HOW DOES AN EVOLUTIONARY PERSPECTIVE INFLUENCE OUR UNDERSTANDING OF SEASONAL AFFECTIVE DISORDER?

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Outline: An evolutionary approach is taken in the paper to explore how this view can increase our understanding of seasonal affective disorder (SAD) as a mental dysfunction, as well as to evaluate the current classification of SAD in the DSM-IV. The first section of the paper examines the evidence for the relationship between seasonality, non-seasonal depression, and SAD. The second part of the paper looks at various evolutionary theories of SAD and how well these support the evidence. The final section looks at how we should define SAD, and seasonality, in terms of mental dysfunction, and examines the importance of a flexible model which takes account of changing environmental contexts.

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'Lethargics are to be laid in the light, and exposed to the rays of the sun (for the disease is gloom)' Aretaeus, 2nd century AD.

Introduction

It has long been recognised that mental disorders show seasonal variation. Even in ancient times, Hippocrates wrote on the relationship between seasonal climactic conditions and manias and melancholias (Zilboorg 1941). More recently, such a form of depression was 'rediscovered' by Rosenthal et al. (1984), which they called 'Seasonal Affective Disorder' (SAD). SAD is an affective disorder characterised by psychological and behavioural changes, such as low mood, increased sleeping, and increased appetite (Rosenthal et al. 1984). These changes occur in winter with remittance in the summer. Rosenthal et al. (1984) termed this as 'winter-type' SAD. Although there is some evidence for the presence of a summer-type form of SAD, it is less common, and so, for the purposes of this dissertation, only the winter form of SAD will be referred to.

Anecdotal evidence suggests that Seasonal Affective Disorder could be an extreme form of 'seasonality' which is present along a continuum in the normal population (Kasper et al. 1989). If this were the case there could be good reason to suspect that SAD, as an extreme form of seasonality, once played an adaptive role in human evolutionary history (Eagles, 2004; Davis and Levitan, 2005). However an alternative theory which has been used to explain the data proposes instead that SAD is and always has been an evolutionary maladaptive trait which has not yet been naturally selected out of the normal population (Sher 2000). This picture is further

confused by the *Diagnostic and Statistical Manual of Mental Disorders-IV* classification of SAD being a subtype of other affective disorders except with a seasonal pattern. This could cause a problem for the evolutionary theories of SAD which try to provide an account of SAD without reference to it necessarily being a pathological disorder (Nesse, 2000; Block 2008).The primary aim of this dissertation is to examine the experimental evidence and literature focusing on SAD from an evolutionary perspective, as well as to provide an evaluation of how an evolutionary viewpoint has influenced our understanding of SAD. This paper is divided into three sections. The focus of the first section looks at the evidence which suggests that it is reasonable to view SAD from an evolutionary viewpoint. One of the factors which needs to be considered is the presence of seasonality within the normal population, which would be support for the view that SAD is only one extreme of this symptomatic continuum, with the other extreme being a complete absence of seasonality. If this is the case, the presence of such variation within the general population is indicative that seasonality and SAD cannot simply be either a random mutation, a recessive deleterious gene, or a present gene which is highly selected against. Thus the high prevalence of seasonality would suggest that seasonality has, or did have until recently, an evolutionary purpose. Another basic factor which is more directly important to the evolutionary theorist is establishing that SAD is primarily a genetically transferred predisposition, rather than environment having a direct influence. There have been many genetic studies which support this hypothesis, which are reviewed by Sher et al. (1999). This again supports the need to look at SAD using an evolutionary approach.

The second section lays down two major theories which take an evolutionary approach to SAD; the first of these views seasonality as a purely maladaptive trait which has only appeared

due to our ancestral movements into the northern and southern hemispheres from Africa around 150,000 years ago (Sher, 2000). An alternative theory proposes that seasonality was once adaptive in our ancestral environment because it improved reproductive success and energy conservation (Davis and Levitan (2005); Eagles,2004). At the end of this section both theories are evaluated with reference to the experimental evidence put forward in the first section to determine how well they explain the evidence. This part concludes by discussing the relevance of an evolutionary theory of low mood and depression (Nesse, 2000; Keller and Nesse 2005) which proposes that low mood once served an adaptive purpose. Looking at individual symptoms of low mood in relation to precipitating factors may help to clarify the potential evolutionary purpose of low mood found in seasonality.The final section of this dissertation discusses SAD using a more philosophical outlook, concerned with how SAD should be diagnosed and described in terms of mental disorder and dysfunction. I argue that we need to find the point when seasonality becomes SAD which can be found with relation to the part seasonality has played in evolutionary history, as well as the interaction between seasonality and the environment. Finally I will summarize how the evolutionary approach has increased our understanding of SAD and the implications this has for future research.

Part One- An evolutionary viewpoint of SAD is coherent

i) What is SAD?

Rosenthal et al. (1984) suggested that SAD be diagnosed by the following criteria: a history of major affective disorder, at least two consecutive previous years in which the depressions developed during fall or winter and remitted by the following spring and summer, absence of any

other axis I psychiatric disorders, and absence of any clear-cut seasonally changing psychosocial variables that would account for variability in mood and behaviour. In this sense SAD is typically understood as the extreme end of a continuum of normative seasonal variation in mood and behaviour; or 'seasonality' (Gordon, Keel, Hardin, & Rosenthal, 1999). However, according to the DSM-IV criteria, SAD is not regarded as a separate disorder, rather it is described as a "seasonal pattern," adjectival modifier of some forms of seasonally recurrent mood disorders; that is, as a variant of categorical mood disorder. Clearly there is a need to find a way to link these descriptions together empirically, though it has been suggested that there is more evidence for the former view of 'seasonality' than categorical SAD (Bauer and Dunner, 1993). The symptoms of SAD in the winter are characterized by sadness, anxiety, irritability, decreased activity, difficulties at work, social withdrawal, increased appetite, decreased libido, and an increase in sleep (Rosenthal et al. 1984). However there are many 'atypical symptoms' of SAD, which do not occur in other mood disorders such as non-seasonal depression, which are thought to be the hallmark physiological signs of SAD. These include anergia, hypersomnia, increased appetite, carbohydrate craving and weight gain (McCarthy et al., 2002) which are particularly notable because they contrast with typical symptoms of non-seasonal depression such as insomnia and weight loss. Therefore it should be expected that the atypical symptoms present in SAD should be highly explainable by looking from a human evolutionary perspective.

ii) What is seasonality?

In contrast to SAD, 'seasonality' is used to describe the degree to which seasonal changes influence mood, energy, sleep, appetite, food preference, and the wish to socialize. Seasonality

can be viewed as a dimension ranging from absence of seasonal changes to extreme changes with the seasons, with the most extreme peak of high seasonality being SAD. There is much empirical evidence which supports the hypothesis that seasonality of mood and behaviour is common throughout the population (Kasper et al.,1989;Rosen et al.,1990; Terman, 1988). For example, although only 1-2% of the US population suffer from SAD, an estimated 14.3% suffer from a milder form of SAD called 'Subsyndromal Seasonal Affective Disorder' (Avery et al. 2001). two Thus seasonality forms a continuum within the population, and an average or moderate degree of seasonality in an individual is seen as 'normal'. Although the DSM-IV is correct in defining SAD as a sub-type of depression with a seasonal pattern, I would argue that a key dimension of SAD which the DSM-IV does not mention is a link to seasonality, and therefore an important factor from a causal point of view is missed out. An important aspect of seasonality is its presence in the normal population, which should support an evolutionary perspective and highlight the importance of our understanding of SAD as a disorder related to seasonality.

iii) Distribution of SAD in the normal population

SAD as a phenomenon isn't evenly spread throughout the population. In many samples fewer than 20% of patients with SAD are male (Pjrek et al., 2004, Rosenthal and Wehr 1987). There is in many samples the often criticised fact that there may be a form of bias due to the nature of sampling; for example females may report SAD to clinicians more often than males. However even in a study which measured seasonal variations of symptoms in a healthy population (Schlager, Schwartz and Bromet, 1993), significant symptom variation was observed amongst the 314 women but not the 1556 men, although in this study care must be taken in

extrapolating data from seasonality variations in symptoms to full SAD . Furthermore SAD occurs almost exclusively in women during their childbearing years (Rosenthal and Wehr 1987). The most consistent finding in the literature regarding the spread of SAD throughout the population is that SAD occurs more frequently at higher latitudes. A major hypothesis is that SAD is triggered by photoperiodic variation (Rosenthal and Blehar, 1989) This refers to the fact that the amount of daylight hours vary throughout the year; with more hours of daylight in the summer when the days are longer, and fewer hours of daylight in the winter when the days are shorter. It is the decrease in the hours of daylight which is thought to trigger SAD in the winter by some biological mechanism, analogous to that which occurs in some species of non-human animals such as squirrels (Mrosovsky 1989). Since photoperiodic variation over the seasons is larger closer to the poles it has been hypothesised that with an increase in latitude there is an increase in SAD. This is supported by two major studies in the USA and Norway, which have stimulated many further studies into prevalence and latitude (Lingjaerde et al., 1986; Potkin et al., 1986). However a review of the literature by Mersch et al. (1999) found that the mean prevalence of SAD is twice as high in North America than in Europe. Over all prevalence studies, the correlation between prevalence and latitude was not significant. However a significant positive correlation was found between prevalence and latitude in North America, while in Europe there was a trend in the same direction but much smaller. From this the conclusion can be drawn that the influence of latitude on prevalence seems to be small and other factors, such as climate and genetic vulnerability, can be expected to play a more important role. Thus although the light hypothesis appears to be an important explanation of the etiology of SAD, there are clearly other factors, such as genetics which will now be discussed, which

influence the etiology of SAD.

The findings from the spread of SAD in the general population provide some of the strongest evidence that there are underlying evolutionary factors driving SAD. Particularly the evidence that females patients greatly outnumber male patients, and that this occurs almost exclusively during childbearing years. This supports the theory that seasonality was once a reproductive adaptation. Many studies have found a correlation between prevalence and latitude, but I would argue that even if the light hypothesis is correct, this is a good example where the genetics behind SAD interact with the environment; that is, the mechanism that evolution uses to initiate seasonal changes in behaviour is related to photoperiodic variation. The immediate trigger of SAD could be the change in light intensity, but the reason that lack of light causes SAD is because of deeper evolutionary causes.,

iv) Genetic heritability of SAD

There is emerging evidence that one or more genetic factors may be important in the vulnerability to or protection from SAD. From an evolutionary viewpoint it is crucial that SAD has a highly heritable component. For SAD to work under the laws of natural selection, the phenotypic variation in SAD must be expressed in genetic variation and vice versa. For example, if high seasonality was at one point an advantage, those with a high seasonality phenotype would be more likely to survive to reproduce, but there would only be an advantage to the offspring of those individuals if the phenotype for high seasonality was expressed genetically, so that it could be passed on to that generation, allowing selection to continue.. One study which investigates this has sought to determine whether genetic selection within the Icelandic population over centuries might have played a role in their adaptation to the long arctic winter

(Magnusson et al. 1993). These authors studied rates of seasonal depression in native Icelanders and in a group of Canadians, who were wholly descended from Icelandic emigrants. Both native Icelanders and emigrated Icelandic descendants were found to have much lower rates of SAD than populations along the east coast of the US, despite living at more northerly latitudes. This is consistent with a genetic model of SAD and suggests possible genetic protective factors in the Icelandic population. Such a protective genetic factor might also contribute to the Mersch et al. (1999) findings that European studies have less of a correlation between prevalence of SAD and latitude than US studies. Not only does this study illustrate how vulnerability to SAD is heritable, but it also emphasises the lack of influence the environmental factor of latitude has. It seems, at least in this study, that the genetic influence over the participants overrides the environmental influence.

The largest study of heritability of SAD was a survey of a cohort of 4639 adult twins in Australia (Madden et al 1996). *The Seasonal Pattern Assessment Questionnaire* (Rosenthal et al. 1987) was used to study the degree of seasonal changes in sleep length, social activity, mood, weight, appetite and energy level of the participants. Using genetic analysis it was found that for most of the items, the dizygotic correlation was just less than one half the monozygotic correlation, implying an influence of genetic effects. Multivariate analysis demonstrated that genetic effects exerted a global influence across all changes in behaviour measured and accounted for at least 29% of the variance in seasonality in males and females. Overall, the genetic predisposition to seasonality was most associated with the so-called "atypical" vegetative symptoms of depression, such as increased food intake, weight gain and increased sleep. Again this shows a strong link between the atypical symptoms of SAD and genetic heritability. .

Because genetic heritability doesn't account for all the variation in seasonality, it is likely that seasonality is controlled by many genes (much like height). So although the results of this study indicate a genetic predisposition to vulnerability to sensitivity in seasonal changes, it has not directly evaluated the heritability of SAD, which as a more extreme phenotype could potentially be less heritable than the trait of seasonality. Interestingly one study found that the genetic factors influencing the female pattern appear to explain more of the variance (69%) than those influencing the male counterparts (45%) (Jang et al., 1997). Davis and Levitan (2005) suggest that this may be because specific genetic factors might increase resistance to seasonal behavioural changes in the male, whose role as a ehunter' occurred all year round, while the role of the female 'gatherer' was directly tied to the seasons.

It has only been very recently that research has been able to potentially identify specific polymorphisms of genes which could be implicated in specific traits of SAD. This has been done by using molecular genetics in the form of association gene studies. Association studies search for correlations in the population between a DNA marker and a disorder. If persons with a disorder have an increased frequency of a specific allele, or genotype, it may mean that the gene contributes to vulnerability to the disease (Sher, 2001). There are several candidate genes involved in neurotransmitter metabolism which have been implicated in the biological pathways in which genetic variation is likely to affect liability to SAD and treatment response, especially in polymorphisms of serotonin. Serotonin has been implicated in the development of SAD as many symptoms such as overeating, carbohydrate craving, weight gain and oversleeping can be related to seretonergic dysfunction. Positive findings come from Rosenthal et al.(1998) who found that a serotonin transporter promoter polymorphism (5-HTTLPR) was associated with

SAD and with higher levels of seasonality. Later, Sher et al. (1999) reported that in the general population, individuals who had at least one *s* allele had higher global seasonality scores than those who were homozygous for the *l* allele. This at first glance appears to provide strong evidence that SAD is not only heritable but implicated with certain behaviours, especially atypical behaviours such as increased appetite, eating and weight gain. However there have also been several studies which have reported no significant results (Ozaki et al., 1996; Lezinger et al.,1999).

Melatonin has also been implicated in the pathogenesis of SAD; according to the 'melatonin hypothesis' SAD may be a result of abnormal secretion of or sensitivity to melatonin, and phototherapy may modify melatonin secretion (Rosenthal et. al 1984). Although there is an abundance of literature attempting to prove and disprove the melatonin hypothesis, there are to date no reported genetic studies relating to melatonin metabolism. This is definitely an area which should be looked into for future research. Overall this early work has been encouraging, and molecular genetics has the potential to provide much more conclusive evidence that SAD is genetically inherited through generations. It can also provide more specificity as to which genes affect which symptoms of SAD and the heritability of such symptoms .

Proving that SAD has a genetic component rather than being the result of environmental influences alone, is essential for an evolutionary approach. If there is a genetic component, as the evidence so far points towards, this suggests that it was once important for aspects of seasonality to be passed on as an echo of their usefulness in certain environments. However at present each of these studies must be considered preliminary and needs to be replicated in much larger samples before firmer conclusions can be drawn. from association gene studies.

I propose from the above evidence that, firstly, it is likely that SAD is present on the continuum of seasonality, due to the shared 'atypical' symptoms which are present in both. Secondly, it appears that variation in seasonality, and to a lesser extent SAD, can be accounted for at least in part by genetic heritability. Thus there must be genes which regulate seasonality in the general population, and which have been inherited from our distant ancestors. This provides good reason to expect that looking at SAD from an evolutionary viewpoint can tell us a great deal about the etiology of SAD.

Part Two- Evolutionary viewpoints of SAD.

"The goal of research in evolutionary psychology is to discover and understand the design of the human mind. Evolutionary psychology is an approach to psychology, in which knowledge and principles from evolutionary biology are put to use in research on the structure of the human mind. It is not an area of study, like vision, reasoning, or social behaviour. It is a way of thinking about psychology that can be applied to any topic within it." - *Cosmides & Tooby, 1997*

Now that a preliminary background has been set out which suggests the evolutionary perspective has the potential to increase our understanding of the etiology and epidemiology of

SAD, several theories are described below which help to explain some of the key findings from research into SAD.

i) Seasonality as a failure of adaptation

One theory which looks at SAD in evolutionary terms is the hypothesis that seasonality is the result of a failure of adequate adaptation. Therefore it is a disadvantage to those living at high latitudes; that is, it decreases the probability of survival. The main proponent of this hypothesis is Leo Sher (2000).I support Sher's view that there is enough evidence to suggest that genetic factors play an important role in the etiology of seasonality, which is something this theory relies on. There is evidence that the development of man's biological rhythms may be related to the unique properties of equatorial dawn and dusk twilight zeitgebers (external time cues) (Kern 1998). This has probablyoccurred through millions of years of evolution and adaptation, so that the biochemical and physiological systems of human beings are at an optimal level to function for survival under equatorial environmental conditions. However since man migrated out of Africa an estimated 150,000 years ago, the genetic sensitivity to light as a means of the body 'time-keeping'already in place has had insufficient time for meaningful evolutionary changes to occur to adapt to more temperate latitudes. Sher (2000) supports the idea that a genetic susceptibility to seasonal changes in mood and behaviour exists and is a genetic predisposition to an insufficient adaptation to temperate and high latitudes. It is at first difficult to see how daily biorhythms which are controlled by the equatorial zeitgeibers can be modified to account for seasonal variations. However this can be argued as follows: the body is programmed to wake up at dawn, and go to sleep at dusk. The measurement for this is the light intensity, which is also supported by studies relating to SAD and light therapy (Lewy et al.1998). Then in the winter at

temperate latitudes, when light intensity is lower and dawn is later and dusk is earlier, if the body is still sensitive to light variations and this is the mechanism used for time-keeping, physiologically the body will be in a constantly sleepy state, somewhat analogous to constant 'jet-lag'. This explains very clearly certain factors of seasonality, including the lethargy and hypersomnia. However it difficult to explain some of the other symptoms, such as increased appetite and weight gain.

Sher (2000) explains the individual variation in susceptibility to SAD by proposing that when humans migrated out of Africa they could be broadly classified into three groups according to their genetic structure affecting seasonality. Firstly, individuals who were very sensitive to seasonal change (who did not survive). Secondly, individuals who were moderately sensitive to seasonal change (some of which did not survive and some whose descendants are now more susceptible to SAD) and thirdly, those with low sensitivity to seasonal changes whose descendants survived and flourished. Although this gives an account of possible variation in the degree of seasonality, I would argue that this section is contradictory. If humans, after evolving for millions of years in the equatorial region, had such an optimally genetically programmed physiological time-keeping mechanism which was unable to adapt in the 150,000 years since migration, then it is highly questionable how variation arose affecting seasonality in the first place. I would argue instead that it is possible to explain this variation by saying that when at the equatorial regions perhaps not all of the human population were adapted to use light intensity as a means of time-measurement, but used an internal body clock, independent of external time cues. Indeed there is evidence that the body, without external time cues (Scheer et al., 2007). It could also be the case that the variation occurred once people had migrated out of Africa, as

Eagles (2004) suggests.

Evidence in support of the theory of seasonality as a failure of adaptation, which Sher (2000) notes is the association found between high level of education and high degree of seasonality has been found (Saarijarvi et al., 1999)). Saarijarvi et al. propose that this is likely to be because educated people might be better at recognising their seasonal symptoms and so this is a sampling bias. However Sher (2000) puts forward another explanation; that historically, among those humans who were moderately sensitive to seasonal changes, only the most intellectually capable would have been able to survive despite their disadvantage to temperate latitudes. Thus those more intelligent individuals would have been the ancestors of those with high seasonality. Eagles (2004) has added to this hypothesis as an example that Scotland, which is a small country at a temperate latitude, has produced a hugely disproportionate number of important inventions, such as the telephone, television, and penicillin, compared to larger, more equatorial countries. However this appears to be a more tenuous link at best. Eagles (2004) also points out that cyclothymia, a common feature of seasonality, has long been associated with increased creativity (Courtet 2003). Ironically, if SAD does have an association with a higher level of intelligence, through its original disadvantage allowing only the most intelligent of those moderately affected by seasonality to survive, this association may now in the modern dayconstitute an evolutionary advantage through seasonality (Eagles, 2004). This highlights the importance of the interaction of evolution with environment; at one time in evolutionary history a trait may be selected against, but at another time it will be selected for. This link with increased intelligence might also explain why seasonality is still present in the general population today.

Looking at the evidence that SAD appears to be an extreme form of seasonality, which exits

on a continuum in the normal population, as well as the genetic factor of seasonality, this theory does seem plausible. However much of the evidence for it is indirect, relies on speculation, and is difficult to test, compared to the theories which suggest that seasonality may once have been more adaptive for reproduction and survival in the temperate and high latitudes (Davis & Levitan 2005). One of the key points on which the 'failure of adaptation'theory relies is that seasonality is a maladaptive trait at these latitudes, which has not yet been selected out due to an insufficient time period. However Eagles (2004) points out that many other striking examples of evolutionary change have occurred in the 7500 generations or so since man migrated from the equator, for example skin colour. The severely weakens the above arguments. Evidence from the Icelandic study (Magnusson et al. 1993) in which it appears that genes providing immunity from SAD also add another dimension to this argument. If seasonality is such a disadvantage, then surely it should have been selected out in the general high latitude populations, such as with the Icelanders. .

ii) Seasonality as an adaptation

The other main evolutionary viewpoint that some theorists, such as Eagle (2004) and Davis and Levitan (2005), support is that seasonality evolved as a specific adaptation to living in a higher latitude environment. The 'seasonality as an adaptation' theory suggests that seasonality plays a part in improving likely survival of offspring and improving reproductive fitness with the various changes in behaviour providing mechanisms, such as energy conservation. To be more specific, it is useful to look at certain 'atypical' characteristics of SAD which mark it out from non-seasonal depression- notably increased appetite and hypersomnia.

Seasonality was a once useful phenomenon in terms of reproductive cycles and energy

conservation There is evidence not only that birth rates are seasonal, and vary by geographical location, but that peak birth rates correspond to the season of optimal reproductive success (see Davis and Levitan (2005) for a review). Furthermore seasonal birth patterns are more pronounced in women with SAD than in the general population (Pjrek et al.2004). Because humans have the capacity to reproduce all year round, reproduction should only be constrained by environmental effects, such as a general food shortage. Because at high latitudes there is a seasonal food shortage, reproduction should be favoured at reproductively advantageous times of year to compensate for this (Bronson 1995). This could be another reason why it appears that the prevalence of SAD increases with latitude.

Seasonal Affective Disorder also appears to affect a greater majority of women than men. If seasonality effects reproduction, and if reproduction is a greater cost for females in terms of energy constraints, then birth rates should show seasonal variation (Bronson 1995). There is good reason to believe that female reproduction is influenced by energy constraints more than males. In females, onset of puberty and ovulation cycle is influenced by restricted food intake and intense exercise. Davis and Levitan (2005) propose that these stressors would have occurred together in societies that had seasonal access to food sources and would have had to move in search of food and water during the season where resources were limited. In comparison, environmental pressure has little effect on male reproduction potential as spermatogenesis requires less energy and so is not dependent on seasonal food resources.

In modern times, the effect of season on birth rate has disappeared, probably due to modern influences such as food being more available all year round, as well as the introduction of birth control and family planning which has allowed couples to decide when to have children.

However there is some evidence from the mid 19th to early 20th century which supports the hypothesis that there is a season of births, for example James (1990) in a review of the pattern of births in European and North American countries found a seasonal trend with a major peak in February-March with a minor peak in September and a trough in summer. This has been supported by a study of births in a Canadian community in the 19th century which found a peak in February and a trough in July-August (Lummaa and Tremblay 2003). Furthermore, James (1990) found that the magnitude of this seasonal pattern correlated positively with latitude with the peak shifting from February to March in higher latitudes. It is advantageous for the cycle of pregnancy to have occurred at these times for several reasons, which affect the survival chances of the mother, the offspring, and the other members of the group.

Final trimester pregnancy and lactation are also highly demanding in terms of energy on the female. Although pregnancy is nine months long, the most energy demanding periods are during the final trimester and over early lactation whilst breast-feeding demands are high. Furthermore during the final trimester and shortly after birth the female will be less able to contribute to securing resources for others in the group (Davis and Levitan, 2005). Seasonality may have been a mechanism which helped to avoid conceptions ninemonths prior to unfavourable times of the year, when the energy requirement for giving birth and lactation would be at its highest and also lack of contribution would be most impacting for other members of the group.

There is much evidence that early environmental conditions, such as poor maternal nutrition during pregnancy, can affect birth weights, which in turn have life-long effects on adult survival, such as susceptibility to chronic disease (Barker 1994). There is also much evidence which suggests that early environment can also affect reproductive potential of the offspring. Lummaa

et al. (2003) have conducted a series of studies which examined how early developmental conditions are related to various downstream indices of reproductive success. Lummaa and Tremblay (2003) found evidence that early environment influences future reproductive success. Using data collected in the 19th century in a Canadian population, they found thatearly environment of the female offspring influenced her own long-term reproductive success as measured by number of live-born children, the number of grandchildren, and her age at last delivery. This provides good evidence that early environmental conditions influence not only the current offspring but span several generations of reproductive success. Thus not only is there evidence that birth rates are seasonal (James 1990), but that giving birth at certain times of year provides a reproductive advantage. Mothers which conceive in may/June and give birth in the late winter/spring would have had access to food supplies during most of their pregnancy. Late pregnancy and lactation would occur after the annual harvest, which requires the greatest energy expenditure, and so the female's lack of contribution would have the least impact (Davis and Levitan, 2005).In addition to this, it has been suggested that seasonality could be a mechanism which helps females to maximise the efficient use of energy resources by the physiological changes themselves (Eagles 2004). For example, some of the changes which occur during winter depression are very similar to those which occur during a normal pregnancy, such as changes in food preference, with women preferring more sweet tasting foods high in carbohydrate. (Skinner et al. 1998). Eagles (2004) points to several studies which show that the dietary intake increases during pregnancy, mainly from carbohydrates (Sacco et al. 2003). Another similarity between SAD and pregnancy is change in sleeping patterns. Santiago et al. (2001) found that sleep time increases during the first trimester of pregnancy, normalizes during the second trimester and

decreases during the third trimester, accompanied by a decrease in daytime alertness. Eagles (2004) suggests that this signals that the autumn through to winter was the preferred time for pregnancy, and this is reflected by women burning less energy, eating more carbohydrate, sleeping more and becoming more passive during the day. This would have enhanced the in utero environment of the offspring and made the mother fatter with more energy reserved to be able to continue lactation for longer.

As SAD is highly heritable, the pattern of birth rates in SAD patients should mimic the seasonal pattern of birth found by James (1990) and Lummaa and Tremblay (2003) (Davis and Levitan 2005). One season-of-birth study of SAD patients reports an unexpected increase of births in May (Pjrek et al. 2004). Another study which measures season of birth more indirectly is Pjrek et al. (2007) which measured season of birth in the siblings of SAD patients to test the parental conception habits hypothesis. They found a significant deviation between the birth distribution of the siblings and the general population calculated on a monthly basis. There were fewer births than expected in the first and fourth quarter of the year and an excess of births in the second and third quarter. There were no significant differences between the group of SAD patients and their siblings regarding their birth patterns as calculated by months or quarters. This study provides further support for the hypothesis of specific parental conception habits underlying the birth seasonality in SAD. However Davis and Levitan (2005) point out that since SAD only became a diagnostic criterion in the 1980's, a time when birth control and family planning has greatly decreased birth patterns in Western society, it will be difficult to find stronger or more direct data to test this hypothesis.

The above arguments which propose that seasonality appears to have had an adaptive

purpose when our ancestors migrated from the equatorial regions into higher latitudes with harsher winters and limited food sources seem to be well supported with strong evidence that; firstly there is, or was until the last 100 years when family planning was introduced, a seasonal birth rate pattern. Secondly that the early in utero environment of the foetus affects the probability of adult survival as well as reproductive success which spans several generations. And thirdly, that peak birth rates correspond to the season of optimal reproductive success. There is also some, although limited, evidence that women with SAD continue to show the traditional seasonal pattern of birth. However there have been few studies which measure this aspect directly and this is an area where further research could be used to support this hypothesis. However even if the SAD as a 'failure of adaptation' theory is less empirically supported than 'SAD as an adaptation', it is surely correct in stating that, in the modern era, seasonality poses a reproductive disadvantage. Six months per annum of low mood and social withdrawal will not enhance the opportunity of finding a mate, and genetic selection may have played a major part in determining the relatively low levels of SAD in northerly areas such as Iceland. Thus although seasonality may have once been a useful trait for reproduction, the pressures which were faced by females in terms of energy constraints are less applicable in the modern era, making SAD a maladaptive heritable trait, rather than an adaptive one.

The above theories all appear to focus of certain 'atypical' traits of SAD, such as hypersomnia and increased appetite. However ,surely for seasonality to have once been adaptive, all of the physiological, psychological, and behavioural changes which are apparent, including low mood, should have some corresponding evolutionary purpose. One thing that the above theories do not appear to address directly is the possible adaptiveness of the psychological low mood which is

apparent in SAD. It is possible that the low mood present in SAD is a by-product of the other symptoms of seasonality. For example, being lethargic and having carbohydrate cravings, similar to being pregnant, might be maintained by hormones which also produce low mood. Or the states of lethargy and having increased appetite in themselves might trigger low mood. However there is some indirect evidence that the low mood present in SAD does have an adaptive purpose. For example Nesse (1999, 2000) has argued that low mood may have had adaptive value. Thus in the next section I will be taking a closer look at the hypotheses that non-seasonal depression and low mood had, and possibly still have, adaptive functions, and how SAD and depression, as another affective disorder, are related.

iii) Low mood in SAD

Nesse (2000) has suggested that depression is an adaptation. Although the typical symptoms of non-seasonal depression and SAD are slightlydifferent, they are both types of depression, and both have symptomsoflow mood and anxiety . Thus by looking at Nesse's work, I attempt to adapt and apply this theory of low mood having an adaptive purpose in non-seasonal depression to it having an adaptive purpose in SAD, along a similar vein as the previous section, but by promoting group cohesiveness at more difficult times of year.

For example, Eagles (2004) has extrapolated from the theory in which depression is described as a "yielding subroutine" in the context of rank theory in human groups. In this theory,submissiveness avoids disputes and promotes group cohesiveness (Stevens and Price, 2000). In male–female pairs, the submissiveness derived from winter depression may also have promoted pair-bonding, which would increase the survival chances of a mother and her infant. Prolonging the pair-bonding for the maximum possible period of time would make it more likely

that the father would continue to provide protection and food, especially at a time when the mother would be less able to provide these things for herself and her offspring. (Eagles, 2004) Also before birth, the lack of activity and low mood of the woman would keep the woman more sheltered from danger to bear and care for her children. Clearly, it would remain advantageous if the strength of pair-bonding endured into the following summer when conception may have occurred once more. As discussed earlier, one reason why males are less affected by seasonality than females is that a less active and socially withdrawn male, whilst being adaptive for conserving his own energy, would be less proficient as a provider to the pregnant female (Eagles 2004). In this way, the evolutionary perspective can be used to show how ancestral social environmental pressures also influenced the evolution of seasonality. Keller and Nesse (2005) in a consideration of whether low mood can act as an adaptation look for evidence in which subtypes of depression have different symptoms which match the precipitants of the depression. They looked at certain causes of depression, such as romantic loss, social isolation, failure at an important goal, stress, and wintertime and see how symptoms manifest in each case. For example failure at an important goal is expected to elicit symptoms of fatigue, pessimism, self-reproach and sadness, whilst wintertime is associated with fatigue, pessimism, high appetite and more sleep. Each aspect of low moodis an adaptive mechanism suited to a particular situation, and is elicited depending upon what the cause of the depression is. For example, the purpose of crying is to elicit empathy and comforting behaviours from observers. It elicits empathy (Labbot et al., 1991), and may help strengthen bonds between people (Frijda, 1986). They hypothesize that one function of crying in adults is to solicit help and to strengthen weakened social networks. Thus more crying is expected when the precipitant involves a lack of social support. By applying this

to SAD, I propose that one aspect of low mood found in SAD, namely crying,may have been an adaptive mechanism for a female to elicit help from other members of a group or from a male when she was unable to spare the energy to search for food and water due to heavy pregnancy.

Fatigue, a common symptom of SAD, can also be seen as adaptive and signals depletion of energetic resources and motivates energy conservation. Fatigue decreases exertion, conserves resources, and reduces goal pursuit in ways that should be adaptive when future effort is unlikely to pay off or when the environment is generally less favourable, such as might have occurred during the winter among human ancestors. In winter-time in the northern climate, the most available food was that which was stored, and therefore easily available, there was less need for exploration which expends energy. Decreased activity would be useful in preventing a female from wondering too far from the group in cold and potentially dangerous conditions, especially if she was already pregnant. Even if stored food sources were scarce, a pregnant female, due to her increased energy demands, may have lost more energy by trying to find food than could be potentially gained. On the other hand members of a group, especially the males, would have been able continue to search for food as they were able to gain more from the energy equation, especially if they were better at hunting animals which would have still been available in the winter-time. Thus looking at the evolutionary viewpoint, we can see how the seasonal symptom of fatigue would have been more beneficial evolving in females than males.

Pessimism is another possible adaptive symptom of SAD, in that it diminishes initiative and withdraws the organism from efforts towards unreachable goals (Kilnger, 1975). Thus during winter, pessimism would have been a mechanism which might prevent the female (and some males) from foraging if there was little to be gained,.

The results from the Keller and Nesse (2005) study indicate that the symptoms of normal low mood differ depending on the kind of situation that precipitated the low mood. This supports my argument that low mood is also an adaptive part of SAD, rather than a by-product, because these symptom differences correspond to those we might expect if the symptoms were partially differentiated to respond to the specific situations that recurred during our ancestral past. Keller and Nesse also support this hypothesis, and statethat this apparent solution-to-problem structure in nature suggests adaptive design. It was found thatfatigue and pessimism were higher in response to failure, stress, and wintertime depression, when these responses would have conserved energy in our ancestral past at times when vigour and initiative would have been maladaptiveIt can be concludedthat the existence of these predicted subtypes provides the strongest evidence to date that low mood is a useful defence shaped by natural selection. This also helps to explain why depression (and SAD) is so prevalentHowever low mood may be activated more often or more intensely than seems necessary because false alarms are often less costly than failing to respond when a response was warranted (Nesse, 2001). SAD could clearly be a such a case, especially in the modern era; pessimism, fatigue, increased appetite and general low mood evolved to deal with contexts different from those we face today, which may explain why such a level of seasonality may be maladaptive now.

Dividing the evolutionary perspective into symptoms brings us closer to linking SAD as a subtype of depression and SAD as an extremity of the seasonality continuum. There is good evidence which has been established from a range of studies that seasonality is present in the normal population (Kasper et al. 1989, Mersch et al. 1999; Schlager et al. 1993). There is also good evidence that SAD is associated with depression (Oyane et al. 2008). It is known that in

non-seasonal affective disorders, much like SAD, there are no clear cut off points between ‘‘normality’’, cyclothymia, and mild bipolar affective disorder (Eastwood et al. 1985). As supported by Keller and Nesse (2005), low mood is not maladaptive in and of itself, it is largely dependent upon the environmental context as to whether low mood should be called depression. Thus I suggest that much is dependent upon how we classify what a dysfunction and a disorder is, as to how SAD should be classified in terms of being on a continuum of seasonality, and whether this should be included in the DSM-IV. An added difficulty to this is the possibility that SAD, as a trait influenced by many genes, could tend to show high levels of maladaptive genetic variance (Houle, 1998; Charlesworth and Hughes, 1999). Seasonality could have been adaptive to our ancestors in the past, but be maladaptive and classified as SAD now because of a simple mismatch between the ancestral environment and how we live in the modern era. However it could be the case that the current phenomenon of SAD is a recurrent mutation which has led to dysfunction of mood regulation mechanisms. This is still a problem for my argument, as it could be an alternative which does not require there to be a relationship between SAD and seasonality. However I would propose that there is still the strong supporting evidence of varying seasonality within the normal population.

Part Three- How should we classify SAD?

“Mental illness is nothing to be ashamed of, but stigma and bias shame us all.”-Bill Clinton

i) Problems with the DSM-IV classification

It is difficult to find the point at which to stop calling the degree of seasonality, ‘seasonality’, and start calling it SAD. By looking at SAD as a mismatch between the current environment and our

ancestral environment, it would be easier to view SAD as being the point above a peak optimum threshold level, with that threshold varying with level of seasonality and adaptation corresponding to the environment. In the ancestral environment, a higher level of seasonality would have been more appropriate in the environmental context, and so SAD would occur at a higher level of seasonality than in modern times (see Figure).

If a higher level of seasonality was an adaptive mechanism, as much of the evidence suggests, and furthermore today it is clearly a maladaptive mechanism, because the threshold level has lowered,, then it must be questioned how we classify SAD, and whether the DSM-IV definition is sufficient. According to the DSM-IV, SAD is not considered a disorder or syndrome on its own. Instead, SAD is considered a pattern specifier, or subtype, of another mood disorder diagnosis. For example, an individual may be diagnosed as having a major depressive

Figure showing theoretical model of interaction curves of modern and ancestral environments, with level of seasonality, adaptation to environment, and thresholds of SAD

episode with a seasonal pattern. However this clearly doesn't take account of certain factors. Primarily looking at SAD from an evolutionary viewpoint doesn't necessitate that SAD be related to a separate mood disorder at all in terms of causes, but rather it could be described as a phenomenon in and of itself I would argue, that based on what can be learned using the evolutionary viewpoint of the causes, it is useful to make a distinction between SAD, where extreme seasonality is the cause, and depression with a seasonal pattern. Although this may appear to be a small distinction, the implications for treatment would be significant, due to the differing precipitating factors . I would also argue that the DSM-IV does not highlight the 'atypical' symptoms of SAD which are markedly different from depression, including increased sleep compared to insomnia, and increased appetite compared to decreased appetite.

ii) Is SAD, or seasonality, a mental disorder?

A further issue of diagnosis has been raised by Horowitz and Wakefield (2007) in relation to depression, but it can also be applied to SAD. Whilst they acknowledge that depression certainly exists and can be a devastating condition, the large increase in diagnosis of depression is less likely to be caused by more people getting depression, but by psychiatrists taking normal sadness and reclassifying it as depression. As Nesse (2000) argues, low mood can be adaptive, so surely if the degree of depression is correct in relation to the context of causation, then this low mood

cannot be seen as a mental disorder. Horowitz and Wakefield stress the importance of distinguishing between abnormal reactions due to internal dysfunction and normal sadness brought on by external circumstances. For example, if someone has a close friend who dies suddenly and unexpectedly, this person might cry for two weeks, feel extremely low, fatigued, and have disturbed sleep and little appetite. According to Nesse this would be part of the normal grieving process and has stemmed from its once adaptive use of eliciting comfort and strengthening social ties. However if someone had all these symptoms with no precipitating cause then this would be more likely a problematic dysfunction and should be termed depression and a mental disorder.

The evolutionary viewpoint of SAD has clearly allowed us to see the adaptive function that SAD once played. Does this mean that we shouldn't be calling SAD a disorder at all, in the same way that Horowitz and Wakefield argue that normal sadness shouldn't be called a disorder? In an interesting and recent paper, Block (2008) tries to put these views into perspective. Block argues, as Wakefield does, that dysfunctions should be understood in Darwinian terms as failures of naturally selected functions. Block proposes that Darwinian theory is not only relevant to the understanding of the disrupted functions, but it also sheds light on the disruption itself, as well as on the harm that accompanies the disruption. The arguments stress a core feature of Darwinism that take into account the environmental relativity of functions and dysfunctions. Block also focuses on a close relationship between social judgements and dysfunctions. According to Block, if SAD positively influences reproductive success of humans in higher latitudes, then SAD is not a dysfunction, and hence not a disorder, in places Scandinavia or Alaska. Also people from equatorial Africa, lacking the capacity to develop mild SAD, do not suffer from a

dysfunction, because they do not gain advantage by having SAD.

However if someone from Scandinavia without the capacity for SAD moves to equatorial Africa, according to Wakefield's 'harmful dysfunction analysis' (HDA) (Wakefield 1992) this would be seen as adysfunctional individual taking advantage of a dramatically changed environment. However Block argues that the absence of the biological mechanism that leads to SAD is not dysfunctional in the new environment, and its absence cannot be dysfunctional, because its presence would not be functional. So they would be in the same position as the person from equatorial Africa who lacks the capacity to develop SAD. If someone from equatorial Africa with SAD moves to Scandinavia, Wakefield would see this as the same for the first case; there is a dysfunction, but the dysfunction is not harmful. However Block argues that there is no longer any dysfunction, because they would be benefiting from the changed environment.

Block's (2008) account appears to make more intuitive sense. It does not make sense to suggest dysfunction exists if there is an absence of any harm, and likewise it does not make sense to suggest there is a dysfunction if the person is benefitting in the new environment from a trait which was once useless or even disadvantageous. There is clearly a strong relativity depending upon the how the trait and environment interact. This provides good support for my main argument that the line between SAD (being a harmful dysfunction) and seasonality (being a neither harmful nor a dysfunction, and sometimes an advantage) can be drawn, depending upon the environmental context.It is clear that SAD, in the modern world, is not only useless but in many cases harmful. As Sher (2001) and Eagles (2004) point out, symptoms of lethargy, social withdrawal and low mood for part of the year will make it less likely that modern SAD sufferers

would find a mate and reproduce, and so the situation has reversed. Using Block's distinction, in the new environment SAD is a dysfunction, and thus should be classified as a mental disorder. However it is important to note that many of the above studies are primarily relevant to Western culture, as almost all of the studies were conducted in the U.S or Europe. Thus the results cannot be generalised to other cultures in which there may still be situations and environments where a higher level of seasonality may still be adaptive and so wrong to class it as a dysfunction.

It is less clear cut, however, whether seasonality should be called a dysfunction. For example even those who claim to not be seasonally affected or that their seasonality isn't severe enough to cause them problems benefit from light therapy.(Partonen & Lönnqvist, 2000). Thus even mild seasonality can compromise optimal health. Block (2008) also emphasises the connection between social values and dysfunction. If seasonality is relatively normal at high latitudes, and there is little or no perceived harm then it is difficult to see why less severe seasonality should be called a disorder. If we did this might result in 'universal diseases' syndromes, like ageing that decrease everyone's health yet persist because they give an overall reproductive advantage (Nesse 1999).

Conclusion

The aim of this dissertation has been to provide empirical evidence, not only that it is reasonable to look at SAD from an evolutionary viewpoint, but by looking at such theories of SAD we can learn much about the etiology of the disorder.

I conclude that, although it is difficult to either prove or disprove whether seasonality evolved as a failure of adaptation, the theory that seasonality, including the accompanying low

mood, evolved because it was advantageous for reproduction and energy conservation is high latitudes is more fits the evidence better. Certainly the variation in seasonality in the current general population is more explainable by the latter view. I don't believe that these views are necessarily mutually exclusive, however. Although this has not been argued previously, the two theories could fit together if it was the case that moderate levels seasonality were originally a disadvantage, however survival with this disadvantage promoted natural selection in higher levels of intelligence (Eagles 2004), which then became a reproductive advantage, so that moderate levels of seasonality were secondarily selected for after intelligence.

Although the current DSM-IV classification of SAD as a subtype of affective disorder, I think that the classification should be expanded to account for current research which examines SAD from a causal view of the symptoms, which have a strong evolutionary component linked to seasonality. It is my view, as I have put forward, that there is much more empirical evidence supporting the view that SAD exists as a highly genetically heritable trait, and this needs to be accounted for. Although SAD is a subset of depression, this additional classification allows more specificity towards seasonal symptomsRather than looking at each disorder as a whole, looking at the context and the precipitants of low mood and other symptoms of non-seasonal depression and SAD can give us a greater understanding of how each evolved as a mechanism designed to be of use in specific situations, and can give us a clearer understanding of how to adequately treat each condition.

In the final part of the dissertation I discussed how SAD and seasonality should be classified. The evolutionary perspective, which is centred heavily on Darwinian principles, takes adaptation to a specific environment to be of utmost importance. Although evolutionary theories propose

that seasonality was once an adaptive trait for humans living in the ancestral environment, it is evident that in the modern environment SAD has become a disadvantage. Thus I have concluded that SAD should be seen as a disorder. One area of future research which I think would help to increase our understanding of SAD is further research into molecular genetics. Although this method of study has only started being used recently, some positive results have already been found (Rosenthal et al. 1998). Further work using molecular genetics could help to identify which genes are related to what symptoms of SAD.

There is also much debate on what role melatonin has in SAD. Studies of light therapy (Lewy et al. 1998) have had several positive results, however no genetic studies relating to melatonin metabolism have to my knowledge been reported which could help resolve the debate and support the already positive findings of light therapy.

A potential study could be to measure birth rate patterns in women with SAD who are not taking contraceptives. If women with SAD show the traditional seasonal pattern of birth with a peak in early spring, this would help to support the evolutionary hypothesis that SAD was a mechanism to improve reproductive success in our ancestors. Furthermore if their offspring then show a similar exaggerated pattern in comparison to the general population this would provide even greater supporting evidence.

Further research which would help to directly support my argument from this dissertation would be to find more evidence of seasonality within the normal population. Longitudinal studies may also present a useful way of measuring how seasonality changes over time within individuals, and how this can be a predicting factor in SAD.

One of the most importantpositive influence and implication which evolutionary theories have had already is in de-stigmatising SAD. If we have an understanding that SAD is not simply a diagnosis with no cause or prognosis, but is a part of human evolution which is no longer relevant to modern living conditions, then it won't be seen simply as an 'abnormality', but as a complex trait which is present in varying degrees within the normal population.

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