is evident in the text. Depression itself was listed as a psychological mechanism, although elements of depressive affect were classified both under the category heading "Affective reactions" (as 'psychotic depressive reaction') and "Psychoneurotic Disorders". In keeping with the psychodynamic perspective, the manual did not list symptoms required for diagnosis, but gave a general description of the disorder. The DSM-I was not revised until 1968, when the DSM-II was published to ensure consistency with the ICD. This manual had 182 categories, more or less consistent with psychodynamic approaches. This second revision was an attempt by the committee, of which Spitzer was a part, "to put down what it judges to be generally agreed upon by well-informed psychiatrists today" (APA 1968: vii *quoted in* Kutchins and Kirk 1997). The third edition was somewhat of a revolution, and requires picking up the trace of another trend beginning at the turn of the 20th century.

Biological Depression

Sigmund Freud is the figurehead of psychoanalysis; Emil Kraepelin is the figurehead of biological psychiatry. Kraepelin was a German psychiatrist who developed a classification of mental disorder modelled on the model of infectious diseases: that of the specificity of 'manic-depressive insanity'— which included depression – and 'dementia praecox'. He emphasized the discontinuity between diseases, and also the distinction between normality and disease. Needless to say, compared to the more continuum-based ideas of disorder in Freud's work, Kraepelin's ideas were somewhat contradictory, especially when one looks at the position of symptoms (Young 1995: 96). "Kraepelin sees symptoms as properties or attributes of disease-like mental disorders, while psychoanalytic writers see symptoms as polymorphic expressions of individual lives and psyches" (Young 1991: 177). That said, Kraepelin's later work was not an important reference in American psychiatry until the 1970s (Healy 1997; Young 1995). What prompted this resurfacing? I will draw from David Healy's *The Antidepressant Era* (1997) as well as Kirk and Kutchin's work on the development of the DSMs (1992; Kutchins and Kirk 1997) to provide a partial answer.

The co-production of depression and antidepressants

David Healy, in *The Antidepressant Era*, paints a picture where "depression as it is known to or understood by the public in the 1990s was all but unknown as recently as thirty-five years ago" (Healy 1997: 4). He hinges the change on the 1962 FDA amendments in the United States. He focuses on the following actors throughout the 20th century: government, psychiatrists, pharmaceutical companies, and the public. On the government side, he traces several pieces of American legislation that shaped the pharmaceutical landscape – all of which are closely tied to public outcries related to pharmaceuticals. He keeps an American focus because of the focus of the pharmaceutical companies on the American market. The United States government enacted the Food and Drug Act (FDA) in 1906 in response to issues with the private industry's production of food and drugs. Following the production of antibiotics in the 1930s, a Food, Drug, and Cosmetics act was passed in 1938 following many deaths due to a particular preparation of an elixir containing sulfanilamide. This act prohibited

misleading labels and the preparation of any compound until it had been proven safe. In 1951, legislation allowed for prescription-only status of some drugs. Following the deformities linked to thalidomide, in 1962 legislation was passed that required drugs to be proven both safe and efficacious before being put on the market (Healy 1997).

During the initial trials of what would become antidepressant medications in the 1950s, pharmaceutical companies had not had much interest in the matter because in the early part of the century in America nearly all diagnoses for inpatients were 'schizophrenia,' on which the companies had focussed their marketing. The work of epidemiologists in the 1960s, however, suggested that many of the "seemingly psychoneurotic complaints presenting to primary care physicians might stem from unrecognized and untreated depressive illness" (Healy 1997: 229). In order to reach the unrealized market for depression, however, the pharmaceutical companies needed to publicize not only the antidepressants but also the idea of depression. In fact, Merck distributed 50,000 copies of a book entitled *Recognizing the Depressed Patient* internationally (Healy 1997: 76).

But this would not have been possible had it not been for the sedimentation in the 1950s of the diffuse elements into the randomized clinical trial, which was the vehicle for proving the efficacy of these antidepressant compounds. And this still might not have been enough to create depression in the sense that we know it today, were it not for the decisions taken by the APA in the 1970s (Healy 1997).

A particular style of reasoning and the spell of science

Here I will turn to the work of Kutchins and Kirk, which centres on the DSM-III revolution by 'an invisible college' in 1980 (1997). During the 1960s and 1970s the psychiatric profession faced serious legitimacy problems as it came under attack from groups such as the gay and anti-psychiatry movements. The funding given to psychiatry had diminished since the 1950s; there was economic incentive for psychiatry to be perceived as a science (Young 1995). Just as Oppenheim argues that psychiatrists in the late 19th century used the rhetoric of nerves and/or degeneration to legitimize themselves, Kirk and Kutchins argue that a group of psychiatrists/researchers in the 1970s used the

rhetoric of science to lend legitimacy to the psychiatric enterprise as a whole (Kirk and Kutchins 1992). To paraphrase the story of this legitimation as told by Kirk and Kutchins: on a dark and stormy night in the history of psychiatry, an 'invisible college' took over the workings of American psychiatry – this takeover involved the adherence to what is termed the 'neo-Krapelinian credo.' Whereas the DSM-I and the DSM-II were not perceived as a collection of scientific work on mental disorders – rather they were "administrative codebooks put out by a small, obscure committee" (Kutchins and Kirk 1997: 247) – the third edition of the manual aspired to be the pinnacle of scientific work with respect to mental disorders (1997).

The new scientific outlook on psychiatry was embodied by the editor, Spitzer, although he refused to be identified as a neo-Kraepelinian. He initiated a shift in diagnosis from the psychodynamic to biomedical approach in order to shift psychiatry's focus from the clinic to research, a more 'scientific' endeavour. This new motivation is entirely consistent with the biological models of therapy, but not with psychoanalysis. Needless to say, many did not approve of the change in focus. With respect to depressive disorders, Spitzer did concede two disorders that worked within a psychodynamic framework: "dysthymic disorder" (Healy 1997: 236) and the qualifier "with melancholic features" (Kirk and Kutchins 1992). The DSM more than tripled in size in terms of pages, and included reliability studies imbued with an aura of objectivity and science with 'kappa statistics,' circumventing the more difficult issue of the clinical relevance and validity of the diagnoses (Kirk and Kutchins 1992).

The ensuing versions of the DSM continued this trend. The DSM-III-R listed 292 categories in much the same way as the previous version (Kirk and Kutchins 1992). The DSM-IV aimed for consistency with the DSM-III-R, and thus categories were not to be changed unless there was evidence proving that they should indeed be changed (Frances *et al.* 1989).

> Biology here is construed as a key symbol uniting disparate meanings and embodying cultural ideals of science and progress in U.S. medicine (Gaines 1992: 171).

Anthropological analogies

> Constructivism thus agrees with Evans-Pritchard in recognizing the kinship of anthropological and historical research (Gaines 1991: 241).

Both history and anthropology might provide case studies contextualized in one time period and in one geographical location. While the historian may 'have the matter of attitude' that makes a more constructionist account, so also may the anthropologist. By presenting the terms of other idioms of distress in the terms of a different culture, anthropologists may implicitly (or explicitly) question the inevitability of depression and psychiatric diagnoses in the DSM as a whole. Examples of this include Ong's study of possession in Malaysian factories (1988), Farmer's study of *move san* among rural Haitian women (1988), Davis' study of nerves among Newfoundland women in fishing villages (1986), and Finkler's (1985) study of nerves among rural Mexican women (all above reviewed in Richters 1996), as well as the experience of *nevra* among first generation Greek immigrant women in Montreal (Lock 1991).

And the DSM-IV

Cross-cultural differences such as these are recognized in the DSM-IV, however they are sidelined in an appendix in a way that reinforces the distinction between the 'cultural' entities in the appendix and the 'scientific' entries in the main body of the text. This distinction between culture and biology is reinforced by several authors in the *DSM-IV Sourcebook*, including anthropologists. In an article by anthropologist and physician Arthur Kleinman, he explains that although major depressive disorder hinges on the affective experience of depression, "in most societies, most people suffering clinical depression do not complain mainly of sadness. Instead they talk about fatigue, headaches, backaches, stomach upset, insomnia, loss of appetite, and so on" (Kleinman *et al.* 1997: 868). He comments that this may lead to a visit to primary health care, rather than mental health care and "usually their depression is neither diagnosed nor effectively treated" (Kleinman *et al.* 1997: 868). Although he highlights that both emphases on emotion and body are clearly "cultural" (Kleinman *et al.*1997: 868), he argues that both

emphases are manifestations of an underlying depression of which there is "convincing evidence" supporting its worldwide distribution (Kleinman *et al.* 1997: 869). This last assumption is also apparent in his work in the key reference book for the anthropology of depression – *Culture and Depression* edited by Arthur Kleinman and Byron Good (1985). In this book, Kleinman and Good outline their method of joining the different 'constitutions' of depression cross-culturally and an underlying biological pathology (Good and Kleinman 1985: 492). Although this seems like a social constructionist account, it does not hold that 'depression need not have existed,' because they assume some form of underlying depressive disease[17]. Kleinman speaks of the important cultural and social forces in shaping depression but ultimately relies on the assumption of a universal biological underpinning, as does Jackson (see Kleinman and Good 1985: 7; also Gaines 1991: 250), rather than on the co-production of cultural and biological facts.

In the *DSM-IV Sourcebook* chapter dealing with "Cultural considerations in the diagnosis of mood disorders," the anthropologist Spero Manson takes a different perspective. He enumerates several issues that may prevent the applicability of depression cross-culturally (1997). He mentions all three issues of 'translation' that I presented in the interlude, including that of language, especially of affect; that of body and experience, for example the cultural specificity of the mind-body distinction; and that of cultural significance of the terms involved with respect to 'pathology' or 'abnormality' (Manson 1997). This chapter arguably takes a rather ironic commitment to the social construction of depression-as-biology, because although he presents depression as contingent, he presents no possible solution (1997: 918); I am left with the impression that "[w]e are nevertheless stuck with it [depression], it forms part of our way of thinking which will evolve, perhaps, in its own way, but about which we can do nothing much right now" (Hacking 1999: 19-20).

Summary for depression, time and space

[17] Kleinman and Good differentiate between disease, which is the underlying pathology, and illness, which is the experience of the disease. I do not use these terms in the rest of my argument because, in attempting to use the models provided by Fleck (1979 [1935]) and Young (1995), I am arguing for the co-production

I have shown how depression is related to various historical traces, and have commented on some problems of 'translating' those traces into the language of depression. Furthermore, I have shown that many of these traces involve different biology, treatment, and general social context.

2. Critiques of depression

In this literature review I have so far presented studies that take a historical or an ironic commitment to the construction of depression in the DSM-IV, a commitment that takes seriously the idea that 'depression need not have existed' (Hacking 1999: 19-21). In this section some of the studies presented actively state that 'depression is quite bad as it is', which represents a 'reformist' or 'unmasking' level of commitment in Hacking's typology. The reformist agrees with the ironist that there is not much we can do about depression, even if it needs to exist, but goes farther than the ironist and argues that there are some modifications that could make depression less of a bad thing. The unmasker, on the other hand, does not strive to change depression or other conditions but "to undermine them by exposing the function that they serve" (Hacking 1999: 20). Some authors presented in this section go further and support the statement that we would be better off without depression altogether.

Universalization

Sushrut Jadhav uses Atwood Gaines' sickness history approach[18] to undermine the indiscriminate cross-cultural application of depression (1996, 2000). His argument hinges on the DSM-IV's definition of depression being integrally related to the particular Western origins of depression. His study does not take a historical level of commitment to the construction of depression, but takes more of an 'unmasking' approach. He presents four fundamental problems with depression being a universally valid disorder,

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of the biology and the experience of depression rather than leaving 'disease' as an ahistorical fact unsuitable to anthropological inquiry.

[18] Atwood Gaines explicitly positions his 'sickness history' of the French illnesses *fatigue* and *triste/fatigue tout le temps* as a cultural constructivist approach. This sickness history "is a culture's historical experience with a particular form of distress which frames both folk and lay understandings and responses to contemporary experiences and concerns" (1991: 248). He re-presents Jackson's work on acedia, tristia, and melancholia to illustrate how *fatigue* and *triste* are the products of dynamic cultural histories. He presents this sickness history to undermine the idea of a unitary Biomedicine.

which I regroup in terms of the three issues of translation outlined in the interlude of the historical section. I will illustrate each of these three fundamental problems by alluding to other anthropological works on depression.

Translation: language

First there is the issue of the translation of language, "with attendant problems of translating emotion-related vocabulary" (Jadhav 1996: 269), that is inextricable from the 'interactive kinds' involved with the language of emotions and mental disorder. How to translate depression when certain groups, for example the Kaluli, do not have any relevant equivalent of depression whatsoever (Schieffelin1985: 107)? Is it possible and appropriate to compare depression, an internalzing term associated with loneliness, unhappiness, and despair in North America, with *yuu tsu* in Japan, an externalizing term associated with ideas of rain, darkness and clouds (Marsella 1980)?

Even when there exists a possible analogy for depression in another language, there may be key differences. For instance, an "essential feature" of a Major Depressive Episode, which is essential for the diagnosis of Major Depression is "a period of at least 2 weeks during which there is either depressed mood or the loss of interest or pleasure in nearly all activities" (APA 1994: 320). However, in certain cultural groups, such as the Ifaluk, the ethnopsychiatric category most likened to western depression does not include the symptom of 'joylessness' (Lutz 1985). Another symptom that is difficult to translate is that of guilt. In an early cross-cultural study of depression, Teja *et al.* compared the Indian and British experiences of depression (1971). The incidence of feelings of guilt was comparable in both locations, at odds with the assumption that guilt is largely found in areas with Christian heritage. Upon closer examination, 'guilt' in India was linked to a diffuse reference to "karmas", and the "individualized guilt" of the British sample was rare in the Indian samples. Although the incidence of the symptom was the same cross-culturally, there was a qualitative difference between the understanding of the term 'guilt' in the two locations. This qualitative difference does not make the symptom incidence amenable to comparison between the groups. These differences in subjective experience may ultimately preclude a meaningful comparison of depression and possible analogies

in other cultures – "translation is *the* central concern if culture is taken seriously" (Kleinman and Good 1985: 38).

Translation: body

This last issue of translation is also associated with problems of the translation of body and experience, including "cross-cultural variations in the definitions of selfhood" (Jadhav 1996: 269; *see also* Heelas 1981). In *Culture and Depression*, Catherine Lutz argues that, among the Ifaluk, emotion is not distinguished from cognition (Lutz 1985: 65). Lutz argues that emotions should be treated as "culturally constructed judgements" (1985: 65); and as such the symptom/disorder depression is not universal. The unsituated use of translated terms might obliterate key differences in the experiences and bodies (Raguram *et al.* 2001). For instance, depression requires a particular view of the self (Jadhav 1996). Certain peoples have normative selves[19] that may be drastically different from the normative psychological self of North America. With the Maori, emotions are experienced through various organs, not in a 'mind' (Smith 1981: 150). In Dinka religion, suffering is externalized, and the 'self' is believed to acquiesce to agents external to the individual (Harris 1989: 602). It is difficult to see the applicability of the largely psychological western tradition of Depression to the Maori or Dinka.

Translation: social context

The final issue of translation is that of the role of normality and pathology. One of the key contributions of anthropologists in this regard is noting that the presence of negative connotations of depression is not always the case, or that the stigma may vary cross-culturally (Weiss *et al.* 2001). A mental disorder must involve some distress or social dysfunction in order to be understood as a disorder – otherwise, any unusual mental process could be classified as a disorder (although this could be argued for the established diagnoses). The *DSM-IV* stipulates that a mental disorder is a "*clinically significant* behavioural or psychological syndrome or pattern" that is associated with distress or disability (1994. Emphasis added). Obeyesekere argues that in Sri Lanka, depressive

affect is not understood as an illness associated with distress or disability, but rather is the representation of one's disattachment to the world, a positively valued state in Buddhism where the source of all suffering is understood to be attachment (1985: 147). Sri Lankans "can generalize their despair from the self to the world at large and give it Buddhist meaning and significance" (1985: 140). Another example is with the Thai peasants, who also do not understand grief as illness but through the rubric of religion (Kleinman and Good 1985: 37).

However, if depression and antidepressants have been co-produced in a particular cultural context, but elements of that context are in the process of becoming global attributes – what will happen to these local ways of experiencing and expressing selves and misfortune?

> [H]as the osmosis of cultures, for example, lead to a more unified way of expressing experience of illness in terms of denoting depression? Many of the conditions previously considered as culture specific and fairly independent from depression tend now to be associated with depression [i.e. brain fag in Africa, and neurasthenia in China]"(Sartorius 1986: 10).

Medicalization

Kirk and Kutchins, professors of social work, "trace how the psychiatric profession struggles with various political constituencies to create categories of mental disorder and to garner support for their official acceptance" in their book *Making us crazy: DSM, the psychiatric bible and the creation of mental disorders* (Kutchins and Kirk 1997: 16). They are not constructivists in the sense of many of the anthropologists presented in this section, because they are arguing that the DSM is the result of bad science rather than arguing that the political and social processes are always inseparable from science in the production of knowledge. I include them here because they expose several ideas key to other critiques of depression as medicalization. Medicalization is the encoding, in biomedical terms, of various aspects of human experience. Kirk and Kutchins focus on

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[19] I use the concept of "normative" self (Rose 1996: 3) to indicate that although there are heterogeneous 'selves' *intra*culturally, these selves are subject to a common "normativity", or to hegemonic discourses,

two aspects of this medicalization: the 'increasing pathologization of everyday behaviour', and how the 'DSM pathologizes those who are undesirable and powerless.' I will use this division to structure this section.

Pathologization of everyday behaviour

Several critics argue that depression, along with other psychiatric diagnoses, represent the 'pathologization of everyday behaviour', which involves the encoding of human suffering – often embedded in life histories, families, and political, cultural, and economic contexts – in medical discourse, such as that of the DSM-IV. Some very strong critiques of the medicalization involved with psychiatric diagnoses emerged in the anti-psychiatry movement of the 1960s and 1970s. Other studies that blur the boundaries between unmasking and rebellious commitment involve several critiques involving the notion of social control, in which mental disorders were argued to be serving the purposes of an elite. The DSM in particular has been criticised as "basically a political and social device for protecting the status quo, for maintaining a social order that supports exploitation and injustice" (Rothblum *et al.* 1986 *quoted in* Kirk and Kutchins 1992: 11).

On the other hand, several of the historical studies presented in previous sections argue from a more 'enabling' view of diagnosis – using a more Foucauldian version of power where the diagnoses may be used positively or negatively, diagnoses may enable "the articulation of social, moral, and cultural debates" (Lutz 1995: 533; Oppenheim 1991). However, the issues of medicalization, as Rabinbach remarks, might not have had the same implications in the 19th century, as conditions were able "to move fluently between science and literature [which] reveals the tendency of nineteenth-century thinkers to equate the psychological with the physical and to locate the body as the site where social deformations and dislocations can be most easily observed" (1992).

Sociologists have also argued similarly, often taking a reformist commitment to depression and its diagnosis. In Karp's study of a self-help group for affective disorders,

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within that culture.

he states that there was a definite ambivalence felt about labels associated with diagnoses (1992: 150). However, "most persons [in the group] were thankful for being able to name what they experienced" (1992: 149), although it is important to note that the culture of this particular group valued the diagnoses. Karp stated that his respondents rarely traced the cause of their depression beyond themselves and their immediate experiences to social and cultural organization and change as a whole (1994: 363), and concludes that the loosening of social connections are integrally related to the experience of depression in America. Similarly, Joan Busfield argues that the idea of diagnosis as presented in the DSM-IV should not be dropped entirely because it does not explicitly specify pathology, but that we should strive for more social explanations rather than stay with individualist ones (Busfield 1996).

Two clinical psychologists also comment on the ambivalent attitude of the women in their study towards their diagnosis of depression – on the one hand, it enabled treatment and the reinterpretation of experiences, but it also came with some stigma that the women overcame in their narrative accounts through the comparison of depression to physical illness such as diabetes (Gammell and Stoppard 1999: 117). However, some of the women in their study did not integrate the diagnosis of depression comfortably in their lives, because they did not like the association of depression with weakness (1999: 120). Gammell and Stoppard conclude by outlining the positives and negatives of the medicalization of women's distress. The positives included the validation that 'something is really wrong with me' (Stoppard 2000: 194), access to treatment, and less personal responsibility for getting better. The negatives included disempowerment, social situations slighted, perception as a person at-risk for reexperincing the distress, and medicalization. They finish by concluding that, overall, the diagnosis is 'bad as it is' because the treatment with antidepressants was ultimately disempowering (Gammell and Stoppard 1999: 125).

And ethnicity

> The belief that intellect and responsibility entail depression has been common since the Romantic period, with its emphasis on the terrible price to be paid by

> melancholic genius and morbidly heightened sensitivity. Depression was the result of the higher biological and social role of the white man; it became a symbol of his responsibility and superiority. Even in the Seventeenth century, depression implied refinement, as Ben Johnson tells us in *Every Man in His Humour*. (Littlewood and Lipsedge 1997: 96)

Kutchins and Kirk also argue that this medicalization is especially detrimental to certain groups of people. They show how racism is entrenched in the history of mental disorders and continues to be entrenched in the DSM-IV (1997). In studies done in the early part of the 19th century the general consensus emerged that depression could not occur in indigenous peoples and it was claimed that "the Negro mind does not dwell upon unpleasant subjects: he [sic] is irresponsible, unthinking, easily aroused to happiness, and his unhappiness is transitory, disappearing as a child's when other interests attract his attention" (Green 1914 *in* Littlewood and Lipsedge 1997: 66). Some people believed that the lack of depression was due to a missing 'sophisticated' psychological vocabulary, thus the depressive affect in non-western cultures was frequently associated with somatization. However, this association waned somewhat with studies with many groups, which concluded that depression was experienced among these groups *via* somatic idioms, although the culture had a language rich in psychological terms. When Kleinman worked in China and Taiwan in the 1970s, he came to the conclusion that the 'somatic' diagnosis of neurasthenia was more appropriate than the diagnosis of depression in this part of Asia (1986: 95), because "[d]epressive feelings ... are not simply suppressed by Chinese [through somatization] and expressed by Americans, but rather are different feelings" (1980: 171, *see also* Kleinman and Kleinman 1985).

And gender

> Psychiatry turns women's discontent with patriarchy and protests against the social order into mental symptoms of underlying pathology. The interests of patriarchy are then served very well (Russell 1995:155)

Janet Stoppard, a clinical psychologist, began her work on depression when she noticed the overrepresentation of women among depressed patients (Stoppard *et al.* 2000). She

now focuses on the inadequacy of current theories explaining the gender differences between men and women in North America, Britain, and Australia. Her focus is on research on depression, and is a rather reformist focus: "if theories of depression are to have emancipatory potential for women, research needs to be informed by a perspective in which women's experiences are viewed as a valid sources of knowledge in their own right" (Stoppard 1999: 87). Stoppard critiques all psychological theories for not taking into account the body; conversely she critiques biological theories of depression (variously based on hormones, neurochemicals, or genetics) for not taking into account women's experiences.[20] She posits a material-discursive approach that involves the concurrent examination of women's embodiment (which in her book, she traces through a lifecycle framework of adolescence, marriage and motherhood, and aging) with the ways in which women draw on "shared cultural beliefs and practises" to construct "meaning, identity, and subjectivity" with respect to their depression in terms of their embodied experiences (Stoppard 2000: 22). Likewise, Richters also evidences a reformist level of commitment, by arguing that the role of the anthropologist is to amplify the protest that may be evident in women's narratives of distress such as depression as these may "be an effective means for women to get at least (part of) their view on reality accepted" (Richters 1996: 240).

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[20] Her arguments are worth repeating because they criticize both sides for not taking an approach wherein the biological, social and psychological are understood to be co-produced. As such they are a useful corrective for the particular focus of this essay: the definition in the DSM-IV. Psychosocial models are gender-blind in that they posit a general stress-diathesis model; women-centred approaches in psychology have often the examination of either 'feminine' personality traits or a more relational approach to the definition of the 'self' (including the 'silencing the self' hypothesis of Jack, D.C. (1991)); social models of depression examine the meaning of social factors, such as affordable housing, child care, poverty, employment, social support in the aetiology of depression. She divides this latter model into two streams: one, typified by the work of Brown and Harris' *Social Origins of Depression: A study of psychiatric disorder in women* (1978), overcomes problems with the stressful event model (namely that not everyone with the same event get depressed) by examining the *meaning* of the events, rather than a simple event/non-event, in people's lives. Although Brown and Harris focussed on women, they were doing this anticipating developing a more generalized theory. This contrasts with the work of McGrath *et al.* who examined what social factors specific to women's lives, such as the 'double day' of work, gender inequities in pay rates, abuse, and poverty related to single parenthood (1990). Stoppard also critiques the narrow way in which the word 'social' is used in these mainstream researches, and advocates a more historical account taking into account structural and material conditions, that is at the same time embedded in the meaning of individual lives (Stoppard 1999: 84).

Big Pharma

'But I'm *not* a Depression, I tell you!' said Variously-Named Distress. 'I'm a—I'm a'

'Well! *What* are you?' said Pharma Rhetoric. 'I can see you're trying to invent something!'

'I – I'm stress,' said Variously-Named Distress, rather doubtfully, as s/he remembered the number of changes s/he had gone through that century.

'A likely story indeed!' said Pharma Rhetoric in a tone of the deepest contempt. 'I've seen a good many stresses in my time, but never one meeting such DSM-IV criteria as that! No, no! You're Depression; and there's no use denying it. I suppose you'll be telling me next that you have never been reduced by an antidepressant!'

'I *have* been reduced with an antidepressant, certainly,' said Variously-Named Distress, who was very truthful, 'but stress is reduced by antidepressants as much as is depression, you know.'

'I don't believe it,' said Pharma Rhetoric; 'but if stress is reduced, why then it's a kind of Depression, that's all I can say.'

This was such a new idea to Variously-Named Distress, that s/he was quite silent for a minute or two, which gave Pharma Rhetoric the opportunity of adding, "You are reduced by antidepressants, I know *that* well enough; and what does it matter to me whether you're stress or Depression?[21]'

[21] I should note that the marketing strategy of pharmaceutical companies has largely involved the rhetoric of differentiation between different disorders (see Greenslit m.s. for the differentiation between Prozac and Sarafem, which are both names for fluoxetine, although packaged differently for depression and premenstrual syndrome respectively, by Eli Lilly). One has to wonder, however, that when advertisements for compounds such as Zoloft list 4 indications, whether this differentiation is anything but a (successful) rhetorical marketing strategy (Healy 1997).

A common thread in studies criticizing depression as medicalization involves the level of involvement of the pharmaceutical industry in depression. This is most clear in Healy's work (1997), but is also criticized in the doctoral work of Greenslit, who resists the idea that, due to direct to consumer advertising in the United States, learning about depression may only involve learning about antidepressant drugs (m.s.). This is even mentioned in *The Noonday Demon*, where it is bluntly stated: "news that depression is a chemical or biological problem is a public relations stunt "(Solomon 2000: 399).

Summary for the critiques of depression

I have presented some reasons why depression, as defined in the DSM-IV, along with antidepressants and a particular biology, is 'quite bad as it is.'

3 Case Studies of antidepressant use in North America and Britain

> We believe in the chemistry of depression with a stunning fanaticism (Solomon 2001: 430).

In the historical studies in the previous sections, variations on 'nerves' are perhaps traces of depression (Oppenheim 1991). Also, variations on 'nerves' are possible anthropological analogies for depression (Lock 1991; Richters 1996; Kleinman1986). These variations seem to be an example of how, given certain active assumptions, the body can provide 'natural symbols' through which this distress is expressed. In the DSM-IV's definition of depression, however, it is a different sort of 'natural symbol' that appears – one that is based on molecular biology. In the previous section I presented some critiques of this particular biology. Furthermore, in anthropology, there are several fascinating recent accounts of the impact of molecular biology, namely genetics and neurochemistry, on the definition of the 'self' (Rabinow 1996; Rose m.s.).

In light of all this theory, do people actually interpret their distress in terms of a biological (genetic or neurochemical) depression, using the 'natural symbols' produced in biological psychiatry? I know for certain at least one friend who does, but how generalized is this? Who is the "we" to whom Solomon is referring in the quote at the beginning of this section? A few anthropological, sociological, and psychological case

studies of depression among 'whites[22]' with depression in Canada, the United States, and Britain provide some tentative answers to this question. I argue that the answer has to do with antidepressants, however there is not enough information to suggest this with any certainty. Even put together, these case studies leave me with the impression of the Cheshire cat – the overall picture somehow escapes the eye.

Narratives of depression

Two qualitative case studies show that at least some groups do interpret their depression in terms of biochemistry. David Karp, a sociologist, did work on depression in the late 1980s and early 1990s in the urban United States. He first spent nearly two years as a participant-observer in a self-help group for affective disorders, and then conducted 35 interviews with people who had been diagnosed and treated by mental health care practitioners. In a New Brunswick town, Deanna Gammell, together with her supervisor in clinical psychology Janet Stoppard, interviewed nine women who had been diagnosed with depression and prescribed antidepressants.

Both studies found that there was invariably a time when the participants had no name for their experiences, and that "initial interpretations of their problem focused on their situations rather than on their selves" (Karp 1994: 355). Gammell and Stoppard note that, like the women in Vivenne Walters' study of health issues important to Canadian women (Walters and Denton 1997), the women in their study often normalized their distress before it was named by a mental health care professional.

Often, it was a mental health care practitioner who first gave a name to the distress – eight of the nine women in the study by Gammell and Stoppard had not labelled themselves as depressed until they were diagnosed (Gammell and Stoppard 1999: 116), although the women in the study now claimed that they were depressed long before the diagnosis. In this latter study, the researchers argue that the "verbal content of the women's accounts became more "medicalized" at the point when they began to describe

[22] That the respondents were 'white' was specified in all the studies described in this section. The one exception was in Karp's interviews, in which one of the 35 interviewees was 'East Indian'.

seeking help from a health professional because of their feelings of ill health" (Gammell and Stoppard 1999: 121), and that at this point tended to differentiate their 'true' selves from the depression (118) following the disease model of mental illness.

In the narratives of participants in both studies, depression was intimately linked to the participants' biochemistries. In the American study, Karp "heard the rhetoric of biochemical cause again and again in the [self-help] group meetings" (Karp 1992: 153), although it was often raised in discussions involving personal blame. As such, the rhetoric of biochemical cause went hand in hand with a rhetoric of victimization involving the unpredictable fate of biochemistry (Karp 1992: 153-154). However, this victim stance was also enfeebling and so the biochemical causes were only presented as partial. As mentioned by Gammell and Stoppard, this biological model enabled group members to draw a distinction between a 'true self' and a 'illness self' (Karp 1992: 155), such that the 'true self' might not be subject to the victim-stance of the 'illness self'. In the Canadian study, "[a]ll the women stated that their depression was caused by a "chemical imbalance in the brain," the explanation for depression given by their physician (Stoppard *et al.* 2000: 90).

It is difficult to generalize on the use of neurochemicals from any of the studies, because each of these findings is based on narratives that are elicited in a particular study context or (in the case of the self-help group) microculture. It is difficult to tell how much information in the Gammell and Stoppard study was affected by the framing of the study – physicians do not invariably explain the exact diagnosis and treatment rationale to the patient, although this seemed to be the case in the study (1999). Furthermore, the women patients' use of medicalized vocabulary, such as 'disease' and 'cure' might have been related to the researchers' framing of the study in terms of diagnosis and treatment (Gammell and Stoppard 1999: 121). With respect to Karp' study with the self-help group, this particular group has a stated goal "to educate the patient and the public on the biochemical nature of mania and depression" (group publication cited in Karp 1992: 153). However, these findings are very different from the findings from two other studies of depression.

Yvette Scattolon did the research component of her Ph.D. in clinical psychology among rural women in New Brunswick who had never sought treatment for depression (1999). She found that only 3 of the 14 women who were part of the study referred to a biological cause for their depression/distress. Scattolon found that the most common way of overcoming depression was 'getting on with life' despite any poverty or adverse family circumstances. Women named daycare services and self-help groups to be two very important things that would help them in their coping. Likewise, Sushrut Jadhav, a psychiatrist and anthropologist, examined the causes of depression mentioned by recently diagnosed men and women in a working-class English city (m.s.). There was so little mention of biochemistry and/or neurochemistry in these accounts that, although he had originally though it might be a cause mentioned by the interviewees, he discarded the notion altogether in his analysis.

Why these differences in the invoking of neurochemical causes? The date of the study does not seem to be relevant, as the studies are interspersed from the late 1980s to the late 1990s. The diagnosis does not seem to be the cause of the difference either, because each of these studies focussed on a group of individuals with depression. The participants in Karp's, Gammell's and Jadhav's studies had been diagnosed with depression by a physician before admittance into the study. Because Scattolon wanted to study the experiences of women before they had sought professional help, the women in her study were not diagnosed by a physician; however, to counteract the idea that these women were not depressed, she had them complete the Beck Depression Inventory and found that "the majority of women interviewed fell within the moderately to severely depressed range" (1999: 192).

There are several differences in the sample that might be at play: geographical location, economic means, and level of education. Both Karp's and Gammell's study involve urban and, in Karp's study, well-educated participants (32 out of the 35 people interviewed had a college education). Jadhav's study was among working-class Britons in an urban area who were presenting to a hospital. Scattolon's study involved rural women

with variable levels of education, with about a third not having completed high school, with the rest having completed high school and half of these having done courses at the university level. But what would these geographical and educational differences change? For one, accessibility perhaps, with the women in Scattolon's study living in remote areas, with marginal economic means having difficulty obtaining childcare and/or transportation to rural health care centres. This doesn't seem to be the case, however, with Jadhav's sample. The level of education was mentioned in all of the studies, perhaps as an indicator of economic means or scientific sophistication. However, the biomedical viewpoint has been widely disseminated in Canada and Britain -- most of the women in Scattolon's study were aware of the medicalized accounts of depression although they chose not to use them in their own accounts (Scattolon 1999: 140), and the 'Defeat Depression Campaign' from 1991 to 1996 in Britain raised awareness of the medical model of depression (Paykel *et al.* 1998; cf Jadhav and Littlewood 1994)[23].

A more likely candidate is perhaps involvement with the medical profession. As Yu-Wen Ying mentions in her study of "Explanatory models of major depression and implications for help-seeking among immigrant Chinese-American women" (1990), the conceptualization of the distress as psychological or physical greatly influenced the appropriateness of self- and family-help versus medical services. In other words, by sampling people who visit physicians, one may be biasing one's sample towards those people who believe their distress is physical. Doctors may be associated with antidepressant drugs, and if antidepressants are not desired or seen as suitable the doctor may not be consulted (Scattolon 1999). In Scattolon's study, "the women understood their depressive experiences as being a part of their lives, something to be endured and coped with. In keeping with this understanding, they explained these depressive experiences as arising out of the particular circumstances of their everyday lives, conditions [i.e. poverty, abuse, confidentiality issues in rural areas] which could not be changed by seeking professional help" (Stoppard 2000: 188):

[23] Clearly there is not a unidirectional flow of information; Allan Young has shown in a related article on stress how vernacular knowledge is reproduced as scientific knowledge (1980).

> I still think that my problem would have been that I had no money, I don't even care for help. I know what my problem is. But if I went to the doctor, he probably would have said, well, you're depressed or something. And I probably would have said, yeah, and said, what's the use, whatever, you know. I know I am depressed and I don't know how you can help there. Are you going to give me some money? (Stoppard *et al.* 2000: 93).

Jadhav's study, however, involved people who visited a psychiatrist for their troubles, but only a few even tangentially mentioned elements of biochemistry. However, Jadhav emphasized that (physical?) nerves were a common explanation. So what makes the physical attribution a neurochemical one in particular? Perhaps it is the involvement of antidepressants. Both the studies in which participants emphasized neurochemistry also involved participants who had taken psychopharmaceuticals. In Karp's study of the self-help group, every participant that he had met had at some point taken psychotropic medications (1992: 162); in Gammell's study, all but one of the interviewees had taken antidepressant medication (Gammell and Stoppard 1999). Conversely, Scattolon's study was among women who had never sought or received professional help (including antidepressants) for their distress, and Jadhav's study was conducted among those recently diagnosed and who were presenting to a psychiatry department for the first time.

Surveys of depression

While these studies shed light on the use of neurochemical models of mind among those who are depressed, in chapter three we will be looking at vernacular documents, of which some have been written by people having suffered from depression, and others not. There are at least two studies to examine 'public attitudes' towards depression, although neither of them are in Canada. The first, illustrates (a) that there is some overlap in 'depression' in the vernacular and in the DSM-IV, that (b) this distress is seen as suitably treated by the medical profession to some extent and (c) there are very different attitudes to different medications and treatments. This first study was conducted in Australia, and involved interviews with over 1000 people as part of a randomized Population Survey Monitor of the Australian Bureau of Statistics in August 1995 (Jorm *et al.* 1997). The

interview was based on a vignette of 'Mary's' or 'John's depression (based on DSM-IV criteria). Participants were asked the question "What would you say, if anything, is wrong with Mary/John?" To which 39% replied depression, and 22% stress (participants were allowed to give multiple answers). When asked, "How do you think Mary/John could best be helped?" the most common responses were to see a doctor (44%), see a counsellor (23%), and to talk with friends and family (20%). The participants were then asked whether a series of medications, treatments and professionals would be helpful or harmful. All of the medications were rated more harmful than helpful except the category 'vitamins, minerals, tonics and herbal medicines,' which was perceived as more helpful (57%) than harmful (3%). By contrast, antidepressant medications were perceived as less helpful (29%) than harmful (42%).

The second study was part of the 'Defeat Depression Campaign' in Britain, and involved monitoring changes in public attitude over the course of the campaign (Paykel *et al.* 1998). This study also demonstrates (a) that depression is a frequently used term, and also that (b) biological interpretations of depression are not the most salient cause of depression in the public. The study involved approximately 2000 people in 1991, 1995, and 1997. About one quarter of the sample said that they had experienced depression themselves, and over half of the sample said that they knew someone else who had experienced it. Many causes of depression were listed, of which 'Biological changes in brain' came in twelfth position each year, with just over a third of the participants agreeing (participants were allowed multiple answers, and bereavement, unemployment, financial problems and stress were the top four reasons mentioned, with approximately 80% of the sample agreeing that these were a cause of depression). To the statement that people suffering from depression should be offered antidepressants, only 16% of respondents agreed in 1991, although this increased to 24% in 1997. The proportion of respondents who thought that antidepressants were very/fairly effective also rose from 46% to 60%. Conversely, approximately 90% of the sample thought that people suffering from depression should be offered counselling, and 86% of respondents thought that it was very/fairly effective.

Beyond North America, England, and Australia

What do antidepressants do where depression does not have as culturally-salient a history? Arthur Kleinman studied the effectiveness of antidepressants in treating a subset of patients in 1970s China (1986). These patients were diagnosed with neurasthenia by Chinese practitioners, and with major depressive disorder by western-trained psychiatrists. Kleinman states that in the first session, when the antidepressants were given, the patients were told what 'depression' was, in simple terms, and were told that the antidepressant drugs would alleviate the depression. This mirrors the information given to patients in North America when antidepressant treatment is begun. The results of the study indicated that these drugs were quite effective in treating the symptoms, with 82% of patients reporting some improvement. Most patients did not feel that their illness, neurasthenia, had been helped – the social impairment associated with neurasthenia had not improved (Kleinman 1986: 91). Furthermore, most patients in this sample in China did not accept the diagnosis of depression. A few patients even commented that the medication must be "antineuasthenia medication" (Kleinman 1986: 164), not antidepressant medication. These patients did not interpret their illness in terms of depression, and did not think antidepressants relevant. Insofar as the small presence and heavy stigmatization of depression, this is rather similar to the situation in Japan. Indeed, pharmaceutical companies did not even bother getting their products on the Japanese market because the market was perceived to be too small (Landers 2002). However, in the late 1990s, 'education' about depression has increasingly taken place via media such as television shows and newspaper ads, and has presented depression using the phrase "*kokoro no kaze*, which loosely translated means 'the soul catching a cold'" (Landers 2002: A10) instead of the more stigmatized term. Supposedly depression is now 'catching' (Landers 2002). As in Canada, 'informational' sites about depression, such as the website utu-net.com, are presented as unbiased sources of information even when they are funded by pharmaceutical companies such as GlaxoSmithKline (Landers 2002). This education of depression that is funded by pharmaceutical companies tends to involve the use of neurochemical bodies.

Conclusion

The definition of depression in the DSM-IV has been co-produced with antidepressants, and a particular biology and spell of science – all within a particular style of reasoning. It is possible that the efficacy of antidepressants facilitates a particular experience of biology, namely neurobiology, in depression.

CHAPTER 3

CLAIMS FOR THE EFFICACY OF ANTIDEPRESSANTS

Outline of this chapter's journey

> Any attempt to legitimize a particular approach as the correct one is at best of limited value, since it is inextricably bound to a thought collective. Neither the style characteristic of opinions nor the technical skills required for any scientific investigation can be formulated in terms of logic. This sort of legitimization is therefore possible only where it is actually no longer required, namely among persons whose intellectual constitution is thought-styled in common (Fleck 1979 [1935]: 35).

The value of antidepressant therapies is clear in the self-authenticating and self-vindicating style of reasoning of one thought collective[24]: biological psychiatry. I have shown how antidepressants are inextricable from depression and a particular biology as they have all been co-produced in this style of reasoning. Another epistemic thing (Rheinberger 1997) that has been co-produced in this web is 'efficacy'. I outline this particular configuration of efficacy in the first section of this chapter as well as how this configuration attributes value to antidepressants *within* this style of reasoning. However, many therapies for depression do not originate within this thought collective. To provide a useful foil for the claims made about pharmaceutical antidepressants, I will focus on St. John's Wort. St. John's Wort is an herb, used 'for centuries in traditional medicine' for depression, that exploded onto the North American market in the mid-1990s. I will examine how value is attributed to both pharmaceutical and herbal antidepressants *outside* this particular thought collective of biological psychiatry. To do this, I will first explain my methodology (Section 2). I will then examine the value imbued by science (Section 3) as well as explain the value imbued by other realms (Section 4) and how these different realms are juxtaposed (Section 5). Finally, I will comment on how these different realms of value might affect the experience of the neurochemical body (Section 6).

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[24] See page 9 for a definition of thought-collective.

Authenticity and Realms of Value

> Consumers want authenticity but don't know what that is. – Michael Langenborg, Vice president of marketing for the tea company *Traditional Medicinals* (MarketResearch.com 2001).

The notion of authenticity, as defined by Walter Benjamin (1968 [1936]), is one that is useful for examining the claims made about the efficacy of antidepressants in vernacular documents within Canada. Benjamin argues that reproductions of works of art lack one thing that an original has: authenticity, including a particular social history and a 'presence' in time and space. This authenticity bestows an aura on the original – the aura entailing temporal and spatial distance that is unapproachable (Benjamin 1968 [1936]: 243; see also Appadurai 1986: 41)[25]. With respect to antidepressants, one may ask: what is this original, unapproachable entity? I argue that, in vernacular documents, there are three main regimes of value that are invoked to bestow an aura of authenticity on antidepressants: science, nature/history, and personal experience[26]. Sometimes this magical aura must be related to the antidepressants very explicitly, in a way similar to the workings of contagious magic. The extent to which the different auras can be attracted and the extent to which this relationship must be explicitly built are different for herbals and antidepressants in significant ways.

SECTION 1: Efficacy

Efficacy and Pharmaceutical Antidepressants in a Particular Thought-collective

> One of the characteristics of the modern era is that nothing can be said to be discovered until it has been through the process of a randomized trial (Healy 1997: 5).

Efficacy, in biological psychiatry, is practically synonymous the randomized controlled trial (RCT). The RCT, although originating in the move for therapeutic reform as early

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[25] It is because of this unapproachability that I switch from 'spell' in chapter two (within the thought collective of biological pscyhiatry) to 'aura' in chapter three (outside the thought collective of biological pscyhiatry.

[26] These three realms of value were extracted from analyses of the vernacular documents included in this section; as they were also spontaneously used by participants at a 'townhall consultation' held by the

as during the 1950s in the United States, was only institutionalized in government regulations for new drugs in Canada, the United States, and Britain in the 1980s (Marks 1997). However, a RCT by itself is not sufficient to constitute 'efficacy'. It must also be corroborated with biological research: for depression this involves neurobiology and biochemistry. Finally, this efficacy must also be observable in clinical practice. The juxtaposition of the RCT, the biological research, and the clinical research validate the efficacy in a sort of 'harmony of illusions' (Young 1995 *from* Fleck 1979 [1935]) or a gestalt of entries in a crossword puzzle (Haack 1999). Thus, the intervention must also be persuasive in order for it to be relevant (Young 1977: 195). For antidepressants this seems to require the merging of statistical, laboratorical, and clinical styles of reasoning.

The RCT

> '...and even Stigand, the patriotic archbishop of Canterbury, found it advisable—'
> `Found WHAT?' said the Duck.
> `Found IT,' the Mouse replied rather crossly: `of course you know what "it" means.'
> `I know what "it" means well enough, when I find a thing,' said the Duck: `it's generally a frog or a worm. The question is, what did the archbishop find?'
> The Mouse did not notice this question, but hurriedly went on...

Despite the 'harmony of illusions,' the use of the RCT is contentious in biological psychiatry, especially in the case of antidepressants. There have been many recent critiques of it in mainstream and well-known scientific journals such as *Science, JAMA, and the British Journal of Psychiatry* (Enserink 1999; Walsh *et al.* 2002; Moncrieff 2002; Leuchter *et al.* 2002; Kupfer and Frank 2002). In the clinical practice guidelines for major depressive disorder, published by the APA, several of these critiques are presented (2000). One critique relates to the publication bias that may keep negative results from public journals—one meta-analysis demonstrated that the strongest predictor of outcome

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Natural Health Products Directorate of Health Canada, they seem to be reasonably valid for 'natural health products' as well.

in a comparative trial with SSRIs was sponsorship (Freemantle *et al.* 2002). Another factor is the question of outcome measures in the RCT:

> a report of "efficacy" could refer to symptom reduction (e.g., reduction in the frequency or severity of major depressive disorder symptoms), response (e.g., reduction in major depressive disorder symptoms below a threshold), or prevention of relapse. Data often come from short-term (6- to 12- weeks) efficacy trials that may not reveal whether treatments are effective over the medium- and long-term (APA 2000: 43).

"It" may vary more than simply 'frog' or 'duck.' However, the most-accepted measure of efficacy is a "50% reduction in symptoms (usually on a 17-item Hamilton Depression Rating Scale [HDRS][27]; this is the standard for approval of antidepressants by regulatory bodies" (Kennedy *et al.* 2001: 38S). In other sources, there are also concerns about whether the statistics are done on the 'intent to treat' group rather than just the trial completers because if trial participants leave the study because of side effects, this affects the efficacy of the antidepressant drug (Moncrieff 2002).

The Placebo Effect

The most important issue presented in the APA guidelines, however, is the placebo effect. The 'placebo effect' is the catch-all explanation for the patients in the placebo arm of the RCT who 'get better[28].' The placebo effect has been blamed for both inflating and diminishing the 'true efficacy' of antidepressants (APA 2000: 43). Two aspects of the design of the RCT are questioned in particular: the double-blind and the 'enriched' design. There is also some debate as to whether the RCTs are really double-blind because of the distinctive side effects of certain antidepressant medications (Greenberg and Fisher 1994). Indeed, the difference between response rates for placebo and antidepressant are reduced if an 'active placebo' – a placebo that produces these same distinctive side effects – is used instead of an inert one (Moncrieff *et al.* 1998). For instance, psychologists Kirsch and Sapirtein conducted a meta-analysis of nineteen

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[27] This Hamilton Depression Rating Scale is what was developed on the basis of the effects of imipramine (Healy 1997; see chapter two).

antidepressant drug trials and concluded that 75% of the efficacy of antidepressants could be attributed to the placebo effect (Enserink 1999). The remaining 25% could be due to the unblinding effects caused by the distinctive side effects[29]. Furthermore, the whole situation is made more complicated by an often-unstated protocol—the 'enriched deesign'— for RCT with placebos (Trussel 1999). In this design, all participants are given a placebo during the first period of the trial; only those who do not respond to the placebo in this first period are then randomized into the treatment or the placebo arm. Thus, the trial as reported only includes those participants who did not respond to a placebo. However, Walsh *et al.* found that this design did not affect the results of the trials (2002).

The placebo itself has been reconfigured by its participation in the RCT and the styles of reasoning in biological psychiatry: it has come to be understood in terms of neurobiology. Leuchter *et al.* suggest that the brain function involved with placebo responders, as measured by quantitiative electroencephalography, has different brain activity in the early part of treatment, as opposed to placebo non-responders and medication responders and non-responders (2002). This may have to do with the self-authentication of the styles of reasoning in biological psychiatry – everything is assessed in the terms of brain biology and chemistry. We have also seen this self-vindication of the neurobiological associations of depression in a specific style of reasoning in biological psychiatry with respect to atypical depression. It was concluded, in the studies examined in chapter two, that the larger placebo effect in atypical depression reflects underlying biological differences between the mechanism of atypical and non-atypical depression. However,

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[28] There is some debate as to the numbers of placebo responders in these RCT, with an estimated 30% of patients in the placebo arm of RCT responding. This ranges from approximately 12% to 50% with a gradual increase in this rate over the years (Walsh *et al.* 2002).

[29] In a meta-analysis of Prozac trials, Greenberg argues that Prozac's efficacy was linked with the severity of side effects – "[w]e found that the more side effects, the better it did" (Enserink 1999). Interestingly, in studies of St. John's Wort, the rates of side effects were linked to the choice of control arm; in studies comparing St. John's Wort to imipramine, side effects for St. John's Wort were approximately 60%, whereas in studies comparing St. John's Wort to placebo the side effects sometimes were under 10%. A meta-analysis of the safety of St. John's Wort reports that in studies that compared St. John's Wort with placebo, only 4.1 percent reported side effects, whereas in studies that compared St. John's Wort with antidepressants, 19.8 reported. (Stevinson and Ernst 1999).The efficacy may also have to do with increased patient compliance due to fewer side effects – for instance in a study where 63% of those on imipramine

could this be the reflection of the fact that if one's mood is reactive then perhaps a positive interaction with the doctor and research staff might elevate one's mood more than if one's mood is less reactive? Is this 'atypicality,' in fact, selecting for responders to general treatment such as this positive interaction, rather than a specific treatment with a different underlying biology? This reflects a focus on the neurobiological "mechanisms of action" (Kupfer and Frank 2002: 1854).

Before its use in the RCT in the 1950s, the placebo was simply an "inert sham given to individual patients" (Kaptchuk 1998: 1723). After its incorporation into the RCT, it "became a hodge-podge of non-linear, difficult to quantify, remnants collected under the rubric of the dummy control of an RCT. Anything that threatened the fastidious detection of a predictable cause and effect outcome was conveniently disposed of in a repository labelled the 'placebo effect'" (Kaptchuk 1998: 1723). As such, the placebo effect subsumed various elements, including any natural remission, regression to the mean, conditioning behaviours, imagination and hope, concurrent interventions and therapeutic activities, and patient-practitioner relations (1724). In other words:

> Any health outcome that does not fit biomedicines' primary explanatory model of physical cause-effect reasoning can operationally be dumped into the wastebasket of placebo effects (Kaptchuk *in* Horrigan 1998: 102).

The placebo is now a 'grab-bag' of anything 'other' than the biological mechanism of action of the antidepressant[30].

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and 39% of those on St. John's Wort experienced side effects, the discontinuation rates were 16% and 3%, respectively (BBC NEWS 2000b).

[30] Several anthropologists have mirrored this biological: other dichotomy in their explanations of how various healing interventions might work. The initial term in the dichotomy, variously named 'curing' (Kleinman 1980), 'technical efficacy' (Sevigny 1999), or the effects of 'specific medical treatment' (Moerman 1979), is related to biology and the efficacy of biomedicine and for the most part is completely left as natural and unproblematic. The other type of efficacy is generally the one that is perceived as more suitable for the anthropological endeavor (Moerman 1979: 60) or is the one to which only anthropological methods can shed light (Kleinman 1980). This is variously termed 'healing' (Kleinman 1980), 'symbolic efficacy' (Sevigny 1999), or the effects of 'general medical treatment (Moerman 1979). Indigenous (Kleinman 1980; Moerman1979) and/or 'alternative' (homeopathy – Sevigny 1999) health practices are argued to mostly be efficacious in terms of the second type of efficacy (Kleinman 1980: 359, 360;

Value, efficacy and non-pharmaceutical antidepressants

This particular version of efficacy, one that centres on the RCT, now sets the tone for assessing the efficacy of any other treatment. This is seen in the clinical practice documents published by the American and Canadian psychiatric associations (2000 and 2001). In the Canadian Psychiatric Association's *Clinical Guidelines for the Treatment of Depressive Disorders* (2001), the article on psychotherapy emphasizes using RCT to assess the efficacy of therapies as diverse as group therapy, marital therapy, and self-help groups (Segal *et al.* 2001). Recommendations for practice are given based on the framework of evidence-based medicine, which is based on RCT as the final arbiter (see Table 4).

Table 4: Chart explaining evidence for treatment recommendations in the Canadian Psychiatric Association's *Clinical Guidelines for the Treatment of Depressive Disorders* (2000: 36S).[31]

Level of evidence	**Criteria**
1	Metaanalysis or replicated randomized controlled trial (RCT) that includes a placebo condition
2	At least 1 RCT with placebo or active comparison condition
3	Uncontrolled trial with 10 or more subjects
4	Anecdotal case reports

Line of Treatment	**Criteria**
First-line	Level 1 or Level 2 evidence plus clinical support
Second-line	Level 3 evidence or higher(a) plus clinical support
Third-line	Level 4 evidence or higher (a) plus clinical support
Not recommended	Level 1 or Level 2 evidence for lack of efficacy

(a) Treatment with higher levels of evidence may be listed as lower lines of treatment due to clinical issues such as side effect or safety profile.

An example radio show

This gold-standard is clearly shown by constant invocation of the RCT as the final arbiter of value for any treatment. Take, for example, this CBC transcript from a news segment

Moerman 1979; Sevigny 1999). This second type of efficacy is also the one where the placebo effect is foregrounded (Moerman 1979; Kleinman 1980: 362).

[31] St. John's wort is recommended on the basis of 'level 1 evidence' for minor depression (Kennedy 2001: 55S).

headline from June 1st, 1998, at 10pm entitled: 'Your Health: St. John's Wort'. It presents a RCT study, at its inception, for St. John's Wort. I will present the controversy ensuing from the result of this RCT in Section Three. This transcript provides an excellent introduction to St. John's Wort as well as an illustration of the status of RCT as final arbiter. Here is the cast of characters, taken from the transcript available on CBC.CA:

Host: *ALISON SMITH*
Guest: *DR. BRIAN GOLDMAN, "The Health Show"*
SHARON KATZ, Claims Depression Alleviated By Herb
DR. JOEL RASKIN, Centre For Addictions And Mental Health
DR. JONATHAN DAVIDSON, Study Director

Here is the transcript for the show:

ALISON SMITH: A study is being launched to test whether a herbal remedy can help treat depression. The test will involve St. John's Wort, an herb that is available in Canada. Researchers hope to evaluate its usefulness by comparing it to a drug that is currently used to deal with the effects of depression. For more on the story, here's the "Health Show's" Dr. Brian Goldman.

DR. BRIAN GOLDMAN / "THE HEALTH SHOW": Sharon Katz is a 20- year survivor of breast cancer. But when she suffered a bout of depression, it was more than she could take.

SHARON KATZ / CLAIMS DEPRESSION ALLEVIATED BY HERB: You don't do anything. You don't want to do anything, you want to die.

GOLDMAN: Her doctor recommended an anti-depressant, but Katz opted for a herbal remedy called St. John's Wort. And now she's letting the company that makes the brand she takes use her story.

KATZ: I got my life back. I started doing things I had done before, I started investigating new things. Um I could be with my friends again.

GOLDMAN: St. John's Wort, an extract of the Hypericum plant, has been used in liquid and pill form in Europe for centuries as a folk remedy for anxiety and insomnia. North American use is starting to skyrocket. According to a 1997 survey by the Non-Prescription Drug Manufacturing Association, as many as 600,000 Canadians have used St. John's Wort. And it's attracting the attention of psychiatrists like Dr. Joel Raskin, head of the Depression Clinic at the Centre For Addictions And Mental Health in Toronto.

DR. JOEL RASKIN / CENTRE FOR ADDICTIONS AND MENTAL HEALTH: People are taking it. I think as psychiatrists, we need to assess its usefulness.

GOLDMAN: Several studies have shown St. John's Wort to be more effective than placebo, and in some cases as effective as anti-depressant drugs. But questions regarding its effectiveness have led to a new study to try and end the debate. This press conference pegged to the American Psychiatric Association meeting in Toronto, announced a new study by the U.S. National Institute of Mental Health.

DR. JONATHAN DAVIDSON / STUDY DIRECTOR: If it doesn't work out, well we've answered a very important question, because it is on everybody's minds.

GOLDMAN: Meanwhile, with many brands of the herbal remedy as close as the nearest pharmacy and health food store, some researchers worry about the risks of side-effects. Known side-effects include headaches, nausea and allergic reactions. In high doses, it can also cause photosensitivity, or skin rashes when skin is exposed to the sun's rays.

> RASKIN: People assume because it's natural that it's safe, and that there are no side-effects or no problems with it. Yes it's natural, but so is arsenic and that can have problems.
>
> GOLDMAN: The American study will compare St. John's Wort to an anti-depressant from the family of drugs that includes "Prozac" [and to placebo]. Researchers hope to recruit more than 300 volunteers. It'll be at least three years until scientists learn whether an ancient remedy is the latest weapon in the battle against depression. For CBC News, I'm Dr. Brian Goldman in Toronto.

Efficacy and traditional medicines in North America

What are some of the issues in applying 'efficacy,' as produced in part in the style of reasoning of biological psychiatry, to treatments not produced by that thought collective? One issue is that the efficacy of the treatments will only be visible in terms of this particular version of efficacy. In Van der Geest's and Whyte's compilation of 'pharmaceutical anthropology' (1988), Etkin states that the investigation of indigenous medicinal plants, to date, has been "constrained by a bioscientific perspective that circumscribes as appropriate inquiry or medicinal plants only pharmacologic investigations" (1988: 317) and that this can conceal more relevant assessments of both efficacy and effect. For instance, side effects are often noted in clinical investigations of new drugs, but in different cultural contexts it is important to note in whose worldview do certain effects rate as being merely secondary (see also chapter two for comments on the culturally salient 'its' – rather than simply frogs and worms). Furthermore, even if the RCTs are used to assess the efficacy of 'natural' medicines, there is no guarantee that positive trials will be persuasive because there may not be any corroborative biological theories or clinical data (Patel 1987; Richardson 2000):

> Resistance to information that directly contradicts conventional wisdom is generally high. It may be useful to recall, in this context, that as recently as 1919 it was 'not unusual for well-known physicians to get up and leave when medical papers were being read which emphasized the germ theory of disease' (Kao and Kao 1979 in Patel 1987: 669).

Anthropological comments on efficacy

> No longer was it sufficient for a therapy to work: it had to be better than a placebo (Kaptchuk 1998: 1724).

Placebo-centricity, along with a particular assumed biological aetiology of mental disorder, makes sense in the web that has been woven for depression, antidepressants, biological psychiatry, and efficacy. Clearly, neurobiology does not *need* to be involved even in this web[32]. The expectation that there is an intermediary between the cause and the effects is one that is embedded in the particular mechanistic tone of much of scientific thought and is only one possible model of causality (Needham 1976).

There are other ways in which antidepressants might be thought to 'work.' As Ted Kaptchuk comments about humoral medicines, medical interventions transform by resonating with and evoking the "unfolding of preexisting potentials" (in Horrigan 2001: 102), rather than acting on any particular biological mechanism. Furthermore, rather than 'working' at the level of the individual, brain, or population, antidepressants could be imagined to 'work' at the level of the family and/or community (Lewis 2000: 181; Boddy 1988: 12). Rather than having a clinician determine the efficacy, the patients, their caregivers, or the community might have the final say on whether a treatment worked (Lewis 2000: 181-4; Kleinman 1980:354).

However, these comments open up many questions as to the application of the particular notion of efficacy to non-biomedical therapies in North America. Which types of efficacy are important? As Gilbert Lewis asks, do we advocate a double standard of efficacy, where one type of efficacy is used for biomedical therapies but not others? (1993) I do not attempt to answer the question but, rather, document the types of claims that are made for pharmaceutical and herbal antidepressants.

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[32] The commonality of biology was invoked to explain the effectiveness of antidepressants; however, the dose of antidepressant required varies widely across ethnicities, and antidepressants in other cultures may be more effective than in North America (Escobar and Tuason 1980: 49) or less effective (Kleinman 1986: 164) for non-biological reasons.

SECTION 2: Methodology[33]

Before looking at the claims that are made for St. John's Wort and pharmaceutical antidepressants, it may be useful to outline how I came to analyse these particular claims. For this project, I have relied mostly on unobtrusive observation in the form of archival research. In this archival research, I have looked at vernacular documents that present claims for the efficacy of herbal and pharmaceutical antidepressants. Documents provide a useful site for investigating the types of claims that might be made about antidepressants' efficacy because there is no risk of spurious explanations used to answer the anthropologists' questions; all the claims are made 'spontaneously' for the purposes of the research.

A hypothesis

The initial site for research was an Internet site. The website depression.com was selected because it was the first 'hit' listed in response to a search on Google.ca for 'depression' in December of 2000. The first informal stage of analysis involved reading through the information provided on treatment options for depression on this website and reflecting on how this information was organised. The formal stage of the analysis used a modified version of grounded theory: thematic analysis. Thematic analysis first involved 'open coding'; in other words, the information provided for each treatment was broken down into groups and then these groups were labelled. It then involved 'axial coding' or establishing links between these groups. Based on the categories and information presented on this website, I developed a tentative theory of the types of claims made about different types of antidepressants. I also reflected on the possible reasons for the particular claims made, including that of government regulations and marketing rhetoric.

Gathering more material

I chose to further sample other sources of information– those given out on the bottles at pharmacies and health food stores in Montreal. According to statistics on the U.S. market in 2000, these two types of stores represent over 60% of the total market for

[33] My ability to articulate the types of analysis and issues of validity are largely due to the exceptionally clear presentation of the methodology section in Yvette Scattolon's doctoral thesis (Scattolon 1999). The

herbal supplements – and St. John's Wort was the fifth best-selling herb (MarketResearch.com 2000; see Table 5).

Table 5: Share of U.S. Herbal Supplement sales by retail outlet type, in percentages (MarketResearch.com 2001)

Type of retail outlet \ year	2000	1998	1996
Health and natural product stores	50.1	36.1	38.8
Mass market	26.9	47.2	44.1
-- Mass merchandisers (e.g., Wallmart)	(10.4)	(22.6)	(19.2)
-- Drugstores	(10.3)	(20.7)	(20.4)
-- Food stores	(6.2)	(3.9)	(4.5)
Direct Selling	17.6	12.6	13.2
Mail Order/ internet	5.4	4.1	3.9

Since 2000, I have been collecting and perusing pamphlets about St. John's Wort, however, in April 2002 I did a more thorough survey of the types of information available in two pharmacies and two health food stores, all either in the downtown area of Montreal or on 'the Main' street. One pharmacy had almost no information, so I did not include it in my analysis. I compared and contrasted the information from the three remaining sites to that of depression.com, in order test my hypotheses. Then, because I was looking at advertisements and pamphlets for St. John's Wort found in the stores, I needed a comparison for pharmaceutical antidepressants. For contrast, I did a content analysis of the advertisements for antidepressants in *The Canadian Journal of Psychiatry* from 1982 to 2002. There were many similarities between the advertisements for herbal and pharmaceutical antidepressants, however, the differences led to a foray into both the vagaries of the history of Canadian advertising regulations and advertisements for pharmaceutical antidepressants. During the discourse analyses of these materials, I found that the trope of the implicit and explicit ranking of evidence along the lines of evidence-based medicine recurred. I consulted the practice guidelines for depressive disorder issued by the American and Canadian psychiatric associations (2000 and 2001, respectively) to find out if evidence-based medicine is also involved there.

terms used in the analysis section and the organization of the criteria for validity are drawn from her work.

Testing

By May of 2002, I had looked mainly at commercial sites, so when a friend, who was recovering from a hospitalization for depression, recommended a best-selling book *The Noonday Demon,* I decided it would be an ideal non-commercial site for testing my hypotheses. By this time, I was also fascinated by the interpretations made about the results of randomized control trials for St. John's Wort. Thus, I did some more unobtrusive observation, namely 'following the fact' of the (negative) results of large, American clinical trials testing the efficacy of St. John's Wort for depression. I followed the fact in newspaper articles, on the radio, and in postings on the Internet. These sources of information led me to substantially revise the hypotheses I had previously developed.

Throughout the course of my research, several other types of research became integrated into this analysis. Throughout the course of this research, I also had the advantage of meeting people who were knowledgeable about St. John's Wort – when I went to local pharmacies and health food stores to document the information available on the packaging of the products, some store personnel (including pharmacists) were quite interested in my project and were very helpful in telling me the sort of information available in their store. Finally, during the write-up stage of this project, I had a chance to test the 'face validity' of some of my hypotheses – namely assessing whether these hypotheses might make sense to those making the claims. I participated in a 'townhall consultation' on the *Standards of Evidence* document (Health Canada 2002) to be used in the new Office of Natural Health Products in Montreal at the end of November 2002. While I went as an observer to find out what types of claims were being used in the evidence document, I ended up more as a participant observer. All attendees of the conference were seated in groups of eight for discussion, and at my particular table there were the presidents of two small Montreal herbal product companies, an operations manager for a manufacturer of herbal products, a marketing consultant, and three government officials from Health Canada. In addition to discussions with these other participants during the breaks, I took on the role of the group 'secretary,' documenting the group discussions. This gave me a chance to process the discussion and to clarify comments. In one discussion section, our group pondered the question: "If a traditional-

use product has been studied scientifically but the scientific evidence does not support its traditional use, should a traditional use claim still be acceptable?" (Health Canada 2002: 23). Thus, while recording the discussion I was able to ask the group questions of clarification that were 'incidentally' also related to my hypotheses. This last project also gave me a chance to think of my findings with respect to St. John's Wort in terms of the claims that were being made about 'natural health products' in general.

I should note that although neither the website nor book are Canadian, they are both available in Canada. A second note is that I tried to conduct most of the research in English, so I could avoid issues of translation. However, there are a few pamphlets that did not have English versions available. In those cases, if I quoted from them, I list the original French statement in a footnote. Also, in the natural food stores, although I began asking about the types of products available for depression in their store, the language of conversation often ended up as a blend of French and English or, in one case, ended up entirely in French.

One limitation of my research is that the vernacular sources that it analyses are mostly commercial. However, I get the impression that most of the sources of information about St. John's Wort, and to some extent antidepressants as well, *are* commercial[34]. The production of information about these substances is intimately linked to their value as commodities. While there have been many exposés of the extent to which the pharmaceutical industry influences both consumers and physicians, the situation for St. John's Wort is little different when it comes down to commercial involvement, albeit on a totally different scale. In addition to all of the informational pamphlets produced, herbal product industries also sponsor workshops within health food stores; at one of the health-food stores I visited, one of the clerks told me that most of what she knew she had just learned because a Quebec herb company had recently come in to the store to do a workshop on depression (Fieldnotes April 8th). However, the commercial involvement for both pharmaceutical and herbal antidepressants is made complicated by government

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[34] Nathan Greenslit presents some quandaries about 'when finding out about depression means only finding out about pharmaceutical antidepressants (m.s.).

regulations. Pharmaceutical antidepressants cannot be truly advertised directly to the consumer for two reasons: they are prescription-only and also indicated for a Schedule A disorder. These 'Schedule A conditions' listed in the Canadian Food and Drugs Act are conditions, such as cancer, anxiety, hypertension and depression, for which nobody may advertise to the public. Technically, advertising for herbal antidepressants should also avoid the word 'depression.' For the most part, especially on the label, the advertisements comply. In 2000, the bottles would talk about "stability of mood" (Voegel), "nervous disorders", "anti tension" (Jamieson), and "insomnia, neuralgia, and nervous tension" (Jamieson). Some had different types of claims, including "Traditional Healing Medicine for improvement in symptoms of nervous tension and restlessness due to fatigue or nervous excitability" (Wamplole) and also included a drug identification number (DIN). However, by the spring of 2002 there was at least one brand that had "Anti-Depressant" written in bold at the top of the bottle (Swiss Herbal). When I asked the natural products specialist at the pharmacy about this difference, he stated that he thought it was illegal but because the regulations were changing there was no reprimand because claims for osteoperosis had been showing up in the previous six months, as well (Fieldnotes April 8th 2002). However, the marketers of herbal medicine in general avoid this stipulation of not making 'depression' claims for their product by making the claims (i) in pamphlets, not on the label, and (ii) not making claims about their product directly, but about St. John's Wort in general. I was informed by a small business president that there are few repercussions because this is a 'loophole' (Fieldnotes November 28th).

SECTION 3: THE AURA OF SCIENCE

> The Queen turned crimson with fury, and, after glaring at her for a moment like a wild beast, screamed `Off with her head! Off--'
>
> `Nonsense!' said Alice, very loudly and decidedly, and the Queen was silent.

Interpretative flexibility[35]

> There are only two kinds of therapies – those that work and those that don't work.
>
> – Robert Califf, of the Hypericum Depression Trial Study Group (BBC 2002).

---

[35] (Epstein 1996: 333)

In this particular web of efficacy spun in biological psychiatry, how does St. John's Wort fit in? In the middle of April 2002, several Canadian news sources listed headlines such as "St. John's Wort poor remedy for depression, study finds" in the *National Post* (Vallis 2002) and "Depressed? Skip St. John's Wort: Study" on CBC.CA (CBC News 2002). News sources in the United States (Vedantam 2002a; Hall 2002) and Britain (BBC NEWS 2002) had similar headlines. These headlines were all prompted by a study published in the April 10th issue of the *Journal of the American Medical Association* (*JAMA*) entitled "Effect of *Hypericum perforatum* (St John's Wort) in Major Depressive Disorder" (Hypericum Depression Trial Study Group 2002). The outcome of this study was summed up by the director of the National Centre for Complementary and Alternative Medicine who stated that the "primary message of this particular study is St. John's Wort does not seem to help patients with major depression with moderate severity" (CBC News 2002) – a rather unambiguous statement.

The controversy

> The Queen had only one way of settling all difficulties, great or small. `Off with his [the Cheshire cat's] head!' she said, without even looking round.....
>
> The executioner's argument was, that you couldn't cut off a head unless there was a body to cut it off from: that he had never had to do such a thing before, and he wasn't going to begin at HIS time of life.
>
> The King's argument was, that anything that had a head could be beheaded, and that you weren't to talk nonsense.
>
> The Queen's argument was, that if something wasn't done about it in less than no time she'd have everybody executed, all round. (It was this last remark that had made the whole party look so grave and anxious.)
>
> Alice could think of nothing else to say but `It belongs to the Duchess: you'd better ask HER about it.'

How to "interpret" a beheading? How to "interpret an RCT, used in biopsychiatry to settle all therapeutic controversies, large or small"? Other than the CBC article, which does not describe more than the study itself, all of the news articles proceed to present the arguments of various actors in support of or against the conclusions of the 2002 study.

First, the articles present defenders of the herb who state that the study was a waste of money anyways because it was testing the efficacy of St. John's Wort for something that it has never been claimed to treat: moderate to severe depression; most claims by advocates of the herb have been in terms of mild to moderate depression (Vedamtam 2002a; Vallis 2002; Hall 2002). Secondly, the defenders argue that neither St. John's Wort nor the accepted pharmaceutical antidepressant (Zoloft) fared better than the placebo on the main measure of the study: the rates of full remission were 24% for St. John's Wort, 25% for Zoloft, and 32% for placebo – so there is no reason to conclude that St. John's Wort is ineffective because, by the same token, Zoloft should be declared ineffective (Vallis 2002; Vedantam 2002a).

In a classic example of self-vindication, these arguments were rebutted by the researchers involved in the study. They argue that the general inefficacy of Zoloft was probably due to the low dosages of Zoloft used in the study (Vedantam 2002) but that, in this study, Zoloft was shown more effective than St. John's Wort because Zoloft did better on 'secondary measures'(Vallis 2002; Hall 2002; Vedantam 2002a):

> Zoloft did bring more relief than the fake pills on some measures, and doctors deliberately kept dosages low in order to minimize risk of side effects. As for St. John's wort, however, the researchers found "a complete absence of trends suggestive of efficacy" (Hall 2002).

Finally, the researchers say that they cannot comment on St. John's Wort's efficacy in mild depression because the study did not examine mild depression (Vallis 2002).

In turn, "[s]upporters of St. John's wort, who include some psychiatrists and the herbal products industry, lashed out at these conclusions and accused the researchers of selectively focusing on results that fit their preconceived opinions" (Vedantam 2002a). The advocates of the herb argue that the Zoloft dosage (100mg) is the middle of the recommended dose range, etc..

The 2002 study was notable for two reasons. One was that this study compared St. John's Wort to both a placebo and a pharmaceutical antidepressant (sertraline or Zoloft);

the previous thirty or so controlled trials had either compared St. John's Wort to a placebo or to a pharmacological antidepressant but not to both. The previous studies done with placebos were criticized because even if St. John's Wort was found to be superior to placebo, there was no way of knowing if the herb was comparable with pharmaceutical antidepressants. The previous studies done with pharmacological antidepressants were criticized because if St. John's Wort was shown to be comparable to a pharmaceutical antidepressant such as imipramine, "St. John's Wort may not have proven to be nearly as good as imipramine in the treatment of this cohort of patients, but imipramine may have proven to be nearly as bad as St. John's Wort" (Deltito and Beyer 1998: 349). Other criticisms of prior trials have ranged from the size of the trial or the appropriateness of dosages or controls to comments that the trials were led by inexperienced researchers, or comments about the not taking measurements of the plasma levels of the antidepressant used as a control (Shelton *et al* 2001)[36]. There are even implications that some studies demonstrating the efficacy of St. John's Wort have been doctored because the side effect rate for the herb is below what one would expect as a baseline rate even without drugs (Deltito and Beyer 1998), and "[w]ith nearly 30 controlled comparisons in depression, the lack of any published negative reports suggests systematic biases in evaluation or reporting" (Shelton *et al.* 2001: 1984).The second reason for the relevance of the 2002 study was that it was designed to be *the* authoritative American trial – the study was the result of a six million dollar (US) project sponsored by the US National Centre for Complementary and Alternative Medicine (NCCAM) (Vedantam 2002a).

The imbalance

> Both products failed – only one got the headlines as being a failure. Both products have been shown in previous studies to be effective. This study doesn't

[36] The majority of the early trials for St. John's Wort are published in German and were virtually unknown in North America until the mid 1990s. In reading the American literature, one gets the impression that these earlier studies, "mostly from Europe" (Hall 2002) or "virtually all conducted in Germany" (Deltito and Beyer 1998), are not good enough for American standards. One reason is simply that the German studies do not use DSM-IV criteria put out by the American Psychiatric Association but use, rather, the criteria that most of the international community uses: the International classification of Diseases (Deltito and Beyer 1998). The criticism seems to extend further than this, however.

> invalidate Zoloft – it certainly doesn't invalidate St. John's wort. – Mark Blumenthal, founder of the American Botanical Council (Vedantam 2002).

> The findings cast doubt on marketing-fueled folk wisdom that has helped turn extracts of a short, yellow-flowering weed, formally known as Hypericum perforatum, into a consumer-health phenomenon with $180 million [US dollar] in sales in 2000. – News article (Hall 2002).

Here, as in Joseph Dumit's study of brain technologies (m.s.), Steven Epstein's study of HIV drugs (1997), and Evelleen Richard's study of Vitamin C (1988), the same scientific 'fact' is interpreted by different actors in nearly opposite ways. By following this 'fact' in newspaper publicity, and the fact that the negative findings are differently attributed to pharmaceutical and herbal antidepressants, I have shown that herbal and pharmaceutical antidepressants have different access to the value of 'science.' In a way, Zoloft is untouchable as an antidepressant while St. John's Wort, conversely, "has not earned its membership yet in the club," as stated the lead researcher in the 2002 *JAMA* article (Hall 2002).

I am not focussing on the scientific studies themselves because, as Evelleen Richards concluded in her study contrasting the assessment of Vitamin C and interferon for Cancer in the 1980s, the imbalance does not necessarily occur at the level of the study design and data crunching (Richards 1988: 684-685; *see also* Jones 2000). I will argue that the imbalance has something to do with the social life of St. John's Wort and its current baseline associations in the North American vernacular. I will provide an answer to this question by analyzing several vernacular sources of information available on these antidepressants from a website, books, advertisements, and product packaging. Foucault comments that one element of excluding certain elements of a discourse is by "a division and a rejection" (1971: 216). The 'science': 'other' dichotomy is one way of *a priori* excluding St. John's Wort from having a stable aura of science.

Divide and Conquer: the website Depression.com

The website was selected by a quick (0.18 second) search for "depression" in Google.com (8Dec00), a popular search engine. This search resulted in over two million hits, with the first hit being www.depression.com (see Figure 4).

Figure 4: First hit for search for 'depression' in Google.com.

Depression.com | Latest **Depression** News, Information & ...
... Health Highlights. How does **depression** affect the brain? Read about the biochemical roots of this illness. ...
Description: Gives an overview on types of **depression** and provides advice about living with a depressed person....
Category: Health > Mental Health > Disorders > Depression
www.depression.com/ - 29k - Cached - Similar pages

The site is a commercial site and a 'channel' of a company that sells prescription drugs, herbal medicines, and many other health-related products. The homepage provides a link to the 'health library' providing information on depression. I followed this link and found that the section on treatments was divided into two categories: Antidepressant Medications[37] and Non-Drug therapies (see Table 6). Each item listed in Table 6 is an embedded link to another page.

Table 6: Treatment pages listed on depression.com, in two categories.

Antidepressant Medications	**Non-Drug Therapies**
SSRIs	Cognitive Therapy
Tricyclics	Psychotherapy
Ludiomil	Support Groups
Remeron	Exercise
Effexor	Herbal [includes **St. Johns' Wort**]
Serzone	Dietary Supplements
Wellbutrin	Alternative
Desyrel	Phototherapy
MAO Inhibitors	Electroconvulsive Therapy

Divide and conquer: The best-seller the Noonday Demon

The Noonday Demon: An Atlas of Depression is written by Andrew Solomon (2001), a well-educated journalist who has struggled with depression. It is an approximately 600-

[37] Throughout this site and many of the other vernacular documents listed below, 'antidepressant' refers exclusively to pharmaceutical antidepressants, except in the section on herbal medicines where 'herbal antidepressants' and 'pharmaceutical antidepressants' were contrasted.

page book that was a *New York Times* best-seller. It is divided into twelve chapters with many on specific topics such as: populations, addiction, suicide, history, poverty, politics, evolution, and a general account of depression and the author's struggles and experiences. The third and fourth chapters of the book are entitled "Treatments" and "Alternatives", respectively, and contain a wide array of information on various treatment options presented (see Table 7), ranging from reports of scientific studies to personal stories of experiences with particular treatment options including the author's musings on the efficacy.

Table 7: Treatments listed in *The Noonday Demon*, in two chapters

Treatments	**Alternatives**
Talking Therapies	Exercise and diet
- (psychoanalysis)	Repeated transcranial magnetic stimulation (rTMS)
- (Cognitive-behavioural therapy [CBT])	Eye movement desensitization and reprocession (EMDR)
- (Interpersonal therapy [IPT])	Light-Box Therapy
	New Age Massage
Antidepressants	Outward Bound
- (SSRIs)	Hypnosis
- (tricyclics)	Sleep changes
- (MAOIs)	**St. John's Wort**
- (atypical antidepressants)	SAM-e
	Homeopathy
Electroconvulsive therapy (ECT)	Dance Therapy
	Support Groups
Faith	Surgery
	Ndeup ceremony

There are a few differences between the categorization of depression.com and that in *The Noonday Demon*. The main difference is that the depression.com site listed only antidepressants under the first category, whereas Andrew Solomon also listed psychotherapies, ECT, and faith. Except for 'faith,' Andrew Solomon's list of treatments is based on what is available through mainstream psychiatrists and psychologists as opposed to other mental health care practitioners.

The place of St. John's Wort

The use of dichotomization in these two popular sources of information for depression is not spurious; the myriad health strategies found in North America are often slotted into one of two categories: on the one hand, medicine (which I call 'biomedicine') and on the 'other' hand, 'alternative', 'complementary', 'traditional' or 'natural' medicine. This dichotomization of biomedicine: alternative tends, through analogy, to situate the categories within a political and moral economy whereby one term is given more value (Simone de Beauvoir 1989 [1949]). In the case of health care styles, the polarization of the hierarchical binary is very clear by the qualifiers (Haraway 1991); medicine is the institutionalized norm, and any other healing style may exist only with a qualifying term in relation to this norm (as 'natural' medicine). The qualities and discourses of the more dominant term, biomedicine, set the tone for both terms. While hypothetically speaking there might be an infinite number of ways in which people might make claims about whether and how a therapy works, in the context of therapies in Canada the gold standard is set in comparison to the treatments in the first column.

We can also see this in the presentation of the latest NCCAM study, where it was the inefficacy of St. John's Wort that made the headlines although neither St. John's Wort nor Zoloft were shown to be significantly more effective than the placebo. Perhaps this is because pharmaceutical antidepressants are so entrenched in the culturally authoritative realm of 'science' that they no longer need to prove their efficacy, as that is established *a priori* by their existence in this authoritative realm[38].

I tried to verify this dichotomy in the information provided in the pharmacy, however, I soon realized that it was difficult to make a comparison because there is no consumer packaging or other information presented for the pharmaceutical antidepressants – the drugs come in bulk bottles. The pharmacists duly informed me that they did hand out an information sheet the first time a person came in to fill a prescription. Indeed, this made

[38] 'Curiouser and curiouser', said Alice. This seems to be a case of the self-vindication of my original typologies. To use an analogy of Kuhn's work on scientific collectives, I am an individual trying to learn how to do social science, and so the process of working with a theory as long as possible until it is eschewed for a better solution.

a sort of marketing deal out of providing information – they handed out a computer printout of information, labelled a 'personalized reminder' (with the concluding sentence: "Your Pharmacist, a professional health adviser!"). This information basically consisted of instructions for use (see Figure 5). One pharmacist gave me a copy of the one package insert that he had (see Figure 6) for the one antidepressant that he sold in a pre-packaged box. This package insert also has no information on the efficacy of the antidepressant. It simply lists precautions and warnings. While this would, on the surface, corroborate with the already-present aura of science, it had more to do with its position as a prescription-only substance and the regulation that this entails. Conversely, the wide array of 'natural products' available at the pharmacy (see Figure 7b,c) consisted of many different packagings of St. John's Wort (see Figure 7a). These natural products had more information on their packaging, and there were also promotional leaflets lying around (Figure 1 in chapter 1, also Figure 8) although not nearly as many as at the natural food stores. On the internet and in the best-selling book many types of claims could be made for both types of antidepressants whereas in the pharmacy the types of claims appearing seem largely determined by government regulations (section 5).

The aura of science

Because of this overarching dichotomy in written texts, in which pharmaceutical antidepressants and St. John's Wort are on opposing sides, the way in which claims are made about them with respect to science are very different. I will demonstrate that there are three ways in which the claims invoke the aura of science – RCTs, quotes from scientific authorities, and biochemical mechanisms of action – although how this is done for each type of antidepressant is different.

Auras and the invocation of RCT: "Research Shows"

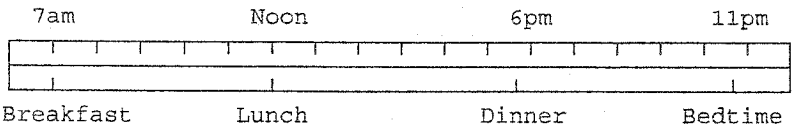
How were RCTs invoked in the claims for pharmaceutical and herbal antidepressants? I had thought, beforehand, that because the pharmaceutical antidepressants were produced as part of a biomedical style of reasoning, the use of RCT to support their efficacy would be expected. That was not the case in either the website or the book.

Figure 5: Pharmacist handout for Remeron, an antidepressant

1 avenue du Mont-Royal est
Montreal, Qc, H2T 1N4 514-284-1865
affilie a Pharmaprix -- visitez www.PharmaprixGuevin.qc.ca

Prepared for: XXXX

Medication : REMERON



This medication is for treating certain mood disorders.
Takes A FEW WEEKS to produce the full effect.

This medication may be taken with or without food.

Important: Follow the instructions on the label.
Do not use more of this product, or more often, than indicated...

In addition to its desired action, this product may entail some undesired effects (side effects):

- it may cause drowsiness or prevent sleep! - be careful until you know how YOU react;
- it may cause nausea or, in rare cases, vomiting;
- it may constipate - drink a lot, eat more fibre;
- it may increase your appetite;
- occasionally, it may cause weight gain;

These side effects do not happen to everyone! If you are bothered by a reaction that you believe is related to your medication (whether it is described above or not), discuss it with your pharmacist.

Avoid alcohol or products containing alcohol while using this medication.

This medication may interact with several other products. Check with your pharmacist before using it with other medications, vitamins or natural products.

If you think that you are allergic to this product or if you have rash, itching, a swollen face or breathing problems, contact your doctor immediately.

YOUR PHARMACIST, A PROFESSIONAL HEALTH ADVISER!

JEAN-FRANCOIS GUEVIN (2002/4/10)

Fiche personnalisée

Personalized reminde

Figure 6: Package insert for Remeron, an antidepressant

REMERON™
(mirtazapine) Tablets

INFORMATION FOR THE PATIENT

Read all of this leaflet carefully before you start taking this medicine.
- Keep this leaflet. You may need to read it again.
- **If you have further questions, please ask your doctor or your pharmacist.**
- This medicine has been prescribed for you personally and you should not pass it onto others. It may harm them, even if their symptoms are the same as yours.

What you should know about Remeron™:
- Remeron™ belongs to a group of medicines known as antidepressants.
- Remeron™ has been prescribed to you to relieve your symptoms of depression.

What you should tell your doctor before taking Remeron™:
- all your medical conditions, including a history of seizures, liver or kidney disease, heart problems, diabetes, low blood pressure, glaucoma (increased intra-ocular pressure), difficulties in urinating as a result of an enlarged prostate;
- any medications (prescription or nonprescription) which you are taking, especially monoamine oxidase inhibitors (e.g., phenelzine sulphate, moclobemide, tranylcypromine sulphate, or selegeline), or any other antidepressants, drugs to treat anxiety;
- if you are pregnant or thinking of becoming pregnant, or if you are breast feeding;
- your habits of alcohol consumption.

How to take Remeron™:
- It is very important that you take Remeron™ exactly as your doctor has instructed. Generally, most people take between 15 mg and 45 mg per day.
- Never increase the amount of Remeron™ you are taking unless the doctor tells you to.
- Some symptoms may begin to improve within about two weeks but significant improvement can take several weeks. Continue to follow the doctor's instructions.
- The tablets should be taken at the same time each day, preferably as a single bedtime dose before going to bed. You should swallow the tablets whole with water. Do not chew them.
- Keep taking your tablets until the doctor tells you to stop. The doctor may tell you to take your medicine for several months. Continue to follow the doctor's instructions.
- Do not take a double dose to make up for forgotten doses.
- If you forget to take your bedtime dose, do not take the missed dose the next morning. Continue treatment at bedtime with your normal dose.

When not to use Remeron™:
- Do not use Remeron™ if you are allergic to it or any of the components (see list of components at the end of this section). Stop taking the drug and contact your doctor immediately if you experience an allergic reaction or any severe or unusual side effects.

Precautions when taking Remeron™:
- Refrain from potentially hazardous tasks, such as driving a car or operating dangerous machines, until you are certain that this medication does not affect your mental alertness or physical coordination.
- Avoid alcoholic drinks while taking Remeron™.

Potential side effects of Remeron™:
- You may experience some side effects such as increase in appetite, weight gain, drowsiness or sleepiness, swollen ankles or feet, occasional dizziness or faintness (especially when you get up quickly from a lying or sitting position) and headache. In rare cases other effects may include seizures, attack of mania, yellow colouring of eyes or skin, rash, abnormal sensation in the skin (e.g., burning, stinging, tickling or tingly) or restless legs. Some side effects are temporary. Consult your doctor if you experience these or other side effects, as the dose may have to be adjusted.
- In very rare cases Remeron™ may cause a shortage of white blood cells, resulting in a lowering of the body resistance to infection. If you have a fever, sore throat, mouth ulcers or any other signs of infection, you should immediately contact your doctor.

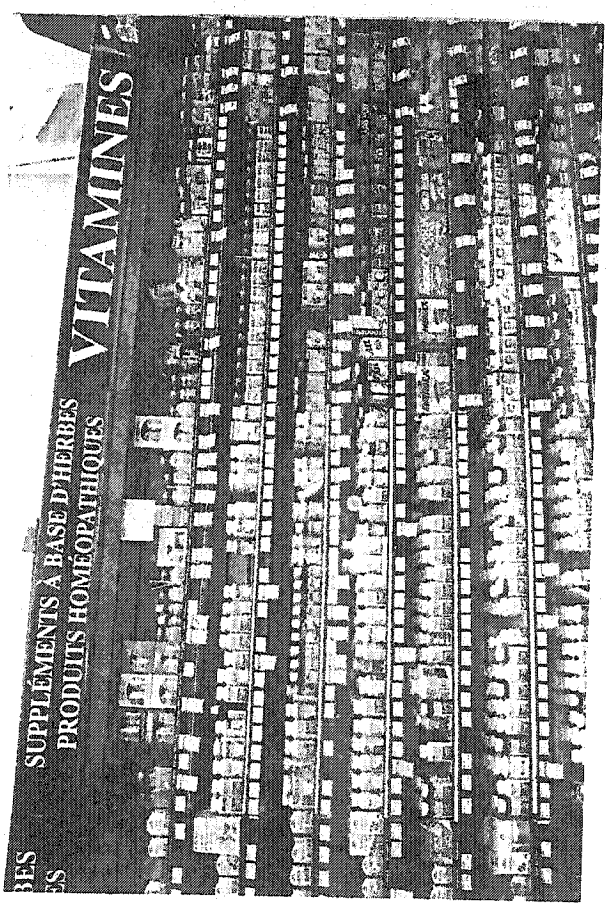
What to do in case of overdose:
- If you have taken a large number of pills all at once, contact your doctor or the nearest hospital emergency department or your nearest Poison Control Centre immediately, even though you may not feel sick. Show the doctor your pack of pills.

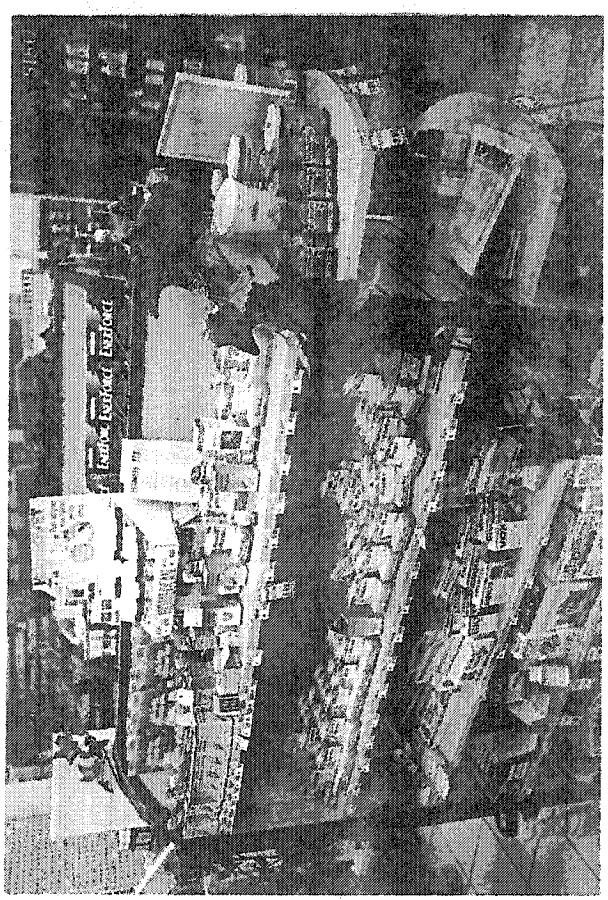
How to store Remeron™:
- Store at controlled room temperature, 15°–30° C (59°–86° F) in the original package.
- Keep Remeron™ out of the reach and sight of children.
- Do not use Remeron™ after the expiry date indicated on the package.

What does Remeron™ contain:
- Remeron™ is available as 30 mg (red-brown scored film-coated tablets). Mirtazapine is the active ingredient.
- Non-medicinal ingredients include: corn starch, hydroxypropyl cellulose, magnesium stearate, colloidal silicon dioxide, lactose monohydrate, hydroxypropyl methylcellulose, polyethylene glycol 8000, titanium dioxide, yellow and red iron oxides.

Who manufactures Remeron™:
Remeron™ tablets are manufactured by Organon Canada Ltd./Ltée.

Figure 7: Natural health product display at pharmacy





Swiss
Natural Sources

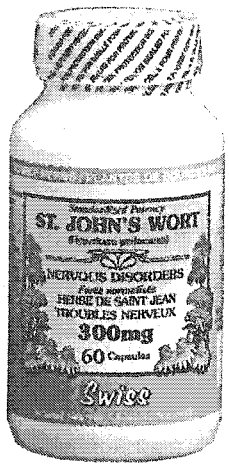
(SH 228)
 # 490037

STANDARDIZED ST. JOHN'S WORT

(Hypericum perforatum)

0.3 % HYPERICIN

For Nervous Disorders



WHAT IS IT?
St. John's Wort is a peren... ... with regular flowers which bloom from June until September. St. John's Wort has been used by humans for centuries for a wide variety of ailments including nervous disorders.

HOW DOES IT WORK?
St. John's Wort contains several active ingredients including hypericin, pseudohypericin, xanthrones and flavonoids. Hypericin and Xanthrones have been shown to contain monoamine oxidase (MAO) - inhibitors which retard the breakdown of the neurotransmitters. Norepinephrine and Serotonin in the brain. Too much serotonin makes people anxious and obsessive, while too little is believed to be the major cause of depression. St. John's Wort has been shown to subtly affect serotonin metabolism and alter brain chemistry similar to anti-depressants without any of the side effects of pharmaceuticals. Clinical trials involving hypericin extract showed improvement in depressive symptoms, including anxiety, apathy, insomnia and depression. In addition, the flavonoids have wound healing and anti-inflammatory activities and current research has recently focused on the anti-viral activities of hypericin and pseudohypericin.

WHY SHOULD I USE ST. JOHN'S WORT?
Herbal remedies for nervous disorders have been used in traditional medicine for centuries. St. John's Wort is one of the most effective and certainly the best researched herbal remedy for nervous disorders. Overall, St. John's Wort is indicated for varied ailments including:

- *Depression, psychological illness, mania, fear, nervous disorders, hysteria*
- *Sedative*
- *Anti-bacterial*
- *Anti-viral*
- *Bed-wetting, childhood nightmares*
- *Gastritis, gastric ulcers, inflammatory bowel disorders*
- *Inflammation*
- *Menstrual cramps*

HOW DO I TAKE ST. JOHN'S WORT?
Swiss Herbal Remedies supplies St. John's Wort 300mg capsules, standardized to contain 0.3% Hypericin.
Adults are recommended to take one capsule daily with food or as directed by a physician.
Avoid excessive exposure to sunlight, tanning or UV sources since hypericin may render the skin photosensitive.

Clinical trials involving St. John's Wort demonstrated improvement in depressive symptoms similar to anti-depressants without any of the side-effects of pharmaceuticals.

TRIPLE QUA ITY ASSURANCE PLUS

Your assurance of Swiss Herbal Premium Quality

A. *Our products are prepared to our specifications in ultra-modern facilities. We demand and get adherence to the highest standards of quality control from raw materials right through the entire manufacturing process. Samples are sent to independent laboratories to verify that our high standard of quality is met or exceeded for purity, potency, stability and freshness.*

B. *ALL SWISS HERBAL products are packed in Tamperproof pharmaceutical grade and recyclable containers.*

C. *We unconditionally guarantee all of our vitamins and supplements for purity, potency and stability for up to three years. To ensure that our rigorous quality standards are met or exceeded, we subject our raw materials and finished products to at least four different tests by independent laboratories. Ask your retailer for details.*

D. *We provide full written disclosure of all of our ingredients, excipients, coatings, binders, fillers, lubricants and disintegrating agents.*

E. *All of our vitamins and supplements are free of preservatives, artificial flavour, starch, added salt, corn and are gluten-free.*

F. *Our commitment to leadership is providing vitamins and supplements that incorporate the most recent findings of modern technology in vitamin therapy. These include nutritional research and clinical studies, involving nutritional research and clinical studies, involving bio-availability, chelation processes, digestive aids and synergistic combination. This commitment of leadership in product development in the vitamin health field sets SWISS HERBAL's premium quality supplements apart and provides creative product alternatives for today's selective health conscious consumer.*

(SH 228) # 490037

Figure 8: 'Informational' pamphlet for Swiss Natural Sources St. John's Wort

On the website, there were clear differences in the use of RCT as an aura-imbuing entity. This is not only illustrated in the introductory page for 'antidepressant medications' but also on the individual pages of each specified treatment. The introduction pages for the 'antidepressant medications' and the 'non-drug therapies' are very different in important ways. The introduction to antidepressants begins with a brief description of what can be expected of antidepressants, with no references to studies done or any other form of validation for that matter; the page simply contains descriptions of what should be expected and instructions on what to ask of a doctor. Conversely, the introduction to non-drug therapies begins with an invocation to "Research": "research shows that for mild depression, non-drug therapies are as effective as antidepressant medications". This introduction also presents excerpts from each of the 'non-drug' treatments' pages including descriptions of studies done, such as "[a] National Institute of Mental Health (NIMH) study showed that after sixteen weeks of cognitive restructuring training, 51% of those with mild to moderate depression reported significant improvement." This plethora of support contrasts strongly with the simple descriptions that were presented in the introduction to antidepressants.

The information presented on the introductory pages of depression.com demonstrates the difference in the auras of science already bestowed on the antidepressants by virtue of their social lives. These differences continued to be present in the subsequent pages (see Table 8). Interestingly, the only antidepressant page that even made reference to a scientific study pertained to the varied rate of sexual side effects amongst studies. With the exception of Cognitive Therapy and Electroconvulsive therapy, all of the non-drug therapies made reference to at least one study, and, furthermore, eighteen studies were described in some detail. SAMe, listed as an herbal therapy, is claimed to be the "subject of more than 100 studies". Likewise, while Andrew Solomon hardly presented any RCT in the chapter on "Treatment," RCT were presented in the section for St. John's Wort (2001).

Table 8: Information Provided on Therapies listed on depression.com

Therapy	Refer to a RCT	Describe a RCT	Time(s) of origin	Quotes
SSRIs	yes	no	1987-1998	no
Tricyclics	no	no	'older'	no
Ludiomil	no	no	1980, 'older'	no
Remeron	no	no	1996	no
Effexor	no	no	1993, 1997	no
Serzone	no	no	1994	no
Wellbutrin	no	no	'newer'	no
Desyrel	no	no	no	no
MAO Inhibitors	no	no	'oldest'	no
Cognitive Therapy	no	no	1976, B.C.E.	many. Grk Phil, 3 M.D.s,
Psychotherapy	yes	yes - 1	no	yes. 1 M.D.
Support Groups	yes	no	no	many. 1M.S., 2 M.D.
Exercise	yes	yes - 5	no	yes.
Herbal - ST. Johns' Wort	yes	yes - 1	'for centuries'	no
Herbal - SAMe	yes	no	1973, 'many years'	no
Herbal - Gingko	yes	yes - 1	no	no
Herbal - Kava Kava	yes	no	1990 US, 'for centuries'	no
Dietary Supplements	yes	yes - 3	no	Paraphrase 2 M.D.s
Alternative -Aromatherapy	yes	yes - 1	no	no
Alternative -Massage Therapy	yes	yes - 1	no	no
Alternative -Music Therapy	yes	yes - 1	in Bible	no
Alternative -Relaxation	yes	yes - 1	no	yes. 1M.D.
Alternative -Acupuncture	yes	yes - 1	no	no
Phototherapy	yes	yes - 2	1980's	yes. 1 (quip)
Electroconvulsive Therapy	no	no	no	yes. 1 M.D.

Quoting scientific authorities

Two other examples of the differences in the 'auras of science' between herbal antidepressants and pharmaceutical antidepressants are in the use of quotes from medical authorities and the explanation of the mechanism of action of the antidepressants. On depression.com, validating quotes were not used at all for the pharmaceutical antidepressants; however, a total of eleven medical doctors and one master of science were quoted or paraphrased in support of the Non-Drug Therapies. In *The Noonday Demon*, probably related to Andrew Solomon's journalism background, the primary link to science was not through referencing RCT but rather through the extensive quotations and paraphrasing of various authorities, including biomedical ones. However, this held true for both pharmaceutical and herbal antidepressants.

Mechanisms of Action

> It [ST. JOHN'S WORT] is thought to work in a similar way to anti-depressants known as serotonin reuptake inhibitors which relieve depression by increasing the availability of serotonin, a neurotransmitter associated with mood (BBC NEWS 1999).

Although the mechanisms of action are posited for both pharmaceutical and herbal antidepressants, they are posited differently, with the most salient being the way in which they are either phrased as fact or probability. On depression.com, the pharmaceutical antidepressants had a specific section entitled "Mechanism of Action," whereas the 'Non-drug therapies" did not have this specific section even though several of these therapies listed mechanisms of action. For the pharmaceutical antidepressants, it was only acknowledged on one of the nine pages that the actual mechanism of action was unknown (see Table 9). Conversely, on the page for the herbal antidepressants, all three possible mechanisms of action listed contained some degree of uncertainty in the phrasing, using phrases such as 'believed to' or 'appears ... can help'. There are several possible reasons for this and one is not differences in scientific findings; there is much speculation on how antidepressants work, but no theory has been 'proven.' One possible reason is that the pharmaceutical antidepressants have been marketed for their specificity – and this commercial site may be using this same strategy to market their pharmaceuticals. The

Table 9: Uncertainty in biochemical mechanism of action

Treatment	Uncertainty in mechanism mentioned?	Mention of biology as possible mechanism of action
SSRIs		"As their name implies, SSRIs selectively keep nerve cells from eliminating the neurotransmitter serotonin."
Tricyclics		"The tricyclic antidepressants selectively keep nerve cells from eliminating serotonin and norepinephrine."
Ludiomil		"Ludiomil selectively keeps nerve cells from eliminating norepinephrine."
Remeron		"Remeron stimulates the release of the neurotransmitters serotonin and norepinephrine."
Effexor		"Effexor and Effexor XR selectively keep nerve cells from eliminating serotonin and norepinephrine, and to a weak degree, from eliminating dopamine..."
Serzone		"Serzone interferes with serotonin activity."
Wellbutrin	yes	"To a weak degree, Wellbutrin (and Zyban) keep nerve cells from eliminating *The actual mechanism of action that is helpful is not known."*
Desyrel		"Desyrel affects serotonin activity."
MAO inhibitors		" Nardil and Parnate [....] interfere with the enzyme monoamine oxidase, which helps clear serotonin, epinephrine, norepinephrine, and ..."
Cognitive Therapy	N/a	[none]
Psychotherapy	N/a	[none]
Support Groups		"Social networks dam the flood of hormones that trigger feelings of depression and stress".
Exercise		It "releases endorphins", "reduces levels of the stress-depression hormone, cortisol, in the blood", and "increases levels of serotonin".
Herbal - St. John's Wort	yes	"More recently, researchers *believe* that the plant is also an SSRI "
Herbal – SAMe	yes	"SAM-e is involved in producing the brain-cell messengers serotonin, dopamine, norepinephrine, and epinephrine [that] *are believed* to play an important role ..."
Herbal – Gingko	yes	"it also *appears* to normalize neurotransmitter levels, and as a result, can help treat depression."
Herbal - Kava Kava	N/a	[none]
Dietary Supplements	N/a	[none]
Alternative - Aromatherapy	N/a	[none]
Alternative - Massage Therapy		"Their blood levels of stress hormones decreased, and they reported improved mood."
Alternative -Music Therapy	N/a	[none]
Alternative - Relaxation response	N/a	[none]
Alternative - Acupuncture	N/a	[none]
Phototherapy	N/a	[none]
ECT	N/a	[none]

presentation of these scientific 'findings[39]' for non-drug therapies reinforces the biochemical aetiology of depression touted on the site, which in a way works as a 'soft sell' for the pharmaceutical antidepressants sold through the site. However, because herbs are not squarely within the cultural constellation of 'science,' they do not have the *a priori* assumption of efficacy in public discourse as do the pharmaceutical antidepressants.

In *The Noonday Demon*, the issue of a biochemical mechanism of action is presented in more complex terms, based on the following argument: although biochemistry may be involved, it is perhaps not the ideal locus for targeting treatment. Nonetheless, throughout the two chapters on treatment, Solomon consistently returns to possible biochemical mechanisms of action for treatments as diverse as psychotherapy, St. John's Wort, and exercise (2001).

SECTION 4: OTHER AURAS

> Other herbal preparations may extract an herb's active component and combine it with foreign, inert material, thus removing the intelligence of the plant from the wisdom of nature – from an *Herbal Actives* pamphlet

Nature (and Kira, a case example)

I have shown how pharmaceutical and herbal antidepressants need to build relations with science in different ways in order to invoke its aura. On a box of Kira-brand St. John's Wort found at a pharmacy, the inscription on the back of the box was almost exclusively an explicit invocation of science:

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[39] Allan Young, in his study of the biology of PTSD (1995), argues that there are three different ways in which the scientists reported the results of statistical tests of significance. If the results met the requirements for the statistical tests of significance, they were represented unambiguously as facts. Conversely, if the results did not meet the tests for significance, they might be reported in one of two ways. In the first way, they might either be reported as 'just having missed statistical significance' or as a 'trend towards significance'; in other words, these were reported as *findings*. In the second way, the results might be dismissed because they had not made statistical significance (*discarded results*). For the antidepressants, the neurobiological mechanism of actions are presented as 'facts,' even though perhaps more accurately they should be presented as 'findings'; for the herbal antidepressants, the mechanisms of action are presented as not even findings – more like hypotheses.

> **You should know ... No other St. John's Wort Extract is the same as LI 160).** The extraction process for LI 160 provides the clinically proven ratio of hypericins, hyperforin and of flavinoids. The case for St. John's Wort is based on the numerous human clinical studies done with LI 160, which demonstrated efficacy – all published in peer reviewed medical journals. **To date, no other St. John's Wort extract has repeatedly shown the same degree of efficacy. Fact:** In Germany, LI 160 is widely recognized as the first line of treatment for mood disorders and figures among the most prescribed preparations – even though a prescription to purchase LI 160 is not required. No other extract of St. John's Wort aproaches [sic] this level of confidence held by German physicians. Studies show that Kira LI 160 is safe and efficacious, not habit-forming, well-tolerated, and has no sedative effects[40] [emphasis in original].

St. John's Wort can access an aura of science, but the relationship of the herb to science needs to be explicitly made. However, St. John's Wort has more of an unconditional aura of another type: 'nature.' Statements referring to the nature of St. John's Wort appear on everything from websites – " because it's natural, it gently helps you effectively regain your emotional balance (Kira website) – to magazine advertisements such as the one I encountered while in a laundromat in a rural centre of Washington State (see Figure 9). On the front of the box of the Kira-brand St. John's Wort, there is a bright yellow sunshine – a metaphor both of nature and of happiness (see chapter 4). Andrew Solomon, likewise, saw an advertisement for Kira-brand St. John's Wort:

> In an advertisement that ran for a while in the London Underground, a blonde woman with an expression of bliss on her face was identified as "Kira, sunshine girl," who was kept in high spirits by the "gently dried leaves" and "cheerful yellow flowers" of Saint-John's-wort. The implication of this ludicrous ad – as if the gentle drying or yellow color had anything at all to do with the efficacy of the treatment – reflects the sappy approach that has made Saint-John's-wort such a popular remedy (Solomon 1999: 147).

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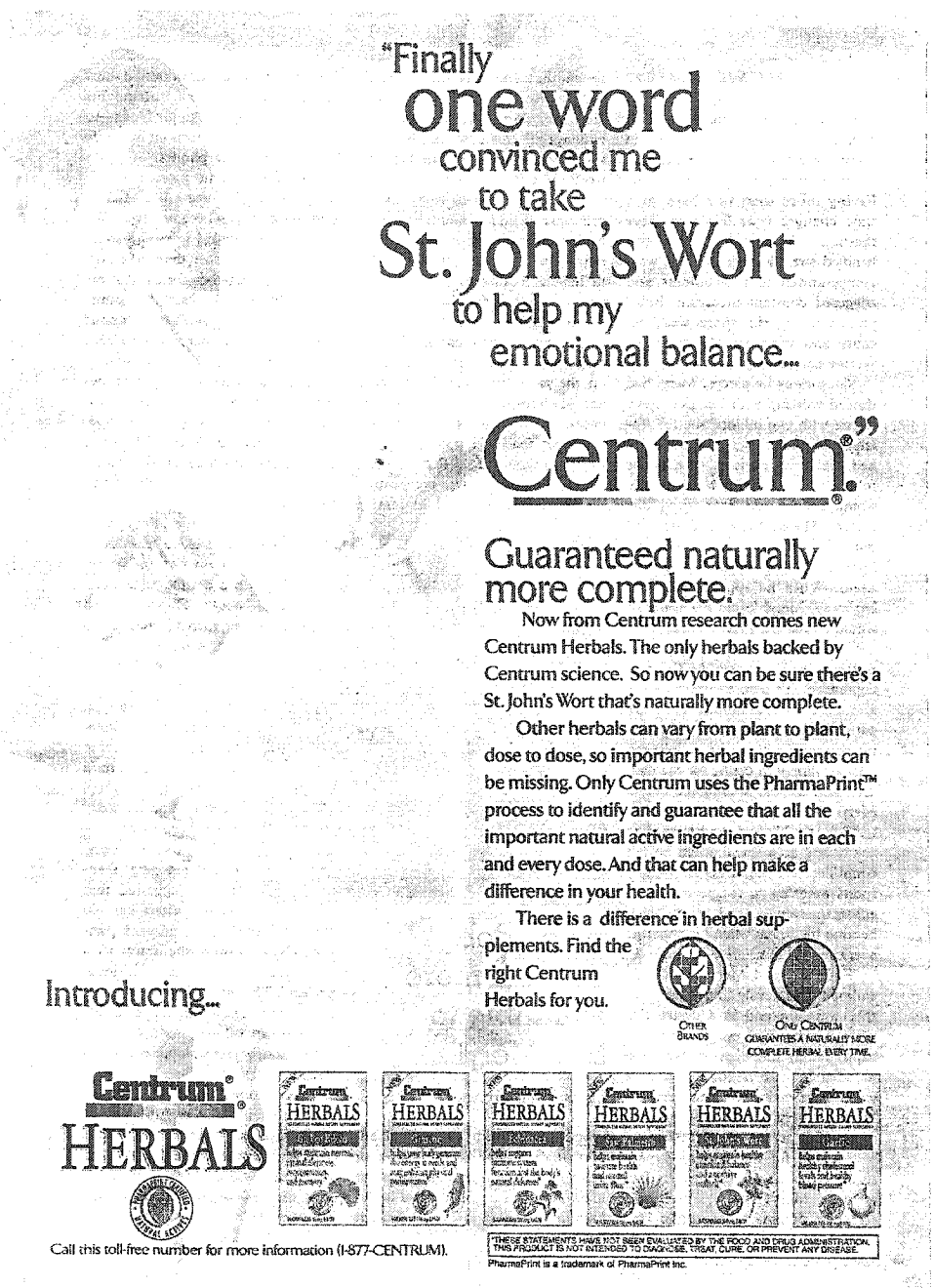
[40] About 10% of this text is translated from the French text written beneath the English version (about 10% of the English version was obliterated by a barcode).

Figure 9: Ad for Centrum St. John's Wort



I do not include Solomon's passage to argue that colour and the invocation of nature are ludicrous – one could also argue that the implication that specific neurotransmitters have anything at all to do with the efficacy of the treatment is equally ludicrous (Healy 1997). I include this Kira ad because, curiously enough, it contains many of the same elements as do the advertisements for antidepressants geared for clinicians in the pages of the Canadian Journal of Psychiatry (see Figure 10). While not explicitly saying 'made with sunshine,' the use of sun and sky imagery in these ads for pharmaceutical antidepressants is just as frequent as the use of graphs, with their aura of science, that imply "made with science". Both are implicit messages meant to validate the product, albeit from different realms of value: 'nature and 'science.' These representations of sun and sky are also far more frequent than any pictorial representation of the brain, although the mention of neurotransmitters, once Prozac hit the market in the late 1980s, was far more frequent than any of the other features described in the content analysis (see Table 10).

Table 10: Content Analysis of Advertisements for Antidepressants in the the first issue of the year (February) of the *Canadian Journal of Psychiatry*, 1982-2002.

Year*	# of ads	sun/sky	graph	brain	pills	neurotramitter mentioned
1982	3		1in 1			
1984	2	1	3 in 1			
1985	5	2				
1986	5					
1987	2					
1988	5	2				"blocking serotonin re-uptake"
1989	4	1				"blocking serotonin re-uptake"
1990	2			1	1	"blocking serotonin re-uptake"; lots
1991	3	1	6 in 1		1	"blocking serotonin re-uptake"
1992	4	1			2	Lots
1993	6	1	1 in 1		1	Lots
1995	6	1				Lots
1996	6		2 in 1			Lots
1997	5	1	1 in 1			Lots
1998	6	1		1	1	Lots
1999	4	1		1		Lots
2000	5	1				Lots
2002	3	2	2 in 2			Lots
total	74	16	16	3	6	Many

* 1983, 1994, and 2001 were not available or had the ads removed.

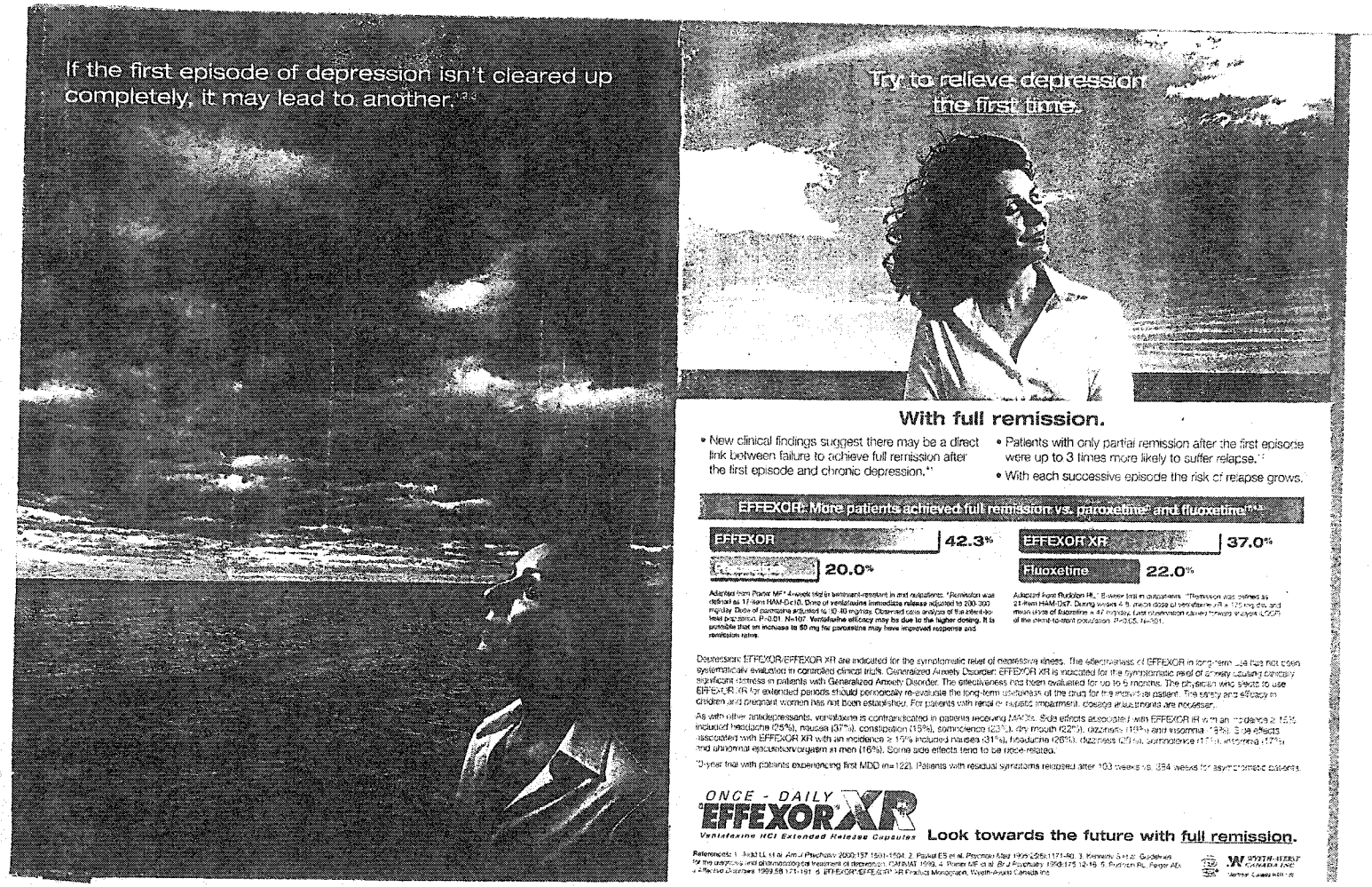


Figure 10: Ad for Effexor, an antidepressant, in the *Canadian Journal of Psychiatry*

Tradition and Ancient Wisdom

In addition to links with nature, there are many claims linking the herb with antiquity. There is no question as to the authenticity of the herb because the herb has its aura derived from ancient sources[41]. On depression.com was the statement "St. John's Wort has been used in traditional herbal medicine for centuries." Likewise, St. John's Wort was introduced in the 2002 newspaper articles as "a weed with a long history of medicinal use" (Vallis 2002) and as a substance "whose medical uses date back to the ancient Greeks" (Hall 2002) that "has been used for many years in folk medicine to treat disorders such as burns, wounds, and depression" (BBC NEWS 1999). Another news story concluded with a quote that stated that the negative results of the 2002 RCT should not invalidate the St. John's Wort because it is "a fascinating herb with a very long history of therapeutic benefit" (Vedamtam 2002). Because of its social life/history, St. John's Wort is more easily imbued with the aura of 'tradition' and 'nature.' Conversely, the depression.com pages for pharmaceutical antidepressants only listed the date of FDA approval and, with one exception,[42] did not contain any mention of the development of the products or other biological treatments for 'depression' during "Greek times" (cf. Solomon 2001)[43].

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[41] This is not unusual for herbal medicines, as often ancient origins are invoked; in a marketing study by the Hartman Group, the "study found that consumers respond best to teas when product branding associates the tea with 1000s years of herbal tradition: (MarketResearch.com 2001). This taste for the things of the past (Baudrillard 1996: 27) is directly related to how Westerners seek "authenticity in the exotic" (1996: 47). This is not restricted to North America. In Miles' study of the mass-marketing of natural medicines in urban Equador, she also states that the packaging of the products "incorporated symbols from healing systems that Ecuadorians consider to be "Indian", ancient" (1998: 212).

[42] One exception to this is that the origin of MAOIs is given on the 'introduction to antidepressants' webpage.

[43] Although the antidepressants are not tied to antiquity, the features of depression are in the DSM-IV and its sourcebooks. In the DSM-IV sourcebooks, the chapters on the melancholic feature specifier and the depressive personality disorder begin by invoking the history of these features. "In summary, melancholic features have a long history" (Rush and Weissenberger 1996: 225). The opening statement in the chapter on depressive personality disorder is: "The concept of depressive personality has a long and rich history" (Philips *et al.* 1996: 799). The authors continue to argue that depressive personality has historically been referred to as "black gall" temperament, melancholic temperament, depressive character, typus melancholicus, dysthymic temperament, characterologic depression, subaffective dysthymia, dysthymic psychopathy, and anankastic personality disorder (PD) with depressive features" (Phillips *et al.* 1996: 799). The same validation is provided in the first section of the chapter on the melancholic feature specifier, entitled 'Historical Perspective on the Concept of Melancholia'. Just like the depressive personality chapter, the authors elaborate on the different reincarnations of melancholia throughout history, and, rather than attempting to understand how these disorders are different, they attempt to validate how some 'core' of the concept has always been present although confounded by cultural problems and different nomenclature. "Historically, the concept of melancholic or endogenous features is longstanding. Until

Personal Experience

Another way of validating antidepressants is through testimony. This is the most common source of validation in *The Noonday Demon*. This might be due to the journalism background of Andrew Solomon but could also be due to his own stance that only those who have been through depression can understand depression and its treatment. Many of the treatments and alternative therapies were presented as therapies that the author had tried and, thus, respected; many treatment sections contained long anecdotes and stories sharing the experiences of others in relation to these treatments. In many situations, the sharing of experience seems to be one of the big ways of validating treatments. Entire books, such as *Living with Prozac and other selective serotonin-reuptake inhibitors* found in the dollar bins of used bookstores in Montreal, are filled with "personal accounts of life on antidepressants" (Elfenbein 1995). Online postings and support group meetings are also filled with these experiential stories (Karp 1992). Finally, advertisements for herbal antidepressants often have a quote from someone for whom the herb has made a world of difference: "I ...have become a total convert to [Flora's] St John's Wort Oil. I have had tremendous success taking it internally for periods of stress and overwork..."(see Figure 1).

Summary

Claims for both pharmaceutical antidepressants and herbal antidepressants build relations between the antidepressants and various regimes of value, including 'science,' nature/tradition, and personal experience. The relations, by a sort of 'contagious magic,' assist the antidepressant in acquiring some of the aura of authenticity of that regime. Pharmaceutical antidepressants often do not even have to explicitly state their relationships to 'science' because, as they have been produced in a scientific style of

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recently, however, conceptual confounding of etiology and phenomenology has been present. In the 1950s and 1960s, the term also began to imply a positive response to ECT and to antidepressant medication". (Rush and Weissenburger 1996: 196). The description of the atypical depression in the DSM-IV itself also situates 'atypical depression' with respect to historicity, although more ambiguously. "'Atypical depression' has historical significance (e.g., atypical in contradistinction to the more classical 'endogenous' presentations of depression) and does not connote an uncommon or unusual clinical presentation as the term might imply" (APA 2000b: 420)

reasoning, they are already imbued with the aura of science; the dominant often becomes unsaid. Conversely, St. John's Wort is more easily validated by 'nature/tradition,' although some pharmaceutical antidepressants are similarly validated. Personal experience is a third, and final, type of value that can function as a source of authenticity and value for both types of antidepressants.

SECTION 5: JUXTAPOSITION OF REALMS OF VALUE

The (Dodo's caucus) race is over, and everyone has won

In all of the sources mentioned so far, most of the text flows seamlessly through these different realms of validation. As such, they are juxtaposed without any need for commensurability. This has also been the case in Canadian government food and drug regulations since the 1950s – substances that may be validated in different ways are segregated into different categories so there is no need to directly juxtapose different methods of validation when regulating any one product. The Canadian Food and Drugs Act has regulated what we would call 'herbal medicines' as either 'food' or 'drugs' since its inception in 1953[44]. Later, a Drugs Directorate guideline emerged for 'traditional herbal medicines' in which a drug identification number (DIN), usually restricted to pharmaceuticals, could be issued to products intended for self-medication "where the efficacy is supported solely by traditional herbal references" (Therapeutic Products Directorate[45] 1995:1). Although the standards were then different for herbs, foods, and drugs, no juxtaposition between scientific and traditional references was needed because they applied to substances in different categories. In 1997, Health Canada initiated an advisory panel on natural health products, and the Standing Committee on health – made up of members of parliament – conducted a review of this regulation. One of the guiding principles adopted unanimously by the members of the Standing Committee on Health is that "NHP are different *in nature* from and must not be treated strictly as either food or pharmaceutical products" (Volpe 1998: chapter 2). This culminated in the government's

[44] For the history section of the Canadian government regulations that follow, the information was pulled off of various pages of the Health Canada website as well as from the summary presented in Chandler 2001.

[45] Although I strive to be as accurate as possible in documenting the proper authors of these government documents, there have been so many changes in the names of the various offices, directorates, and branches that retroactively pinpointing the correct name (when it is not written on the document) is difficult.

acceptance of all the recommendations of the committee, which essentially legislated the NHPD to regulate 'natural health products' using diverse types of evidence and authority. The legislation stipulated the following:

19. NHPs [Natural Health Products] be allowed to make health claims, including structure-function claims [calcium builds strong bones], risk-reduction claims [garlic decreases the risk of cardiovascular diseases] and treatment claims [St. John's Wort is useful in the treatment of mild to moderate depression].
20. Claims be assessed to ensure that there is reasonable evidence supporting the claim.
21. The evidence not be limited to double blind clinical trials but also include other types of evidence such as generally accepted and traditional references, professional consensus, other types of clinical trials and other clinical or scientific evidence.
22. The evidence required vary depending on the type of claim being made, with different evidence being required for structure-function claims and risk-reduction claims for minor self-limiting conditions than for therapeutic or treatment claims
23. The label indicates clearly the type of evidence used to support the claim (Volpe 1998)

Thus, the NHPD has to develop a way of explicitly relating the authorities produced in different styles of reasoning and, in so doing, is codifying claims made about herbal antidepressants.

Links to the DSM-IV

> `Well, I never heard it before,' said the Mock Turtle; `but it sounds uncommon nonsense.'
>
> Alice said nothing; she had sat down with her face in her hands, wondering if anything would EVER happen in a natural way again.

The evidence requirements for the licencing of the NHPs in Canada harkens back to the rhetoric used to defend the choices of evidence taken for the DSM-IV (chapter 2). The input from sixty-five organizations, hundreds of workgroup advisors, as well as the information taken from literature reviews, traditional practice, the ICD-10, and "common

sense" would be incorporated into the changes. Essentially, everything will be taken into account – how could anyone possibly disagree with this? As Kirk and Kutchins satirize:

> Expert opinion will not be enough, but expert consensus will be considered. Systematic reviews of evidence will be required, except where it is limited or not available. Nothing is sacred, but historical tradition is to be respected (1992: 211).

What is black-boxed is exactly *how* these different types of evidence, information, and advice are made commensurable and/or incorporated into a final synthetic decision.

Initial plans for un-black boxing efficacy

How will these recommendations be incorporated in practice? All proposals, at least the ones that are publicly available, involved some sort of tiered system with different levels of claims requiring different 'levels of evidence.' These are very similar to the "levels of evidence table" used as a guiding tool for the Canadian Psychiatric Association (Table 4). One of the first proposals in the minutes of the expert advisory committee meetings was a colour- and letter-coded system. For A level claims, evidence must be obtained from either a well-designed meta-analysis of randomized controlled trials or from at least one well-designed randomized control trial. It was suggested that this be shown on product packaging with a gold symbol. For B level claims, with a silver symbol on the products packaging, evidence must be obtained from at least one well-designed controlled study without randomization or some other well-designed experimental study. C level claims, with a bronze symbol, would require evidence from either (i) descriptive studies, such as comparative studies, correlation studies or case control studies, or (ii) the testimony of expert committees and/or respected authorities. Finally, traditional use claims might be made if the product had been "used in a traditional manner for at least 100 years", and a green symbol would go on the packaging (minutes from meeting held on January 7&8, 2001).

At the "Standards of Evidence" townhall meeting, the situation was even more complicated. References throughout the day to "forms of proof" and the "strength" of the evidence were used in such contexts such that it was clear that the sum of the peer

reviewed scientific evidence was what was used to explain the 'totality of evidence,' with other "forms of proof" much lower on the hierarchy (November 28th, 2002). Products were divided into the following categories, with each category requiring different "forms of proof" (listed from the least to most amount of proof required in terms of Table 4): traditional use, non-traditional but generally accepted, non-traditional with a history of market, and non-traditional anything else.

> `I should like to have it explained,' said the Mock Turtle.
> `She can't explain it,' said the Gryphon hastily. `Go on with the next verse.'

What was particularly interesting was the debate ensuing from a question posed to the table of eight participants, including me, at the Townhall meeting (Fieldnotes November 28th): "If a traditional use product has been studied scientifically but the scientific evidence does not support its traditional use, should a traditional use claim still be acceptable?" (Health Canada 2002: 23). The three small business participants and the marketing representative at first argued that these are two valid ways of knowing, so the traditional label should still be on the bottle. The government workers stated that as consumers, they would want to know that the scientific evidence said that it didn't work. The small business participants countered that the scientific method is fallible – it says one thing one day, another thing the next but that traditional knowledge it built up over hundreds of years. The operations manager spoke of the "culture, experience, years" in the natural health products that one scientific study should not annul. The anthropologist threw in her two cents worth: that it was unlikely that the traditional claim and the scientific[46] trial were actually talking about the same disorder, so the traditional claim should be kept. This resulted in many blank stares. Once the conversation resumed, the small business participants concluded, though, that if the 'totality of scientific evidence,' which meant that there were *many* studies against the efficacy of the product (eg: five to one) then maybe it should be discontinued.

[46] The word 'scientific' was used throughout the day; the word 'medical' was rarely heard.

> `What IS the use of repeating all that stuff,' the Mock Turtle interrupted, `if you don't explain it as you go on? It's by far the most confusing thing I ever heard!' `Yes, I think you'd better leave off,' said the Gryphon: and Alice was only too glad to do so.

Summary

Using the rubric of evidence-based medicine that emphasizes the importance of the RCT, the Natural Health Products Directorate is developing regulations that hierarchize the different regimes of value legislated to validate Natural Health Products.

SECTION 6: Biologies

> In Western herbalism, these herbs are collectively classified as *nervines*, with a subclass called *nervous restoratives* or *nervine tonics* being the nervines of choice for depression (Baumel 2000: 181).

Do St. John's Wort products, when they talk about element of the nervous system, really promulgate the same neurochemical body as do the pharmaceutical antidepressants? While neurochemicals are mentioned, it is difficult to tell if the overall impression is more of one of 'nerves' or 'neurochemicals'. Take for instance one of Jamieson's products: "Jamieson Neural Sources Neurosome ™ St. John's Wort." On the label, it is written that it is indicated for pain of neuralgia, 'nervous excitability, tension and restlessness due to fatigue', and insomnia and sleeplessness.

I argue that St. John's Wort does not yet facilitate the neurochemical body as much as pharmaceutical antidepressants, although it may facilitate other embodied understandings in term of nerves. However, as St. John's Wort is increasingly being compared with and associated with pharmaceutical antidepressants, and science in general, the herb is increasingly facilitating this neurochemical understanding of the body. At both natural health food stores I visited, the first clerks with whom I spoke used neurochemical

terminology when speaking with me about St. John's Wort and other herbs[47]. However, at the first store the clerk brought out the store's naturopath because she thought that the naturopath would be interested in my question. The naturopath, one of the more experienced employees(in terms of time worked), dismissed my question about products that might be useful for depression. She said that she looked for the *cause* of depression, which could be different in each case (Fieldnotes April 8th). Thus, the neurochemical-depression as an entity in and of itself was not relevant. When I revisited the second store to take the photographs of the product packaging, the manager of the store was there to make sure that we[48] understood that help for depression did not come in a pill because she did not want us leaving with the impression that this was the solution (Fieldnotes April 10th, 2002). More than anything else, she talked about interpersonal relations and loneliness as factors in depression(April 10th, 2002). Neither the more experienced naturopath nor the store manager were comfortable with or used neurochemical terminology; both relatively new store personnel were fluent in and used this terminology.

This change in associations with St. John's Wort is also mirrored in a book that all four of the store personnel with whom I spoke recommended. The *Prescription for Nutritional Healing* (Balch and Balch 1990 and 1997) was what they first referred me to, and both stores had at least one dog-eared copy on hand. This book is written in encyclopedic format and has 'depression' as one of the entries. The two editions in the stores, from 1990 and 1997, contained rather different information although they were both written by Balch and Balch– one an M.D. and the other a C.N.C.. The first edition (1990) does not spend any time describing depression in terms of DSM categories and does not mention St. John's Wort at all. Neurotransmitters are briefly mentioned in the context of how various foods affect their levels. The description of depression in the second (1997) was longer by about 50%, and most of this increase was due to the description in some detail of depression as diagnosed in the DSM-IV. Two other major changes are that St. John's

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[47] I should also mention that at this particular health food store, the clerks in the supplement section (which encompasses an entire floor of the store) all wear white laboratory coats – the aura of science is invoked in many different ways!

[48] Not a royal 'we' – a friend of mine was with me taking the pictures of the labels.

Wort mentioned, as are the pharmaceutical antidepressants with rather long descriptions of their side effects. The only sentence mentioning St. John's Wort frames it in terms of pharmaceutical antidepressants: "St. John's Wort acts in the same way as monoamine oxidase (MAO) inhibitors do, but less harshly" (Balch and Balch 1997: 225). In the following statement version, the emphasis on neurotransmitters is much stronger: "perhaps we will abandon the catchall category called depression and diagnose people according to their particular chemical imbalances" (Balch and Balch 1997: 224).

CHAPTER 4

CONCLUSIONS: PLOTS AND SUBPLOTS

> And here Alice began to get rather sleepy, and went on saying to herself, in a dreamy sort of way 'Do cats eat bats? Do cats eat bats?' and sometimes, do 'bats eat cats?' for, you see, as she couldn't answer either question, it didn't much matter which way she put it.

Summary

The complexity of depression does not lend itself to an analysis involving causal statements, and to some extent this story has been one of tracing webs with various combinations of depression-antidepressants-neurochemistry rather than defining a linear, causal plot. In the introduction, I began by pondering if antidepressants facilitate (at least the *expression of*) experiences of neurochemistry. The presence of experiences of neurochemistry published in individual narratives of depression (Solomon 2001; Wurtzel 1995) and in case studies of depression (Karp 1993, 1994; Gammell and Stoppard 1999) indicate that, at least to some extent, antidepressants do facilitate the expression of these experiences. In these narratives, neurochemistry may either be an embodied experience (such as implied by my friend) and/or used as a way of presenting the self in the best possible light (Karp 1992: 158). Rather than focussing on individual treatment narratives, however, I have chosen to examine two other 'sites' of cultural production in order to investigate what might be involved with antidepressants' facilitation of experiences of neurochemistry. I first examined the social life of antidepressants in terms of their co-production with depression. I then perused North American documents providing 'information' on antidepressants.

This choice of anthropological 'sites' has not been particularly satisfying, because they are very difficult to contextualize. Many studies of biomedical categories in anthropology manage to contextualize the work, whether by a clear physical location (e.g., Veteran's Unit for post-traumatic stress disorder in Young 1995), social community (e.g., support groups for families of children with Down's Syndrome in Rapp 2000b), one 'fact' in particular (e.g., brain scan results for chronic fatigue in Dumit m.s.), or some

combination of the above combined with clearly identified interview respondents (e.g., interviews pertaining to amniocentesis in Rapp 2000a; interviews pertaining to menopause in Lock 1993). I have tried to explore a biomedical category (depression) by examining information readily available to consumers of antidepressants – however it is not clear to whom this information is addressed and by whom it is read. What might be a useful follow-up project would be to do a small longitudinal study of a few individuals who are depressed, and investigating what sources of information, if any, seem to affect and effect their experiences with depression.

The co-production of depression and antidepressants

In this thesis, I have explored depression's co-production with antidepressants, neurochemistry, and efficacy in a particular style of reasoning in biological psychiatry. None of these factors can truly be disentangled from each other or from the social and cultural context in North America in the second half of the 20th century. This has been re-iterated recently by sociologist Alain Ehrenberg in his book *La Fatigue d'être soi: Dépression et société* (1998); he argues that depression is a constellation of antitheses to the ideal 'self,' in terms of both actions and characteristics, in Western societies. Antidepressants work to solidify and reify this antithesis as depression.

However, to what extent is depression, especially depression on neurochemical terms, relevant in Canada? The case studies presented at the end of chapter two hint that the relevance is intimately tied to antidepressants. These antidepressants, in part because they are tied to a rhetoric of neurochemistry, are also the site of cultural resistance (Greenslit m.s; *see also* Figure 11) and cultural irrelevance (Scattolon 1999).

> "Indeed the idea that there might be a depression that drugs could treat had in one sense to be invented as had the idea of an antidepressant – and part of the problem to this day is that neither of these inventions has fully worked, at least not with the public at large" (Healy 1997: 4).

Figure 11: 'Culture Jam' in *Adbusters* (May/June 2001). *Adbusters* lists the following credits: 1. Graphic from Zoloft Advertisement, Pfizer Inc., 2. Text from *The Chemical Basis of Mental Illness: a Theory in Search of Evidence* by Jonathan Leo, and 3. Photo by Martin Parr

And specificity

> In an ideal world, specific patient profiles would determine specific antidepressant treatments. – opening sentence for the Canadian Psychiatric *Association's Clinical Guidelines for the Treatment of Depressive Disorders* chapter four: Medications and Other Biological Treatments (Kennedy *et al.* 2001)

Neurochemistry serves well in the rhetoric of advertising, and it also serves well in the rhetoric of biological psychiatry. In biological psychiatry, there is an ideal for antidepressants in clinical practice: "an ever more sophisticated application of differential therapeutics based on a more rigorously refined differential diagnostic system" (Deltito and Beyer 1998: 346; *see also* APA 2000: 67). Not only the system of therapeutics and diagnosis but also its rationale must be specific: "in any event, molecular precision is required in explanations that are to be candidates for truth: they must work in terms of anomalies in receptors understood in terms of their specific shapes, or in terms of anomalies in receptors understood in terms of their channels and the like" (Rose *in press*). Mental disorder, because it is due to little tangible elements of biology, is best treated with little tangible elements of science: pills (Van der Geest and Whyte 1989: 346).

This specificity works not only for pharmaceutical antidepressants but also for 'natural' antidepressants. Indeed, the *Prescription for Nutritional Healing* that was recommended as a source of information on depression by the personnel of both natural health food stores that I visited, states that "as we learn more about this disease in all its complexity, perhaps we will abandon the catchall category called depression and diagnose people according to their particular chemical imbalances" (Balch and Balch 1997: 224).

Claims made for the efficacy of antidepressants

> Vindication and refutation occur only on that site; value in a mission is something else. All the jokes about military gadgetry hinge on this banal fact. If people opposed to conventional medicine had a sense of humor, and if the rest of us didn't feel that jokes about disease were sick, then they could make exactly the

> same jokes about medical research that we peaceniks make about weapons research. The military like to advertise their gadgets as working with surgical precision. When was the last time they were in a surgery? (Hacking 1992a: 59).

Likewise, in the third chapter, I explored the claims made for the efficacy of pharmaceutical and herbal antidepressants in terms of their social lives. I argue that pharmaceutical antidepressants, are generally assumed to be effective *a priori* through their association with mainstream biomedicine, require fewer types of claims to establish their value in terms of 'science.' Because they have a more or less permanent aura of science, they are also less likely to be discredited by negative RCT. I also argued that the pharmaceutical antidepressants' privileged access to the aura of science might facilitate experiences of the neurochemical body because of the web of associations. I argued that herbal antidepressants, having more of an aura of 'nature' and 'ancient-ness' by virtue of their social lives, are somewhat less associated with neurochemistry. However, as marketing rhetoric and evidence-based medicine increasingly associate St. John's Wort and 'science,' this may change.

And evidence-based medicine

> In summary, we find the accumulated evidence regarding the efficacy of St. John's Wort to be inconclusive (Deltito and Beyer 1998).

At the time Deltito and Beyer wrote that statement in a scientific journal, the recent negative results from RCTs on St. John's Wort had not yet emerged; based on exclusively positive trials, they concluded that there was not enough evidence to make a judgement. Conversely, on the same evidence, many respected herbal texts rated the herb as 'efficacious' (Chandler 1996: 350) or at least 'likely effective' (NaturalDatabase.com 2000: 984). Take the two further examples from the opposing camps:

> As long as these types of products remain available to the public without the protection of adequate, controlled and unbiased studies, taking them is like playing Russian roulette with your health. – Robert Califf, author of the NCCAM-sponsored RCT for St. John's Wort (*quoted in* BBC 2002).

> As miraculous as SJW has been for so many depressives, it would be an even greater miracle if it were the only herb that could perform such antidepressant wonders (Baumel 2000: 181).

In Germany, where there is a strong tradition of herbal medicine, even among physicians, the RCTs demonstrated the efficacy of St. John's Wort. In North America, where there has not been a strong tradition of herbal medicine in medical practice, the RCTs demonstrate the inefficacy of St. John's Wort in the eyes of many. No matter how statistical are the tools by which efficacy is assessed, the value judgements that have been made in order to give the tools something with which to work seem to be the most salient factor in determining the efficacy of antidepressants.

Pinning down the efficacy of antidepressants in RCT

> The chief difficulty Alice found at first was in managing her flamingo: she succeeded in getting its body tucked away, comfortably enough, under her arm, with its legs hanging down, but generally, just as she had got its neck nicely straightened out, and was going to give the hedgehog a blow with its head, it WOULD twist itself round and look up in her face, with such a puzzled expression that she could not help bursting out laughing: and when she had got its head down, and was going to begin again, it was very provoking to find that the hedgehog had unrolled itself, and was in the act of crawling away: besides all this, there was generally a ridge or furrow in the way wherever she wanted to send the hedgehog to, and, as the doubled-up soldiers were always getting up and walking off to other parts of the ground, Alice soon came to the conclusion that it was a very difficult game indeed.

While this debate shows no sign of abating in North America – until perhaps St. John's Wort is no longer a best-selling drug – there are some reasons why RCTs for St. John's Wort are important in assessing the value of St. John's Wort. I do not mean that they are important in making a definitive judgement on the efficacy of St. John's Wort, but rather for changing the social life of St. John's Wort. In this process of being subjected to an RCT, the context for which the treatments are used may be drastically changed. This was seen in the NCCAM-funded RCT testing St. John's Wort for *moderate to severe*

depression, instead of the *mild to moderate* depression that had been advocated by the herb's advocates[49]. With other treatments, such as Tibetan treatments, this change in context might be even more drastic – with crucial ingredients in multi-ingredient medicines unable to cross the borders and the difficulty of translating Tibetan diagnoses into biomedical diagnoses – and may criminalize practitioners using the substances (Adams m.s.).

Furthermore, through their participation in these RCTs, different realms of values are juxtaposed. While it is only St. John's Wort that is on trial, its aura of 'nature' and its social history with associations with traditional medicine mean that, in a way, natural and traditional medicines as a group are on trial. The overall message through the institutionalization of these RCTs in the context of evidence-based medicine is that 'nature' can be assessed by 'science,' but the reverse cannot happen.

> "knowledge [or, I argue, value] hierarchies are rarely "accidental" in their origins: They tend both to build upon and reinforce social cleavages based on other markers of difference – class, formal education, race, gender, sexuality, and nationality" (Epstein 1996: 352).

Through this implied comparison between 'science' and 'traditional medicine,' these RCTs may institutionalize the notion that non-biomedical systems have an equivalent 'theory' as that of (the archetypal) biomedicine, with their corroborating elements striving to avoid internal contradiction (Farquhar 1987, 1994; Lewis 1993). However, this perceived equivalence may enable new symbolic relations:

> Natural Medicine transcends the usual dichotomies created between science and nature and modernity and tradition – a dichotomy that tends to stigmatize those most closely linked with the latter of these dichotomies. By doing so it provides a 'double action' commodity of powerful symbolic potency. Natural medicines take science, the cultural symbol of the elite and powerful, and use it to validate the (sometimes lost) natural world of the peasant farmer and rural dweller (Miller 1998: 221).

Concluding remarks

The 'actual' efficacy of antidepressants

> `What do you know about this business?' the King said to Alice.
>
> `Nothing,' said Alice.
>
> `Nothing WHATEVER?' persisted the King.
>
> `Nothing whatever,' said Alice.

Many people over the past few years have asked me what I do. When I reply that I am looking at different ways of claiming that antidepressants work, I tend to get one of the following reactions. One is a personal narrative of that person's experience of depression. While I am often saddened by the many difficulties that people experience, at least I feel comfortable responding. The second reaction is sort of a blank look and a change of topic, sometimes with the question "Isn't that depressing?" However, a third runs along the lines of the question "So do antidepressants really work? See my friend/relative is on them but I don't really believe that they work" – and I am expected to give an authoritative answer to the question.

I feel that I only disappoint with either deflection such as "Has it helped your friend/relative?," blanket answers such as "I definitely think that they work for some people" or recourse to personal experience such as "I have [such and such a friend] who swears by them, but they didn't seem to do anything for another friend." I am uncomfortable with giving an authoritative answer, although I could easily spout off the 50-60% cited in many places, because it doesn't seem like that answer will be particularly useful to any particular individual beyond what s/he already knows. I am all for whatever treatment works for any particular person. However, I do think that RCTs are very useful. They promise to make unnecessary, or to at least reduce, the trial and error process with treatments, which in depression can be a long and devastating process. I also think that the power of RCTs stems from dealing with very specific, delineated categories – to what extent are depression or mood disturbances treated in herbal traditions suited to becoming specific and delineated problems? A comparison between antidepressants and placebos might be helpful (chapter 3, sections 1 and 2).

Antidepressants and Placebos

> There was no label this time with the words `DRINK ME,' but nevertheless she uncorked it and put it to her lips. `I know *something* interesting is sure to happen,' she said to herself, `whenever I eat or drink anything; so I'll just see what this bottle does.

To what extent do the antidepressants themselves matter in the claims made in vernacular North American literature? To what extent are the antidepressants marketed via realms of value, and to what extent are the realms of value marketed via antidepressants? Clearly there are some cases where what is being consumed are the values, symbols and images surrounding the consumption of a drug, superseding the significance of the actual drug (Montagne1988: 420). While working in a large city in northern Mexico in 2000, I was informed by several of my students, mostly business executives, that taking antidepressants was 'all the rage' in upper class circles. They speculated it was because it made people not only fashionable but also 'modern.'

In addition to making people modern, antidepressants seem to make some people less depressed. Is it the antidepressant's chemical formula that brings about these effects or simply its various auras of value that bring about these effects?. With substantial rates of response to placebos – negatively termed 'dummy medicine' (BBC 2002), 'sugar pills' (Vallis 2002), 'inert substance' (Vedantam 2002a), 'dummy pills' (Hall 2002), 'fake pills' (Hall 2002), and 'dud pills' (Vedantam 2002b) in the newspaper articles presented in chapter 3 – are antidepressants simply placebos with the right social life that are shown to "work" on an empirical level because of their side-effects?

From Efficacy to Risk

But perhaps my talk of efficacy is outmoded. The age of risk management is institutionalized in government regulations, in which efficacy and safety are inseparable and it is only the overall 'risk' of the health product that is considered (Health Canada 2002). The new Natural Health Products Directorate in Health Canada is doing risk

management by looking at the totality of experience (Fieldnotes November 28th, 2002), and has explicitly stated that NHP are lower risk drugs (Fielnotes November 28th, 2002). Might the placebo be the ultimate low-risk drug?

> In science, just as in art and in life, only that which is true to culture is true to nature (Fleck 1979 [1935]: 35).

APPENDIX: LIST OF ABBREVIATIONS

APA	American Psychiatric Association
DSM	*Diagnostic and Statistical Manual of Mental Disorders*
RCT	Randomized Controlled Trial
WHO	World Health Organization

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