Changes in dream frequency, vividness and intensity in subjects taking Selective Serotonin Reuptake Inhibitors

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Compulsory declaration

This work has not been previously submitted in whole, or in part, for the award of any degree. It is my own work. Each significant contribution to, and quotation in, this dissertation from the work or works of other people has been attributed, and has been cited and referenced.

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Abstract

Various sources of evidence suggest that dream frequency, intensity and vividness are increased with the use of selective serotonin reuptake inhibitors (SSRI's) commonly prescribed for depression and anxiety. In this study the effects of SSRI use on dream frequency, intensity and vividness in psychiatric patients was examined through a comparision of the dream characteristics of an SSRI- medicated patient group vs. an unmedicated patient control group. Each group comprised 20 patients recruited through state psychiatric outpatient facilities affiliated with two research universities. Age and gender were evenly spread across the two groups. Psychiatric disorders represented were generalised anxiety disorder, panic disorder, obsessive-compulsive disorder and social phobia. Patients completed a questionnaire consisting of a most recent dream report and self-report likert-scaled questions regarding dream frequency, intensity, vividness and memorability. Two independent raters rated the dream reports for intensity on a likert scale. Self-reported visual vividness was significantly higher ($p=0.027$, effect size .86) among SSRI users compared with controls, whereas self reported dream frequency and emotional intensity as well as independent raters assessment of dream intensity were not significantly different across the two groups. Findings of increased dream vividness without increases in dream frequency complement the results of an earlier study. Since serotonin is suppressed during REM sleep, these findings cast further doubt upon the notion of an isomorphic link between REM sleep and dreaming and argue for the search for a more sophisticated model of neurotransmitter modulation of sleep-cognition.

1. Introduction

A number of case studies and anecdotal reports have shown increased dream frequency and intensity in patients taking Selective Serotonin Reuptake Inhibitors (SSRI's) for a variety of psychiatric conditions (Pace-Schott, Gersh, Silvestri & Hobson, 2001). Patients describe dreams that differ markedly from the norm in vividness or intensity, with one patient stating that her dreams "were so exiting she couldn't wait to fall asleep to find out what will happen" (Markowitz, 1991, p. 432). This is an unexpected finding given that SSRI's suppress REM sleep by up to 80%, and given the presumed association between dreaming and REM sleep (Hobson, Pace-Schott & Stickgold, 2000a). In this paper it is intended to explore the debate concerning the relationship between REM and dreaming, as well as to test the proposition that dreaming frequency and intensity are indeed increased by SSRI use in the psychiatric population. An outline of normal sleep architecture and physiology will serve as a backdrop to a detailed discussion of the most prominent theories of dream mechanisms, together with the evidence either supporting or contradicting the theories. This will be followed by a discussion of the literature on the influence of SSRI's on dreaming in order to show its relevance for the most established model of dream mechanisms. The next section will explore the action of various neurotransmitters involved in sleep and dreaming in order to avoid oversimplification in understanding the effects of psychiatric drugs on sleep and dreaming. The chapter will conclude with a discussion of problems with the research evidence as part of a general discussion on dream research methodology, and conclude that despite these problems, there is good reason to persevere with the dream research enterprise.

Normal sleep architecture is characterized by two alternating states – Rapid-Eye Movement (REM) and Non-rapid eye movement (NREM) sleep. There are between three and six REM periods in a typical night's sleep with the first occurring around 90 minutes after sleep onset (SO). REM sleep occupies around 25% of total sleep time. There are four stages of NREM sleep, labelled stages 1 through 4, corresponding to increased depth of sleep (Rechtschaffen & Kales, 1968). Stages 1 and 2 are sometimes called light sleep and stages 3 and 4 deep sleep or slow wave sleep (SWS), from which it is difficult to awaken (Perry & Young, 2001, Strauch & Meier, 1966). In NREM most physiological processes as measured by EEG and EMG are subdued compared with both waking consciousness and with REM (Schredl, 2000). NREM stages are characterized

by lower heart rates, blood pressure and respiratory rates. By contrast REM is characterized by a more aroused physiological state with rapid eye movements, which are the main identifying feature of the REM state. In REM there is also a dramatic reduction in muscle tone, 'near total paralysis of the postural muscles' and genital engorgement (Kaplan & Saddock, 2003, p.758).

1.1. Explanatory models of sleep and dreaming

1.1.1. REM sleep and dreams

It was initially supposed that dreaming and REM sleep were synonymous (Solms, 2002, Strauch & Meier, 1996), so much so that REM came to be known as 'dream sleep' (Domhoff, 1966). David Foulkes found in the early 1960's that dream reports could also be obtained from NREM sleep, though he noted that these NREM dreams appeared qualitatively different from REM dreams, being more thought-like and realistic or plausible, and lacking the elaborate narrative or bizarre qualities we commonly associate with dreaming (Foulkes, 1962; Monroe, Rechtshaffen, Foulkes & Jensen, 1965). It is now widely acknowledged that around 75-90% of dreams occur in REM sleep but also that some form of dreaming does occur in NREM sleep, and it has become increasingly apparent that despite reports of qualitative differences between REM and NREM dreams, there is sufficient dreamlike mental activity occurring in NREM to pose a challenge to the accepted assumptions around dreaming and REM as isomorphic entities. Since this debate forms the backdrop to the present research, it will be necessary to examine the three most prominent models of dream physiology in some detail.

1.1.2. Hobson's REM-dreaming paradigm

Hobson has spent much of his research career since the 1960's developing models in the field of sleep and dreaming, beginning with his early activation-synthesis model in which dreams were considered an epiphenomenon of REM. This early model proposed that firing of pontine cholinergic cells activated the forebrain during REM sleep, and that this random activation of sensory and motor systems was then synthesized to produce dreams (McCarley & Hobson, 1979). Hobson and his fellow researchers had shown that REM sleep could be induced in rats by cholinergic agents through an experiment in which REM was increased after injecting cholinergic agents into the pedunculopontine region of the brainstem (Hobson et al, 2000). The component of the activation-synthesis model that accounts for dreaming was built on the work of Azerinsky and

Kleitman in the 1950's who had first noticed an association between REM sleep and dreaming (Hobson et al, 2000a). Hobson based his dreaming model on the assumption that this association represented an isomorphism. Therefore, in this model, dreams were hypothesized to originate as a result of 'noise' generated by the random activity of these cholinergic pontine neurons (Strauch & Meier, 1996). Accordingly, the content of dreams was held to be intrinsically meaningless, or 'motivationally neutral' (McCarley & Hobson, 1979, p.86), turning on their heads widely held views of dreams as meaningful, wishful or necessary, whether from psychodynamic roots or 'folk' belief. Despite some disagreement in the field at that time, this theory and its basic premise of an isomorphic relationship between REM sleep and dreaming became the received view in sleep and dreaming research. This is despite the fact that opponents of the activation-synthesis model such as David Foulkes consistently pointed out that NREM sleep also yielded dream reports, in many cases indistinguishable from those found in REM, particularly toward the later stages of the night.

1.1.2.1. Differences between REM and NREM dreams

The debate around the existence of NREM dreams for the most part focused on whether these dreams are really dreams or simply incidents of sleep mentation. Failure to replicate research findings that supported one or the other position has been attributed to differences in what qualified as a dream (Herman, Ellman & Roffwarg, 1978). It was argued by Hobson and others (Hobson et al, 2000a, Monroe et al, 1965) that independent raters can distinguish the average REM from NREM dream since REM reports are longer and more vivid and NREM dreams more similar to waking thought. The typical REM dream is described as hallucinatory, bizarre, delusional, lacking in self-reflection and orientational stability, and as having increased emotion. These qualities Hobson claimed are found in the majority of REM but rarely in NREM dreams (Hobson et al., 2000a). Some researchers claimed that the longer the NREM dream, the more closely it resembles the REM dream, but that NREM dreams are often shorter. They claim that if the researcher controls for dream length, the other differences tend to disappear (Strauch & Meier, 1996, Cavallero, 2000). A number of authors have effectively countered this by saying that it is precisely *because* these NREM dreams are more mundane and thought-like that they are shorter, not the other way round. Dreams that lack narrative complexity or bizarre imagery will of necessity require fewer words to describe them (Hobson et al 2000a, Nielsen, 2000a).

Antrobus (1983, 1993) postulated that reported differences between REM and NREM dreams might simply be an artifact of differences in ease of retrieval due to physiological differences in attention and memory across the two states. Acetylcholine is suppressed in NREM, which would support his hypothesis, as it is strongly implicated in memory retrieval. NREM is also accompanied by a far lower level of brain functioning overall than is REM (Schredl, 2000). Conduit, Crewther and Coleman (2000) point out that all we *can* assert is that there appear to be differences in the ability to recall NREM dreams on awakening. This view is consistent with the observation that arousals from slow wave sleep (NREM stages 3 and 4) are associated with disorientation and disorganized thinking but also with 'amnesia for events that occur during the arousal' (Kaplan & Saddock, 2003, p.756). Hobson et al. (2000a) concede that differences could be on the basis of differences in memory processes across the two stages but also state this is unlikely and still needs to be proved.

Nowadays few authors would dispute the fact that *on average* there are distinct differences between REM and NREM dream reports, but many have continued to challenge Hobson to account for the fact that it *is* possible to obtain dreams that are indistinguishable from REM dreams, from NREM sleep. Hobson, Pace-Schott & Stickgold (2000b) have argued that where dreaming does occur outside REM, it occurs in the parts of NREM that are closest in time to REM, and therefore subject to overlap with REM states. Vogel (2000) refutes this, stating that NREM dreaming is not increased during the pre-REM period as Hobson suggests. Nielsen (2000a) also showed that the most vivid NREM reports can be obtained from early sleep onset stages, or stage I sleep (90 minutes prior to the first REM stage) and that these are often indistinguishable from REM dream reports. In response to mounting evidence that NREM dreams are not simply carryover effects from REM, Hobson and his team have developed a more sophisticated model, called the AIM model, or Activation-Input-Modulation model, which posits a three dimensional model of dreaming, and have now (mostly) modified their claims to assert only that dreaming is more *likely* to occur in REM, as the physiological characteristics of this state are most favourable to the production of dreaming.

1.1.2.2. Hobson's AIM model

In this revised model, the 'Activation' dimension refers to the brain's activation level, being greater when we are awake than asleep, and greater in REM than in NREM. The second dimension, Input, refers to sources of information, this includes feedback sources of input. Modulation refers to the changes in neurotransmitter levels that influence brain states, most importantly cholinergic, serotonergic and noradrenergic innervation or suppression (dopamine is not considered an important component). It was shown through experiments with rats that serotonin and noradrenalin reduced REM sleep, and that injections of serotonin agonists reduced REM in cats (Hobson et al, 2000a), similarly, an almost universal suppression of REM has been found in humans using antidepressants whose mode of action is to increase levels of the amines serotonin and noradrenalin (Galliard, Nicholson & Pascoe, 1994). The cholinergic-aminergic mediation of REM and NREM has thus become generally established.

Critics of the revised AIM model have argued that there is no empirical support for a three dimensional model (why three for example and not four [Hartmann, 2000]), and that the model is just that – a model, though a plausible description, it is not a scientific explanation of dreaming (Antrobus, 2000). Perhaps the strongest criticism that is leveled at the AIM model is that in much of his writing Hobson still clings to the old REM=dreaming isomorphism (Hartmann, 2000) despite the evidence for vivid dreaming outside REM. For example, in their response to various authors' commentary on one of their articles (Hobson et al. 2000a), Hobson et al (2000b) still maintain their stated preference for the term 'sleep mentation' as opposed to 'dreaming' for dreaming that occurs outside REM sleep on the grounds that dreaming occurs more often in REM and that dreams outside REM are *usually* more mundane.

1.1.3. Solms - REM and dreaming are dissociable states

1.1.3.1. The lesion studies

The strongest argument against Hobson's REM-dreaming isomorphic model came from lesion studies that showed that it is possible to have REM sleep without dreaming as well as dreaming in the absence of REM sleep (Solms, 1997). While Solms subscribes, along with Hobson, to the monist perspective that argues for the necessity of searching for psychophysiological correlates for phenomenological experience, these studies convinced Solms that whatever the psychophysiological correlate of dreaming might be, it could not be REM sleep. This

notwithstanding the fact that dreaming is far more commonly observed in conjunction with REM than it is with NREM. Solms' review of the extant literature, together with his own clinico-anatomical data showed that there are two lesion sites which are known to obliterate dreaming. These are either bilateral lesions in the deep white matter surrounding the anterior horns of the lateral ventricles or lesions of the parieto-temporal-occipital junction (PTO) in either cerebral hemisphere (Solms, 1997). Solms reported 100 cases of loss of dreaming in total, of which 94 were PTO, some (83) unilateral and some (11) bilateral, and the remaining 16 were around the anterior horns of the lateral ventricles, always bilaterally. In addition, Solms argued that lesions in the brainstem areas that obliterate REM do not cause cessation of dreaming. Solms therefore claimed that REM and dreaming are doubly dissociable states (Solms, 2000a). Aside from subjective reports of cessation of dreaming (which might arguably be an artifact of morning forgetting of nighttime dreaming) Solms (2000a) presents as additional evidence the fact that during experiments in which subjects with such forebrain lesions were repeatedly awakened during REM, there were no dream reports elicited. Solms points out that this recent experimental evidence had however already been anticipated by reports in the psychiatric literature to the effect that prefrontal leucotomy to control the hallucinations and delusions in schizophrenic patients also led to global cessation of dreaming, a finding which took some time to reach the dream research community (Solms, 2000a). Almost 1000 instances have been reported of loss of dreaming following these ventromesial frontal lesions.

1.1.3.2. The role of the forebrain and dopaminergic circuits

On purely common sense grounds it seems unsurprising that lesions in the PTO area – an area in which visual association takes place - render mental imagery in the form of dreaming impossible. These patients also report impaired mental imagery in waking life. The role of the frontal ventromesial area in dreaming is less self-evident, and lends itself to theoretical exploration. Solms points out that there are strong connections between this area of the frontal lobes and limbic structures, and that damage along this pathway leads to reduced initiative, imagination and ability to plan ahead. The ventromesial quadrant of the frontal lobes is also the target of antipsychotic medication. Antipsychotics designed to inhibit activity along this pathway result in the reduction of dreaming together with reduction of positive psychotic symptoms such as hallucinations and delusions (Solms, 2000a). Experiments with L-dopa, which activate this pathway, have been shown to cause unusually vivid dreaming without corresponding changes in

REM physiology (Solms, 2000a). Solms also argues that limbic forebrain stimulation can invoke (REM-like) dreaming in the absence of changes in REM frequency, duration or intensity. He thus poses an alternative theory for dreaming generation: that dreaming is attributable to activation of mesocortical dopaminergic projections from the ventral tegmental area, a collection of structures and networks known as the 'seeking' system in Panksepp's terminology (Solms, 1997).

Hobson et al. (2000b) have countered that the pathway from the ventral tegmental area through the limbic system to the ventromesial area of the frontal lobes is not exclusively dopaminergic, and that to focus on this neurotransmitter to the exclusion of others is too narrow an approach. It has long been thought that levels of dopamine do not change significantly between waking and sleeping which may account for its disregard in Hobson's AIM model despite the link between the positive symptoms of psychosis and dopamine, which might otherwise have suggested a possible role for dopamine in dreaming. Hobson et al. (2000b) argue that stimulation of dopamine production does not always enhance dreaming; for example administration of sulpiride, an antipsychotic and dopamine *antagonist* enhanced dream recall, contrary to expectations. The Hobson team (2000b) also pointed out that research into the dreaming of Parkinson's disease patients has produced equivocal results, with a variety of changes in dreaming reported. This unexpected finding is hypothesized to be due to the fact that there are different brain distributions of the different receptor subtypes from one individual to another, whose respective roles are as yet not completely understood.

It is not clear that Solms does in fact claim an exclusive role for dopamine in dreaming. In summarizing his views regarding the normal dreaming process, he states that since limbic seizure activity in NREM sleep increases dreaming, and since dreaming is more likely in a more aroused brain-state (REM) and that stimulation of dopaminergic appetitive circuits can stimulate dreaming, these lines of evidence converge, suggesting 'that *anything* (my emphasis) that arouses the sleeping brain can initiate the process of dreaming.' (Solms, 1997, p.244). In a more recent text, Solms (2001) suggests an interplay between dopaminergic, cholinergic, serotonergic and noradrenergic systems in dream generation, as suggested by Gottesmann's monoaminergic disinhibition hypothesis. Gottesmann (2001) argued that despite the fact that dopamine levels are steadily maintained throughout the sleep-wake cycle, the relative suppression of other aminergic neurons during REM (such as noradrenalin and serotonin) might act to increase the influence of dopamine relative to other neurotransmitters. He argues that receptors that inhibit the release of

dopamine when activated are inactive during REM sleep, thus resulting in a relative excess of dopamine. Gottesmann concludes that any account of dreaming must consider a central role for dopamine. Perry and Piggott (2001) propose a similar model of dreaming that could account for differences in REM and NREM dreaming based on relative levels of dopamine, serotonin, noradrenalin and acetylcholine, which they have dubbed the 'multitransmitter hypothesis'.

The view that dopamine levels remain relatively steady across the sleep-wake-cycle has recently been challenged by evidence that although forebrain dopamine is higher during waking than REM or slow wave sleep, dopamine release in the nucleus accumbens of rats is highest during REM sleep, as compared with waking and slow wave sleep (Lena, Muffat, Deschaux, Parrot, Sauvinet, Suaud-Chagny, Renaud & Gottesmann, 2004). This is an important finding given the reported involvement of the nucleus accumbens in the positive symptoms of schizophrenia (Lena et al, 2004).

1.1.3.3. Challenges to the forebrain dream generator theory

Solms's claim that lesions in the ventromesial quadrant of the forebrain cause cessation of dreaming is now almost universally accepted, though it has been pointed out that lesions here only caused cessation of dreaming around 85% of the time. According to Ogilvie, Takeuchi, and Murphy (2000) this leaves a problem regarding the other 15%, which they argued should also experience cessation of dreaming if this area is obligatory for dreaming to take place. Solms (1997) had however already pointed out that like many other neuropsychological (and indeed neurological) symptoms, cessation of dreaming is typically experienced in the acute stage, and may recover to some degree in the chronic stage. In patients with prefrontal leucotomy, those in whom dreaming returned had a poorer prognosis, as this tended to coincide with the return of psychotic symptoms (Solms, 1997). The finding of cessation of dreaming with lesions in the ventromesial region is now generally accepted. But is it the case that bilateral ventromesial or PTO lesions disturb the process of dreaming itself, or rather the ability to structure the experience into a verbal report and/or the motivation to supply this report? Doricchi and Violani (2000) point out that between dream and dream report, various activities must take place, involving memory, language, affect, motivation as well as visual-spatial imagery. These are not fragmented processes but involve many parts of the brain working together (Greenberg, 2000). Lesions in pathways subserving any of the areas of the brain involved in the dream report production process could

theoretically result in the inability to obtain dream reports. Feinberg (2000) has suggested that patients with lesions in either of Solms's proposed lesion areas would also be entitled to memory deficits that could interrupt the dream report production endeavour.

Solms (1997) seems to have anticipated some of these criticisms in pointing out that loss of dreaming does not correlate with either loss of memory or loss of speech. Patients with either bilateral ventromesial or PTO lesions would not be typically described as densely amnesic either; it would be far more likely to expect such patients to produce a disorganized or sparse dream report than to positively *claim* that they no longer dream. In addition, patients' claims regarding loss of dreaming are backed up by laboratory studies in which REM awakenings in brain-damaged patients who claimed to no longer dream, produced no dream reports, thus validating their claims (Domhoff, 2003). Therefore Solms' finding of cessation of dreaming with forebrain lesions has been a substantial contribution to current dream theorizing, as demonstrated by Gottesman's (2000, p.942) statement that forebrain generators of dreaming 'now seem self evident'.

1.1.3.4. Pontine lesions – cessation of REM without cessation of dreaming?

Solms (1997) claim for evidence that pontine lesions causing cessation of REM do not necessarily cause cessation of dreaming is by contrast not universally accepted (Gottesman, 2000). Various authors argue that this claim cannot be adequately tested since to eliminate a pontine area large enough to cause cessation of REM would either eliminate consciousness (Domhoff, 2003) or at best severely curtail the ability to communicate (Hobson et al., 2000a). The prevailing view seems to be that this is probably not a testable proposition and that insufficient evidence exists to support the view that cessation of REM does not lead inevitably to cessation of dreaming. Solms concurs that this is difficult to establish empirically (2000c), nevertheless he maintains that a small number of his patients demonstrated unequivocally that dreaming was maintained where brainstem lesions had been shown to eliminate REM (personal communication, 2005). Solms also points out that it has *never* been shown that loss of dreaming occurs in conjunction with cessation of REM (Solms, 1997).

1.1.3.5. Summary of Solms' viewpoint

To contrast the essence of Solms' position with that of Hobson, Solms subscribes to a 'one-generator' model of dreaming, in which regardless of which stage of sleep, the mechanisms that generate dreaming are the same. Hobson argues there is one generator for REM dreaming, which he has elaborated in some detail and a separate mechanism for NREM dreaming, which remains relatively unexplored in his model. Moreover, Solms argues that the mechanisms which generate dreaming are not brainstem mediated, but forebrain mediated as suggested by the lesion studies. Solms concedes along with other authors that NREM dreams *are* on average more thoughtlike, but authors differ in terms of what this implies for the theory of dreaming, depending on their theoretical framework. Theorising in the field *has* to take account of the fact that at least 10% of NREM dreams are indistinguishable from REM dreams, particularly at sleep onset and during later portions of the night. It also has to take into account the fact that lesion studies have demonstrated that REM and dreaming are dissociable states.

1.1.4. Nielsen – an attempt to bridge the two positions

1.1.4.1. Nielsen's covert REM model

Over and above the challenge to accepted notions regarding REM and dreaming, many authors now see sleep stages as far more fluid and overlapping than previously thought, with transitions between some sleep stages showing physiological features of both REM and NREM (Feinberg 2000). Domhoff (2000) points out that EEG monitoring of individual subjects rarely supports the neat delineation of stages that have been the model of sleep physiology for some time. Nielsen (2000a) reports that there are 'intermediate' sleep states that occupy between 1 and 7 percent of sleep time in normal subjects and between 10 and 40 percent in psychiatric conditions such as psychosis. REM deprivation leads to an increased time spent in these intermediate states of sleep. A REM rebound phenomenon may also result following REM deprivation, in which there are repeated attempts to enter REM during NREM, as well as longer REM episodes and decreased time to the first REM period (REM latency). His research showed that NREM mentation was more similar to REM mentation when it was obtained within 5 minutes of a REM period (81%) than after 10 minutes of a REM episode (3.8%). Building on these observations, Nielsen (2000a) argued that dreams reported in NREM might be due to what he terms 'covert' REM activity occurring during the NREM stage of sleep, thus returning to the REM=dreaming model via changes to the definition of NREM. By covert REM, Nielsen therefore means that parts of REM physiology, including some eye movements, intrude into NREM sleep. Nielsen thus attempted to

bridge the conflicting theories by postulating these covert REM processes that occur at the boundaries of REM/NREM transitions and at sleep onset.

1.1.4.2. Some criticism of the covert REM model

Ogilvie et al (2000) have criticized the covert REM model on the basis that in sleep onset there are no REM periods and it is highly unlikely that REM mechanisms are responsible for any vivid dreaming that might occur in this stage of sleep. These authors state if you want to retain the REM = dreaming model, a more plausible account would be that we are not fully asleep at this stage, and thereby attempt to explain any mentation on the basis of waking cognition rather than covert REM. Cicogna and Bosinelli (2001) claim that this model does not account for dream reports obtained in the first stage of slow wave sleep preceding REM as intrusion of elements of REM physiology is least likely at this point. Feinberg (2000) points out that stage 4 sleep is the 'polar opposite' of REM in terms of physiology and yet Stage 4 sleep has produced vivid dreams; a finding he argues refutes the covert REM hypothesis. Nielsen nevertheless demonstrates that sleep architecture is far more fluid that the models would have us believe and that transitions between stages are not characterized by abrupt changes in the physiological measures that characterize them, but that they blend into each other. Where sleep is disrupted, particularly when REM sleep is suppressed, the boundaries may become even more blurred between REM and NREM.

1.1.4.3. Covert REM a return to REM=dreaming?

Hobson has supported Nielsen's theory of covert REM since it once again reinforces the REM=dreaming model. Responding to the observation that sleep stages are less neatly defined, he was to state "All sleep is REM sleep – more or less" (2000b, p.252). By contrast, critics of Nielsen's theory have effectively pointed out that Nielsen is taking a tautologous route in simply redefining what constitutes REM in order for it to explain the presence of dreaming during NREM sleep. Nielsen himself defines covert REM as 'any episode of NREM sleep for which some REM processes are present but for which REM sleep cannot be scored with standard criteria' (Nielsen, 2000a, p. 861). This is merely a redefinition of sleep stages to suit the theory. Solms (2000c) points out that thus far all we can say is that some of the features which together collectively define REM, also sometimes occur in NREM, but that a finding of transient EEG de-synchronisation during NREM does not indicate a covert interval of REM – there are other states

in which this physiological activity takes place. Experimental evidence for Nielsen's theory is still lacking (Gottesman, 2000), however this new formulation has helped force researchers to clarify their talk regarding aspects of sleep, dreaming and REM and generated new hypotheses which can be tested in support or otherwise of his theory

1.1.5. Further theorizing on the relationship between REM and dreaming

1.1.5.1. Time of night as a determinant of dream quality

A number of authors have noted that dreams tend to become more vivid and lengthy as the night progresses, so that dreams in stage 4 late in the night have higher word counts and vivid imagery than those in the corresponding sleep stage earlier in the night. Feinberg (2000) showed that REM in later stages of the night produced longer word counts than earlier REM stages whereas length of time in a particular REM stage had no effect on dream word count. Domhoff (2000) showed that NREM reports after the third REM period of the night were more similar to REM reports than to earlier NREM reports on a range of Hall and Van de Castle's content indicators. This finding was noted by Herman et al. (1978) to be a possible confounding factor obscuring or exaggerating differences in REM vs. NREM dreams, since researchers often lump together the REM and the NREM periods, for example comparing a dream from Stage 4 NREM with an early evening REM period. Rather than viewing this as a factor to be controlled for, time of night is now taking a more central role as a determinant of the quality of sleep mentation. Fosse, Stickgold and Hobson (2004) have recently published their findings in this regard, which support an early vs. late stage of night dichotomy as highly significant in determining the quality of sleep mentation. In this study, a distinction is made between 'directed thoughts' and 'hallucinations' in order to categorise different types of dream mentation. Their findings were that while in the initial stages of sleep, directed thoughts make up the majority of NREM mentation and hallucinations are the preserve of REM sleep, but as the night progressed, the presence of hallucinations in NREM sleep increased while directed thoughts decreased, and in REM the presence of directed thoughts remained low, while the presence of hallucinations increased. Hallucinations remained more common overall in REM as expected. Importantly, it was not possible to statistically distinguish the mentation of early night REM with late night NREM sleep mentation in terms of the amount of either directed thinking or of hallucinations. The fact that early vs. late stage of night was a better predictor of the quality of sleep mentation than REM vs. NREM clearly has important ramifications for dream generation theory.

1.1.5.2. The role of cerebral activation

Pagel (2000) has stated that the dream research literature has shown that there are better correlations between dreaming and 'cerebral activation' than with dreaming and REM. As early as 1970 Zimmerman reported that different auditory awakening thresholds (which he used to define light vs. deep sleepers) were predictors of NREM dreaming. Following NREM laboratory awakenings, light sleepers reported dreaming 71% of the time, vs. the deep sleepers who reported dreaming after only 21% of NREM awakenings. Zimmerman also found higher awakening thresholds in stages 3 and 4 than in stage 2 and REM sleep, and also higher awakening thresholds earlier in the night than later in the night. These findings converge to indicate that it is the depth of sleep as measured by awakening threshold that is predictive of dreaming. Also of significance was that the light-sleepers' NREM mentation was described as more dreamlike than that of the deep sleepers in his study, and in addition, within the light sleeper group there was no significant difference in the 'dreamlike' quality of NREM mentation as compared with REM mentation, which is contrasted with differences of between $p=0.2$ and $p=0.002$ that were recorded for deep sleepers. The significance of these findings was such that Zimmerman concluded that it was probable that heightened cerebral arousal, and *not* sleep stage is the physiological substrate of dreaming. Yet the REM = dreaming paradigm persisted into the 21st century in the face of such a highly competitive model. The cerebral activation model of dreaming is consistent with Nielsen's observation that there are 'features' of REM that occur in NREM. These features are indicative of this activation, which happens to be more characteristic of REM than of NREM. An activation component has always been part of Hobson's theoretical model, though it does not appear to have occupied as central a position as the REM/NREM dichotomy. Both general cerebral activation and time of night have now been shown to be better predictors of the quality of dream mentation.

Kahan (2000) argues that if differences in REM vs NREM dreaming boil down to a question of quantitative differences, then a one-generator model would suffice. However if qualitative differences do exist, she argues we do need a two-generator model, one for REM and another for NREM. Notwithstanding the fact that Kahan (2000) believes these differences to be quantitative in nature, it does not follow that qualitative differences necessarily imply different mechanisms. There are clearly competing theoretical approaches to a two-generator model for the presence of qualitative differences, such as the comparative differences in acetylcholine-driven memory

ability producing differences in dream reporting ability rather than dream, the role of cerebral activation, time of night, and the multitransmitter theories of Gottesmann (2001) and Perry and Piggot (2000), which would place these qualitative differences on a continuum corresponding with as-yet- unknown levels of the different neurotransmitters involved in sleep and dreaming.

1.1.6. Serotonergics – further evidence against the REM=dreaming paradigm?

The fact that there are still attempts to redefine REM sleep in an effort to reconcile the existence of NREM dreams with the predominant theory shows that there is still controversy regarding the extent to which REM sleep and dreaming are related, what constitutes REM, as well as what constitutes dreaming. Given the substantial co-occurrence of narrative type dreaming and REM sleep it would be unexpected to encounter a substance that suppresses REM while *increasing* REM-like dreaming; such a finding would clearly support claims that REM sleep and dreaming are less interdependent than is strill supposed by many. Assuming such qualitative differences between REM and NREM dreams do exist, one would be unlikely to characterize the vivid and intense dreams anecdotally described by SSRI users as typical NREM dreams. Pace-Schott et al. (2001) were therefore correct in assessing this finding as unexpected, leading them to postulate the possibility of REM rebound as the result of disturbed sleep architecture in SSRI patients characterized by suppression of REM sleep. Hobson et al (2000b, p. 1029) described the finding of increases in dream intensity with SSRI use as "consistent with the notion of cholinergic rebound". There is currently no empirical evidence for this. Also of importance, Perry & Perry (1995) have shown that *defective* cholinergic transmission rather than an increase in cholinergic activity has for some time been associated with visual hallucinations, both in the attenuated cholinergic transmission of Lewy body disease, as well as in a range of recreational and prescription substances with anticholinergic properties, most notably those containing scopolamine and atropine. The following section will explore the evidence for reports of increased dream frequency with antidepressant usage.

1.2. Neurotransmitters involved in sleep and dreaming

The neurotransmitters most studied with respect to sleep and dreaming are serotonin, acetylcholine, noradrenaline, and dopamine. According to Hobson et al (2000a) , in REM sleep forebrain acetylcholine is increased, and serotonin and noradrenaline decreased; in NREM the opposite occurs. Serotonergic activity originating in the raphe nuclei projects to the pons, where it

inhibits cholinergic activity, an effect that is believed to block or delay REM sleep which is cholinergically driven. Reciprocally, changes in acetylcholine immediately lead to changes in the levels of dopamine, serotonin and noradrenalin (Thase, 1998). In sleep, aminergic weakness necessarily implies cholinergic strength. The mutual exclusivity of high acetylcholine with (relatively) high serotonin and noradrenalin only applies to the sleep state however – in waking it is possible to have high release of all 3 neurotransmitters simultaneously (Gottesman, 2000). Of all the amines, it is thought that only dopaminergic neurons continue to fire at waking levels during REM, though as mentioned, new evidence points to a more differentiated role for forebrain dopamine across the various sleep wake states (Lena et al, 2004). Hobson et al. (2000a) note that more recent research also points to the possible involvement of neuropeptides such as glycine and adenosine, as well as suggesting a role for glutamate, GABA, and other substances in modulating REM sleep.

Since the REM-NREM cycle is thought to be characterized by changes in the neurotransmitters serotonin, noradrenaline and acetylcholine, drugs that cause alterations in levels of these neurotransmitters would be expected to cause alterations in sleep physiology whether directly or indirectly, and, depending on one's theoretical viewpoint, on dreaming as well. It will therefore be instructive to examine the available literature on drugs that cause changes in any of these neurotransmitters, and which are known to alter REM-NREM sleep physiology, dreaming or both.

1.3. Antidepressants and their effects on sleep and dreaming

1.3.1. The SSRI's – first line treatment in depression

The Selective Serotonin Reuptake Inhibitors (SSRI's) are the drug therapy of choice for the treatment of depression, replacing the older tricyclics and monoamine oxidase inhibitors (MOAI's) due to a far lower incidence of unwanted side effects (De Vane, 1992). The SSRI's are also simpler to administer relative to the older drugs and boast an improved safety profile; the MOAI's have fallen into some disfavour due to the risk of hypertensive crises leading to possible brain haemorrhage, a risk which necessitated onerous dietary restrictions. The SSRI's are less toxic in overdose than both older classes of drug and are safer in use with renal-impaired patients (DeVane, 1992; Rosenbaum, Fava, Hoog, Ascroft & Krebs, 1998; Richelson, 1994). The

improved side effect tolerability has also meant that fewer patients are inclined to drop out of treatment prior to experiencing the therapeutic benefits of the drugs (Boyer & Feighner, 1998).

1.3.2. Other uses of SSRI's

Though initially designed as antidepressants, it was soon noted that SSRI's were also effective in treating anxiety symptoms in depressed patients, a finding that led to the hope that these drugs might also prove useful in the treatment of anxiety disorders such as panic disorder and social phobia (Boyer & Feighner, 1998). The SSRI's have been argued to be more effective at treating certain anxiety disorders such as panic disorder than were the older drugs and are now commonly prescribed as a first line of treatment for a range of anxiety disorders, including PTSD, OCD, panic, social phobia, and generalized anxiety disorder (Ballenger, 1998; Kaplan and Saddock, 2003). In addition, the SSRI's are often prescribed for eating disorders, though according to Kaplan and Saddock (2003, p.745), no medication has so far provided 'definitive improvement of the core symptoms of anorexia nervosa'.

1.3.3. Mechanisms of serotonergic antidepressants

The discovery of the link between increased serotonin and relief of depression was serendipitous, like many other psychopharmalogical discoveries, and for this reason, less is known about the mechanics of the alleviation of depression and anxiety symptoms than might be expected (Gorman, 2000). For example, it is still not understood why many patients do not experience alleviation of symptoms until around four weeks after commencement of antidepressant regimes, usually some days or even weeks after steady state plasma levels of brain serotonin have been reached (Snyder, 1986; Hobson, 2001). Gaillard, Nicholson & Pascoe (1994) suggest that the therapeutic benefit of antidepressants may even be due to alterations in mechanisms other than serotonergic ones as suggested by an observed increased in the number of GABA receptors with antidepressant use. All three major classes of antidepressants (monoamine oxidase inhibitors, tricyclics and SSRI's) are designed to increase serotonin levels, though the mechanisms vary. The MOAI's increase serotonin levels through inhibiting the creation of the enzyme that degrades excess synaptic serotonin. The tricyclics act through inhibition of the reuptake of noradrenalin and serotonin by the presynaptic neuron. SSRI's act by selectively limiting only the reuptake of serotonin by the presynaptic neuron, thus leaving more of the neurotransmitter available at the synapse. However, alterations in one neurotransmitter system have effects on levels of other brain

1.3.4. Effects of antidepressants on sleep and dreaming

Roth, Kramer and Salis (1979) performed a review of the available literature on sleep and dreaming at a time when only the MOAI's and tricyclics were available for the treatment of anxiety and depression. Virtually all the antidepressants developed to date have some effect on sleep physiology, either reducing the amount of REM sleep or the time taken to reach the first REM period, and in fewer cases, altering other sleep stages. (REM latency refers to the time taken to the first REM period, and REM density refers to the number of eye movements per unit time). These effects may be relatively short lived or persist for the duration of the antidepressant therapy. In some instances REM rebound occurs on discontinuation, as a result of prolonged deprivation of REM sleep. Of the research considered 'minimally adequate' methodologically in the Roth et al. (1979) review, none were available for review on the monoamine oxidase inhibitors (MOAI's) with regard to either sleep or dreaming, although Nicholson et al (1994) report reduced REM sleep with monoamine therapy, a phenomenon which Jacobs (1977) also observed does *not* correct itself with prolonged administration and in which no REM rebound was observed. A tricyclic antidepressant, amitriptyline, was shown in four studies to cause an initial increased sleep time and increased stage 3 sleep. REM sleep was found to be significantly reduced. On discontinuation of the drug, a REM-rebound phenomenon occurred, in which subsequent night's sleep showed increased time in REM sleep. Two studies looked at dreams and imipramine (another tricyclic drug), one in normal subjects, which found reduction in 'REM-Recall dreams' (note that subjects dream recall was only tested in REM, since REM and dreaming were still considered to be uncontroversially isomorphic at that time). Another study using seven depressed patients showed no difference in frequency of dreaming but an increase in the hostility and anxiety content of dreams after a week on imipramine. In their review Kramer et al. (1979) deplored the lack of systematic work on the study of drug effects on dreaming, stating that not even one drug had been examined in any depth. Despite anecdotal evidence to the effect that the SSRI's sometimes cause dramatic changes in dreaming characteristics, today the situation with regard to available research on psychiatric drugs and sleep physiology and dreaming is still much the same.

1.3.4.1. The impact of SSRI's on sleep and dreaming.

As would be expected given their effect on serotonin levels, the SSRI's strongly suppress REM sleep, although REM is not completely eliminated. Administration of the SSRI's would also be expected to reduce reports of dreaming given the strong co-occurrence of REM sleep and dreaming. However clinical anecdotes as well as a small number of published studies have consistently shown increases in certain dreaming characteristics in patients taking SSRI's, particularly with regard to frequency, vividness and intensity, with SSRI users often reporting that it feels like they dream all night long (Pace-Schott et al, 2001, Hobson, 2001). Research evidence has been mostly restricted to case studies, with limited supporting evidence from clinical trials, which incidentally list abnormal or increased dreaming amongst various other side effects. There is one experimental study in the literature with the specific aim of investigating the relationship between the SSRI's and altered dreaming. This study, although small, found significant increases in dream intensity. The following section will take a look at the case report and clinical trial data before examining the experimental study in more detail.

1.3.4.2. The case report data

Markowitz (1991) discusses 4 case reports in which vivid and intense dreaming was reported with fluoxetine treatment in dysthymic patients. In all four cases the sudden increase in vivid dreaming preceded therapeutic benefit. One patient recorded 'technicolor dreams', where her pre-medication dreams were unremarkable. All reported more vivid and realistic-feeling dreams, more memorable and intense though with unchanged form or content. In his own practice, Markowitz asked a further 18 patients who had switched from tricyclics to fluoxetine whether they recalled more dreams. Only two reported increased recall under fluoxetine (11 percent of the sample). It is not clear whether any of the four more detailed cases had previously been on other antidepressant medication. A study by Armitage, Rochlen, Fitch, Trivedi, and Rush (1995) into dream reports in depressed patients included patients that were taking either fluoxetine or nefazodone. Since these antidepressants have quite different effects on sleep architecture (nefazodone does not suppress REM whereas fluoxetine does [Thase, 1998]) the sample as a whole cannot be viewed as a homogenous group for our purposes. Nevertheless Armitage noted that whereas the sample as a whole showed a decrease in dream recall, those on fluoxetine showed an increase in dream recall together with a suppression of REM sleep. All the patients on

nefazodone showed a decrease in dream recall. Lepkifer, Damon, Iancu, Ziv and Kotler (1995) report four cases in which the administration of fluoxetine in patients coincided with frequent nightmares, which stopped with cessation of treatment. None of the patients had reported nightmares prior to treatment. Three of the patients were diagnosed with a major depressive episode, and one patient was diagnosed with OCD. The patients with major depressive episode were placed on 20mg fluoxetine/day and the patient with OCD on 40mg/day. In addition to the nightmares, the patient with OCD experienced hypnopompic phenomena (confusion of dreaming with reality on emerging from sleep) under fluoxetine, which Hobson (2001) argues is a not-infrequent consequence of long-term use of fluoxetine. Hobson ascribes this to frequent awakenings, suggesting that the SSRI's actually break down the boundaries between REM and NREM sleep. Lepkifer et al. (1995) propose that fluoxetine may cause an imbalance between dopamine and serotonin levels leading to a possible hypersensitivity of dopaminergic receptors, which in turn leads to heightened dopaminergic activity and hence more vivid dreams. The latter theory ties in with Gottesman's (2001) monoaminergic disinhibition hypothesis and Perry and Piggott's (2000) multitransmitter theory discussed above.

1.3.4.3. Clinical trials – impact of SSRI's on dreaming and sleep architecture

In a Scandinavian clinical trial, citalopram was administered in order to test its effectiveness in treating Obsessive Compulsive Disorder (OCD). The drug was administered over a 24-week period, and as an incidental side effect, six percent of the 29 participants reported increased dreaming (Koponen, Lepola, Leinonen, Penttinen & Turtonen, 1997). Despite the fact that such side effects are only reported in a minority of patients, this symptom, together with decreased sleep were listed among the 'most commonly experienced adverse events' (p343) along with nausea and vomiting, and notably, more frequently reported than diminished sexual interest and performance, a well-documented side effect of SSRI use (Kaplan & Saddock, 2003). It is not clear whether patients were specifically asked about increased dreaming or whether this was spontaneously reported. This is important, since many patients would not attach significance to changes in dreaming as being related to the medication and might fail to report such changes. One presumes 'increased dreaming' indicates an increase in dream frequency rather than length or intensity, but cannot be certain that this is the case. The authors note that the increase in dreaming persisted throughout the 24-week study period. A study by Ekselius, von Knorring and Eberhard (1997) comparing the side effect profiles of Citalopram with Sertraline noted 'increased dream

activity' in 4.5 % of patients on sertraline and 5.5% of patients treated with citalopram, both statistically significant, though sample sizes were large. 400 patients with major depression were randomly assigned to the two groups, of whom 308 completed the 24–week study. Again it is not clear whether reports of increased dream activity was spontaneously volunteered or elicited through specific questioning. That increased dreaming is a well accepted phenomenon with SSRI use is evidenced by its inclusion in the latest edition of Kaplan and Saddock (2003), a psychiatric reference text, who note that 'a minority of persons taking SSRI's report recalling extremely vivid dreams or nightmares' (p.1099). In addition Markowitz (1991) noted that the package insert for fluoxetine lists 'abnormal' dreams under the heading 'frequent adverse effects'. It would seem that abnormal or increased dreaming is a fairly well documented side-effect of treatment with SSRI's in a minority of patients, though the clinical trials that have led to this conclusion have not been conducted with the purpose of exploring this relationship but rather to document the side effect profile of the various drugs. For this reason it is not entirely clear which qualitative aspects of the dreams of these patients changed.

In terms of the impact of SSRI's on sleep architecture, Saletu, Frey and Krupka (1991) found in an experiment with 18 volunteers that although REM was significantly decreased under fluoxetine and paroxetine, subjectively sleep quality was reported as unchanged. Lepkifer et al (1995) also argue that although fluoxetine increases REM latency, it does not alter REM density or the total number of hours slept. By contrast Koponen et al (1997) did find a decrease in overall sleep time in their study with citalopram.

1.3.4.4. Experimental data - The Pace-Schott study

A recent study by Pace-Schott, Gersh, Silvestri, Stickgold, Salzman and Hobson (2001) provides the only experimental evidence to date with the express purpose of exploring the phenomenon of SSRI-related dream alteration. Their study comprised 14 normal, paid volunteers and involved 31 days of continuous assessment. The first 7 days were treated as drug-free baseline; during the next 19 the subjects were administered either paroxetine (7 subjects) or fluvoxamine (7 subjects). Both drugs have similar half-lives (the half life of a drug is the time it takes for the blood level of a drug to fall to 50% of its peak). The final 5 days of the study entailed a withdrawal period. REM sleep measures were taken using polysomnography, and subjects were interviewed regarding various aspects of their dream experience, such as intensity and memorability, content variables

such as how many known characters vs. strangers were present, and the bizarreness rating of the dream. The research team found decreased self report measures of dream frequency but increased word count and emotional intensity between baseline and steady state, which is based on approximately 5 half-lives of the drug (De Vane, 1999). Increases in visual vividness with treatment were described as a trend, though not statistically significant; power calculations showed an effect size of 0.65. For the most part their findings are supportive of the anecdotal and case study evidence with regard to qualitative features of the dream experience such as intensity and visual vividness, though clinical reports of increased dream frequency were not upheld by the data.

As this was intended as a pilot study, there are some limits with regard to the scope of conclusions that can be drawn, in that the sample was small, and the subjects were only on the drugs for a total of 19 days, a timeframe that would not be considered therapeutic (although one that makes sense in the context of the time limits of the study, which involved daily monitoring of volunteers taking psychiatric medication). As it turns out, most case reports indicate increased dreaming prior to therapeutic result, and in fact some even suggest that such side effects may provide indication of the likelihood of therapeutic success with the medication (Markowitz, 1991, Nicholson, Bradley, Pascoe, 1994), nevertheless for completeness one would ideally like to delineate all the phases as they are experienced by patients. (In recent correspondence with Pace-Schott, he has stated that his current research is focused on the longer term effects of SSRI's on dreaming, apparently with quite marked effects on dream intensity (private correspondence, 2003)).

Hobson (2001), possibly commenting on the same data, also noted an increase in, rather than a leveling off of abnormal dream content and abnormal sleep architecture with prolonged use of fluoxetine. Hobson also argues that SSRI users spend more time in light sleep near waking levels, and suggests that they dream more and are aware of their dreams more. Hobson's explanation for this is that they awaken more often (Hobson, 2001). Both Hobson and Pace-Schott propose the increased intensity and vividness of dreams noted in this and in other research might be due to a cholinergic driven REM-rebound phenomenon precipitated by prolonged REM suppression. This has not been shown to occur with either the SSRI's or with the older classes of drug (it has already been mentioned that the MOAI's have a prolonged effect on REM suppression which

does not correct itself during treatment, and also that withdrawal of tricyclic therapy leads to REM rebound, but there is no documented evidence of a rebound phenomenon *during* antidepressant use). However Thase (1998) states that antidepressants redistribute REM later into the night (he unfortunately does not state which antidepressants, nor provide a reference for this particular assertion). It is also of interest that paroxetine, which is used in the Pace-Schott et al (2001) study, is the only SSRI with significant anticholinergic properties. The authors reported greater rated bizarreness of dreaming in the paroxetine group compared with the fluvoxamine group. Since cholinergic activity increases the propensity for entering REM, anticholinergic activity should further suppress REM according to the authors' theory, and consequently *decrease* vivid or intense REM–type dreaming. This finding *is* however consistent with reports of visual hallucinations associated with use of anticholinergic drugs discussed above (Perry & Perry, 1995).

The fact that there was no placebo group in this study makes it difficult to draw conclusions regarding intervention with SSRI's in general (as opposed to intervention per se), a limitation recognized by the authors. It has long been reported that subjects can be trained to remember dreams, and that once attention is focused on the importance of dream recall, subjects tend to report more dreams, as shown in laboratory dream series (Strauch & Meier, 1966) thus while a pre-post design has strong merits, inclusion of a control group would have diminished concerns regarding possible experimenter effect. This study is important however in that it is the only experimental study to date to examine the effects of SSRI's on dreaming. The fact that increases in certain measures of intensity were found in their pilot study thus calls for confirmation through replication.

1.4. Dream Research – problems and pitfalls

1.4.1. Determinants of Dream recall

In a 1968 study by Meier, Ruff and Ziegler, it was reported that around 80 of REM awakenings in the laboratory would yield a dream report in normal subjects, though there are large individual differences. Most dreams obtained during laboratory testing through middle of night awakenings would otherwise have been forgotten in the morning since most people remember less than one per cent of their dreams (Domhoff, 2003). Dreams rated as intense are the most likely of these dreams to be remembered, along with longer dreams, and dreams experienced most recently.

Dreams rated as having higher intensity tended to occur during the earlier periods of sleep, and shorter dreams occurred from the last period of sleep. Meier et al. (1968) concluded from this that 'recency, intensity and amount of material interact to influence the amount of morning recall of dreams recorded during the night.' (p.555.) Cohen (1979) found that along with emotionality and vividness, salience of the dream for the individual also affected recall. Hartmann (2000) argues that it is no less difficult recalling one's daydreams than recalling the average dream. To recall what one was thinking on the way to work this morning may be fairly difficult for example, since it is usually of little consequence. The net result is that dreams spontaneously recalled are unlikely to be representative of dream life on average.

1.4.2. Laboratory Dreams

Much argument has been generated regarding the validity of laboratory dreams, with allegations of experimenter effect, laboratory fatigue, the construction of the dream experience through focus on it, and motivational problems on the part of subjects (Hobson et al., 2000a). While not elaborating this area of debate in too great detail, it should be noted that without laboratory dream series, the amount of data at our disposal would be significantly poorer. For example it is only in the laboratory that frequent awakenings from physiologically verifiable sleep stages can reveal the amount of dreams subjects are able to report in contrast with what is retained after normal morning awakenings. And it is only in the laboratory that one is likely to encounter the more mundane thought-like dreams that might otherwise have been forgotten. Nevertheless laboratory studies, while contributing a more representative sample of dreams, remain expensive and time consuming and thus beyond the scope of some researchers.

1.4.3. High vs. Low dream recallers

Because of large individual differences in the ability to recall dreams, 'low dream recallers' are frequently excluded from dream studies, with the result that participants in dream research tend to be high recallers. Nielsen (2000) found differences in the rates of REM vs. NREM dreams in high recallers vs. low recallers, which may account for some differences in the findings where studies failed to replicate one another's results.

1.4.4. The problem of correspondence

Possibly the most intractable methodological problem in dream research is the fact that the dream report is not the dream itself; LaBerge (2000) points out that the word 'dream' is repeatedly substituted for dream report, by both Hobson and Solms. Moreover the event (the dream) occurs in one mental state while the dream report is obtained in another, (waking) state (Kramer, 2000); and as Hobson et al. (2000a) point out, a unimodal vehicle is used to describe a multimodal experience. Memory is 'a constructive rather than a reproductive enterprise' according to Kramer (2000, p.960) and Feinberg (2000) goes so far as to suggest that dreams might be constructed during the process of awakening as memory systems which have been disabled or disconnected during sleep, switch back on. Domhoff (2003) counters the latter concern by citing examples of sleeptalking subjects in whom the post-awakening report reflects the sleeptalking narrative. Failure to obtain dream reports could be due to a lack of skill in pulling together a narrative and communicating it, which in turn may be due to poor language skills, poor organizational skills or poor education. Thus one cannot assume for example that dream report length gives an indication of dream length, or that reported complexity is faithfully communicated in the report (Schredl, 2000).

1.4.5. Defining the dream

Another important, but theoretically surmountable methodological difficulty facing dream researchers is the fact that there is no standard definition of what constitutes a dream. Aristotle defined dreaming as 'the mental activity of the sleeper insofar as he is asleep' (As quoted in Pagel [2000, p.985]). This broad (and intuitive) definition would certainly be viewed as too inclusive by those wishing to postulate a separate mechanism for mentation occurring outside of REM sleep, hence Hobson and Nielsen's more exclusionary criteria. Nielsen (2000a) provides a definition of dreaming (which most authors don't) but his definition might be considered restrictive, including only those dreams that other researchers consider 'apex' dreaming – those that exhibit elements of bizarreness, delusional thinking and hyperemotionality. The benefits of providing a definition are therefore outweighed by the fact that the definition almost always serves to provide support for the theory, and the criteria used to define what does or does not qualify as a dream inevitably affect the results (Kramer, 2000; Kahan, 2000), a point which Nielsen (2000a) himself concedes.

In early laboratory series it became clear to participants what researchers deemed worthy of 'dream' status and what were considered thoughts during sleep, thus influencing what they chose to report on awakening (Herman et al, 1978). Some studies had binary-type ratings for dreams as either dreams or not-dreams and others had sliding scales according to which reports were graded as more or less dreamlike (Herman et al, 1978). Since the debate regarding REM vs. NREM dreams has from the start been focused on qualitative differences between sleep mentation obtained from these two states, it is crucial that researchers make clear from the outset their criteria for inclusion and exclusion. Awareness of this problem surfaced early in the NREM dreaming debate as it resulted in a string of studies in the 1970's being apparently unable to replicate one another's findings (Herman, Ellman & Roffwarg, 1978). Early researchers tended to rely on a common intuitive understanding of what we mean by dreaming, however as soon as researchers stated their criteria it became clear that this was not the case. Researchers in the field have thus tended to evaluate their results against an ever-changing definition of what constitutes a dream. Nielsen (2000b) argues for a theory-free definition of dreaming, or at least a strategy to address the disagreement in this area in order to overcome confusion.

1.4.6. Quality of the research evidence

Schredl, Funkhouser, Cornu, Hirsbrunner & Bahro (2001b) argue that much of the extant dream research is methodologically unsound with questionnaires poorly assessed for reliability prior to administration. In the Schredl et al. (2001b) study, word count was found to be stable over time but most dream content variables were not, and therefore were not considered stable characteristics. Antrobus (1995) points out that findings regarding dreaming and sleep stages in animals have been (erroneously) generalized to humans. Much of the early 'dream' research was conducted on animals, from whom no dream reports were elicited; the assumption was that REM sleep and dreaming are synonymous, therefore conclusions regarding REM sleep were extrapolated to conclusions regarding dreaming

If researchers are to investigate dreaming it is not possible to reduce the enterprise to a search for physiological variables, since there is no agreement on the physiological correlates of the dreaming process. The phenomenological data frequently point to logical areas of investigation, and should therefore not be disregarded. For example, the fact that dreams over a night seem from the dream reports obtained, to be driven by the same emotional mood, suggest a role for the

limbic system in generating content rather than a random access of one's memory banks (Kramer, 2000). We need the subjective self-reports of dream experience to generate the theories that may then be subjected to empirical support through PET scanning, lesion studies or whatever new technology we may apply to the problem.

1.5. The importance of continuing with dream research

Investigations into both the intended and unintended effects of certain medications frequently provide information regarding brain functions. Prior to the introduction of the benzodiazepines for the treatment of anxiety for example, it was assumed that the inevitable result of tranquilizing drugs such as the barbiturates, was sedation. With the advent of the benzodiazepines it was discovered that anxiolytics need not necessarily cause sedation, and the previously assumed connection between brain mechanisms sustaining alertness and those causing anxiety was questioned (Snyder, 1986). Therefore if drugs such as the SSRI's have the effect of causing abnormally vivid and intense dreaming contrary to expectations that are consistent with current sleep and dream architecture theory, such a finding might add another piece to the puzzle of sleep and dream theory. A link between increased frequency or intensity of dreaming and SSRI therapy posits another argument against the simple association of dreaming and REM and requires explanation.

In an article entitled 'Neurological dreaming', Oliver Sacks (1991) talks about dreaming as a barometer of neurological health and disease. Sacks describes how neurological disease may alter dreaming either qualitatively or quantitatively, a fact which he claims is often overlooked by clinicians and one that could be used in diagnosis. In this article he describes one account of vivid dreaming as migraine aura, and noted in another case that visual imagery vanished from the dreams of an Alzheimer's patient. In his studies with Parkinson's patients he found that the first signs of successful response with L-Dopa are vivid dreams, and in one patient, the first signs of impending movement difficulty occurred in his dreams. Solms (1997, p.1) points out that 'many neurological patients complained of specific changes in their dreams, the onset of which they dated to the beginning of their illness', for example, with hydrocephalus, a condition in which cessation of dreaming is typical, patient reports of cessation or return of dreaming may be helpful in monitoring the progress of the disease, since successful treatment with shunting may often result in a return of dreaming in this patient group. The brain mechanisms of dreaming (as

opposed to REM sleep) are an under-researched function, and one which according to Solms (2003, personal communication), has great theoretical relevance for our understanding of the brain mechanisms underlying the positive symptoms of some psychoses. A clearer understanding of the mechanisms and neurotransmitters involved in dreaming may thus prove to be invaluable, and perhaps a deeper understanding of the mechanisms of action of medications such as the SSRI's may pave the way for the creation of more effective and safer psychiatric medications.

2. Method

2.1. Sample

This study is cross-sectional in design, comparing specific dream characteristics from two groups of subjects, one group receiving therapy with SSRI's and a comparable group receiving no pharmacotherapy. Participants were recruited at one of two centers, the first being the outpatient psychiatric department of the state teaching hospital facility associated with the university of Cape Town, (Groote Schuur) and the other being the anxiety disorders unit affiliated with Stellenbosch University. Both universities ethical review boards gave ethical approval. All participants gave written informed consent. Interviews took place between June 2003 and May 2004. It was decided to use psychiatric patients rather than normal volunteers due to both financial and ethical constraints associated with clinical trial research. In addition this choice of sample afforded the opportunity to study patients whose duration of SSRI therapy already extended into what would be considered a therapeutic timeframe, thus providing the possibility of complementing the Pace-Schott et al (2001) findings, in which 19 days of SSRI therapy was administered. A disadvantage of this population is that benzodiazepines are frequently concurrently prescribed; possible implications of this will be discussed in more detail below.

Participants spoke either English or Afrikaans. Although there are 11 official languages in South Africa, the interview protocol was restricted to these two languages for the sake of practicality. Afrikaans is the language most spoken in the Western Cape, so this did not prove restrictive. Since both facilities are state funded to a varying degree, the low to middle socioeconomic groups are over represented, with tertiary education the exception rather than the rule. According to Kramer and Roth (1979) socioeconomic class has a small effect on dream content.

Responses from a total of 54 participants were obtained, and of these it was necessary to exclude fourteen responses. These fourteen exclusions resulted in a total of 40 participants, 20 in each group. Eight of the exclusions were due to discontinuation of SSRI therapy, in most cases for placement on alternative treatment regimes. A further six were excluded since it became apparent that their Axis I diagnosis could result in a potential study confound due to their documented association with dream abnormalities, as will be discussed below.

2.1.1. The relationship between psychiatric disorders and dream abnormalities

With patients in whom the diagnostic criteria for their psychiatric disorder include abnormal dreaming it would be reasonable to expect spontaneous alteration in dreaming in keeping with other treatment benefits where SSRI treatment is successful. Changes in dream characteristics have indeed been documented in association with successful treatment for certain psychiatric conditions (Kirschner, 1999; Ross, 1989). This could create a potential confound for this study. Therefore, this next section takes a look at the range of disorders for which SSRI's are commonly prescribed, with a view to identifying those for which there are no reported abnormalities in sleep or dreaming characteristics for inclusion in this study.

2.1.1.1. The relationship between depression and dream characteristics

Sleep anomalies have been reported in a majority of people with depression. Some researchers place this incidence at more than 90% of depressed patients (Lepkifer, Damon, Iancu, Ziv & Kotler, 1995). Insomnia, hypersomnia, shorter REM latency, early morning awakenings, lack of dreaming and nightmares are some of the conditions reported and included as DSMIV diagnostic criteria for depression (Thase, 1998; Nicholson, Bradley & Pascoe, 1994). A study by Armitage, Rochlen, Fitch, Trivedi, and Rush (1995) found significantly lower dream reports amongst depressed patients than is the norm. For admission to the study, patients had to score 17 or more on the Hamilton depression scale. Only 21 of 82 patients recalled any dreams at all during the study. Those that did dream gave dream reports, which she described as 'bland, short and devoid of any strong emotional content' (Armitage et al, 1995, p.190). In addition, cognitive abilities including memory and concentration may be impaired during a depressive episode (Kaplan & Saddock, 2003). Both the incidence of dreaming and the memory of these dreams are thus likely to be suppressed during a depressive episode. The introduction of depression-relieving treatment, whether therapy or medication, could therefore be expected to lead to a 'spontaneous' improvement in reported dreaming. Kirschner (1999) documented changes in dream content in a depressed patient taking Sertraline, in which he describes her post-medication dreams as closer to norms in terms of aggressive and friendly themes, though he does not explore alterations in dream frequency. Due to the well-established relationship between depression and suppressed dreaming, patients with an Axis I diagnosis of depression were excluded from the study as a potential confound, since successful treatment might be expected to result in increased dream frequency.

2.1.1.2. Relation between anxiety disorders and dream characteristics

In 1979 Kramer and Roth performed a review of the literature regarding dreaming in relation to psychopathology, and found a preponderance of case reports, which were mostly descriptive and without reference to norms. Their review covered dreams in schizophrenic, depressed, alcoholic, mentally retarded and brain damaged patients. Most of the studies that *were* experimental did not control for the presence of medication, and the basis on which diagnosis was made was in many cases not stated. Despite a comprehensive review, they reported no data on the relationship between dreaming and anxiety or eating disorders, with the studies reviewed having focused on psychosis and depression. There does appear to be one study that did look at anxiety and dreaming that is discussed below (Desroches & Kaiman, 1966), this was possibly excluded for methodological reasons. Many of the older studies that have attempted to link dreaming characteristics with psychiatric disorders failed to adequately operationalise either the independent or dependent variables. Ross et al (1989) point out that it is often not stated whether patients are or are not taking psychotropic medication, or whether they suffer from additional co-morbid psychiatric or other medical complaints. It is often quoted that anxiety is related to an increase in frightening dreams, but a search of the literature revealed no recent research on the topic, aside from PTSD. Instead this assertion is made with reference to older studies such as the Desroches and Kaiman (1962) correlational study in which no DSM (or other accepted) criteria for anxiety are used. In this study an intercorrelation matrix is used to show links between certain indicators of anxiety, such as self-report on how often the subject got up in the night to urinate or smoke, or how often in the last week the subject felt jumpy, and how many times during the last week the subject experienced stomach cramps. In this study 30 different intercorrelations were done; of these only 4 were significant; 3 'different' variables correlated positively with the variable 'recall of dreams'; these were 'number of times up last night', 'number of times up at night to urinate' and 'number of times up last night to smoke'. No attempted link was made between frightening dream and traumatic experience, as we do now with PTSD despite the fact that it appears that half the subjects in this study were resident at the war veterans administration center in Tennessee. The disorders encompassed under the rubric of anxiety disorders include very heterogenous syndromes, particularly from the point of view of their relationship with sleep and dreaming disorders. As it is hoped to show, of the disorders classified as anxiety disorders, only PTSD is definitively linked in the literature with changes in dream intensity or frequency of dream recall.

2.1.1.3. OCD and sleep/dreaming changes

REM latency was shown in some studies to be reduced in OCD but other studies showed normal REM latency (Benca, Obermeyer, Thisted & Gillin, 1992). According to a more recent survey by Robinson, Walsleben, Pollack & Lerner (1998) the small database of existing research show neither significant sleep nor dreaming anomalies in OCD. In their own study Robinson et al. (1998) found no significant differences in sleep EEG polysomnography between OCD and normal volunteers. The relationship between OCD and abnormal dreaming was investigated by Sauteraud, Menny, Philip, Peyre and Bonnin (2001), who found no differences between the dreams of OCD and normal patients. 47 dreams were collected from the OCD and 55 from the control group. In a blind rating, two judges found no higher incidence in the dreams of OCD patients of anxiety, failure, sadness or obsessive-compulsive themes than there were in the control patients. These authors also comment on the problem of diagnostic imprecision in previous studies and chose to examine OCD patients since they felt the diagnostic criteria to be less variable. Depression was excluded as a co-morbid diagnosis through use of the Hamilton depression scale, and patients had to engage in ritual behaviour for at least 2 hours a day in order to qualify for inclusion. The authors were struck by the difference in waking mentation and dream mentation in this patient population, with no evidence of either integration of daily events or amongst the OCD patients of any ritual themes into their dreams. Negative themes predominated in both groups but were in fact slightly but insignificantly higher amongst the control group. There were no statistics provided on whether the incidence or frequency of dreaming differed amongst the two groups, but given that there were 10 OCD patients and 11 controls it does not appear that there is a significant difference in dream recall rates (identical dream report rates would therefore have been 50 OCD dreams for 55 control dreams - the actual figures are 47 OCD dreams for 55 control dreams).

2.1.1.4. Panic Disorder and dreaming

Mellman and Uhde (1989) reported a decrease in the number of REM periods in patients with panic disorder compared with controls but no difference in the percentage of time spent overall in any stage of sleep compared with controls and no other abnormalities associated with the REM stages as assessed using EEG. A study by Free, Winget and Whitman (1993) was designed to look at separation anxiety in panic disorder by various means including dreaming. They found

more themes of both 'covert hostility' and separation anxiety in the dreams of 20 DSM-III diagnosed panic disorder patients as compared to 20 psychiatric controls. Unfortunately no measure of dream frequency was obtained. The choice of psychiatric patients rather than normal controls also implies that there are no normative comparisons that can be made from their study regarding dream content. Schredl, Kronenberg, Nonell and Heuser (2001a) investigated the relationship between panic disorder as defined by DSMIV criteria, dream recall and nightmare frequency, and found a relationship between frequency of nocturnal panic attacks and nightmare frequency, but no relationship between the severity of the panic disorder and dream recall or nightmare frequency.

2.1.1.5. PTSD and dreaming.

The topic of PTSD and changes in dream mentation has been widely documented, with recurrent, distressing dreams of the (traumatic) event one of the diagnostic criteria for the disorder (Kaplan & Saddock, 2003). Ross, Ball, Sullivan and Caroff (1989) report that amongst the normal population, nightmares are reported in around 20 to 25 percent of people, at a rate of less than one per year. By contrast 68% of Vietnam veterans reported having nightmares at least once a month. Germain and Nielsen (2003) report a rate of 75% of reported presence of nightmares in PTSD patients. Dreams are described as vivid, affect laden, and disturbing. REM latency was found to be increased and length of REM periods shorter. Ross et al (1989) question whether these episodes of dream mentation should be considered real nightmares or represent a type of parasomnia that could be defined as the re-experiencing of the traumatic incident in sleep. Germain and Nielsen (2003) found differences between PTSD nightmare sufferers and 'ordinary' nightmare sufferers, with the PTSD nightmares occurring earlier in the night as well as an association with more gross body movements and more frequent nocturnal awakenings. (Of relevance for the REM-NREM dreaming debate, Domhoff (2003) points out that the recurring nightmares of PTSD patients occur both in REM and NREM sleep). Treatment with antidepressants such as the monoamines and tricyclics has been shown to significantly reduce the number of nightmares along with the other features of PTSD (Ross, 1989). It should be clear from the foregoing that this subgroup would be singularly unsuitable for any investigation into the link between administration of SSRI's and increased dream intensity or vividness.

At the time of writing there were unfortunately no published studies investigating links between Generalized anxiety disorder as defined by DSM (or other standard) criteria and dreaming. In addition, nothing of relevance could be found relating social phobia with dreaming changes.

2.1.1.6. Eating disorders and dreaming

Kaplan and Saddock (2003) did not note any disturbance in sleep or dreaming associated with eating disorders, however the SSRI's are unlikely to be the first line of treatment for this range of conditions, and for this reason patients with eating disorders were not considered to be a practical group for inclusion in this study.

2.1.1.7. Co-Morbidity in psychiatric disorders

According to Kessler (1998) most people with anxiety disorders have other co-morbid psychiatric disorders, with the strongest co-morbidity relation between anxiety and depression, in particular between depression and generalized anxiety disorder, and between depression and panic disorder. Kessler (1998) argues that with the proliferation of diagnostic categories, people who might previously have been diagnosed with one disorder might now find themselves with multiple diagnoses. This poses a potential confound when using psychiatric patient samples.

2.1.1.8. Dreaming and Psychiatric disorders - Conclusion

There appears to be a paucity of research into the dream characteristics of various psychiatric disorders, which is unfortunate since it might be premature to categorically conclude that there are no differences in dream frequency and vividness in obsessive-compulsive disorder, panic disorder, social phobia and generalized anxiety disorder compared with normal controls. At this stage, the only DSM-IV reference made regarding abnormal sleep or dreaming in anxiety disorders is made with reference to PTSD (Kaplan & Saddock, 2003). The fact that the evidence leans in favor of the null hypothesis makes this group a better choice than depressed patients rather than a perfect choice. The presence of abnormal dreams as one of the diagnostic criteria for PTSD makes this an obvious exclusion from the anxiety disorder group. Patients on SSRI's for generalized anxiety disorder, panic disorder, OCD and social phobia were therefore considered suitable candidates for this study, bearing in mind the potentially confounding factor of a high co-morbidity of depression with many anxiety disorders. The Axis I diagnosis has been used, as supplied by the psychiatrists who had seen the patients.

Table 1: Spread of anxiety disorders included

Axis I	SSRI	No meds
Generalised anxiety disorder	7	10
Panic disorder	5	6
OCD	6	3
Social Phobia	2	1

2.1.2. Other sample characteristics

For the group taking SSRI's, the duration of therapy varied from 1 month to 60 months with a mean of 17 months and a median of 9 months. The age range was 14-53, with the mean and median by group listed in Table 2. There was a fairly even distribution of gender across the two groups, though females are over-represented in the sample as shown in Table 3.

Table 2: Age distribution:

	SSRI	No meds
Mean	34	37
Median	31	34

Table 3: Gender distribution:

	SSRI	No meds
Female	14	13
Male	6	7

2.1.3. Presence/length of dream report

Despite the fact that the Hall & Van de Castle standardized content analysis is not designed to be performed on dreams less than a specified word count, usually 50 (Domhoff, 2003), patients were included whether or not they completed a dream report and regardless of length, since exclusion of those who could not provide a dream report might have obscured possible recall differences between groups.

2.1.4. Medication

Of the 5 SSRI's included in this study fluoxetine (12), citalopram (4), paroxetine (4) and sertraline (1) were represented. No patients were prescribed fluvoxamine. Duration of SSRI treatment ranged from one month to five years, with a mean of 16 months. Thase (1998) notes that of all the antidepressants, the SSRI's are noted for negatively impacting on sleep maintenance, and that insomnia is a frequent consequence. This is just one of the reasons that SSRI prescription is frequently accompanied by concurrent prescription of sedatives such as the benzodiazepines, another is that despite their prescription for anxiety, the SSRI's initially may cause anxiety and agitation (Boyer & Feighner, 1998) making study of the effects of SSRI's alone in patient populations a practical impossibility. Argyropoulos, Sandford & Nutt (2000) state that it is common practice to prescribe benzodiazepines in the initial stages of treatment (usually two weeks) with SSRI's due to widespread findings of initial jitteriness and a temporary increase in symptoms of anxiety. Given their addictive potential it is perhaps not surprising that some patients were still taking benzodiazepines long into SSRI treatment, despite an indication that benzodiazepines were generally prescribed only for the first 2 weeks of SSRI treatment.

Benzodiazepines modulate the activity of GABA neurotransmitters, which may in turn have effects on either transmission or synthesis of other neurotransmitters such as noradrenalin (Gaillard, 1994a). In terms of impact on sleep and dreaming, the benzodiazepines are known to decrease sleep latency and increase REM latency, though REM density is not suppressed overall and no REM rebound is observed. NREM is altered in various ways depending on the specific benzodiazepine concerned, with time spent in some stages increased and others decreased, and with alterations in the physiology of sleep stages (waveforms, body movements and other phasic events) as well as the number of shifts between sleep stages (Gaillard, 1994b). These studies showed no significant reduction in number of dreams recalled, though Perry and Young (2001) report that dreaming is diminished with sedative use in general, under which heading they have included the benzodiazepines. One study noted less dream bizarreness with Nitrazepam (Roth, Kramer & Salis, 1979). Given all of the above, it would have been preferable to exclude patients on benzodiazepines, but difficult in terms of patient numbers in any population outside the volunteer (non-psychiatric) population. To diminish the potential of a confounding presence in this sample, there are equal numbers of patients in each group that are taking benzodiazepines (5 in each group).

2.1.5. Social stressors

As previously mentioned, the Groote Schuur outpatient clinic serves a population group representing largely those of a low socio-economic grouping, and as a result sees many patients who face overwhelming social stressors. These include domestic violence, parents with children in unsafe environments in which gang activity frequently results in bystander injuries, unemployment and substance abuse.

2.2. Procedure

A questionnaire was devised to obtain measures of dreaming frequency, visual vividness and emotional intensity with a view to being able to compare the results with those of the Pace-Schott et al. (2001) study. In addition, each participant was requested to supply a most recent dream report, in order to perform external ratings of dream intensity. This was done according to the format recommended by Domhoff (2003), in which a standardized directive is given the respondent, asking for date of the dream, and asking the respondent to pay attention to all aspects of the dream, such as its setting, familiarity and feelings experienced. The next section is devoted to a discussion of the rationale for the construction of the questionnaire and plans for subsequent analysis.

2.2.1. Operationalising the dependent variables – vividness and intensity

Various authors have made statements regarding dreaming intensity and vividness, whether in relation to drug use, which is the focus of this research, whether in comparing REM with NREM dreams, whether as part of a broader content analysis, or whether in relation to REM rebound (Hobson et al., 2000b). Few of the studies reviewed attempt a definition of either intensity or vividness and fewer still have specified a method by which actual dream reports may be objectively rated for intensity. Most rely on an intuitive understanding of the terms intensity and vividness, though vividness is sometimes qualified as visual vividness and intensity as emotional intensity. In a 1998 study by Schredl and Doll, the terms intensity and emotional intensity are used interchangeably throughout the paper, as they are in the Meier, Ruef, Ziegler and Hall (1968) study. Hobson et al. (2000b) in attempting to answer the question of whether intense dreaming is confined largely to REM sleep, confusingly proceed to discuss dream length as though this were the same thing as intensity. Meier et al (1968) set the criteria which dream intensity (again used interchangeably with emotional intensity) is measured, as including those in

which there was the presence of conflict, misfortune, dramatic events or strong emotion, thus providing a similar working definition to that of Schredl and Doll (1998) in that intensity appears restricted to the emotional sphere. In the Hall and Van de Castle (1966) method of content analysis, dream intensity is not explicitly defined, though it is measured through the identification of modifiers that are used to describe force or expenditure of energy. Various authors repeat the assertion that REM dreams are more vivid and intense than NREM dreams; one should be aware that such usage often relies on an intuitive understanding of the psychological constructs concerned and the meaning may vary from author to author (see Nielsen, 2000, Feinberg, 2000).

The Oxford dictionary (1994, p.898) defines 'vivid' as '1. bright and strong, intense,....2. producing strong and clear mental pictures', whereas 'intense' is defined as 'strong in quality or degree', and 'intensity' as 'the amount of some quality' (p.416). These are fairly useful guidelines, though different from those provided by the authors described above. Whereas vividness and intensity are sometimes used interchangeably in dream descriptions, the Oxford definition appears to assigns a visual component to the definition of vividness ('providing strong and clear mental pictures'). The word intensity is relegated a more quantitative role as descriptor – as the strength, amount or degree of the impression or feeling. Perhaps this explains why the use of both terms is common, as one would presumably wish to capture the strength of emotions as well as the strength of visual imagery when speaking of intensity. Particularly where vividness and intensity are used interchangeably, it seems likely that intensity refers to the strength of a visual impression as well as the strength of a feeling.

2.2.1.1. Self report measures of vividness and intensity

Preferring not to reinvent the wheel, and since this research is intended in large measure to complement the findings of Pace-Schott et al (2001), it was hoped to use the same approach to rating vividness and intensity. In their discussion section, they refer to observing an increase in five out of seven 'intensity related' characteristics of dreaming. These are memorability, visual vividness, amount of sound, emotional intensity, meaningfulness, amount of movement and strangeness, all self-report likert-scaled measures. It is not specified how the authors arrived at this particular set of descriptors as indicators of intensity, though it appears in line the Oxford dictionary meaning in terms of measuring the strength of various impressions – auditory, visual, emotional, motion. The available literature on increased dream characteristics relating to SSRI

use deals specifically with intensity and vividness, and since it was considered important to minimize the number of questions posed to participants, it was therefore decided to restrict the questions to these two most salient components of this scale, emotional intensity and visual vividness, plus the memorability item, due to the reported link between dream intensity and memorability (Domhoff, 2003). Since the aim was to compare our findings, these items were used verbatim from the Pace-Schott et al. (2001) questionnaire. The self-report questions posed to the participants were as follows:

How often do you dream? (every night, most nights, about half the time, occasionally, never).
This is followed by the most recent dream report as set out by Domhoff (2003), whereafter three questions about that dream are posed:

How memorable was your dream?

Most memorable ever 1 2 3 4 5 *can only remember that I did dream*

How visually vivid was your dream?

Most vivid ever 1 2 3 4 5 *not at all vivid*

How emotionally intense was your dream?

Most intense ever 1 2 3 4 5 *not at all intense*

Following this set of directed questions, an open-ended question was posed verbally in which participants were asked if there was anything else they would like to add about their dreams. In this question it was hoped to elicit spontaneous observations regarding changes in dreaming while using SSRI's. It was not possible to ask all participants this question as many participants were not interviewed in person. In retrospect it may have yielded more interesting results had this open-ended question been included in the questionnaire itself from the outset.

2.2.1.2. External ratings of dream intensity

Certain authors are mistrustful of self-report measures (Domhoff, 2003), quantitative researchers generally prefer independent ratings of the dream reports. Domhoff (2003) argues that self-report in dreams is in theory at least, less open to impression management than reports of waking actions, since dreamers do not feel personally responsible for their dreams, as they do their waking actions. Despite this, in content analysis he found that self rated vs. independent rated content variables varied significantly; for example, the number of aggressive or friendly

interactions the dreamer provided does not tally with the description of the dream itself. Domhoff (2003) appears to conclude it is the self-report that is unreliable, but it is also plausible that the dreamer may have experienced an interaction as more or less friendly or aggressive than independent raters might argue them to be. This is even more likely to be the case where descriptors about the dream as a whole are concerned, especially if they are not part of the episodic narrative, such as the general mood of the dream or strength of visual impressions or emotions.

2.2.1.2.1. Precedents for independent ratings of vividness and intensity

Since a number of studies refer to dreams as being more or less vivid or intense than others it was hoped that there was some precedent in independently rating the dream reports as such. This is the case for only a small minority of studies. Markowitz's four patients are described as having newly 'vivid' dreams; this is based on self-report of a qualitative nature. Monroe et al (1965) also argue REM dreams to be more vivid than NREM dreams but provide no definition or method by which this is objectively measured. Aside from the Hall and van de Castle (1966) system which measures intensity, and which will be discussed in some detail, Domhoff (2003) also mentions four studies that had attempted to objectively rate dream intensity. Each of these studies will be briefly examined.

In the earliest of these studies, by Baekeland & Lasky (1968), an assertion is made that REM density is an indirect measure of dream intensity. It seems safe to assume that their definition of intensity simply reflects the then-uncontested view of REM sleep and dreaming as isomorphic. In this way dream intensity and REM density (the quantity of REM sleep) could be equated.

Meier, Ruef, Ziegler and Hall (1968) performed an analysis of the dreams of one subject in which emotional intensity was rated on a four-point scale by the dreamer, with the external rater being the dreamer's psychiatrist. We have mentioned their definition of an intense dream above, as one in which there was presence of impulse, conflict, strong emotion, misfortune and dramatic events, thereby tapping only the emotional sphere. Aside from the problem of their definition being overly restrictive for our own purposes, it is also problematic that the 'independent' rater was the subject's psychiatrist. The subject and the psychiatrist reached an 88% agreement on their ratings of intensity, but the psychiatrist, knowing the patients background, is aware of those situations

which this particular subject is likely to find conflictual or distressing. It is quite possible that a truly independent rater will come to a different conclusion.

Takeuchi, Ogilvie, Ferelli, Murphy and Belicki (2001) created a dream property scale, which purportedly measures vividness. Here vividness is mentioned by comparison with Cohen's (1979) dream categories of emotionality, bizarreness, activity and vividness, which purportedly correspond with the Takeuchi et al scale's four factors of emotionality, bizarreness, activity and impression. Their factor of 'impression', which is said to correspond with Cohen's vividness property is comprised of the following descriptors: fuzzy vs. clear, unfocused vs. focused and foggy vs. clarity. The Cohen (1979) paper refers the reader to an earlier study (Cohen and Macneilage, 1974) for a description of how their four dream properties are defined and measured. Here, the authors describe vividness as 'sensory impact', which broadens the scope beyond visual impact. No further information is provided in this paper as to what questions were framed in order to establish a vividness score, however it is stated that this was obtained through a series of self-report questions.

Trinder and Kramer (1971) operationalised dream intensity as the sum of a number of dream content variables as measured using the Hall and Van de Castle system, which is discussed below. These variables were social interactions, emotions, achievement outcome and 'environmental press', the meaning of which is unclear. These variables were combined to produce what was termed a 'dramatic intensity' scale.

2.2.1.2.2. Hall and Van de Castle's content analysis

The Hall and Van de Castle (1966) content analysis system is perhaps the best-known method of quantitative analysis and has been adopted by Domhoff (2003) as a standard in the quantitative analysis of dream content. Hall and Van de Castle (1966) noted that prior dream research used intensity interchangeably with frequency, usually of a certain type of interaction (They provide an example to illustrate - that someone who has a lot of aggressive interactions in their dreams allows for the interpretation that there is 'intense' aggression in the dream). What they seem to be saying is that intensity per se had not been rated in previous dream research (prior to 1966) but rather it was the intensity of a particular construct that was of interest. Hall and Van de Castle went on to state that 'it may be that…intensity cannot be defined operationally in psychology'

(p.14). Later in the same text however they do just that, as they operationalise intensity using a series of modifiers that are used to describe force or expenditure of energy. The Hall and Van de Castle (1966) system of dream content rating uses the intensity 'modifier' as either high or low intensity. In this way, a word such as 'very' or 'extremely' modifies an emotion such as sadness allowing the emotion to be coded high intensity, or I+ whereas a word such as 'mildly' downgrades the emotion to low intensity or I-. Each intensity modifier is listed as either I+ or I-; in this way a dream could have two I- and three I+ modifiers listed. No subsequent step is taken in the rating process to evaluate a dream as more or less intense than another though it is apparent that should one wish to do so, one simply has to total the modifiers to arrive at a score of for example 1 where there are two negative and one positive modifiers. Intensity modifiers need not refer to emotion, for example a *bright* light attracts an I+ in this system. The number of positive and negative intensity modifiers may be compared with norms, which are separate for males and females.

The original Hall and Van de Castle (1966) text provides an example to illustrate what can or cannot be viewed as an intensity modifier. In this example (Hall & Van de Castle, 1966 p.120), the authors assign an I+ to words such as 'very' or 'extremely'. However they do not allow an I+ for words such as 'furious' since 'simple mention of an emotion usually associated with a strong effect is not sufficient for coding' (p.120). This lends itself to a situation in which one individual might describe an interaction stating the person was 'very cross' and another, arguably more linguistically sophisticated, might describe the same situation stating the person was 'furious'. According to the Hall and Van de Castle system, only the former description may be assigned an intensity rating. This unfortunately undermines the validity of the construct as used by their system. An attempt was therefore made to modify the Hall and Van de Castle method to allow such descriptors, but soon ran into the kind of trouble that presumably led to their exclusion. In an attempt to establish inter-rater reliability on these descriptors, it became clear that a scale would be required in order to establish correspondence of every extant adjective with a degree of intensity – for example agreement could be reached that 'furious' scores an I+, but is 'annoyed' an I-? Is 'cross' neutral? These are not problems that can be satisfactorily resolved, therefore one is forced back on the compromise that Hall and Van de Castle used - the exclusion of these adjectives altogether. The following, very short dream for example would not score any points for

intensity, though raters agreed that on a subjective rating of the dream, this should score highly for intensity.

"It was a dream about me and a friend being raped, and I couldn't reach out to help her and there was no way that I could help her. I wanted to go back into the dream so that I could take revenge."

This dream describing an extremely unpleasant experience scores no points since there is no contextualisation allowed to take part in the rating process, no allowance for negative events (no verbs) such as rape or assault, and no allowance for the repetition of phrases. The dreamer has not told us she felt 'very' scared, which could have scored an I+ but if she had said she was 'terrified' she would have still not scored an I+. Raters agreed that this scores at least a 4 out of 5 on a subjective intensity rating, as we *infer* from the nature of rape per se, as well her intention to take revenge that intense feelings would be taken as given.

2.2.1.2.3. The Schredl and Doll study

Schredl and Doll (1998) attempted an objective investigation into dream emotion using Hall and Van de Castle's rating system. Aside from the fact that emotion, intensity and emotional intensity are conflated in their study, their self-report measurement of dream emotion, both positive and negative, had an extremely poor correlation with independently rated measurement of positive and negative emotion, using the Hall and Van de Castle system. They show that judges consistently under-rate the number of emotions in the dream report, and typically over-rate the amount of negative emotion relative to positive emotions as compared to the dreamers evaluation of the dream. In their study, a comparison was made between self-rating of positive emotion, independent rating of positive emotion and the Hall and Van de castle system's rating of positive emotion. Good inter-rater reliability was obtained on the Hall and Van de Castle system measures, but this had a correlation of only .362 for positive and .2 for negative emotions with the self report measures. External rating on a scaled system of independent rater evaluation produced better correlation with self report measures at .557 for positive emotions and .669 for negative emotions, but inter-rater reliability was not reported. In both cases (external scaled and Hall & Van de Castle rated) the overall amount of emotion and amount of positive emotion was underestimated by external raters as compared with self report (Schredl & Doll, 1998).

Following the original Hall and Van de Castle rules strictly with regard to rating intensity it is easy to see how one could attain very good inter rater reliability, and nevertheless produce meaningless results. This is not to discredit their system in its entirety; the Hall and Van de Castle system is intended as a system of content analysis, and content variables lend themselves better to measurement (e.g. how many strangers vs. known people were in the dream). Domhoff (2003) has shown that using the system, various dream content variables can be demonstrated to have continuity with the dreamer's waking life, therefore demonstrating the validity of the method for content analysis. Intensity is not a content variable however, rather a characteristic of the dream as a whole.

2.2.1.2.4. Approaching an independent rating of dream intensity

With the foregoing in mind, in attempting an objective rating of intensity from the dream report, we took a similar approach to Schredl & Doll (2000), and decided to use a to use a rater-assigned likert scaled intensity score for the dream as a whole. After some discussion among the raters, it was agreed that a four-point likert scale would be used, with the values defined as follows:

1 – Not at all intense
2 – A bit intense
3 – Very intense
4 – Extremely intense

Two raters were employed to analyse the dream reports, and were instructed that the Oxford dictionary definition of intensity would be used, as the strength of some impression. They were explicitly instructed not to restrict this to emotional intensity but also the strength of any impression. Raters were blind as to whether the dream reports came from experimental or control patients. A person other than these raters typed all dream reports up prior to distribution. Inter-rater reliability was first obtained on dreams that had been excluded from the study due to incompatible medication or psychopathology. Inter-rater reliability was calculated using Pearson's r (Howell, 1999) and is considered good at $r=.94$ ($p<0.5$).

2.3. Data Analysis

The self-report likert-scaled data, as well as the externally rated intensity measure were compared between the experimental and control groups using t-tests. Domhoff (2003) prefers to restrict data analysis in dream reports to nonparametric tests such as chi-squared with nominal categories. He argues that nominal systems are better than parametric analysis with ordinal rating scales treated as interval data, in which a score of 4 cannot be shown to be twice a score of 2. On the other hand Durrheim (1999) argues that treatment of the same data by parametric or nonparametric means often yields similar results. For this reason he argues that nonparametric statistics should be reserved for categorical data. We have therefore proceeded as per the Pace-Schott et al. (2001) study, treating the ordinal data as interval data and proceeding with parametric statistics (in their case repeated measure ANOVA was used to compare the likert-scaled responses across the four treatment stages). Independent sample t-tests were performed comparing the responses from the two groups on the four self report and the single external rated intensity measure.

3. Results

A total of 46 dream reports were obtained from the 40 subjects. 5 patients recalled no dream, 3 from the experimental group and 2 from the control group. Three patients contributed more than one dream report, two of whom were taking SSRI's and one without medication.

T-tests were performed using Statistica 6.1 in order to establish whether any differences exist between the SSRI group and the control group on the four self-report variables obtained, as well as the independently rated measure of intensity. For the sake of completeness, the Mann-Whitney test for non-parametric data was also performed. Where data is absent for a particular dependent variable, that case is omitted, hence slightly variable sample sizes from test to test. Reported values are rounded to 3 decimal places.

3.1. Self-report Measures

3.1.1. Frequency

On the self reported measure of frequency, no difference was found between the two groups, moreover there was not even a trend, as can be seen below:

T-Test:

	Mean SSRI	Mean Control	t-value	df	p	Valid N	Valid N
Frequency	2.211	2.474	-0.689	36	0.495	19	19

Mann-Whitney:

	Rank Sum	Rank Sum	U	Z	p-level	Valid N	Valid N
Frequency	349.000	392.000	159.000	-0.627	0.530	19	19

3.1.2. Memorability

On the self report measure of memorability, there was no statistically significant difference

T-Test:

	Mean SSRI	Mean Control	t-value	Df	p	Valid N	Valid N
DMemorable	2.412	2.389	0.057	33	0.955	17	18

Mann-Whitney:

	Rank Sum	Rank Sum	U	Z	p-level	Valid N	Valid N
DMemorable	319.500	310.500	139.500	0.445	0.642	17	18

3.1.3. Visual Vividness

On the self- report measure of visual vividness, there was a statistically significant difference at the p=.05 level both using a t-test (p=0.027) and the non-parametric Mann-Whitney (p=0.029). Effect size was calculated as the difference between the means divided by either standard deviation as per Howell (1999). This was calculated as (2.412-1.722)/0.795 = 0.86. The standard deviation for the control group was used, at .795. Pace-Schott et al (2001) use a slightly different formula, averaging the two standard deviations. Using their formula we would obtain a slightly stronger result. (2.412-1.722/0.779 = 0.88).

T-Test:

	Mean SSRI	Mean Control	t-value	df	p	Valid N	Valid N
Dvivid	2.412	1.722	2.309	33	0.027	17	18

Mann-Whitney:

	Rank Sum	Rank Sum	U	Z	p	Valid N	Valid N
Dvivid	372.000	258.000	87.000	2.178	0.029	17	18

3.1.4. Emotional Intensity

Dreams were not rated as significantly different on the self report measure of emotional intensity. ($p > 0.05$)

T-test:

	Mean SSRI	Mean Control	t-value	Df	p	Valid N	Valid N
Dintense	2.294	2.500	-0.430	33	0.669	17	18

Mann-Whitney:

	Rank Sum	Rank Sum	U	Z	p-level	Valid N	Valid N
Dintense	296.000	334.000	143.000	-0.330	0.741	17	18

3.2. Independent measures

Independent raters assessment of intensity was not significantly different across the two groups.

T-Test:

	Mean SSRI	Mean Control	t-value	df	p	Valid N	Valid N
IndependantIntense	2.304	1.952	1.618	42	0.113	23	21

Mann-Whitney:

	Rank Sum	Rank Sum	U	Z	p-level	Valid N	Valid N
IndependantIntense	572.000	418.000	187.000	1.281	0.200	23	21

3.3. Correlation between measures of self reported intensity vs Independent rating

A self-report measure of overall intensity was calculated using a combination of the three intensity sub-measures, viz; memorability, visual vividness and emotional intensity. This value was correlated with the independent measure of intensity. There was no relationship between the independently rated likert-scaled intensity and self-reported likert-scaled intensity measures (Pearson's r = -.25, p >.05). Broken down by group, the correlation coefficient for the SSRI group was r = –0.30, p>.05 and for the control group r =–0.22, p>.05.

3.4. Qualitative data

23 of the 40 respondents were asked whether there was anything they would like to add about their dreaming. Of these, five had nothing to add. A further six respondents made comments that are not useful for our analysis, such as one report that a person had always had premonitory dreams, another discussing recurring dreams that they had, and the remainder describing sleep rather than dream disturbances.

Of the 12 responses that held useful data, 8 were from patients who were on SSRI medication and 4 who were not. Of these 4, one complained of having mostly upsetting dreams, and another reported that they hardly ever dreamed. The other two who were not taking SSRI's had previously taken fluoxetine, but had terminated therapy some time ago. One claimed that 'I dreamt more vividly when I was on fluoxetine', and the other stated 'I dream less since off fluoxetine, my dreams were more vivid, stronger and more often on the meds'.

The comments from the 8 patients taking SSRI's may be summarized as follows:

Two reported more frequent dreaming

Two reported more 'realistic' dreaming

Two reported more unpleasant dreams and nightmares

One reported more 'complicated' dreams

One stated that she had always had very detailed dreams. This patient had only been on SSRI's for one month.

One reported hypnopompic phenomenon in which her dreams and waking reality sometimes became confused. Her comments were 'Sometimes I wonder if a dream really happened, I can get quite confused sometimes, it can be quite upsetting if I can't work it out.' This person had been taking fluoxetine for 3 years.

Table 4: Qualitative responses by SSRI and therapy duration

Patient	Response	SSRI	Duration
LG	More frequent	Fluoxetine	1 yr, 6 months
PH	More frequent	Citalopram	1 month
PH	More realistic	Citalopram	1 month
EV	More realistic	Fluoxetine	1 year
TK	More unpleasant	Fluoxetine	3 years
DM	More unpleasant	Paroxetine	2 yrs, 6 months
NW	More complicated	Fluoxetine	4 months
MH	Always detailed	Fluoxetine	1 month
AM	Hypnopompic	Fluoxetine	3 yrs

It was unfortunately not possible to pinpoint differences between the different drugs, as there were not sufficient proportions of each drug to carry out statistical analysis. As previously mentioned, the majority of participants were on fluoxetine, since generics of this drug tend to be cheaper and more readily available from State facilities. Nevertheless for interest, a table (Table 4) is provided detailing the responses, the corresponding SSRI and the duration of SSRI therapy. Note that one patient (PH) described dreams that were both more realistic and more frequent; this case is listed twice, once under each response heading.

4. Discussion

4.1. Self Report measures

Of the self report measures that were taken from the Pace-Schott et al (2001) study for use as indicators of intensity, only visual vividness came up as significantly higher for the experimental group ($p = 0.027$), with memorability, emotional intensity and frequency of dreaming emerging as not significantly different across the two groups. The effect size for visual vividness at .86 is considered large according to Cohen's rule of thumb (Howell, 1999), and this should therefore be regarded as a solid finding. Domhoff (2003) in fact argues that effect sizes of more than .40 should be considered large in dream research.

4.2. Independent ratings of intensity

There was no statistically significant difference between the group taking SSRI's and the control group on the measure of independently rated intensity. Despite good inter-rater agreement in terms of what constitutes an intense dream, it was apparent that there is an extremely poor correlation between the dreamers self-report of intensity (as measured using a combination of memorability, emotional intensity and visual vividness) and independently rated assessment of the intensity of the dream report ($r=-.27$). Looking back at the Meier et al (1968) study in which the psychiatrist rated his subject's dreams and was guided by personal history as context, it is instructive that where the process of obtaining inter-rater agreement was lacking in consensus in our own study, it was around precisely the issue of personal salience. Raters frequently felt a dream described some level of misfortune but that the dreamer had not conveyed their own subjective sense of distress. As an example, in this dream the dreamer assigned the highest rating for emotional intensity:

"*I dreamed about my sister's son, he passed away last year. I dreamed he helped me with my kitchen, putting in some new furniture.*"

Since the rater does not know what relationship the dreamer had with the sister's son, and aside from the mention of his being dead, no account of distress was given, the rater assigned this the lowest intensity rating (Not at all intense). This correlates poorly with the self-report measure of emotional intensity. There were three other similar examples. With regard to independent

measures of intensity, the inclination is therefore to revert to Hall and Van de Castle's 1966 initial assertion – that perhaps it is a psychological construct that cannot reliably be objectively assessed in a narrative. This lack of correspondence is in our opinion inherent in the task itself rather than a function of our sample, which would lead us to expect that the same problem would result in any replication attempt with a different sample. Therefore, despite Domhoff's (2003) reservations regarding self report, one is inclined to take the phenomenological route, and assert that at least in this dream series, dreamers have for whatever reasons, not successfully managed to convey their subjective sense of a dream's intensity in their report of the dream. The fact that visual vividness was found to be highly significant whereas frequency, emotional intensity and memorability were not at all significant militates against potential impression management where self – report is concerned.

4.3. Qualitative information

The qualitative responses follow the same pattern as the quantitative data, with two comments regarding increased frequency on SSRI medication (20 % of those that were asked the open-ended question), and a larger number of comments (seven) stating that dreams were more realistic, more vivid, more complicated and sometimes more unpleasant on SSRI treatment. It is of interest here that in Solms' (1997) study, patients used the term 'realistic' interchangeably with 'vivacity' and 'perceptual clarity' in describing their dream imagery. If one considers the comments regarding vividness, realistic, complicated, nightmarish and hypnopompic phenomenon collectively as indicators of more vivid and intense dreaming, comments regarding increased vividness and intensity represent 70 percent of SSRI users that were posed this open ended question. This excludes the two comments regarding increased vividness when taking SSRI's made by control group patients that had previously taken SSRI's, since not all of the control group patients had previously been on medication. None of the control group patients spontaneously described their current dreams in these terms. It should be reiterated that the question was posed directly after the written questions eliciting self report information about dream intensity, vividness and memorability, and it is possible that some degree of experimenter effect was at play.

4.4. Implications

Using similar bases of comparison, this study has replicated the main findings of the work of Pace-Schott et al (2001), in that self reports of visual vividness are higher in users of SSRI's whereas frequency of dreaming is not increased in SSRI users. Unlike the Pace-Schott study we did not find differences in memorability or emotional intensity, even as a trend. It should be stressed that there are substantial differences in our respective studies; the Pace-Schott et al study uses 7 subjects in each group whereas ours uses 20 in each, and that their study uses a repeated measures design for purposes of comparison, whereas ours is cross sectional, using a non-medicated control group for comparison. Finally the mean duration of treatment in our study was far longer at 16 months (compared with 19 days).

The 'modulation' component of Hobson's AIM model would predict that increased serotonin, the primary effect of SSRI administration, should lead to a reduction in frequency as well as vividness and intensity of dreaming through the suppression of REM sleep. The evidence does not support this theory on either count, despite the fact that the SSRI's do strongly suppress REM sleep. Frequency of dreaming on the one hand remains the same across users of SSRI medication and those not taking SSRI's. Self-reported visual vividness is increased in SSRI users, a movement in the opposite direction to what the AIM model predicts, and emotional intensity is not significantly different in our study.

4.5. Limitations

4.5.1. Education and socio-economic factors

The dream reports obtained were taken from individuals who for the most part had no tertiary education, and came from a low SES background. This might potentially impact on the nature of the dream reports obtained. It may be that with a group from low SES communities in which poverty, poor living conditions, substance abuse and gang violence is rife, that it is not unlikely for dream life to take a backseat. One cannot be sure that with a relatively 'unpsychologized' sample, terms such as 'intensity' and 'vividness' are understood in the same way that they might be by, for example, college students. Hobson et al. (2000a) point out that word count is also dependent on the language skills of the individual. This may account for the fact that many of the dream narratives (13 out of the total 46 dreams) were shorter than are usually considered adequate for content analysis (Domhoff [2003] has suggested a 50-word minimum). Domhoff (2003)

prefers to use reports of similar length for content analysis as he argues that word count has correlations with other rating scales (it would be unsurprising to find correlations between word count and level of education for example). Therefore as an exercise, word count was correlated with all the dependent variables (frequency, memorability, visual vividness, emotional intensity), nothing approaching significance was found.

4.5.2. Use of Normal, paid volunteers vs. psychiatric participants

There are advantages to conducting research amongst the psychiatric population; since this is the population from whom the case reports arose in the first place and since they are already taking the drug one wishes to research. The disadvantages are that with the high co-morbidity of depression with anxiety disorders, there may have been patients in the experimental group with co-morbid depression that dreamed more than they did prior to taking SSRI's. In addition there may have been patients in the control group with depression that dreamed less than they would have if they had been undergoing drug or other therapy. Another problem with the psychiatric population is the co-administration of other medication, as well the impossibility of screening out unreported substance abuse, which has a higher incidence amongst the psychiatric population (Kaplan & Saddock, 2003). As Pace-Schott et al (2001) noted however it is costly conducting research of this nature with volunteers, who are paid for their participation and provided expensive drugs. For this reason those authors were forced to treat their study as a pilot, limiting their sample to 14 volunteers, 7 in each group, and consequently limiting the scope of possibilities for statistical analysis.

4.5.3. Complexity of neurotransmitter systems

We have discussed the activity of the aminergic and cholinergic systems as though neurotransmitters operated as discrete entities and exerted unitary effects, for the purpose of illustrating that a substance that suppresses REM would not also be expected to increase dreaming according to the dominant view. This oversimplification may obscure the fact that the various neurotransmitter systems influence one another, for example GABA (an inhibitory neurotransmitter) is known to inhibit activity in the locus ceruleus, which produces noradrenalin (Gaillard, 1994a). These interactions may occur in rather complex ways, for example serotonin release from raphe nuclei inhibits dopamine production in the midbrain but simultaneously facilitates its production in the cortex. The facilitatory or inhibitory influence of one

neurotransmitter system on another is also state dependent, as for example in the noradrenergic activation of dopaminergic neurons during attention but not during sleep states (Gottesman, 1999). Gaillard (1994b) also suggests that since a single neuron may produce both neurotransmitters and neuropeptides it is possible that the peptide acts as a 'co transmitter' to modulate or fine-tune the action of the neurotransmitter at the synapse. Substance P for example is consistently found together with serotonin (Perry & Young, 2001). Increased levels of a neurotransmitter such as acetylcholine in the brainstem, certainly does not imply increased forebrain levels of acetylcholine, indeed it is possible to have decreased levels of cholinergic activity in one part of the forebrain such as the hippocampus, but not other parts such as neocortex, as is found in Alzheimers disease (Perry & Perry, 1995). Gaillard (1994b) thus cautions against simplistic models of the effects of drugs on sleep and dreaming characteristics due to the level of interaction among neurotransmitters and neuronal pathways, leading to 'cascades of changes in which it is difficult to identify the primary effect' (p.336). Gaillard et al (1994) and Gottesmann (1999) also warn against extrapolation of findings in rats and other small mammals (in whom a majority of sleep research has been conducted) to humans, in whom the neuronal system is considerably more complex. At least one study has found a significant discrepancy in the activity of cholinergic enzymes in the human brain as opposed to the rodent (Perry & Perry, 1995).

4.5.4. Heterogeneity of the SSRI's

With respect to the foregoing, two issues are of concern with regard to medication; the first, the concurrent use of benzodiazepines has been discussed, though mitigated by equal numbers in each group. A further concern is that if we are to attempt to establish the neurochemical mechanisms involved in dreaming through a study of their effects in SSRI users, it is not an ideal approach to study heterogeneous drugs. Despite the fact that the SSRI's have in a gross sense, a similar mode of action, these drugs are not identical, for example citalopram is sometimes said to be 'cleaner' in the sense that it has very few direct effects on the levels of neurotransmitters other than serotonin (these effects are called secondary binding properties), whereas paroxetine for example has anticholinergic properties, mild dopamine reuptake occurs with sertraline, and noradrenalin reuptake occurs with fluoxetine. These secondary binding properties are considered important enough to have potential clinical consequences over and above the intended serotonergic effects; moreover, as new receptors and enzymes are better clarified, other effects

may be identified (Stahl, 2000). Perry & Piggott (2000) noted that one anti-Parkinson medication with affinity for D_3 but not for D_1 receptors led to more pronounced increases in dreaming intensity than L-dopa. Different SSRI's also have different strength of effects on different serotonin receptor subtypes (eg. $5HT_{1A}$ vs. $5HT_{2A}$), the functional differentiation of which is still in its early stages. In practice, the Pace-schott et al (2001) study showed that paroxetine and fluvoxamine appear to have behaved quite differently from one another with respect to dream frequency and word count when comparing baseline to initiation of treatment. The result is that any conclusions regarding neurotransmitter impact on dreaming characteristics through SSRI use run the risk of oversimplification of mechanisms that are as yet not fully understood. For this reason it would be recommend that any further investigation of increases in dreaming frequency or intensity be undertaken as part of larger clinical trials targeting specific SSRI compounds. In 1979 Roth, Kramer and Salis lamented that in their review of the literature on the impact of drugs on dreaming, there had been no attempt to examine a single drug in any depth. The demand for newer and better antidepressants is likely to entrench this difficulty, ensuring that for any given patient population, an ever broader range of alternatives is available; research using healthy volunteers thus seems the more promising way to study a single drug.

4.6. Conclusions

Despite apparently strong disagreement, differences in opinion regarding REM and dreaming seem to have been more pronounced in the past than currently. There are currently many common areas of agreement. There is more widespread acknowledgement of the role of the forebrain in dreaming, where Hobson's original activation synthesis model previously gave it the role of responding to random noise from the pontine brainstem, it is now accorded a more complex role. There also seems to be agreement that the later stages of sleep tend to produce more hallucinatory than thoughtlike material regardless of whether that stage is REM or NREM. In a recent article in which Hobson is a co-author (Fosse, Stickgold & Hobson, 2004), the authors go so far as to state that this "…speaks to the variability of cognitive function in any sleep state". (p.302), and further that "the basic EEG differences between NREM and REM do not provide reliable correlates to the cognitive data" (p.302), though variation in muscle activity has been shown to correlate with the different types of sleep mentation. The authors conclude that the biological underpinnings of the changes in cognition from early to late night sleep remain unknown. This appears to represent a major shift on Hobson's part away from a two-generator model of dreaming with REM/NREM

as its psychophysiological correlate, when compared to his earlier statement that dreaming is an 'epiphenomenon of REM sleep' (Hobson, Stickgold, & Pace-Schott, 1998, p.R12). Perhaps surprisingly there is even agreement regarding emotion as the probable shaper of dream content. Many authors (Kramer, 2000; Hobson et al., 2000b) now consider pre-sleep emotion to be the primary shaper of dream content (in the same way it is, Hartmann (2000), argues, for waking daydreams). The role of the limbic system in the production of dream content is now a feature of both Hobson and Solms's dream theorizing, though Hobson cautions against drawing inferences from limbic involvement in dreaming towards assumptions of some sort of emotional processing aspect to dreaming. Finally it is commonly acknowledged that the role of cerebral activation needs to be considered in dream generation theories.

On obtaining a finding of increased dream intensity in their earlier study, the Hobson team (Pace-Schott et al, 2001) posits a REM-rebound phenomenon to explain increases in their self-report measures of intensity with reduced REM. While REM rebound on discontinuation of SSRI's is documented, a rebound effect during SSRI administration remains an as yet untested hypothesis. We conclude that whatever the reason for the increase in vivid dreaming, until this proposition is tested, the findings remain inconsistent with the REM=dreaming position. As far as the cerebral activation model is concerned, Solms (2004, personal communication) points out that the fact that ventromesial and PTO lesions disrupt dreaming also shows that cerebral activation is only one piece of the puzzle, and as a model it is therefore incomplete. The finding that increased serotonin leads to more vivid dreaming would also not have been predicted by theories of cerebral activation, since serotonin is regarded as having largely inhibitory effects (Gottesmann, 2001). There are some promising areas for investigation, such as Gottesmann's (2001) emphasis on relative levels of the amines in sleep and dreaming, and also on elucidating the functions of the various neurotransmitter receptor subtypes. We may yet uncover receptor subtype functions in serotonergic transmission that act in an excitatory rather than an inhibitory manner with investigations into specific SSRI's rather than the class as a whole. The Fosse et al (2004) data on time of night in predicting the quality of sleep mentation also points to promising new areas for research. We concur with Nielsen and colleagues in concluding that the psychophysiological correlates of dreaming remain unclear (Esposito, Nielsen & Paquette, 2004), though if we are to move forward in uncovering these correlates, as Stickgold (2000, p.1011) stated – 'we should trust the data more and the theories less'.

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APPENDIX 1 – Dream questionnaire

Initials ____ **Age** _____ **Gender (M/F)**_____

Date Today _____

Current psychiatric medications	Length of time on these medications	Dosage level per day

Do you take these medications as they were prescribed? (Yes/No)_____

If 'No', please state the average daily dose actually taken _____

How often do you dream? (circle the answer that best describes your recollections of having dreamed, even if you don't remember all the details)

Every night	Most nights	About half the time	Occasionally	Never

MOST RECENT DREAM

We would like you to write down the last dream you remember having, whether it was last night, last month, or last year. But first please tell us the date this dream occurred: _____.
Then tell us what time of day you think you recalled it: _____. Then tell us where *you were when you recalled it:* _____.
Please describe the dream exactly and as fully as you remember it. Your report should contain, whenever possible: a description of the setting of the dream, whether the setting was familiar to you or not; a description of the people, their age, sex, and relationship to you; and any animals that appeared in the dream. If possible, describe your feelings during the dream and whether it was pleasant or *unpleasant. Be sure to tell exactly what happened during the dream to you and the other characters.*
Continue your report on the other side of the page and on additional sheets if necessary.

1. How memorable was your dream?
   most memorable ever **1** **2** **3** **4** **5** *can only remember that I did dream*

2. How visually vivid was your dream?
   most vivid ever **1** **2** **3** **4** **5** *not at all vivid*

3. How emotionally intense was your dream?
   most intense ever **1** **2** **3** **4** **5** *not at all intense*

Thank you very much for participating in this study.