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COMBINAISONS SÉQUENTIELLES DES TRAITEMENTS PHARMACOLOGIQUE ET COGNITIVO-COMPORTEMENTAL POUR L'INSOMNIE CHRONIQUE

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RÉSUMÉ COURT

Cette thèse de doctorat vise à étudier l'efficacité de combinaisons séquentielles d'un traitement pharmacologique et behavioral-cognitif (TBC) pour l'insomnie et à évaluer l'efficacité de l'actigraphie comme outil objectif de mesure du sommeil. Le premier chapitre évalue, dans une étude pilote, différentes séquences de traitements afin d'identifier celles qui semblent les plus prometteuses et le deuxième chapitre évalue plus systématiquement ces séquences de traitement. Les résultats des deux premiers chapitres démontrent que le traitement séquentiel est efficace pour améliorer la continuité du sommeil et que ces améliorations surviennent principalement après l'introduction du TBC. Les résultats démontrent également que l'ajout d'une médication en début de traitement augmente surtout le temps total de sommeil. Finalement, les résultats du dernier chapitre, s'intéressant aux techniques d'évaluation de l'insomnie, démontrent la sensibilité de l'actigraphie comme mesure d'évaluation des effets du traitement. Cette thèse comporte des implications cliniques importantes pour améliorer l'évaluation et le traitement de l'insomnie.

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RÉSUMÉ LONG

Cette thèse de doctorat étudie l'efficacité relative de combinaisons séquentielles d'un traitement pharmacologique et béhavioral-cognitif (TBC) pour l'insomnie. Parallèlement, elle étudie la sensibilité et la précision de l'actigraphie comme outil objectif de mesure du sommeil. Le premier chapitre explore différentes séquences de traitements afin d'identifier le protocole et les séquences qui semblent les plus prometteuses. Le deuxième chapitre établit les meilleures séquences de traitements possibles en modifiant le protocole et les séquences afin de surmonter les difficultés rencontrées dans l'étude exploratoire. Six participants ont été sélectionnés dans la première étude et 17 dans la seconde. La médication utilisée est le zopiclone et le TBC comprend la restriction du temps passé au lit, le contrôle par le stimulus et la thérapie cognitive. La durée du traitement est de 10 semaines. Dans l'ensemble, les séquences de traitements utilisées sont efficaces à court terme pour améliorer la continuité du sommeil jusqu'au suivi de trois mois. Le second chapitre démontre que l'amélioration du sommeil survient principalement à la suite de l'introduction du TBC soulignant ainsi son caractère essentiel. De plus, la médication en début de traitement atténue les effets secondaires de la restriction du temps passé au lit en favorisant l'augmentation ou le maintien du temps total de sommeil. Ainsi, la séquence de traitement combiné (Médication + TBC) suivi de TBC seul semble optimiser l'effet des traitements. Finalement, le dernier chapitre, évaluant la précision et la sensibilité de l'actigraphie au traitement de l'insomnie, démontre que l'actigraphie est un outil de mesure sensible à l'effet du traitement et qu'il doit être utilisé conjointement à un autre instrument de mesure du sommeil. Cette thèse a permis de démontrer que les combinaisons séquentielles d'une médication et du TBC sont efficaces pour traiter l'insomnie. Elle souligne également le caractère essentiel du TBC dans le traitement de l'insomnie chronique et, par conséquent, la nécessité de le rendre plus accessible à la population.

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ABSTRACT

This thesis evaluates the relative efficacy of sequential treatment combinations involving medication and cognitive-behavioural treatment (CBT) for insomnia. Furthermore, it assesses the accuracy of actigraphy as an objective evaluation tool to measure treatment outcome. The first chapter evaluates three treatment sequences in order to determine which ones deserve further investigation in a subsequent study. Using a modified design intended to overcome difficulties encountered in the exploratory study, a second investigation, reported in chapter 2, was then carried out to determine the best treatment sequence. Six participants were enrolled in the first study and 17 in the second one. The medication used was zopiclone, and CBT included sleep restriction, stimulus control, and cognitive therapy. Treatment duration was 10 weeks. Treatment sequences were effective in improving sleep continuity in the short-term and at a 3-month follow-up assessment. The second chapter demonstrates that sleep improvement mainly occurred after the introduction of CBT, suggesting that it is an essential treatment component. Moreover, the use of medication in the first phase of treatment attenuated the secondary effects of sleep restriction by enhancing or maintaining total sleep time during treatment. Thus, it seems that the sequence combining Medication + CBT initially followed by CBT alone optimised treatment effects. Finally, the last chapter, discussing the accuracy and sensitivity of actigraphy, demonstrated that actigraphy is sensitive to treatment response and that it should be used as an adjunct to other sleep assessment tools. This thesis provides evidence that sequential combinations of medication and behavioural therapy are effective in treating insomnia. It also underlines that CBT is an essential component in the treatment of chronic insomnia, thereby emphasising the necessity to increase CBT accessibility to the population.

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AVANT-PROPOS

Les chapitre inclus dans cette thèse rendent compte du travail effectué durant mes cinq années d'études doctorales. Toutefois, ils représentent bien mal l'endurance nécessaire à la réalisation d'une thèse, les différents obstacles rencontrés ainsi que le développement professionnel et personnel qui s'est opéré durant ces années. Les difficultés auraient pu m'empêcher de terminer s'il ne m'avaient pas permis en même temps de découvrir des trésors de professionnalisme, de coopération, de soutien, d'humour, d'amitiés et d'amour. Bref, l'endurance est née des découvertes engendrées par les obstacles et, ce sont ces découvertes qui, au delà du résultat qu'est la thèse, donnent un sens à toutes ces années. Je désire donc remercier tous ceux qui ont contribué à la réalisation de ma thèse tant par support professionnel que personnel.

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FIN

TABLE DES MATIÈRES

LISTE DES TABLEAUX

CHAPITRE 1

CHAPITRE 2

CHAPITRE 3

LISTE DES FIGURES

CHAPITRE 1

CHAPITRE 2

CHAPITRE 3

INTRODUCTION

Étendue de la problématique

L'insomnie fait partie des plaintes les plus fréquemment rapportées aux professionnels de la santé (Morin & Wooten, 1996). Selon des études épidémiologiques, 35% de la population générale a souffert d'insomnie durant la dernière année et 6% à 12% en souffre de façon régulière ou chronique (Ancoli-Israël & Roth, 1999; Ohayon, 1999). D'autres études épidémiologiques se sont intéressées aux facteurs reliés au développement de l'insomnie. Ainsi, le fait d'être une femme (Foley, Monjan, Izmirlian, Hays, & Blazer, 1999; Ohayon, Caulet, & Guilleminault, 1997), de vieillir (Ohayon, 1996), d'avoir un faible niveau d'éducation et un faible revenu (Blais, Morin, Boisclair, Grenier, & Guay, 2001; Ohayon, 1996) de même que le fait d'avoir une histoire familiale et personnelle d'insomnie (Bastien & Morin, 2000; Klink, Quan, Kalterborn, & Lebowitz 1992) favoriseraient le développement de l'insomnie. Toutefois, des études récentes remettent en question l'impact du vieillissement sur le développement de l'insomnie en démontrant que si d'autres facteurs tels que l'état de santé des individus, la prise de médication et les changements normaux du sommeil reliés à l'âge sont pris en considération, alors le vieillissement n'est plus relié à la prévalence de l'insomnie (Foley et al., 1999; Sutton, Moldofsky, & Badley, 2001). L'insomnie chronique se caractérise soit par des difficultés à initier ou à maintenir le sommeil, par un réveil tôt le matin sans pouvoir se rendormir ou par un sommeil non réparateur pendant au moins six mois (International Classification of Sleep Disorders [ICDS], American Sleep Disorders Association [ASDA], 1990). L'insomnie est dite primaire lorsqu'elle n'est pas reliée à une condition médicale, psychiatrique ou à un abus de substance (Ohayon, Caulet, Priest, & Guilleminault, 1997; Simon & VonKorff, 1997).

L'insomnie a des conséquences néfastes tant au niveau de la santé qu'aux niveaux économique et occupationnel, que ce soit pour l'individu ou la société (Gallup Organisation, 1991; Mellinger, Balter, & Uhlenhuth, 1985). En effet, 35% à 40% des individus souffrant d'insomnie rapportent un trouble de l'humeur, d'anxiété, de personnalité et/ou un problème de dépendance ou d'abus d'alcool (Morin & Ware, 1996). Par ailleurs, l'insomnie interfère avec le fonctionnement quotidien de l'individu et affecte sa qualité de vie (Hajak, 2001; Zammit,

Weiner, Damato, Sillup, & McMillan, 1999). De plus, l'insomnie amène des difficultés de concentration et augmente les risques d'accidents (Chilcott & Shapiro, 1996; Kipke et al., 1998). Finalement, les personnes souffrant d'insomnie ressentent un inconfort qui se traduit souvent par un retrait social (Marchini, Coates, Magistad, & Waldum, 1983; Zammit, 1988). Pour la société nord-américaine, les conséquences de l'insomnie se traduisent par une augmentation des coûts et de l'utilisation des services de santé (Roth, 1995; Simon & VonKorff, 1997). L'impact socio-économique de l'insomnie non traitée ou mal traitée est de plus en plus considéré dans les études actuelles. Lors de la Commission nationale sur la recherche sur les troubles de sommeil qui a eu lieu en 1993, les coûts directs (p.ex., visites médicales) de l'insomnie ont été estimés à 15 milliards de dollars américains par année (Roth, 1995). En outre, lorsque les coûts indirects (p.ex., absentéisme au travail) sont cumulés, ils sont estimés à plus de 30 milliards de dollars américains par année (Chilcott & Shapiro, 1996; Stoller, 1994). Par conséquent, il est important de bien comprendre les mécanisme étiologiques de l'insomnie afin de développer le traitement le plus efficace et accessible possible.

L'étiologie de l'insomnie

Depuis une trentaine d'années, plusieurs recherches empiriques s'intéressant à identifier les mécanismes interférant avec l'endormissement et le maintien du sommeil ont été réalisées. Ces études ont permis d'identifer plusieurs mécanismes physiologiques et psychologiques contribuant au développement et au maintien de l'insomnie. Les mécanismes identifiés dans la littérature sont : (a) l'activation physiologique (e.g., Freedman & Sattler, 1982; Monroe, 1967), cognitive (e.g., Lichstein & Rosenthal, 1980) et émotionnelle (e.g., Espie, 1991); (b) les principes de conditionnement classique et opérant (Bootzin, 1972); (c) les croyances dysfonctionnelles (Espie, 1991; Morin, 1993); (d) les mauvaise perception du temps (e.g., Coates, Killen, George, Marchini, & Thoresen, 1982; Edinger & Finns, 1995). Constatant que ces mécanismes ne peuvent de façon individuelle expliquer l'insomnie, deux modèles étiologiques intégratifs ont été élaborés dans les années 90 (Espie, 1991; Morin, 1993).

Le premier modèle (Espie, 1991) explique que l'insomnie est générée par l'interaction entre l'activation du système nerveux central, l'activation des éléments psychologiques tels que les croyances dysfonctionnelles et l'activation environnementale tels que les mauvais indices temporels et situationnels appris par conditionnement classique. Le deuxième modèle (Morin, 1993) présente l'activation physiologique, cognitive et émotionelle comme principal médiateur de l'insomnie. Selon ce modèle, l'activation interagit avec les croyances dysfonctionnelles concernant l'insomnie, les mauvaises habitudes de sommeil et les conséquences perçues de l'insomnie. Ces composantes seule ou en interaction produisent de l'insomnie. Ce modèle souligne l'importance des croyances dans l'insomnie et introduit une distinction entre l'activation cognitive et les croyances elle-mêmes. De plus, il met l'accent sur l'influence bi-directionnelle de ces composantes.

Récemment, de nouveaux mécanismes reliés au maintien de l'insomnie ont été suggérés et influencent la recherche sur le traitement de l'insomnie. Premièrement, un intérêt croissant a été placé sur les conséquences diurnes de l'insomnie (p.ex., fatigue, diminution de performance) afin de comprendre leur rôle dans le maintien des difficultés de sommeil. L'influence de ce mécanisme est pris en compte dans les études s'intéressant à l'amélioration du fonctionnement diurne soit par l'intermédiaire d'une sieste à durée pré-déterminée (Lack & Baraniec, 2002) soit par l'ajout d'un stimulant dans le traitement de l'insomnie (Perlis et al., 2002). Un autre mécanisme a été suggéré à la lumière des différences démontrées entre les évaluations polysomnographiques et subjectives du sommeil (Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997). Ces différences entre les mesures pourraient être dues à un niveau d'activité électroencéphalocorticale élevé qui crée un état d'amnésie empêchant de distinguer le sommeil et l'éveil. Finalement, plusieurs auteurs suggèrent que la présence d'un dérèglement chronobiologique affecterait le rythme circadien justifiant ainsi l'efficacité de la restriction du temps passé au lit dans le traitement de l'insomnie (Espie, 2002; Wohlegemuth & Edinger, 2000). Ce dérèglement expliquerait également la diminution du délai d'endormissement via l'exposition matinale à la lumière vive (Lack, Wright, & Paynter, 1995).

Afin de tenir compte de tous les mécanismes, Espie (2002) propose un modèle intégratif psychobiologique du sommeil normal expliquant le développement et le maintien de l'insomnie. Avec ce modèle, l'auteur rappelle que le sommeil est induit par les principes de rythme circadien et d'homéostasie, que ces principes sont involontaires et capables de réajustement et ainsi, met en lumière le caractère automatique et de plasticité du sommeil. Ces caractéristiques sont centrales dans ce modèle et sous circonstances normales, provoquent, par défaut, un sommeil normal. L'auteur ajoute aussi la notion de défenseurs de sommeil. Ceux-ci incluent tous les mécanismes préalablement mentionnés et sont au nombre de quatre : (a). le sommeil relié au contrôle par le stimulus; (b) le sommeil relié à la désactivation physiologique; (c) le sommeil relié à la désactivation cognitive; et (d) la période diurne favorisant le sommeil de nuit. Ces quatre défenseurs ont pour rôle d'inhiber les stimuli de natures internes (croyances, émotions, physiologiques) ou externes (environnement) qui nuisent à un bon sommeil. Ainsi, ils renforcent le sommeil et celui-ci, les renforce à leur tour. La notion d'influence bi-directionnelle des différentes composantes est toujours présente. Dans ce modèle, l'insomnie est le résultat d'un problème d'inhibition d'au moins un défenseur du sommeil. Si le dysfonctionnement du défenseur se maintient, il y aurait alors perte d'automaticité et de plasticité du sommeil. Le rythme circadien et l'homéostasie seraient affectés et l'insomnie se maintiendrait. Ce modèle présente l'avantage d'expliquer à la fois le sommeil normal, le début ainsi que le maintien de l'insomnie. De plus, il met l'accent sur la compréhension des mécanismes de traitement. Enfin, il pourrait également expliquer les problèmes d'hypersomnie. Finalement, à ce jour, il permet d'intégrer les connaissances empiriques accumulées depuis près de 35 ans dans le domaine de l'insomnie.

Les traitements de l'insomnie

Les options de traitements pour l'insomnie chronique sont pharmacologiques, psychologiques ou alternatifs tels que l'acupuncture ou la photothérapie. À ce jour, quatre méta-analyses ont recensé des études à groupe contrôle dont deux portent sur le traitement pharmacologique (Holbrook, Crowther, Lotter, Cheng, & King, 2000; Nowell et al., 1997) et

deux sur les traitements psychologiques (Morin, Culbert, & Schwartz, 1994; Murtagh & Greenwood, 1995). Smith et al. (2002) ont récemment comparé les résultats de ces quatre méta-analyses afin de faire le point sur l'efficacité des traitements pour l'insomnie.

Les traitements pharmacologiques

Plusieurs agents pharmaceutiques sont employés pour traiter l'insomnie. Ces agents sont sélectionnés pour leurs propriétés hypnotiques et visent à réduire l'activation physiologique et cognitive à l'heure du coucher et durant la nuit (Morin & Wooten, 1996). Les principaux agents pharmaceutiques utilisés sont les antidépresseurs, les benzodiazépines et les agents non-benzodiazépines (Morin & Wooten, 1996). Parmi ceux-ci, les benzodiazépines sont les plus fréquemment employés (Nowell et al., 1997). Toutefois, les agents non-benzodiazépines (zolpidem, zopiclone et zaleplon) présentent plusieurs avantages et sont de plus en plus favorisés.

L'efficacité des traitements pharmacologiques. Dans la première méta-analyse sur le traitement pharmacologique de l'insomnie, 45 études cliniques comparant une benzodiazépine à un groupe contrôle placebo ou à un autre agent non-benzodiazépine ont été répertoriées (Holbrook et al., 2000). Dans la deuxième, 22 études évaluant l'efficacité des benzodiazépines et du zolpidem ont été recensées (Nowell et al., 1997). Les auteurs rapportent que l'efficacité des benzodiazépines, du zolpidem et du zopiclone sont comparables en ce qui a trait aux paramètres de sommeil évalués (p.ex., latence d'endormissement) mais sont supérieures au placebo. Quant au zaleplon, les quelques études effectuées démontrent qu'il diminue la latence du sommeil et augmente le temps total de sommeil (Dietrich & Farr, 1995; Roth et al., 1995; Walsh et al., 1995). Enfin, il semble qu'une médication utilisée seule, qu'elle soit une benzodiazépine ou un non-benzodiazépine, est efficace à court terme et procure un soulagement rapide de l'insomnie bien qu'il y ait peu d'évidences que les gains thérapeutiques se maintiennent après le retrait de la médication (Holbrook et al., 2000; Nowell et al., 1997; Smith et al., 2002). En outre, étant donné l'accessibilité de ce traitement et la facilité avec laquelle on peut y adhérer, peu d'efforts sont nécessaires pour obtenir ce soulagement rapide (Billiard,

1995). Ainsi, il est recommandé d'utiliser la médication pour dormir sur une courte période et pour de l'insomnie transitoire (Kripke, 2000).

Les limites des traitements pharmacologiques. Plusieurs problèmes sont toutefois reliés aux agents pharmaceutiques et limitent leur utilisation. En effet, les benzodiazépines peuvent, à différents degrés, produire de la dépendance, de l'amnésie antérograde, de la somnolence diurne et des problèmes au niveau de la performance psychomotrice (Greenblatt, 1992; Jonas, Coleman, Sheridan, & Kalinske, 1992; Vogel, 1992; Wadworth & McTavish, 1993). De plus, la majorité des benzodiazépines provoque également de l'insomnie de rebond lors du retrait de la médication (Balter & Uhlenhuth, 1992; Greenblatt, Harmatz, Zinny, & Shader, 1987; Roehrs, Vogel, & Roth, 1990; Schneider-Helmert, 1988). L'insomnie de rebond est définie comme une augmentation des difficultés de sommeil relativement à celles présentes au niveau de base (Roehrs et al., 1990). En outre, les benzodiazépines affectent l'architecture du sommeil en diminuant les ondes lentes du sommeil (stades 3 et 4) et le sommeil REM (Goa & Heel, 1986; Pecknold, Wilson, & Le Morvan, 1990). Bien que les agents non-benzodiazépines agissent sur les mêmes récepteurs que les benzodiazépines, ils sont toutefois reconnus pour moins affecter l'architecture du sommeil que les benzodiazépines (Declerck, Ruwe, O'Hanlon, & Wauquier, 1992; Monti, 1989; Scharf, Roth, Vogel, & Walsh, 1994). Toutefois, en raison de la tolérance, les agents pharmacologiques sont reconnus pour perdre de leur efficacité à long terme (McClusky, Milby, Switzer, William, & Wooten, 1991; Morin, Colecchi, Stone, Sood, & Brink, 1999).

Les traitements psychologiques

Les traitements psychologiques, quant à eux, sont basés sur les différents mécanismes étiologiques postulat que le sommeil est un comportement qui peut être influencé par conditionnement et par apprentissages sociaux (Morin, 1993). Leur but est donc de réassocier le lit et l'environnement de la chambre à coucher avec le sommeil et de modifier les croyances dysfonctionnelles concernant le sommeil qui maintiennent le problème d'insomnie. Les principaux traitements psychologiques, principalement de type béhavioral-cognitif (TBC) sont:

(a) le contrôle par le stimulus qui comprend une série d'instructions données afin de diminuer les activités incompatibles avec le sommeil et régulariser l'horaire de sommeil (Bootzin, Epstein, & Wood, 1991) ; (b) la restriction du temps passé au lit au nombre d'heures de sommeil réel (Spielman, Saskin, & Thorpy, 1987); (c) la thérapie cognitive qui vise à modifier les croyances dysfonctionnelles face à l'insomnie (Morin, 1993); (d) la relaxation qui a pour but de diminuer l'activation somatique et cognitive (p.ex., relaxation progressive) (Jacobson, 1977); (e) l'intention paradoxale qui consiste à persuader la personne d'essayer de rester éveillée afin de diminuer l'anxiété associée à vouloir dormir à tout prix (Espie & Lindsay, 1985); et (f) l'éducation à l'hygiène du sommeil comprenant l'enseignement de bonnes habitudes de sommeil (p.ex., éviter la consommation de café) (Hauri, 1982).

L'efficacité des traitements psychologiques. Les deux méta-analyses portant sur l'efficacité des traitements psychologiques ont relevé des études empiriques qui ont utilisé des protocoles expérimentaux ou quasi expérimentaux avec groupe contrôle (Morin et al., 1994; Murtagh & Greenwood, 1995). La première méta-analyse a répertorié 59 études réalisées entre 1974 et 1993. La seconde a répertorié 66 études pour la période s'échelonnant de 1973 à 1993 mais elle comprend également des études non publiées. Les grandeurs d'effet de chaque traitement psychologique sur les variables de sommeil ont été calculées. Les résultats indiquent d'abord que les traitements de contrôle par le stimulus et de restriction du temps au lit seraient les plus efficaces des traitements psychologiques. Ceux-ci produisent une réduction significative de la latence du sommeil et du temps d'éveil durant la nuit à la fin du traitement. Les traitements psychologiques contribuent donc à améliorer la continuité du sommeil. Enfin, les résultats des deux méta-analyses indiquent également que ces changements au niveau de la continuité du sommeil se maintiennent lors des suivis et qu'à ce moment, le temps total de sommeil a aussi augmenté. L'efficacité des traitements psychologiques de l'insomnie a été confirmée par des études empiriques récentes (e.g., Espie, Inglis, Tessier, & Harvey, 2001; Morin, Colecchi et al., 1999; Perlis, Sharpe, Smith, Greenblatt, & Giles, 2001).

Les limites des traitements psychologiques. Malgré l'efficacité à court et à plus long terme des traitements psychologiques, l'amélioration du sommeil découlant des psychothérapies

se manifeste plus lentement que pour la pharmacothérapie (McClusky et al., 1991). De plus, il apparaît qu'entre 20% et 25% des personnes souffrant d'insomnie n'obtiennent pas les gains thérapeutiques espérés (Morin et al., 1994; Murtagh & Greenwood, 1995) ou rechutent à la fin du traitement. En outre, selon Smith et al. (2002), le traitement psychologique est plus difficile d'accès et plus coûteux que le traitement pharmacologique. En effet, le médecin généraliste est le professionnel de la santé le plus facilement accessible pour la population générale. Par conséquent, le premier traitement offert aux personnes souffrant d'insomnie est souvent médical et rarement psychologique. Par ailleurs, les coûts directs reliés aux consultations médicales de même que les médicaments prescrits sont couverts par la majorité des assurances santé alors qu'il en va autrement pour les soins psychologiques.

Les traitements combinés

Le traitement combiné est l'association concurrente d'un agent pharmaceutique avec un ou des traitements psychologiques. L'objectif du traitement combiné est de maximiser les effets thérapeutiques des traitements pharmacologiques et psychologiques et de tirer profit de leurs avantages respectifs et complémentaires.

L'efficacité des traitements combinés. Quatre études ont vérifié l'efficacité du traitement combiné (Hauri, 1997; Rosen, Lewin, Goldberg, & Woolfolk, 2000; Milby et al., 1993; Morin, Colecchi et al., 1999). Les résultats de ces quatre études démontrent que l'amélioration du sommeil survient plus rapidement avec un traitement combiné qu'avec un traitement béhavioral-cognitif (TBC) employé seul. De plus, ces quatre études démontrent que l'approche combinée est efficace à court terme (de 4 à 8 semaines) mais qu'à plus long terme, les résultats sont variables. En effet, malgré cette amélioration à la fin du traitement, les résultats démontrent que le temps total d'éveil, l'efficacité du sommeil ainsi que la continuité du sommeil se sont détériorés lors des suivis sans toutefois retourner au niveau de sévérité initiale. Par conséquent, il semble que l'approche combinée ne produit pas à long terme d'aussi bons résultats que le TBC employé seul.

Les limites des traitements combinés. Plusieurs facteurs peuvent expliquer la variabilité dans les résultats à long terme du traitement combiné. Selon plusieurs auteurs, maximiser les gains thérapeutiques en combinant une médication et un traitement psychologique dépend de la contribution relative attribuée à la médication et au développement d'habiletés (Bandura, 1997; Edinger & Wolhgemuth, 1999; Kendall & Lipman, 1991; Morin & Wooten, 1996; Murtagh & Greenwood, 1995). Selon Bandura (1997), si la médication est utilisée dans le but de développer les habiletés, la perception d'efficacité personnelle de l'individu augmente et l'efficacité du traitement combiné ne devrait pas être affectée. Par contre, si l'amélioration est attribuée à la médication plutôt qu'à la contribution du développement d'habiletés, la perception d'efficacité personnelle demeurera faible et l'efficacité du traitement combiné sera diminuée. Cette théorie a été appliquée à l'insomnie (Edinger & Wolhgemuth, 1999; Kendall & Lipman, 1991; Morin & Wooten, 1996). L'efficacité à long terme du traitement combiné serait plus faible car les participants, consommant une médication, attribueraient l'amélioration de leur sommeil à la médication plutôt qu'à eux-mêmes. Ils ne développeraient pas leurs habiletés à gérer leurs habitudes de sommeil et posséderaient par le fait même, une faible perception d'efficacité personnelle de gestion de leur sommeil. Ainsi, lors du retrait de la médication, les participants ne percevraient pas qu'ils ont développé la capacité d'agir sur leur sommeil et les mauvaises habitudes de sommeil reviendraient pour laisser place à l'insomnie (Edinger & Wolhgemuth, 1999).

Conclure à la supériorité d'une approche combinée est prématuré. En effet, étant donné la disparité méthodologique des quatre études sur le traitement combiné, leur comparaison ainsi que la généralisation des résultats deviennent difficiles. D'abord, la durée du traitement varie de trois à huit semaines et les suivis de cinq semaines (Milby et al., 1993) à 24 mois (Morin, Colechi et al. 1999). Ensuite, la nature des traitements utilisés varie beaucoup d'une étude à l'autre. Ainsi, trois études évaluent la relaxation comme composante comportementale (Hauri, 1997; Milby et al., 1993; Rosen et al., 2000), deux études évaluent le contrôle par le stimulus (Milby et al., 1993; Morin, Colecchi, et al., 1999) et une seule teste un programme de TBC incluant la restriction du temps au lit, le contrôle par le stimulus et les interventions cognitives (Morin, Colecchi, et al., 1999). En outre, tel que recommandé par Kazdin (1998), lorsque

l'objectif de l'étude est d'évaluer l'efficacité d'un nouveau traitement, il importe de le comparer au traitement standard. En considérant que le traitement standard pour l'insomnie est la médication ou le TBC employé seul, il devient nécessaire de contraster l'efficacité du traitement combiné avec au moins l'un des traitements standards de l'insomnie. Parmi les quatre études de groupe, le traitement combiné a été comparé seulement une fois à la médication seule (Morin, Colecchi, et al., 1999), une fois à la relaxation et l'hygiène du sommeil (Hauri, 1997) et une fois au TBC seul (Morin, Colecchi, et al., 1999).

Il semble donc que l'objectif de maximiser la réponse thérapeutique en tirant profit des avantages complémentaires des deux traitements et en produisant un effet plus durable que le TBC employé seul n'a pas encore été évalué de façon optimale. L'objectif n'est donc que partiellement atteint et il apparaît difficile de statuer sur l'efficacité à long terme du traitement combiné.

Les traitements séquentiels

En s'appuyant sur les résultats des études de traitements combinés ainsi que sur les avantages et limites complémentaires de chacun des traitements, il a été suggéré de combiner les traitements pharmacologiques et psychologiques de façon séquentielle (Edinger & Wolhgemuth, 1999; Hauri, 1997; Lichstein & Johnson, 1993; Morin & Wooten, 1996). Selon ces auteurs, l'introduction séquentielle de traitements devrait favoriser à la fois une amélioration rapide du sommeil et un maintien à long terme de cette amélioration.

L'efficacité des traitements séquentiels. L'hypothèse de l'efficacité du traitement séquentiel a été émise et évaluée pour des problèmes cliniques autres que l'insomnie, tels que le trouble obsessif-compulsif (Balkom et al., 1998; Keijser, Hoogduin, & Schaap, 1995), la boulimie (Agras et al., 1992; Treasure et al., 1999), l'obésité (Craighead, 1984), l'hypochondrie (Visser & Bouman, 1992), le trouble panique (Mavissakalian, 1990) ou la dépression (Mercier, Stewart, & Quitkin, 1992). Parmi ces études, quatre utilisent un protocole expérimental de groupe comparant l'efficacité de traitements séquentiels à un traitement utilisé seul (Agras et al.,

1992; Balkom et al., 1998; Treasure et al., 1999; Craighead, 1984). Deux autres études utilisent des protocoles expérimentaux contrebalancés (Keijsers et al., 1995; Visser & Bouman, 1992), alors qu'une autre utilise un protocole à cas unique (Mavissakalian, 1990). Les séquences de traitements étudiées semblent indiquer que l'utilisation d'une médication seule au début du traitement ralentit l'effet du traitement psychologique. Selon ces auteurs, le ralentissement pourrait être dû au fait que la médication soit retirée avant que le TBC n'agisse pleinement, ce qui empêcherait d'attribuer l'amélioration de l'état psychologique au développement d'habiletés personnelles. Parmi les études sur le traitement séquentiel, deux démontrent que le ralentissement observé peut être minimisé à l'aide d'une séquence se divisant en deux parties : la première partie du traitement comprend une médication et un TBC suivi de la deuxième partie, qui se caractérise par un sevrage graduel et le maintien de l'utilisation du TBC se poursuivant à la fin du sevrage (Agras et al., 1992; Craighead, 1984).

Les limites des traitements séquentiels. L'efficacité du traitement séquentiel de l'insomnie n'a pas encore été étudiée. Quelques suggestions concernant les séquences de traitement ont cependant été émises. Premièrement, il est suggéré de débuter le traitement de l'insomnie par une médication afin de produire un soulagement rapide (Morin, 1993; Morin & Wooten, 1996), puis de poursuivre avec un TBC en retirant graduellement la médication. D'autres auteurs proposent de débuter par un TBC et de poursuivre avec une médication si le TBC n'est pas efficace (Wagner, Wagner, & Hening, 1998). Ces derniers auteurs basent leur suggestion sur la présence des effets secondaires des benzodiazépines ainsi que sur la méconnaissance de l'impact à long terme des nouveaux agents non-benzodiazépines. Le traitement séquentiel de l'insomnie chronique apparaît donc comme une option prometteuse utilisant à meilleur escient les avantages et limites des traitements pharmacologiques et psychologiques.

La mesure de l'insomnie

L'intérêt suscité par le développement de traitements efficaces pour l'insomnie a amené les chercheurs à s'intéresser parallèlement aux instruments de mesure de l'insomnie. La compréhension du traitement et de la nature de l'insomnie requière l'évaluation de plusieurs composantes telles que l'histoire personnel du sommeil, le fonctionnement quotidien, les cycles veille/sommeil et également le sommeil lui-même (Sateia, Dodhramji, Hauri, & Morin, 2000). Plusieurs instruments de mesure objectif et subjectif ont été développés pour évaluer ces différentes composantes de l'insomnie. À ce jour, la polysomnographie (PSG) est reconnue comme la mesure standard objective du sommeil et l'agenda du sommeil, comme la mesure subjective standard (Sateia et al., 2000). La PSG procure une description détaillée et précise du temps d'éveil et de sommeil ainsi que de l'architecture du sommeil. Toutefois, cet outil de mesure est coûteux et nécessite l'évaluation du sommeil dans l'environnement artificiel qu'est le laboratoire. À l'inverse, l'utilisation de l'agenda du sommeil est recommandée pour évaluer le sommeil dans l'environnement naturel de l'individu et peut être utilisé sur une plus longue période de temps (Sateia et al., 2000). L'agenda du sommeil possède l'avantage de mesurer la variabilité internuit typique au problème d'insomnie. Toutefois, l'agenda est reconnu comme étant moins précis que la PSG et dépendant de la perception de l'individu qui souffre d'insomnie. En effet, il a été démontré que plusieurs personnes souffrant d'insomnie sur ou sous-estiment leurs périodes d'éveil et de sommeil (Edinger & Fins, 1995). Par conséquent, il apparaît essentiel d'utiliser des instruments de mesure objectifs qui seront plus accessibles et économiques que la PSG et qui évalueront le sommeil dans un milieu naturel sur une plus longue période de temps.

L'actigraphie a été proposée comme mesure objective et ambulatoire du sommeil (Sadeh, Hauri, Kripke, & Lavie, 1995). L'actigraphe est un petit appareil semblable à une montre qui se porte sur le poignet durant la nuit. L'activité motrice est enregistrée de façon continue. La présence de mouvement est interprétée comme du temps d'éveil et l'absence de mouvement comme du temps de sommeil. Un estimé des différents paramètres de sommeil est calculé à l'aide d'algorithmes préétablis. Bien que quelques études se soient intéressées à la

précision de l'actigraphie (e.g., Jean-Louis et al., 1997; Hauri & Wisbey, 1992), une seule a comparé, pour la même nuit, les données de l'actigraphe, de la PSG et de l'agenda (Verbeek, Arends, Declerk, & Beecher, 1994). Par ailleurs, seulement trois études ont évalué la sensibilité des données de l'actigraphie au traitement de l'insomnie (Brooks et al., 1993; Friedman et al., 2000; Schmidt-Nowara, Beck, & Jessop, 1992). Ainsi, avant d'utiliser l'actigraphe comme outil de mesure ambulatoire dans un milieu clinique, il apparaît nécessaire d'évaluer sa précision et sa sensibilité lors d'un traitement pour l'insomnie.

Synthèse de la problématique

L'insomnie chronique est un trouble prévalent ayant de multiples conséquences tant au niveau de la qualité de vie et de la santé des individus que de l'économie. Les différentes études sur le traitement de l'insomnie ont permis de démontrer les points suivants: (a) la pharmacothérapie et le TBC employés seuls ou de façon combinée sont efficaces à court terme; (b) la médication seule ou combinée au TBC procure un soulagement plus rapide de l'insomnie que le TBC seul; (c) les différentes médications possèdent à des degrés divers des effets secondaires de même que certains risques de tolérance et de dépendance; (d) le TBC seul a des effets à long terme plus durables que la pharmacothérapie; et (e) le traitement combiné a des effets à long terme variables.

En s'appuyant sur ces résultats, il semble qu'une séquence de traitements pharmacologique et psychologique permettrait de procurer un soulagement rapide de la même façon qu'une médication seule ou un traitement combiné. Elle pourrait également favoriser le maintien des gains à plus long terme, comme le permet le TBC. En outre, le traitement séquentiel à court terme permettrait d'éviter les inconvénients (p.ex., problèmes de dépendance) liés à la consommation de médication. Les études qui ont évalué cette question à partir d'autres problématiques cliniques reconnaissent qu'il est possible d'optimiser la réponse thérapeutique avec un traitement séquentiel comprenant une médication et un TBC. Par contre, la séquence optimale pour maximiser les gains thérapeutiques à court et à plus long terme n'est pas encore déterminée. Parallèlement, compte tenu des outils actuellement disponibles pour mesurer

l'insomnie, la recherche sur le traitement de l'insomnie requiert le développement d'outils objectifs de mesure du sommeil. Développer un instrument de mesure moins coûteux et ambulatoire permettrait d'améliorer l'accessibilité non seulement au traitement de l'insomnie mais également aux outils de mesure essentiels.

Objectifs de la thèse

Cette thèse de doctorat vise donc à étudier l'efficacité d'un traitement séquentiel pour l'insomnie chronique. Le corps de la thèse comprend trois chapitres. Le premier explore différentes séquences de traitements pharmacologiques et psychologiques afin d'établir le protocole et les séquences qui nécessitent un plus grand intérêt. Le deuxième chapitre est basé sur les résultats du premier. Le protocole ainsi que les séquences de traitement ont été légèrement modifiés afin de surmonter les difficultés rencontrées dans l'étude exploratoire et d'établir les meilleurs séquences de traitement possibles. Finalement, le troisième et dernier chapitre s'intéresse aux techniques d'évaluation de l'insomnie. Cette étude faite en parallèle à la deuxième, permet d'une part de vérifier la précision de l'actigraphie par rapport à la PSG et à l'agenda du sommeil et, d'autre part, de vérifier la sensibilité de l'actigraphie au traitement de l'insomnie.

CHAPITRE 1

LE TRAITEMENT SÉQUENTIEL DE L'INSOMNIE CHRONIQUE: UNE ÉTUDE PILOTE

Résumé

Objectif: Explorer l'efficacité d'un traitement séquentiel pour l'insomnie comprenant une médication et un traitement béhavioral-cognitif (TBC).

Méthode: Six participants souffrant d'insomnie chronique primaire ont été enrôlés dans un protocole à cas unique à niveau de base multiple. Les participants ont été assignés aléatoirement à trois niveaux de base différents (3, 4 et 5 semaines). Les séquences de traitement élaborées d'une durée de 10 semaines sont : (a) une combinaison concurrente de la médication et du TBC; le retrait graduel de la médication s'effectue après la neuvième semaine de traitement; (b) la médication pendant les cinq premières semaines de traitement suivie de l'introduction du TBC à la semaine 4; le retrait graduel de la médication se fait après la cinquième semaine, de telle sorte que les deux traitements se chevauchent durant les semaines 4 et 5 (Médication → Combiné → TBC); et (c) la médication utilisée seule pendant les cinq premières semaines de traitement suivi de l'introduction du TBC seul pour les cinq semaines subséquentes; le retrait graduel de la médication s'effectue dès la sixième semaine (Médication → TBC). Un suivi est effectué trois mois après la fin du traitement.

Résultats: Chaque séquence produit une amélioration significative de l'efficacité et de la continuité du sommeil. Toutefois, ces améliorations surviennent à différents moments durant l'intervention. Les participants dans le traitement combiné ont une amélioration significative de l'efficacité du sommeil (ES) et du temps total d'éveil (TTE) durant la première phase de traitement. Ces améliorations sont maintenues au posttraitement. Les participants dans la séquence où les traitements se chevauchent (Médication → Combiné → TBC) présentent les mêmes améliorations. Les participants dans la séquence Médication → TBC ne présentent pas d'amélioration significative avant l'introduction du TBC dans la deuxième phase de traitement. Des cinq participants ayant répondu au suivi de trois mois, un s'est de nouveau amélioré, trois maintiennent leurs gains et un retourne au niveau de base.

Conclusion: Les résultats suggèrent que le traitement séquentiel est efficace pour l'insomnie chronique primaire.

Running head: SEQUENTIAL TREATMENT FOR CHRONIC INSOMNIA

Sequential Treatment for Chronic Insomnia: A Pilot Study

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Abstract

Objective: To explore the efficacy of sequential treatment involving medication and cognitive behavioural treatment (CBT) for insomnia.

Methods: Six participants with primary chronic insomnia took part in a multiple baseline across subjects design and were randomly assigned to one of three baseline durations (3, 4, and 5 weeks). They received one of the following treatment sequences (10 weeks duration): (a) concurrent combination of medication (zopiclone) and CBT for the 10-week treatment duration (Combined), with gradual medication withdrawal after the ninth week; (b) medication for the first five weeks, with introduction of CBT at week 4 and gradual medication withdrawal after the fifth week resulting in treatment overlap during weeks 4 and 5 (Overlapping); and (c) medication alone for the first five weeks followed by gradual medication withdrawal and CBT alone for an additional five weeks (Medication → CBT). There was a follow-up assessment three months after the end of treatment.

Results: Each sequence led to significant improvement of sleep efficiency and continuity, but the timing of these improvements occurred at different times during the intervention. Participants in the combined sequence had significant improvement in Sleep Efficiency and Total Wake Time during the first phase of treatment and those improvements were maintained at posttreatment. Participants in the overlapping sequence had the same improvements from baseline to the first phase of treatment. Participants in the Medication → CBT sequence did not have any significant improvement before the introduction of CBT during the second phase of treatment. Three participants maintained their gains at the 3-month follow-up, one had additional improvement, one was unavailable for this assessment, and one returned to baseline level.

Conclusion: These results suggest that a sequential treatment is effective for chronic insomnia.

Sequential Treatment for Chronic Insomnia: A Pilot Study

Insomnia is a widespread complaint reported to health professionals and affecting 6 to 12% of the general population on a chronic basis (Ancoli-Israël & Roth, 1999; Ohayon, 1999). The efficacy of drug and behavioural therapies for insomnia has been well documented over the years (e.g., Morin, Culbert, & Schwartz, 1994; Murtagh & Greenwood, 1995; Nowell et al., 1997). Drug therapies are commonly used for insomnia because they are easily accessible and provide rapid relief from insomnia symptoms (Kupfer & Reynolds, 1997; Morin & Wooten, 1996). However, given the potential side effects and problems upon withdrawal (e.g., rebound insomnia), medication is recommended for transient and short term insomnia only (Kripke, 2000). Contrary to drug therapy, cognitive behavioural treatment (CBT) is not associated with such side effects or withdrawal symptoms. CBT is well accepted by patients, promotes good sleep habits, and there is evidence that it is more effective in the long run than medication alone (Edinger & Wohlgemuth, 1999; Espie, Inglis, & Harvey, 2001; Stepanski & Wyatt, 2000). Despite its proven efficacy, about 20 to 30% of insomnia patients do not benefit from CBT (Espie et al., 2001; Morin, Hauri, et al., 1999). Because of the complementary advantages of drug therapy and CBT, their combination seems to be a promising avenue to maximise therapeutic gains.

Four treatment studies have evaluated the effectiveness of the concurrent combination of CBT with benzodiazepines (Hauri, 1997; Milby et al., 1993; Morin, Colecchi, Stone, Sood, & Brink, 1999; Rosen, Lewin, Goldberg, & Woolfolk, 2000). The results of these four studies indicate that medication alone produces quicker results than CBT alone in the first week of treatment. Both treatments, alone or combined, are equally effective in the short-term interval (4-8 weeks). Long-term outcomes are fairly clear for the single therapy modality but are more equivocal for a combined approach. For instance, the initial benefits of drug therapy alone are quickly lost upon discontinuation of medication, while CBT alone produces sustained benefits over time. However, a combined approach does not yield better outcome in the long-term than CBT alone, at least for some patients. Surprisingly, even though CBT usually involves an initial period of mild sleep deprivation, it produces a better long-term outcome than a

combined intervention. Therefore, although a combined treatment approach is promising, it does not seem to lead to the degree of improvement expected.

The variability in the long-term results of these studies could be due to the patients' attribution of their improvement to medication. Indeed, it has been suggested that this attribution effect could undermine the efficacy of behavioural strategies in a combined approach (Morin & Wooten, 1996; Stepanski & Wyatt, 2000). In such cases, individuals would not develop appropriate self-management skills during treatment and when the medication is withdrawn, patients are more likely to relapse (Edinger & Wolhgemuth, 1999; Morin, Colecchi, et al., 1999).

Conclusions about the superiority of a combined approach are still premature, mainly because of methodological differences across the four studies. For example, treatment duration varied from three to eight weeks and follow-up assessment from five weeks (Milby et al., 1993) to 24 months (Morin, Colecchi, et al. 1999). The nature of the treatment options tested was also different across studies. For example, three studies tested relaxation as the behavioural component (Hauri, 1997; Milby et al., 1993; Rosen et al., 2000), two tested stimulus control (Milby et al., 1993; Morin, Colecchi, et al., 1999), and one tested a multi-component CBT program including stimulus control, sleep restriction, and cognitive interventions (Morin, Colecchi, et al. 1999). Moreover, as stated by Kazdin (1998), to investigate whether a new treatment is effective, it should be compared with the standard treatment. If we consider that medication alone or CBT alone are the standard approaches to treat insomnia, then the efficacy of combined treatments should be contrasted with that of these standard treatments. In the four group treatment studies, combined treatment was compared only once to medication alone (Morin, Colecchi, et al., 1999) and only twice to CBT alone (Hauri, 1997; Morin, Colecchi, et al., 1999). Therefore, additional studies are needed to draw conclusions about the relative efficacy of combined treatments.

Finally, beyond these considerations, it is also possible that the variability observed in the long-term outcome was due to the concurrent combination of treatments. The fact that

both interventions were administered simultaneously might have undermined the therapeutic response, possibly through the attribution of therapeutic gains to medication, or by any other mechanism particular to this combination. Therefore, it seems essential to elaborate other ways of combining medication and CBT in order to benefit fully from the respective advantages of both treatments. A sequential introduction of both treatments could produce an outcome superior to that of a given treatment used alone or in a concurrent combination. Indeed, the initial introduction of medication could first allow rapid relief of sleep difficulties. Subsequently, behavioural procedures could be implemented to consolidate those initial gains. Furthermore, such a sequential treatment would more realistically correspond to the present health care system situation, in which medication is easier to access as a first treatment option. This study explores the efficacy of sequential treatment for insomnia and attempts to determine whether a sequential introduction of medication and behavioural interventions is effective and, if so, at what point in time symptom relief appears?

Method

Participants

Participants were recruited through newspaper advertisements or physician referrals. Inclusion criteria were: (a) being between 30- and 50-years old; (b) reporting insomnia, defined as sleep onset latency, wake after sleep onset, or early morning awakening equal or superior to 30 minutes at least 3 nights a week for the past 6 months; (c) reporting significant distress or daytime impairments as evaluated by the *Insomnia Severity Index* (2 or higher on a 1 [*not at all*] to 4 [*very much*] Likert scale); and (d) cessation, at least one month prior to treatment, of any sleep or other psychotropic medication that can interfere with sleep. Exclusion criteria were: (a) presence of another sleep disorder such as sleep apnea or circadian rhythm disorder; (b) evidence that insomnia is related to a medical condition; (c) presence of major depression, anxiety disorder, alcohol/substance abuse or any other severe psychopathology as diagnosed with the Structured Clinical Interview for DSM-IV (SCID-IV; First, Spitzer, Gibbon,

& Williams, 1997); (d) currently involved in psychotherapy; and (e) regular use of a medication interfering with sleep. These criteria are consistent with the *International Classification of Sleep Disorders* (American Sleep Disorder Association [ASDA], 1990) and the DSM-IV (American Psychiatric Association [APA], 1994).

Fifty-two prospective participants underwent a telephone screening. Forty-five individuals were then excluded for the following reasons: age (n = 14); medication interfering with sleep (n = 8); mild insomnia (n = 4); refusal to use medication (n = 4); insomnia resulting from another medical condition, psychological disorder or night shift (n = 8); not interested anymore by the study (n = 7). Subsequent assessments included a semi-structured sleep history interview, a SCID-IV evaluation (First et al., 1997), and a physical evaluation. One additional participant was excluded because of generalised anxiety disorder. Note that participant 4 had to work night shifts during the last week of treatment.

The final sample included 6 participants (three men and three women) with a mean age of 47.5 years (*SD* = 7.92, range 34 - 50). Five participants were married and one was divorced. The average education level was 16.7 years (*SD* = 1.63). Four participants were working and two were unemployed. The average insomnia duration was 15.5 years (*SD* = 9.2) and the mean age of insomnia onset was 30.7 years (*SD* = 8.96). Five participants presented sleep-maintenance insomnia only and one mixed insomnia. Of the 6 participants, one had previously tried zopiclone (a non benzodiazepine hypnotic agent), four had tried a benzodiazepine and one had tried both for sleep. Three of them also had tried an over-the-counter pill. Participants were free of any sleep medication before entering in the study.

Design and procedures

A multiple baseline across subjects design was used (Barlow & Hersen 1984; Ladouceur & Bégin, 1993). Treatment duration was 10 weeks. Participants were randomised to a baseline duration of either three, four or five weeks in order to control for historical events and participants' maturation. For each baseline, one of the following treatment sequences was applied: (a) concurrent combination of medication and CBT for the 10-week treatment

duration (Combined), with gradual medication withdrawal after the ninth week; (b) medication for the first five weeks with introduction of CBT at week 4 and gradual medication withdrawal after the fifth week, resulting in a treatment overlap during weeks 4 and 5 (Overlapping: Medication → Combined → CBT); and (c) medication alone for the first five weeks followed by gradual medication withdrawal after the fifth week and CBT alone for an additional five weeks (Medication → CBT). The introduction of CBT varied across treatment sequences to control for a possible order effect. Another assessment was conducted two weeks following treatment (posttreatment). Finally, a follow-up was conducted three months after posttreatment. Participants completed all measures four times during the experimentation: at baseline, midway during treatment, at posttreatment, and at the 3-month follow-up assessment period.

Measures

Initial screening and evaluation. The initial screening included a 20-minute phone interview administered to determine participants' eligibility. A subsequent multi-measure pre-treatment evaluation was composed of a semi-structured sleep history interview to diagnose insomnia, the SCID-IV (First et al., 1997) to evaluate the presence of psychological disorders, and a physical examination.

Sleep measures. Participants monitored their sleep/wake patterns using daily sleep diaries which were completed from baseline through the 10-week treatment period, two weeks after the end of treatment, and at the 3-month follow-up. From these diaries, an estimate was computed for total wake time (TWT), total sleep time (TST), and sleep efficiency (SE: ratio of total sleep time divided by time in bed). Participants also monitored type and dosage of medication used.

Actigraphy. Participants wore an actigraph during the first two weeks of treatment and for another two consecutive weeks at posttreatment. The actigraph is a small, watch-like device worn all night on the wrist of the nondominant hand. Motor activity is recorded on an

ongoing basis. The presence of movement is interpreted as wake time and absence of movement is interpreted as sleep time. The actigraph used in this study, is from the IM System Inc. Company. Data were recovered and scored (TST, TWT, and SE) with the software of IM system Inc. Company. Contrary to polysomnography, no adaptation night is necessary (Jean-Louis et al., 1997; Sadeh, Hauri, Kripke, & Lavie, 1995). Although the use of actigraphy is still controversial, there is an increasing interest in its validity as a movement-based predictor of wakefulness and sleep (Sadeh et al., 1995; Verbeek, Klip, & Declerck, 2001). Moreover, actigraphy is a useful non-intrusive tool for assessing sleep in a natural environment and produces reliable information about patients' adherence to behavioural procedures (Sadeh et al., 1995). Note that one actigraph was damaged by water during treatment.

Psychological assessment. Participants completed self-report inventories before, at the middle, after the treatment, and at the 3-month follow-up. First, the *Insomnia Severity Index* (*ISI*; Morin, 1993) includes seven questions about participants' sleep difficulties. The seven items evaluate the severity of sleep-onset, sleep maintenance, early morning awakening problems, satisfaction with current sleep pattern, interference with daily functioning, noticeably of impairment attributed to the sleep problem, and level of distress caused by the sleep problem. The ISI has adequate psychometric properties and has been shown to be sensitive to changes in clinical trials of insomnia (Bastien, Vallières, & Morin, 2001). The *Dysfunctional Beliefs and Attitudes about Sleep scale* (*DBAS*; Morin, 1993) includes 30 items assessing sleep beliefs about the perceived causes and consequences of insomnia and about sleep requirements expectations. The DBAS has adequate psychometric properties (Blais, Gendron, Mimeault, & Morin, 1997; Espie, Inglis, Harvey, & Tessier, 2000; Morin, Stone, Trinkle, & Mercer, 1993). The *Attributional Questionnaire* (*AQ*) is an 8-item scale based on Weiner's motivation and performance attribution theory (Weiner, 1985). Participants rated on a 0 *(not at all)* to 100 *(extremely)* scale if changes in their sleep were due to the physician, the psychologist, the medication, the behavioural procedures, the modification in beliefs and attitudes, self-effort or luck. The eighth item assesses perceived self-efficacy. The AQ was completed at posttreatment and at the 3-month follow-up. Psychometric properties of this

questionnaire have not been tested yet. The *Beck Depression and Anxiety Inventories* (*BDI*; Beck, Epstein, Brown, & Steer, 1988; *BAI:* Beck, Steer, & Garbin, 1988) were used to monitor anxiety and depressive symptoms throughout the treatment.

Additional measures. The participants' perception of treatment acceptability was assessed using the *Treatment Acceptability Scale* (Morin, Gaulier, Barry, & Kowatch, 1992) adapted for sequential treatment. Participants completed this questionnaire at baseline, at the middle and after treatment, as well as at the 3-month follow-up.

Compliance measures. Adherence to treatment protocol was evaluated with sleep diaries, actigraphy, and significant other's independent rating of medication intake. Based on their sleep diaries, a weekly percentage of adherence to each behavioural procedure and medication intake was computed for each participant. Then the average of this percentage of adherence was also computed. Actigraph measures of time in bed were used to estimate the adherence to CBT procedures (i.e., sleep restriction).

Treatment

The CBT administered in this study is outlined in a treatment manual (Morin, 1993) and includes five 50-minute individual therapy sessions. For the Combined sequence, CBT sessions were administered every two weeks. For the Overlapping sequence, CBT sessions were delivered at weeks 4, 5, 6, 8, and 10. Finally, for the Medication → CBT sequence, CBT sessions were administered weekly during the last five weeks of treatment, with no overlap between treatments. The behavioural component included stimulus control instructions and sleep restriction procedures. Participants were instructed to go to bed only when sleepy at night, use the bed only for sleep and sex, get out of the bed whenever unable to fall asleep or return to sleep, maintain a regular arising time in the morning and avoid daytime napping. The sleep restriction procedure consisted of curtailing the time spent in bed to the actual sleep time. To begin, a *sleep window* was determined using the average of total sleep time reported by participants in their sleep diaries. The sleep window was gradually increased, contingent upon reaching a sleep efficiency of 85 % or more. The cognitive component was aimed at

altering dysfunctional beliefs and attitudes about sleep and the impact of sleep loss on daytime functioning. Finally, the educational component included didactic presentations on sleep hygiene and a review of basic facts about changes in sleep patterns over the life span. Strategies to prevent relapse were reviewed during the last treatment session.

The medication treatment included 20-minute individual consultation sessions with a physician. These consultation sessions were designed to monitor insomnia symptoms, treatment response and side effects, and to adjust the medication dosage and frequency of use. No specific behavioural recommendations for improving sleep were given during those sessions. For the Combined sequence, there were five consultation sessions occurring every two weeks. For the Overlapping sequence and the Medication → CBT sequence, the five consultation sessions occurred weekly during the first phase of treatment. One additional session with the physician was administered during medication withdrawal. The medication used was zopiclone (Imovane), a non-benzodiazepine hypnotic agent which has a rapid action and a relatively short half life (4 - 6 hours). Its efficacy for insomnia is comparable to benzodiazepines, but it is associated with few secondary effects and has a low potential for rebound insomnia (Wadworth & McTavish, 1993; Wagner & Wagner, 2000). The dosage used varied from 3.75 mg to 7.5 mg and was determined according to participant response to medication. The medication was prescribed for nightly use and was taken 30 minutes before bedtime. At the end of treatment, the dosage was gradually reduced. The time necessary to complete withdrawal depended on the initial dosage and the presence of withdrawal symptoms. It varied between one and two weeks.

Therapist

The CBT sessions were led by the first author a licensed clinical psychologist who had previously treated a minimum of five clinical participants using this protocol before beginning the study. A general medical practitioner with 20 years of experience in treating patients with sleep disorders performed initial physical examinations, led the medication treatment sessions, and supervised the medication withdrawal. A manual was also used for the medication

session. This manual outlined the structure and topics that had to be covered in each session and information that was not allowed to be discussed (e.g., behavioural procedures). Sessions were recorded and randomly reviewed by the study coordinator to ensure adherence to protocol. The initial psychological screening was conducted by two independent licensed clinical psychologists.

Data analysis plan

Data obtained from sleep diaries were divided according to each treatment phase such as baseline (three to five weeks), first five weeks of treatment, second five weeks of treatment, posttreatment (two weeks), and follow-up (two weeks). Thus, five series of sleep diary data were available for each sleep variable for each participant. These data were used as repeated measures. Given that each measure is dependent on the previous (serial dependency), such data are usually highly correlated. This autocorrelation can lead to a misinterpretation of the results and to an increased probability of type I error (DeProspero & Cohen, 1979; Jones, Weinrott, & Vaught, 1978). In addition, the design used and the small number of participants did not allow for the typical comparison of means without inflating the type I error as well. Sleep diary data for each participant were thus analysed separately with autoregressive analysis [see Gorsuch, (1983) for more details on autoregressive analysis]. This type of times series analysis has recently been recommended for use with single case study (Rivard & Bouchard, 1998; Ellis, 1999; Heath, Kelly, & Longstaff, 2000). The autoregressive analysis produces an estimation of the autoregressive coefficient and controls for serial dependency. The first step of this analysis is to extract the autocorrelation from the data set to get residual data due to the experimental effect (treatment) or to error. Then, each small series of residual data (e.g., baseline, first five weeks of treatment) is modelled according to a standard regression. Predictors of change tested between paired series of residual data are change in level, time effect, and slopes. Slopes can be a fraction ($1/x$), a linear ($ax + b$), a quadratic ($ax^2 + bx + c$), a logarithmic ($\log_{(x)}$), or a square root ($\sqrt{x}$) equation. The predictor of change with the highest R^2 was identified and selected to be entered in the regression. The final equation found to be significant represents the relationship between the paired series and provides a percentage of the data variance explained by the equation [i.e.,

R^2; see Tabachnik & Fidell (1996) for more details on regression]. Autoregressive analyses were performed for each sleep variable for each participant and under five comparisons: (a) baseline vs. the first five weeks of treatment; (b) the first five weeks vs. the second five weeks of treatment; (c) the second five weeks of treatment vs. posttreatment; (d) baseline vs. posttreatment; and (e) posttreatment vs. follow-up. In order to decrease type I error, the alpha for slope and time effects was adjusted to .01 (familywise) and to .05 for autocorrelation.

The Friedman's non parametric test (χ^2) was performed on self-report measures and actigraphic data, and Pearson's correlation analyses among *AQ* items at posttreatment were calculated. In addition, clinical significance of outcome was examined at posttreatment and at the 3-month follow-up according to the following four criteria: (a) reaching a SE higher than 85%; (b) obtaining a total change in SE higher than one standard deviation of sleep efficiency before treatment for all participants; (c) obtaining an ISI total score below the clinical threshold (<14/28); and (d) reaching a TST higher than 6.5 hours. Participants meeting three or four criteria were considered to have an excellent treatment response. Those who met two criteria were considered to have a moderate treatment response. When only one criterion or less was met, participants were considered to have a minimal treatment response.

Results

Sleep diary data

Figure 1 shows the daily change in SE for each participant through each phase of the study. Visual inspection of SE over time showed that participants presented three to seven nights of insomnia during baseline. Also, night-to-night variability in baseline data was experienced by all participants. The smallest of the night-to-night variability may be observed for participant 1. These observations confirmed the presence of insomnia before treatment. A closer look at baseline data suggested that no change was observed in the insomnia profile before treatment was introduced. For participants 1 and 2 in the Combined treatment, the

night-to-night variability diminished after three weeks of treatment and those improvements were maintained at posttreatment. Participant 3 in the overlapping sequence presented a linear change during the first phase of treatment while, for this same period, participant 4 experienced a level change. In addition, their night-to-night variability was reduced during the second part of treatment and maintained at the subsequent assessment periods. Participants 5 and 6, who received the Medication → CBT sequence, did not seem to present improvement during the first phase of treatment. During the second phase through posttreatment, participant 5 experienced a lower degree of night-to-night variability. Participant 6 did not seem to improve. Autoregressive analyses were performed on SE, TWT, and TST to determine if there was significant improvement between treatment phases (See Tables 1, 2, and 3).

Combined sequence. Both participants improved significantly in SE and TWT from baseline to the first phase of treatment ($ps < .001$) and had a significant deterioration in TST ($p < .001$). For participant 1, no further change was observed before posttreatment during which an additional reduction in TST was obtained ($p < .0001$). For participant 2, all variables were stable during the second phase of treatment, except TST which improved ($p < .001$). No further improvement was observed at posttreatment.

Overlapping sequence. Participant 3 improved in SE and TWT during the first phase of treatment ($ps < .01$), and in TWT as well as TST ($ps < .01$) during the second phase of treatment. Even though it remained under baseline level, TWT worsened at posttreatment ($p < .001$). Participant 4 improved in SE and TWT during the first phase of treatment and in TST ($p < .001$) during the second phase of treatment only. All other variables remained stable at posttreatment.

Medication → CBT sequence. Both participants (5 and 6) improved in SE and TWT ($ps < .01$) when CBT was introduced during the second phase of treatment only. Participant 5 had no further change whereas participant 6 improved in TST during the second phase of treatment and posttreament ($ps < .05$, and $< .01$, respectively).

Follow-up

Three-month follow-up sleep diary data were available for five participants (1, 2, 4, 5, and 6) (See Table 1 and Figure 1). Comparison of these data with posttreatment data revealed that SE and TST remained stable for participants 1, 2, and 4 and that TWT worsened ($p <$ .001) for participant 1 only. Participant 5, while remaining improved over baseline level, had significant deteriorations in SE and TWT ($ps < .01$ and .001, respectively). Participant 6 had an additional improvement in SE ($p < .01$).

Actigraphic data

Actigraphic data were available for six participants for the first two weeks of treatment and for four participants at posttreatment (See Table 2). These data revealed a significant decrease in time in bed for all participants ($p < .05$). Also, although not significant, TST improved for participant 1 and decreased for participants 4, 5, and 6. SE improved for participants 1 and 5, while it remained stable for participant 4 and worsened for participant 6. TWT improved for participants 1, 4, and 5, while it increased for participant 6.

Psychological assessment

Means and standard deviations for all self-report measures at the four assessment periods (baseline, middle of treatment, posttreatment, and 3-month follow-up) are presented in Table 3. Friedman's non parametric test showed a significant decrease in insomnia severity from baseline to posttreatment as evidenced on the ISI measure completed by the participants, the clinician, and the significant others ($\chi^2_{(2)}$s = 10.17, 9.33, and $\chi^2_{(1)} = 6.00$, respectively, $ps <$ .01). DBAS means decreased from baseline to posttreatment for each treatment sequence but did not reach statistical significance. There was a trend for the DBAS and the anxiety symptoms to be lower at midtreatment than at posttreatment and a trend for the depressive symptoms to be higher at the same time for participants (5 and 6) in the Medication → CBT sequence only.

Attributional effect

Pearson correlation analyses were performed between the *Attributional Questionnaire* items and the change scores from baseline to posttreatment for sleep variables (SE, TST, and TWT). Results showed that increases from baseline to posttreatment in SE were positively related to an attribution of sleep change to beliefs ($r = .89, p < .05$). Also, an increase in TST from baseline to posttreatment was positively related to the attribution of therapeutic gains to the physician ($r = .91, p < .05$).

Treatment compliance, satisfaction, and clinical improvement

The average weekly observance of treatment procedures was 82.6% ($SD = 8.44$). Sleep diary reports of medication intake indicated that participants 3 and 5 took one pill more than prescribed during the first week. Also, participants 1 and 5 took one pill more per week than prescribed during the withdrawal period. The medication intake at the 3-month follow-up corresponded to the significant others' reports. Actigraphic data also suggested that participants followed CBT procedures by the reduction observed in time spent in bed.

Based on the criteria outlined above, five participants (1, 3, 4, 5, and 6) reached an excellent treatment response and one a moderate response (participant 2) at posttreatment. At 3-month follow-up, participant 4 still met criteria for an excellent treatment response, participants 1, 2, and 5 for a moderate response, and participant 6 for a minimal response.

Discussion

The present findings indicated that the sequential combination of drug and behaviour therapies for chronic insomnia is effective in improving sleep in the short-term. Each sequence led to significant improvement of sleep efficiency and continuity. For the combined sequence, improvements in SE and TWT occurred during the first phase of treatment but TST

was also reduced during this period. The overlapping sequence provided the same improvements during the first phase of treatment and, in addition, TST was stable for that period. Participants (5 and 6) in the Medication → CBT sequence did not show any significant improvement before the introduction of CBT during the second phase of treatment. Actigraphic results corroborated those obtained in the sleep diary for two participants (1 and 5) only. There was a positive relation between sleep improvement and the attribution of these changes to beliefs. Also, insomnia severity and mood were improved for all participants. Half of the participants (2, 4, and 6) maintained their gains at the 3-month follow-up.

Taken together, these results suggest that sleep changes occurred at different times during treatment. In fact, improvements occurred after CBT introduction, a manipulation that was altered across treatment sequences. These findings are consistent with earlier reports (McClusky et al., 1991; Milby et al., 1993; Morin, Colecchi, et al., 1999; Rosen et al., 2000) on the importance of integrating CBT as an essential component of insomnia treatment. Also, when medication is combined with CBT, withdrawing medication before ending CBT enhances treatment effectiveness. Indeed, during the first phase of treatment and at posttreatment, participants in the Combined (participants 1 and 2) and the Overlapping (participants 3 and 4) sequences had similar improvements, with the exception that those in the Combined sequence worsened on TST. Therefore, the Overlapping sequence should be further investigated by varying its duration and the introduction of each treatment during this period. Interestingly, participants reported being satisfied and more confident in being able to sleep, regardless of sleep improvement obtained and of the treatment sequence received. Thus, this satisfaction may be more associated with their overall improvement in mood or insomnia severity than with a particular treatment component.

Actigraphy is a potentially useful instrument to measure the participants' adherence to treatment procedures (Sadeh et al., 1995). However, the variability of actigraphic data underlined the importance of conducting additional studies to investigate the accuracy of actigraphy in treatment studies (e.g., Wicklow & Espie, 2000; Verbeek et al., 2001).

Actigraphy should be compared to polysomnography and sleep diary using a large number of participants.

The study design poses some methodological limitations. First, given the absence of a placebo or waiting list control group, it is difficult to directly compare treatment sequences. However, the design used in this study suggests that if changes in sleep always follow treatment implementation, then those changes are likely due to treatment rather than other factors such as history or maturation. Second, results should be interpreted cautiously given the small sample size. Third, the mild insomnia severity of most participants (1, 2, 4, 5, and 6) reduces the likelihood of detecting improvement, since real sleep changes could be hidden by a possible ceiling effect.

In conclusion, this study is the first to investigate the sequential combination of drug and behavioural therapies for insomnia. Additional clinical trials should be conducted with a larger sample of individuals with more severe insomnia. A more detailed monitoring and analysis of attributional effect would also be useful. The treatment sequence might be modified based on the results obtained in this study. The Overlapping sequence that entailed two weeks of concurrent therapy should be increased in duration. Also, the Medication → CBT sequence must be adapted to preclude having participants withdrawing from medication before beginning CBT.

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Table 1

Predictors and direction of change between treatment phases for each sleep variable and participant in the three treatment sequences

	Baseline vs. Phase 1			Phase 1 vs. Phase 2			Phase 2 vs. Posttreatment			Posttreatment vs. FU-3		
	Predictor of change	R^2	Direction of change	Predictor of change	R^2	Direction of change	Predictor of change	R^2	Direction of change	Predictor of change	R^2	Direction of change
Combined sequence (n= 2): Sleep efficiency												
P 1	Level	21.95^{***}	+	None		0	None		0	None		0
P 2	Level	37.95^{***}	+	None		0	Root	14.62^{**}	-	None		0
Total Sleep Time												
P 1	Level	26.92^{**}	-	None		0	Level	37.70^{***}	-	Time	18.10^{**}	0
P 2	Level	32.83^{***}	-	Root	19.10^{***}	+	None		0	None		0
Total Wake Time												
P 1	Level	37.74^{***}	+	None		0	None		0	Level	39.2^{***}	-
P 2	Level	62.77^{***}	+	None		0	root	25.99^{***}	-	None		0
Overlapping sequence (n =2): Sleep efficiency												
P 3	Root	50.10^{***}	+	Time	56.67^{***}	0	None		0	N/A	N/A	N/A
P 4	Level	33.30^{***}	+	None		0	None		0	None		0
Total Sleep Time												
P 3	None		0	Level	21.56^{**}	+	None		0	NA	N/A	N/A
P 4	None		0	Time	19.10^{***}	0	Quadratic		+	None		0

(to continue)

Table 1 (continued)

	Baseline vs. Phase 1			Phase 1 vs. Phase 2			Phase 2 vs. Posttreatment			Posttreatment vs. FU-3		
	Predictor of change	R^2	Direction of change	Predictor of change	R^2	Direction of change	Predictor of change	R^2	Direction of change	Predictor of change	R^2	Direction of change
Total Wake Time												
P 3	Level	46.08**	+	Level	58.12**	+	Level	39.79**	-	NA	NA	NA
P 4	Fraction	36.33***	+	None		0	None		0	None		0
Medication → CBT sequence (n = 2): *Sleep efficiency*												
P 5	None		0	Quadratic	17.23***	+	Time	18.53**	0	Fraction	45.97**	-
P 6	None		0	Fraction	14.34**	+	None		0	Level	52.20**	+
Total Sleep Time												
P 5	None		0	None		0	Linear		+	Time	43.23*	0
P 6	Time	18.64*	0	Linearly	19.60**	+	None		0	None		0
Total Wake Time												
P 5	None		0	Linear	11.02**	+	Time	22.92**	0	Level	34.40***	-
P 6	None		0	Fraction	8.85**	+	None		0	None		0

Note. P1 to P6 = Participant 1 to participant 6; + = improved; 0 = stable; - = worsened; N/A = Not available; Phase 1 = first five weeks of treatment; Phase 2 = second five weeks of treatment.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 2

Means (M) and standard deviations (SD) for actigraphic data at each assessment period for each participant

	Time In Bed (minutes)		Total Wake Time (minutes)		Total Sleep Time (minutes)		Sleep Efficiency (%)	
	Baseline	Posttreatment	Baseline	Posttreatment	Baseline	Posttreatment	Baseline	Posttreatment
	M (SD)	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*
Combined sequence								
P1	411.69 (16.99)	379.00 (100.41)	75.31 (44.66)	18.00 (5.66)	336.38 (44.24)	361.00 (106.07)	81.62 (10.88)	95.00 (2.83)
P2	397.38 (46.28)	N/A	66.62 (35.25)	N/A	330.77 (42.44)	N/A	83.38 (8.15)	N/A
Overlap sequence								
P3	536.00 (.)	N/A	96.00 (.)	N/A	440.00 (.)	N/A	82.00 (.)	N/A
P4	503.00 (57.20)	447.43 (29.46)	158.75 (31.95)	140.21 (29.02)	344.25 (42.20)	307.21 (41.95)	68.63 (4.57)	68.57 (6.70)
Medication → CBT sequence								
P5	399.43 (40.43)	391.71 (40.55)	83.57 (36.81)	69.14 (33.51)	315.86 (70.81)	309.71 (73.43)	78.29 (10.69)	81.71 (10.09)
P6	515.71 (58.31)	465.23 (10.97)	112.86 (64.86)	143.85 (36.39)	402.86 (45.10)	321.38 (40.73)	78.71 (10.64)	69.15 (8.13)

Note. P1 to P6 = Participant 1 to participant 6; N/A = Not available.

Table 3

Means (M) and standard deviations (SD) of self-report measures for each treatment sequence

	Baseline		Mid treatment		Posttreatment		FU-3	
	M	(*SD*)	*M*	(*SD*)	*M*	(*SD*)	*M*	(*SD*)
Combined (n = 2)								
ISI-participant	15.00	(0.00)	11.50	(0.71)	6.00	(1.41)	10.00	(7.07)
ISI-clinician	11.50	(2.12)	6.50	(2.12)	5.50	(0.71)	N/A	N/A
ISI-other	13.00	(1.41)	N/A	N/A	7.50	(3.54	11.00	(5.66)
DBAS	50.14	(17.73)	29.14	(12.07)	19.43	(17.22)	30.43	(21.82)
BDI	8.00	(1.41)	5.50	(2.12)	6.50	(3.54)	6.50	(0.71)
BAI	7.50	(9.19)	4.00	(2.83)	6.00	(7.07)	4.50	(4.95)
Overlapping sequence (n = 2) [1]								
ISI	19.50	(4.95)	15.50	(0.71)	4.50	(3.54)	10.00	(0.00)
ISI-clinician	13.00	(1.41)	8.50	(3.54)	3.00	(1.41)	N/A	N/A
ISI-others	19.50	(0.71)	N/A	N/A	10.50	(2.12)	13.00	(0.00)
DBAS	52.54	(8.29)	46.04	(3.73)	26.04	(16.31)	25.00	(0.00)
BDI	10.50	(10.61)	7.50	(6.36)	3.00	(4.24)	0.00	(0.00)
BAI	4.50	(2.12)	1.50	(2.12)	0.50	(0.71)	2.00	(0.00)
Medication →CBT sequence (n = 2)								
ISI	16.00	(1.41)	14.00	(2.83)	9.00	(4.24)	11.00	(0.00)
ISI-clinician	9.50	(2.12)	4.50	(2.12)	4.50	(2.12)	N/A	N/A
ISI-others	15.00	(0.00)	N/A	N/A	11.00	(0.00)	12.50	(3.54)
DBAS	41.00	(13.68)	32.35	(15.58)	36.70	(23.05)	27.57	(8.15)
BDI	5.50	(4.95)	8.50	(9.19)	5.00	(5.66)	5.50	(6.36)
BAI	4.50	(3.54)	3.50	(4.95)	5.50	(6.36)	7.50	(6.36)
Total (N = 6)								
ISI	16.83[a]	(3.13)	13.67[a]	(2.25)	6.50[b]	(3.27)	10.40	(3.58)
ISI-clinician	11.33[a]	(2.16)	6.50[b]	(2.74)	4.33[b]	(1.63)	N/A	N/A
ISI-others	15.83	(3.06)	N/A	N/A	9.67	(2.50)	12.00	(3.46)
DBAS	47.89	(11.99)	35.84	(12.04)	26.96	(18.03)	28.20	(11.87)
BDI	8.00[a]	(5.73)	7.17[a]	(5.27)	4.83[b]	(3.87)	4.80	(4.21)
BAI	5.50	(4.76)	3.00	(2.97)	4.00	(5.06)	5.20	(4.66)

[1] Follow-up assessment was available for one participant only in the Overlapping sequence.

Note. CBT = Cognitive Behavioural Treatment; ISI = Insomnia Severity Index; DBAS = Dysfunctional Beliefs and Attitudes about Sleep Scale; BDI = Beck Depression Inventory; BAI = Beck Anxiety Inventory; N/A = Not available. Means in the same row that do not share the same subscripts (a and b or 1 and 2) differ at $p < .05$.

Figures Caption

Figure 1. Daily change of sleep efficiency for participants in each treatment sequence.

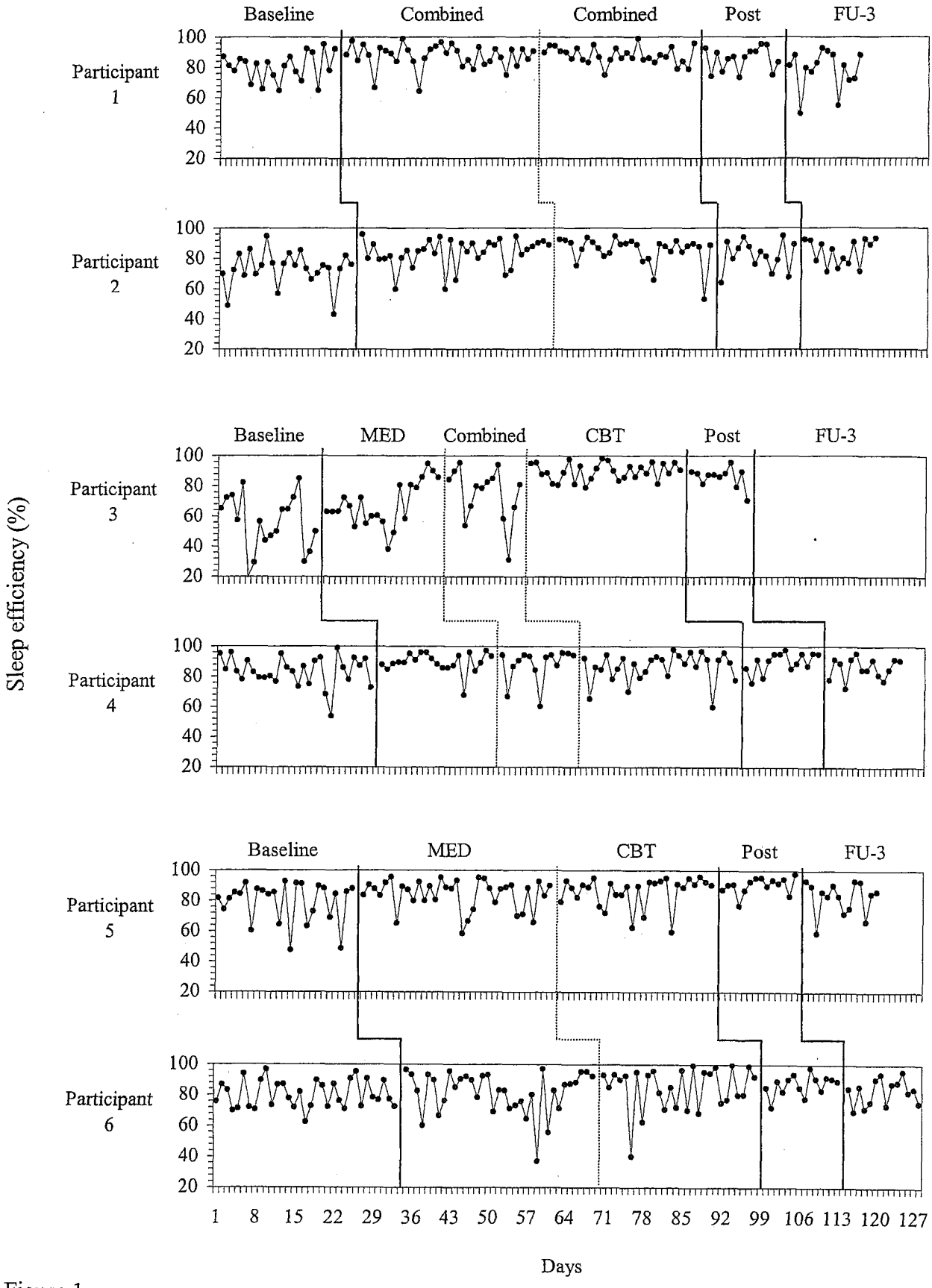
Baseline
Combined
Combined
Post
FU-3
Participant 1
Participant 2
Baseline
MED
Combined
CBT
Post
FU-3
Participant 3
Participant 4
Baseline
MED
CBT
Post
FU-3
Participant 5
Participant 6
Sleep efficiency (%)
100
80
60
40
20
1 8 15 22 29 36 43 50 57 64 71 78 85 92 99 106 113 120 127
Days

Figure 1.

CHAPITRE 2

COMBINAISON SÉQUENTIELLE DES TRAITEMENTS PHARMACOLOGIQUE ET COGNITIVO-COMPORTEMENTAL POUR L'INSOMNIE CHRONIQUE

Résumé

Objectif: Évaluer l'efficacité relative de séquences de traitement comprenant une médication et un traitement béhavioral-cognitif (TBC) pour l'insomnie chronique primaire.

Méthode: Dix-sept participants souffrant d'insomnie chronique primaire ont été enrôlés et assignés aléatoirement à trois niveaux de base différents (3, 5 et 7 semaines). Les séquences de traitement testées d'une durée de 10 semaines sont : (a) la médication utilisée seule pendant les cinq premières semaines de traitement suivi de l'introduction du TBC et du retrait graduel de la médication après la neuvième semaine de traitement (Médication → Combiné); (b) la médication combinée au TBC durant les cinq premières semaines de traitement suivi du retrait graduel de la médication et le TBC persiste seul durant les cinq dernières semaines (Combiné → TBC); et (c) un TBC seul offert durant les 10 semaines. Un suivi est réalisé trois mois après la fin du traitement.

Résultats: Chaque séquence de traitement produit une amélioration significative du sommeil mais à des moments différents durant l'intervention. Pour la séquence Médication → Combiné, la majorité de l'amélioration du sommeil se situe au niveau du temps total d'éveil (TTE) et est obtenue après l'introduction du TBC. Pour la séquence Combiné → TBC et le TBC seul, la majorité des variables du sommeil s'améliore dès la première phase de traitement. Neuf participants maintiennent leurs gains thérapeutiques au suivi de trois mois, un présente une amélioration additionnelle et trois une détérioration.

Conclusions: Ces résultats suggèrent que le traitement séquentiel est efficace pour traiter l'insomnie chronique primaire. Une séquence de traitement commençant par un traitement combiné suivi d'un TBC seul semble fournir les meilleurs résultats.

Running head: SEQUENTIAL COMBINATIONS OF DRUG AND BEHAVIOURAL

Sequential Combinations of Drug and Behavioral Therapies for Chronic Insomnia

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Abstract

Objective: To evaluate the relative efficacy of three sequential treatments involving medication and cognitive behavioural treatment (CBT) for chronic primary insomnia.

Methods: Seventeen participants with primary chronic insomnia took part in a multiple baseline across subjects design and were randomly assigned to one of three baseline durations (3, 5, 7 weeks). They received one of the following treatment sequences (10 weeks duration): (a) medication (zopiclone) used alone for the first five weeks, followed by combined medication plus CBT for five more weeks (Medication → Combined); (b) combined treatment for the first five weeks, followed by CBT alone for an additional five weeks (Combined → CBT); or (c) CBT alone. There was a follow-up assessment three months after the end of treatment.

Results: Each treatment sequence produced significant sleep improvements but the timing of those improvements occurred at different points during the intervention. For the Medication → Combined sequence, most improvement was in Total Wake Time (TWT) and occurred only after the introduction of CBT. For the Combined → CBT sequence and the CBT alone, most sleep variables improved during the first phase of treatment. At the 3-month follow-up, therapeutic gains were maintained for 9 participants, improved for one participant and deteriorated for three participants.

Conclusions: These results suggest that a sequential treatment is effective for chronic insomnia. A sequence beginning with a combined treatment followed by CBT alone seems to produce the best outcome.

Sequential Combination of Drug and Behavioural Therapies for Chronic Insomnia

Insomnia is a widespread complaint reported to health professionals and affecting 6 to 12% of the general population on a chronic basis (Ancoli-Israël & Roth, 1999; Ohayon, 1999). The efficacy of drug and behavioural therapies for insomnia has been well documented over the years (e.g., Morin, Culbert, & Schwartz, 1994; Murtagh & Greenwood, 1995; Nowell et al., 1997). Drug therapies are commonly used for insomnia because they are easily accessible and provide rapid relief from insomnia symptoms (Kupfer & Reynolds, 1997; Morin & Wooten, 1996). However, given the potential side effects and problems upon withdrawal (e.g., rebound insomnia), medication is recommended for transient and short term insomnia only (Kripke, 2000). Contrary to drug therapy, cognitive behavioural treatment (CBT) is not associated with such side effects or withdrawal symptoms. CBT is well accepted by patients, promotes good sleep habits, and there is evidence that it is more effective in the long run than medication alone (Edinger & Wohlgemuth, 1999; Espie, Inglis, & Harvey, 2001; Stepanski & Wyatt, 2000). Despite its proven efficacy, about 20 to 30% of insomnia patients do not benefit from CBT (Espie et al., 2001; Morin, Hauri, et al., 1999). Because of the complementary advantages of drug therapy and CBT, their combination seems to be a promising avenue to maximise therapeutic gains.

Four treatment studies have evaluated the effectiveness of the concurrent combination of CBT with benzodiazepines (Hauri, 1997; Milby et al., 1993; Morin, Colecchi, Stone, Sood, & Brink, 1999; Rosen, Lewin, Goldberg, & Woolfolk, 2000). The results of these four studies indicate that medication alone produces quicker results than CBT alone in the first week of treatment. Both treatments, alone or combined, are equally effective in the short-term interval (4-8 weeks). Long-term outcomes are fairly clear for the single therapy modality but are more equivocal for a combined approach. For instance, the initial benefits of drug therapy alone are quickly lost upon discontinuation of medication, while CBT alone produces sustained benefits over time. However, a combined approach does not yield better outcome in the long-term than CBT alone, at least for some patients. Surprisingly, even though CBT usually involves an

initial period of mild sleep deprivation, it produces a better long-term outcome than a combined intervention. Therefore, although a combined treatment approach is promising, it does not seem to lead to the degree of improvement expected.

The variability in the long-term results of these studies could be due to the patients' attribution of their improvement to medication. Indeed, it has been suggested that this attribution effect could undermine the efficacy of behavioural strategies in a combined approach (Morin & Wooten, 1996; Stepanski & Wyatt, 2000). In such cases, individuals would not develop appropriate self-management skills during treatment, and when the medication is withdrawn, patients are more likely to relapse (Edinger & Wolhgemuth, 1999; Morin, Colecchi, et al., 1999).

Conclusions about the superiority of a combined approach are still premature, mainly because of methodological differences across the four studies. For example, treatment duration varied from three to eight weeks and follow-up assessment from five weeks (Milby et al., 1993) to 24 months (Morin, Colecchi, et al. 1999). The nature of the treatment options tested was also different across studies. For example, three studies tested relaxation as the behavioural component (Hauri, 1997; Milby et al., 1993; Rosen et al., 2000), two tested stimulus control (Milby et al., 1993; Morin, Colecchi, et al., 1999), and one tested a multi-component CBT program including stimulus control, sleep restriction, and cognitive interventions (Morin, Colecchi, et al. 1999). Moreover, as stated by Kazdin (1998), to investigate whether a new treatment is effective, it should be compared with the standard treatment. If we consider that medication alone or CBT alone are the standard approaches to treat insomnia, then the efficacy of combined treatments should be contrasted with that of these standard treatments. In the four group treatment studies, combined treatment was compared only once to medication alone (Morin, Colecchi, et al., 1999) and only twice to CBT alone (Hauri, 1997; Morin, Colecchi, et al., 1999). Therefore, additional studies are needed to draw conclusions about the relative efficacy of combined treatments.

Finally, beyond these considerations, it is also possible that the variability observed in the long-term outcome was due to the concurrent combination of treatments. The fact that both interventions were administered simultaneously might have undermined the therapeutic response, possibly through the attribution of therapeutic gains to medication, or by any other mechanism particular to this combination. Therefore, it seems essential to elaborate other ways of combining medication and CBT in order to benefit fully from the respective advantages of both treatments. A sequential introduction of both treatments could produce an outcome superior to that of a given treatment used alone or in a concurrent combination. Indeed, the initial introduction of medication could first allow rapid relief of sleep difficulties. Subsequently, behavioural procedures could be implemented to consolidate those initial gains. Furthermore, such a sequential treatment would more realistically correspond to the present health care system situation, in which medication is easier to access as a first treatment option.

A pilot study using a single-case design has already explored this issue with six individuals presenting insomnia of mild to moderate severity (Vallières et al., 2001). Three treatment sequences were evaluated: (a) a consecutive combination of medication alone and CBT alone with no overlap between treatments; (b) a consecutive combination where medication was withdrawn two weeks after the introduction of CBT, thus creating a treatment overlap; and finally (c) a concurrent combination of CBT and medication. Results of this study suggest that treatments combinations are effective in the short term. Sleep efficiency improved from baseline (mean of 72%) to posttreatment (mean of 87%) for all participants, while total wake time improved for five participants, and total sleep time improved for the two participants in the overlapping sequence only. Half of the participants maintained their therapeutic gains at the 3-month follow-up. Even though therapeutic gains were significant, the magnitude of sleep improvement was small. This could be due either to the fact that participants experienced only mild to moderate insomnia at baseline or to the choice of treatment sequences.

The present study further examined the efficacy of new treatment combinations with a sample composed of participants with more severe insomnia. The treatment combinations that

were used in the pilot study were modified in order to maximise their impact on sleep. The first sequence in the pilot study (Medication → CBT) was modified to become a sequence of medication alone followed by the combination of medication and CBT (Medication → Combined). This sequence is more representative of what currently takes place in clinical practice. In the same way, the second sequence in the pilot study (Medication → Medication + CBT → CBT) was modified to begin with both treatments presented concurrently followed by CBT alone (Combined → CBT). Given that the overlapping sequence in the pilot study was the only sequence to increase total sleep time, modification was undertaken in this study to optimise this effect. Finally, the concurrent treatment combination was replaced with CBT alone in order to provide a better comparison point. The impact at the moment of transition of these sequential treatment combinations and to CBT alone would be evaluated. It was hypothesised that both treatment sequences and CBT alone would be effective in the short term. In addition, it was predicted that sleep improvement would occur during the first phase of treatment only when the sequence begins with both treatments. An exploratory investigation of the patient attribution of therapeutic gains would also be undertaken to examine its possible impact when medication and CBT were combined.

Method

Participants

Participants were recruited through newspaper advertisements or by physician referrals. Inclusion criteria were: (a) being between 30- and 50-years old; (b) reporting insomnia, defined as sleep onset latency, wake after sleep onset, or early morning awakening equal or superior to 60 minutes at least 4 nights a week for the past 6 months; (c) reporting significant distress or daytime impairments as evaluated by the *Insomnia Severity Index* (score of 2 or higher on a 1 [*not at all*] to 4 [*very much*] Likert scale); and (d) cessation, at least one month prior to treatment, of any sleep or other psychotropic medication that could interfere with sleep. Exclusion criteria were: (a) presence of another sleep disorder such as sleep apnea or circadian

rhythm disorder; (b) evidence that insomnia was related to a medical condition; (c) presence of major depression, anxiety disorder, alcohol/substance abuse or any other severe psychopathology as diagnosed with the Structured Clinical Interview for DSM-IV (SCID-IV; First, Spitzer, Gibbon, & Williams, 1997); (d) currently in psychotherapy; and (e) regular use of a medication interfering with sleep. Although more stringent, these criteria are consistent with those of the *International Classification of Sleep Disorders* (American Sleep Disorder Association [ASDA], 1990) and the DSM-IV (American Psychiatric Association [APA], 1994).

A total of 142 individuals responded to the advertisements. Of these 142 people, 58 were excluded because they reported taking a medication, suffering from pain disorder, anxiety, or depression. Therefore, 84 individuals were judged suitable for the study and underwent a telephone screening. Of the 84 people, 14 were not interested anymore and 34 were excluded for the following reasons: medication interfering with sleep (n = 15); insomnia secondary to a psychopathology, a medical condition, or noise (n = 7); circadian rhythm disorder resulting from night shift (n = 3); currently in psychotherapy (n = 4); over 50-year old (n = 5). The remaining 36 people underwent subsequent assessments including a semi-structured sleep history interview, a SCID-IV evaluation (First et al., 1997), a psychological screening as well as a physical evaluation. An additional 19 individuals were excluded because they were not interested anymore in the study (n = 5); they did not meet insomnia criteria (n = 5); their insomnia was secondary to a psychopathology, alcohol abuse, a medical condition, noise or previous night work experience (n = 9). Note that one participant dropped out after one week of treatment.

Thus, the final sample included 17 participants (7 men and 10 women) meeting ICSD (ASDA, 1990) and DSM-IV (APA, 1994) criteria for primary chronic insomnia and 16 who ended the treatment. Their mean age was 41.6 years (*SD* = 5.7; range from 34 to 50). Eight participants were married, five were divorced or separated and four were single. The average education level was 15.2 years (*SD* = 3.0; range from 10 to 19 years). Fourteen were working and three were unemployed. The average insomnia duration was 11.8 years (*SD* = 6.2) and the mean age of insomnia onset was 29.8 years (*SD* = 7.7). One participant presented sleep-

onset insomnia only, nine sleep-maintenance insomnia only and seven mixed insomnia. No participant had a history of psychiatric disorder, chronic pain or health problems known to interfere with sleep. Of the 17 participants, one had never taken any sleep medication, one had previously tried zopiclone alone, and another had tried zopiclone combined with an over-the-counter-pill and homeopathy. Fourteen had already used a benzodiazepine for sleep. Among these 14 participants, 5 had tried benzodiazepines alone, 1 had combined a benzodiazepine with an over-the-counter pill, 4 with an anti-depressant, 3 with zopiclone, and 1 with alcohol. All participants were free of any sleep medication before entering the study.

Design and procedures

A multiple baseline across subjects design (Barlow & Hersen, 1984; Ladouceur & Bégin, 1993) was used to evaluate the efficacy of the different treatment sequences. Treatment duration was 10 weeks. Participants were randomised to a baseline duration of either three, five or seven weeks in order to control for historical events and participants' maturation and then to one of the treatment sequences. Baseline duration were longer than in the pilot study (Vallières et al., 2001) in order to better distinguish the introduction of treatment and enhance observation of sleep change. For each baseline, one of the following treatment conditions was applied: (a) medication used alone for the first five weeks, followed by medication combined with CBT for an additional five weeks and gradual medication withdrawal after the ninth week (Medication → Combined); (b) medication combined with CBT for the first five weeks, followed by CBT alone for an additional five weeks and gradual medication withdrawal after the sixth week (Combined → CBT); or (c) CBT alone for the 10 weeks of treatment. The moment of CBT introduction varied across treatment sequences to control for a possible order effect. Another assessment was conducted following treatment (posttreatment). One month after posttreatment, a CBT booster session was offered to participants who wanted it. Finally, a follow-up was conducted three months after posttreatment. Participants completed all measures four times during the experimentation: at baseline, midway during treatment, at posttreatment as well as at the 3-month follow-up assessment period.

Measures

Initial screening and evaluation. The initial screening included a 20-minute telephone interview administered to determine participant's eligibility. A subsequent multi-measure pre-treatment evaluation was composed of a semi-structured sleep history interview to diagnose insomnia, the SCID-IV (First et al., 1997) to evaluate the presence of psychological disorders, and a physical examination.

Sleep measures. Participants monitored their sleep/wake patterns using daily sleep diaries which were completed from baseline through the 10-week treatment period, two weeks after the end of treatment, and at the 3-month follow-up. From these diaries, nightly averages of total wake time (TWT), total sleep time (TST), and sleep efficiency (SE; ratio of total sleep time to time in bed) were computed. Participants rated their sleep soundness and restedness on a 1 *(poor)* to 5 *(extremely)* Likert scale. From these ratings, a score of sleep quality was derived (mean of soundness and restedness every day). Participants also monitored type and dosage of medication used.

Polysomnography. Participants underwent three consecutive nights of sleep laboratory evaluation during baseline, two consecutive nights at mid-treatment (week six), and two consecutive nights at posttreatment. The polysomnographic montage included electroencephalographic, electromyographic, and electro-oculographic monitoring. Sleep stages, respiratory disturbance, and limb movements were scored according to standard criteria (Rechtschaffen & Kales, 1968) by an experienced clinician blind to participants' treatment sequence. Respiration (air flow, tidal volume, and oxygen saturation) and anterior tibialis electromyographic readings were recorded during the first night to detect sleep apnea or periodic limb movements. Outcome measures [Wake After Sleep Onset (WASO), Sleep Onset Latency (SOL), TWT, TST and SE] were based on the average of baseline nights two and three, middle nights four and five, and posttreatment nights six and seven. To allow an adaptation to the laboratory, data from the first baseline night were not used in computing baseline means.

Psychological assessment. Participants completed self-report inventories before, at the middle and after treatment as well as at the 3-month follow-up. First, the *Insomnia Severity Index* (*ISI*; Morin, 1993) includes seven questions about participants' sleep difficulties. The seven items evaluate the severity of sleep-onset, sleep maintenance, early morning awakening problems, satisfaction with current sleep pattern, interference with daily functioning, noticeably of impairment attributed to the sleep problem, and level of distress cause by the sleep problem. The ISI has adequate psychometric properties and has been shown to be sensitive to changes in clinical trials of insomnia (Bastien, Vallières, & Morin, 2001). The *Dysfunctional Beliefs and Attitudes about Sleep scale* (*DBAS*; Morin, 1993) includes 30 items assessing sleep beliefs about the perceived causes and consequences of insomnia and about sleep requirements expectations. The DBAS has adequate psychometric properties (Blais, Gendron, Mimeault, & Morin, 1997; Espie, Inglis, Harvey, & Tessier, 2000; Morin, Stone, Trinkle, & Mercer, 1993). In addition, the *Attributional Questionnaire* (*AQ*) is an 8-item scale based on Weiner's motivation and performance attribution theory (Weiner, 1985). Participants rated on a 0 *(not at all)* to 100 *(extremely)* scale if changes in their sleep were due to the physician, the psychologist, the medication, behavioral procedures, modification in beliefs and attitudes, self-effort or luck. The eighth item assesses perceived self-efficacy. According to Bouchard, Bastien, and Morin (1999), this last question alone is enough to assess the participant's perception of self-efficacy. The AQ was completed weekly throughout the treatment and at each assessment period to detect any fluctuation in perceived self-efficacy and attribution of clinical improvement. Psychometric properties of this questionnaire have not been tested yet. However, given the growing importance in the literature accorded to the potential negative attributional effect on the development of skills management when medication and CBT are combined, it seemed necessary to develop and use an attributional measure.

To assess the presence of variation in mood state throughout the treatment, the *Beck Depression Inventory* (*BDI*; Beck, Steer, & Garbin, 1988), which evaluates depressive symptoms, and the *Penn State Worry Questionnaire* (*PSWQ*; Meyer, Miller, Metzger, & Borkovec, 1990), which assesses the tendency to worry, were administered at each assessment

phase. The French versions of both questionnaires possesses reliable psychometric properties (For *BDI* : Bourque & Beaudette, 1982; for *PSWQ* : Gosselin, Dugas, Ladouceur, & Freeston, 2001).

Additional measures. The participants' perception of treatment acceptability was assessed using the *Treatment Acceptability Scale* (Morin, Gaulier, Barry, & Kowatch, 1992) adapted for sequential treatment. Participants completed this questionnaire at baseline, in the middle and after treatment as well as at the 3-month follow-up. In addition, withdrawal symptoms were assessed using the *Withdrawal Symptoms Evaluation Scale* (*WSES*). This self-report measure was adapted to the medication used in this study and is based on the French version of the *Benzodiazepine Withdrawal Symptoms Scale* (Bourin, 1988). It includes 10 items in the participant version and 3 items in the physician version. This questionnaire was completed by the physician as well as by the participants during medication withdrawal.

Compliance measures. Adherence to treatment protocol was evaluated with three modalities. First, participants were asked to monitor on their sleep diaries their medication intake and adherence to CBT procedures. A weekly percentage of adherence to each behavioural procedure and medication intake was computed for each participant. Then, the overall average of this percentage of adherence was also computed for all weeks combined. Second, a pill count was conducted by the treating physician at each consultation visit. Third, an independent rating of medication compliance was obtained from the subject's significant other.

Treatment

The CBT administered in this study is outlined in a treatment manual (Morin, 1993) and includes 50-minute individual consultation sessions. For the Medication → Combined sequence, CBT was administered in 5 weekly sessions during the last five weeks of treatment. For the Combined → CBT sequence, 10 CBT sessions were administered, weekly through the 10-week treatment. Finally, for CBT alone, there was one treatment session every two weeks for a total of five CBT sessions. Regardless of sequential combination, the content of each

session was the same. However, when there were ten sessions, each component was discussed for a longer period of time. CBT treatment featured behavioural, cognitive, and educational components. The behavioural component included stimulus control instructions and a sleep restriction procedure. Participants were instructed to go to bed only when sleepy at night, use the bed only for sleep and sex, get out of the bed whenever unable to fall asleep or return to sleep, maintain a regular arising time in the morning and avoid daytime napping. The sleep restriction procedure consisted of curtailing the time spent in bed to the actual amount of time asleep. To begin, a *sleep window* was determined using the average of total sleep time reported by participants in their sleep diaries. The sleep window was gradually increased, contingent upon reaching a sleep efficiency of 85 % or more. The cognitive component was aimed at altering dysfunctional beliefs and attitudes about sleep and the impact of sleep loss on daytime functioning. Finally, the educational component included didactic presentations on sleep hygiene and a review of basic facts about changes in sleep patterns over the life span (Morin, 1993). Strategies to prevent relapse were reviewed during the last treatment session as well as in the booster session.

The medication treatment included 20-minute individual consultation sessions with the physician involved in the study. These consultation sessions were designed to monitor insomnia symptoms, treatment response and side effects, and to adjust the medication dosage and frequency of use. For the first treatment sequence (Medication → Combined), there were 10 weekly consultation sessions with the physician. For the second treatment sequence (Combined → CBT), there were 5 weekly consultation sessions administered during the first five weeks of treatment. One additional session with the physician was administered during medication withdrawal. There was no consultation session with the physician for CBT alone. The medication used was zopiclone (Imovane), a non-benzodiazepine hypnotic agent which has a rapid action and a relatively short half life (4 - 6 hours). Zopiclone was the only non benzodiazepine hypnotic agent available in Canada when the study was undertaken. Its efficacy for insomnia is comparable to benzodiazepines, but it is associated with few secondary effects and has a low potential for rebound insomnia (Wadworth & McTavish, 1993; Wagner & Wagner, 2000). The dosage used varied from 3.75 mg to 7.5 mg and was

determined according to participant response to medication. The medication was prescribed for nightly use and was taken 30 minutes before bedtime. At the end of treatment, the dosage was gradually reduced. The time necessary to complete withdrawal depended on the initial dosage and the presence of withdrawal symptoms. It varied between one and three weeks.

Therapists

The CBT sessions were led by the first author a licensed clinical psychologist who had previously treated several clinical patients using this protocol. The original treatment manual (Morin, 1993) was adapted for a treatment duration of five sessions. A general medical practitioner with 20 years of experience in treating patients with sleep disorders led the medication treatment sessions and supervised the medication withdrawal. A manual outlining the structure of the medication session was used. This manual included the topics that had to be covered in each session as well as the information that could not be discussed with the participant (e.g., behavioural recommendations). Sessions were recorded and randomly reviewed by the study coordinator to ensure adherence to protocol.

Data analysis plan

Data obtained were divided in smaller series according to each treatment phase such as baseline (three to seven weeks), first five weeks of treatment, second five weeks of treatment, posttreatment (two weeks), and follow-up (two weeks). Thus, five series of sleep diary data were available for each sleep variable for each participant. These data were used as repeated measures. Given that each measure is dependent on the previous (serial dependency), such data are usually highly correlated. This autocorrelation can lead to a misinterpretation of the results and to an increased probability of type I error (DeProspero & Cohen, 1979; Jones, Weinrott, & Vaught, 1978). In addition, the design used and the small number of participants did not allow for the typical comparison of means without inflating type I error as well. Sleep diary data for each participant were thus analysed separately with autoregressive analysis [see Gorsuch (1983) for more details on autoregressive analysis]. This type of times series analysis has been recommended for use with single case study (Rivard & Bouchard, 1998; Ellis, 1999; Heath,

Kelly, & Longstaff, 2000). The autoregressive analysis produces an estimation of the autoregressive coefficient and controls for serial dependency. The first step of this analysis is to extract the autocorrelation from the data set in order to get residual data due to the experimental effect (treatment) or to error. Then, each small series of residual data (e.g., baseline, first five weeks of treatment) is modelled according to a standard regression. Predictors of change tested between paired series of residual data are change in level, time effect, and slopes. Slopes as predictors can be a fraction (1/x), a linear (ax + b), a quadratic ($ax^2 + bx + c$), a logarithmic ($\log_{(x)}$), or a square root ($\sqrt{x}$) equation. The predictor of change with the highest R^2 was identified and selected to be entered in the regression. The final equation found to be significant represents the relationship between the paired series and provides a percentage of the data variance explained by the equation [i.e., R^2; see Tabachnik & Fidell (1996) for more details on regression]. Autoregressive analyses were performed for each sleep variable for each participant and under five comparisons: (a) baseline vs. the first five weeks of treatment; (b) the first five weeks vs. the second five weeks of treatment; (c) the second five weeks of treatment vs. posttreatment; (d) baseline vs. posttreatment; and (e) posttreatment vs. follow-up. In order to decrease type I error, the alpha for slope and time effects was adjusted to .01 (familywise) and to .05 for autocorrelation.

Self-report questionnaires, attributional questionnaire (*AQ*) and PSG data were analysed with the Friedman's non parametric test (χ^2) for repeated measures (baseline, mid-treatment, and posttreatment), and with the Kruskal-Wallis test for between group comparisons. For the AQ, correlation analyses were also performed among items and between change scores from posttreatment and baseline.

In addition to these analyses, clinical significance of outcome was examined in the middle of treatment, at posttreatment, as well as at the 3-month follow-up according to the following four criteria: (a) reaching a sleep efficiency higher than 85%; (b) obtaining a total change in sleep efficiency higher than one standard deviation of the mean of sleep efficiency before treatment for all participants; (c) obtaining a ISI total score below the clinical threshold (<14/28); and (d) reaching a TST higher than 6.5 hours. Based on these four criteria, four

categories of treatment response were created; (1) excellent treatment response; (2) moderate treatment response; and (3) minimal treatment response. Participants meeting three or four criteria were considered to have an excellent treatment response. Those who met two criteria were considered to have a moderate treatment response. When only one criterion or less was met, participants were considered to have a low treatment response.

Results

Sleep diary data

Figures 1, 2, and 3 show the daily change in SE for all participants through each phase of the study. Visual inspection of SE over time showed that all participants presented between four and seven nights of insomnia during baseline. Also, there was extensive night-to-night variability in the sleep patterns of all participants during baseline, a classic feature of insomnia (with the exception of participant 6). These observations confirmed that participants exceeded diagnostic threshold for insomnia at baseline (APA, 1994; ASDA, 1990). A closer look at baseline data showed that no change occurred in the sleep of participants before treatment was introduced. At that moment, participants 1, 2, and 3 in the Medication → Combined sequence presented a gradual increase in SE. Participant 2 seemed to present a reduction in night-to-night variability after three weeks of treatment. By the end of the second phase of treatment, sleep efficiency was more stable and these changes were well maintained at posttreatment and FU-3 for participants 2, 3, and 5. Three participants (7, 8, and 10) in the Combined → CBT sequence made a gradual change during the first phase of treatment. During the second phase of treatment, participants 6, 8, 9, and 10, presented a lower level of night-to-night variability. In addition, participant 7 returned to baseline level during the eighth week of treatment.

All participants, who received CBT alone showed some improvement of sleep efficiency but only toward the end of this first phase of treatment. During the second phase of treatment, the night-to-night variability was diminished for all participants. These changes were well

maintained for participants 11, 13, 14, and 16. Participant 15 worked night shifts during the last two weeks of treatment and presented a SE reduction at that time. Autoregressive analyses were performed on SE, TWT, and TST to confirm these visual observations and to determine if there was significant improvement across treatment phase (See Tables 1, 2, and 3). A summary of the number of participants improved on sleep efficiency is presented in Table 4.

Medication → Combined sequence. Results for this treatment sequence are presented in Table 1 and Figure 1. Overall, results indicated that participant 4 improved on all three sleep variables and that participant 1 improved on SE and TWT after the introduction of medication alone. Three participants (1, 3, and 5) improved on SE and TWT after the introduction of CBT, while four (1, 2, 4, and 5) had a decrease in TST during the second phase of treatment. Finally, participant 2 did not improve on any sleep variables during treatment.

For participant 1, SE and TWT significantly improved when medication was introduced ($ps < .0001$). When CBT was added to medication, SE continued to increase while TWT continued to decrease ($ps < .01$). During the same period, however, TST worsened significantly ($p < .0001$). These variables remained stable at posttreatment. For participant 2, no improvement was observed from baseline to the first phase of treatment. When CBT was introduced, TST significantly declined and then increased during posttreatment ($ps < .001$). No further change in any sleep variables was observed for this participant. Participant 3 did not experience improvement during the first phase of treatment. This participant had to stop the medication at the sixth week of treatment because of side effects (e.g., nausea). In the second phase of treatment, a significant improvement in SE was obtained ($p < .001$) as well as in TWT ($p < .01$). All variables remained stable at posttreatment. For participant 4, all variables improved following the introduction of medication ($ps < .01$). When CBT was introduced, TST worsened ($p < .01$). All variables significantly worsened at posttreatment ($ps < .0001$), even though they remained improved over baseline level. Participant 5 showed no significant change on any sleep variables before the introduction of CBT. Then, SE and TWT improved ($ps < .001$). Also, TST worsened during this period ($p < .01$). From the second phase of treatment to posttreatment, SE improved ($p < .01$) while TST and TWT remained stable.

Combined → CBT sequence. Results for this treatment sequence are presented in Table 2 and Figure 2. All five participants improved on SE. Four participants (6, 8, 9, and 10) improved on TWT during the first phase of treatment, while TST remained stable during this same period. Sleep remained improved for these four participants when medication was withdrawn, but the other participant (7) returned to baseline level.

For participant 6, SE and TWT significantly improved (*p*s < .001 and .0001, respectively) while TST worsened significantly following the introduction of the combined treatment ($p < .01$). During the second phase of treatment, all sleep variables remained stable while SE and TWT returned to their baseline level during posttreatment (*p*s < .01). For participant 7, a significant increase in SE was made during the first phase of treatment ($p < .001$). When medication was gradually withdrawn, during the second phase, SE, TWT, and TST returned progressively to their baseline levels (*p*s < .01). From this second phase of treatment to posttreatment, all variables remained stable except for TWT that significantly worsened ($p < .001$). Participant 8 showed improvements in SE and in TWT after the introduction of treatment (*p*s < .001). No further change in any variables was observed before posttreatment. Participant 9 presented significant improvement in SE and TWT during the first phase of treatment (*p*s < .001). These three variables remained stable through posttreatment, and TST improved during this period ($p < .001$). Participant 10 showed improvements in SE and TWT during the first phase of treatment (*p*s < .01). There was no further change before posttreatment, when SE increased and TWT decreased (*p*s < .001). During the same period, a significant deterioration in TST was also observed ($p < .0001$).

CBT alone. Results for this treatment sequence are presented in Table 3 and Figure 3. All participants improved in SE and four of them (12, 14, 15, and 16) improved in TWT during the first phase of treatment. Three participants (12, 14, and 15) also showed a decrease in TST during this period. During the second part of treatment, one participant (11) improved further on SE and TST while the other participants remained stable.

Participant 11 improved significantly in SE ($p < .0001$) from baseline through the first

five weeks of treatment. During the second phase of treatment, SE and TST improved (*p*s < .0001). There was no further change from the second five weeks of treatment through posttreatment. For participant 12, significant improvements were observed in SE and in TWT (*p*s < .01) from baseline to the first phase of treatment. During the same period, TST was significantly reduced (*p* < .01). During the second phase of treatment, all sleep variables remained stable. At posttreatment, SE and TWT worsened (*p*s < .01), although these variables did not return to their baseline level. Participant 13 experienced a significant increase in SE after the initial introduction of CBT (*p* < .01). No further change was observed. Participant 14 improved in SE and TWT (*p*s < .0001), while TST worsened (*p* < .01) during the first phase of treatment. All variables remained stable during the second phase of treatment. At posttreatment, SE and TWT worsened (*p*s < .01), but none of these variables returned to baseline level. Participant 15 improved on SE and TWT (*p*s < .0001) from baseline to the first phase of treatment, but TST deteriorated (*p*s <.001). This participant began working on night shift during the seventh week of treatment. When comparing the first and the second five weeks of treatment, SE and TWT significantly worsened (*p*s < .001). All variables remained stable at posttreatment. For participant 16, SE and TWT improved after the introduction of CBT (*p*s < .01). From the first to the second phase of treatment, sleep variables were stable. Even though SE and TWT worsened (*p*s < .01 and .001, respectively), they did not return to their baseline level at posttreatment.

Follow-up

Three months follow-up sleep diary data were available for 13 of the 16 participants (see Tables 1, 2 and 3 as well as Figures 1, 2 and 3). Comparison of these data with posttreatment data reveals an additional improvement in SE for participant 2 in the Medication → Combined sequence, for participant 7 in the Combined → CBT sequence and for participants 11 and 15 in the CBT alone condition (*p*s < .001 and .0001, respectively). Although some improvements were still evident relative to baseline levels, a significant deterioration was observed for one participant in each sequence (3, 10, and 14) (*p*s < .01 and < .0001, respectively), while all other participants remained stable on this variable. Also, a significant improvement in TST

was obtained for one participant (2) in the Medication → CBT sequence, for two participants (7 and 10) in the Combined → CBT sequence, and for five participants (11, 12, 14, 15, and 16) in the CBT alone condition (*p*s < .01). TST for all other participants remained stable. TWT worsened and returned to baseline level only for participant 10 in the Combined → CBT sequence ($p < .01$). TWT significantly improved for participant 7 in the Combined → CBT sequence and for two participants (11 and 15) in the CBT alone condition (*p*s < .0001).

Polysomnographic data

Means and standard deviations for all PSG measures at the three assessment periods (baseline, mid-treatment, and posttreatment) are presented in Table 5. Note that participant 7 in the Combined → CBT sequence, who relapsed when medication was withdrawn, was removed from the analyses data set at posttreatment because of extreme data (see Roth, 1994). Friedman non parametric tests showed significant improvement in SE and TWT from pre- to mid-treatment for the Combined → CBT sequence ($\chi^2_{(2)}$s = 6.53 and 6.00, respectively, *p*s < .05) and for CBT alone ($\chi^2_{(2)}$s = 6.00 and 6.00, respectively, *p*s < .05). These changes were maintained at posttreatment. Despite the presence of a decreasing trend in TWT for participants in the Medication → Combined sequence, this was not significant. In addition, Kruskal-Wallis non parametric tests revealed that SE was higher and TWT lower in the Combined→ CBT sequence than in the two other sequences at mid-treatment only ($\chi^2_{(2)}$s = 6.77 and 6.30, respectively, *p*s < .05). No difference was observed across treatment sequences and over time in any of sleep stages.

Attributional effect

Figure 4 presents the weekly evolution of the attribution of therapeutic gains to medication, behaviour, and beliefs for each treatment sequence. Visual inspection indicated that during the first phase of treatment, participants in the Medication → Combined sequence attributed more their sleep change to medication than those in the Combined → CBT sequence. During the second phase of treatment, this difference between treatment sequences

were no longer present. Non parametric tests showed that the attribution of treatment gains to medication was significantly higher for the Medication → Combined sequence than for the Combined → CBT sequence and CBT alone at mid-treatment ($\chi^2_{(2)}$ = 11,56, p < .01). Also, Friedman's non parametric test showed that for the Medication → Combined sequence, attribution to medication was significantly higher at mid-treatment than at posttreatment ($\chi^2_{(2)}$ = 7.60, p < .04). At posttreatment, Pearson correlation analyses performed on the *Attributional Questionnaire* scores showed that increases in perceived self-efficacy were positively related to an increase in the attribution of therapeutic gains to changes in behaviours and beliefs (rs = .87 and .70, respectively, ps < .05).

Psychological assessment

Means and standard deviations for all self-report measures at the four assessment periods (baseline, mid-treatment, posttreatment, and 3-month follow-up) are presented in Table 6. The results showed a significant decrease in insomnia severity from baseline to middle of treatment for each treatment sequence as evidenced by the ISI measures completed by the participants ($\chi^2_{(2)}$s = 7.68, 7.60, and 11.57, respectively, ps < .02 and p < .003). The ISI measures completed by the clinician and the significant others showed a decrease in insomnia severity from baseline to mid-treatment for the Combined → CBT sequence only ($\chi^2_{(2)}$ = 6.63 and 6.50, respectively, ps < .05). These changes were maintained at posttreatment and follow-up. There was also a significant decrease in the DBAS scale from baseline to middle of treatment and posttreatment for the Combined → CBT sequence and for CBT alone ($\chi^2_{(2)}$s = 9.58 and 8.40, respectively, ps < .008 and < .05). Visual inspection of the DBAS means for the Medication → Combined sequence showed a tendency towards an increase from baseline to middle of treatment and towards a decrease at posttreatment. Depressive symptoms (*BDI*) and the tendency to worry (*PSWQ*) were reduced for each sequence, but these changes reached statistical significance for CBT alone only from baseline to mid-treatment ($\chi^2_{(2)}$s = 6.00 and 6.35, respectively, ps < .05). The *Withdrawal Symptom Evaluation Scale* completed by both the physician and the participants indicated that only participant 3 reported withdrawal symptoms (e.g., nausea). Finally, all participants who were taking zopiclone reported

experiencing a metallic bitter taste that was not particularly disturbing as a side effect.

Treatment compliance and clinical improvement

The average weekly observance of treatment procedures in the Medication → Combined sequence varied from 100%, when there was only medication, to 67.6% when CBT was added. The average treatment observance in the Combined → CBT sequence varied from 100% in the first week to 72.8% at the end of treatment. Sleep diary reports of medication intake indicated that five participants (3, 5, 6, 8, and 9) took one half pill or two pills fewer than prescribed. Also, participants 4 and 5 took one pill more than prescribed at week 5. Finally, during gradual withdrawal, participants 4 and 8 took a half pill fewer and stopped their intake earlier than prescribed. The medication intake at the 3-month follow-up corresponded to the significant others' reports. In CBT alone, the average treatment observance varied from 54.8% to 80%.

Clinical outcome ratings were computed at the middle of treatment, at posttreatment, and at the 3-month follow-up assessment. Based on the criteria outlined above, eight participants (3, 4, 5, 9, 10, 11, 13, 16) met criteria for a minimal treatment response, six (2, 6, 7, 12, 14, and 15) achieved a moderate response, and two (1 and 8) had an excellent treatment response by the middle of treatment. At posttreatment, six (4, 5, 6, 7, 11, and 15) had reached a minimal treatment response, five (1, 8, 9, 13, and 16) a moderate response, and five (2, 3, 10, 12, and 14) an excellent response. At 3-month follow-up, four of the 13 participants (8, 10, 13, and 15) met criteria for a minimal response, three (4, 7, and 9) for a moderate response and six (2, 3, 6, 12, 14, and 16) for an excellent one. Two participants (7 and 12) asked to receive the CBT booster session one month after posttreatment. One of these participants was in the Medication → Combined sequence while the other was in the CBT alone.

When questioned at posttreatment regarding their satisfaction with the treatment, participants reported being satisfied and more confident about being able to sleep, regardless of the treatment sequence received. This observation was supported by the absence of a

significant difference among participants on the *Treatment Acceptability Scale*. Furthermore, only participant 3, in the Medication → Combined sequence, would have preferred to receive one of the other sequences.

Discussion

The present findings indicated that the sequential combination of drug and behaviour therapies is effective for chronic insomnia. Each sequence led to significant improvement of sleep efficiency and continuity, but at different points in time. For the Medication → Combined sequence, most of the improvement obtained was a reduction of TWT after the introduction of CBT, but TST was also reduced during this period. For the Combined → CBT sequence, most variables improved during the first treatment phase and TST remained stable up to posttreatment. All participants in CBT alone improved significantly on all sleep variables during the first phase of treatment; however, three participants also experienced a reduction of TST. In the second phase of treatment, all variables remained stable. Polysomnographic measures corroborated sleep diary results. The attribution of therapeutic gains varied across treatment sequences and over time. Also, mood was improved and worry was diminished for all participants. Follow-up results suggest that therapeutic gains were maintained for most participants in the Combined → CBT sequence and in CBT alone. Long-term results of the Medication → Combined sequence were more variable.

Taken together, these results extend our preliminary findings (Vallières et al., 2001) and provide additional evidence that the sequential combination of behavioural and drug therapies is effective for chronic primary insomnia. They suggest that an initial combination of CBT and medication optimises treatment effects while protecting against a reduction in total sleep time. With this treatment sequence, patients do not suffer from an initial sleep reduction, as is observed with CBT alone. Results of the Medication → Combined sequence were not as favourable as expected during the first weeks of treatment considering that medication is known to be rapidly effective (Holbrook et al., 2000). This rapid effect was not necessarily

absent in the sequence. Indeed, visual inspection of participants 1, 2, and 4 showed an increase in sleep efficiency during the first week. However, these changes may have gone undetected because of the analysis used. Regardless, these surprising results underline the importance of introducing CBT when the medication is still effective and of testing this in a controlled group.

Overall, these results further corroborate earlier reports (Hauri, 1997; McClusky et al., 1991; Milby et al., 1993; Morin, Colecchi, et al., 1999; Rosen et al., 2000) on the importance of integrating CBT as an essential component of insomnia treatment. Indeed, in the present study, most of the sleep continuity improvements occurred only following the introduction of CBT. Finally, follow-up results indicate a stability in sleep continuity for the Combined → CBT sequence. In addition, any reduction in total sleep time experienced during treatment was recovered, and even improved, at the 3-month follow-up. This rebound/delayed effect has already been observed in other treatment studies involving CBT (e.g., McClusky et al., 1991; Morin, Colechi, et al., 1999). Interestingly, these results suggest that a short combination of both treatments, followed by CBT alone, could yield better long-term outcome than a concurrent combination of both treatments. However, if CBT is as effective as the Combined → CBT sequence in the long run, then the principal advantage of this sequence would be observable during the first weeks of treatment. Therefore, using this treatment sequence would be a better choice for patients who might benefit from this early advantage. However, the patients' characteristics indicating who would benefit from this advantage need to be determined.

Another issue that emerges from this study pertains to the number of consultation sessions needed to deliver treatment. It seems that five treatment sessions provided on a bi-weekly basis are enough to produce both short- and long-term improvements. Indeed, CBT alone led to better short-term and long-term results than the Medication → Combined sequence, even if they both involved five treatment sessions. In addition, the Combined → CBT sequence and CBT alone seemed equally effective even if the former sequence involved a total of 10 CBT sessions. Thus, given that behavioural and cognitive procedures were

introduced at the same time, regardless of sequence, it would appear that elapsed time between CBT sessions and the overall duration of treatment are essential to produce sleep improvement. This concurs with the result obtained by Edinger, Wohlgemuth, Radtke, and Marsh (2000) on the dose response effect of CBT. They demonstrated that only one or two CBT sessions which they consider as low treatment dose, over eight weeks of treatment were necessary if there was sufficient time to implement treatment recommendations during CBT sessions. With this in mind, the Combined → CBT sequence would probably have been as effective with five sessions administered over 10 weeks.

Exploratory findings on attributional effects demonstrate that participants in the medication alone present the highest attribution gains to medication. Also, participants with sleep deterioration during medication withdrawal, present the highest overall attribution of therapeutic gains to medication. Therefore, exploratory findings raise two questions. First, has the attribution gains to medication an impact on sleep improvement? Second, what would be the mechanism underlying this impact on sleep? A putative mechanism might work in such a way that changes in beliefs and behaviours are related to a lower attributional effect to medication which in turn is related to an increase in sleep efficiency. These findings concurs with the notion advanced in the literature (Morin, Hauri, et al., 1999; Stepanski & Wyatt, 2000) that a sequential treatment in which CBT is continued following medication withdrawal provides a better opportunity to integrate behavioural procedures and to develop greater confidence in controlling sleep. In the absence of a comparison condition in which CBT was discontinued at the same time as medication, this interpretation remained tentative.

According to the present findings, sequential treatment presents several advantages for clinical practice. First, a patient waiting to receive CBT or a patient who has just begun CBT could benefit from an early and short trial on medication. Indeed, this treatment sequence could minimise the initial reduction of sleep time when CBT is introduced first. Second, the degree of satisfaction and the high degree of compliance suggest that sequential treatment would be well accepted by patients in clinical practice.

Other treatment sequences, even more representative of clinical realities, could be tested. One possibility, would be a treatment sequence beginning with medication alone, followed by a combined treatment, and then medication withdrawal while CBT is continued. This sequence would look like the overlapping sequence in the Vallières et al. (2001) study. Another possibility would involve testing the Combined → CBT sequence with one treatment session every two weeks or, testing a sequence applying CBT first and, if the patient does not respond to treatment, adding medication. Additional studies are needed to investigate these treatment implementation models.

The study design poses some methodological limitations. First, given the absence of a placebo or waiting list control group, it is impossible to directly compare treatment sequences. However, the multiple baseline across subjects design used in this study allows to conclude that, if changes always follow treatment implementation, then those changes are likely due to treatment rather than to other factors such as history or maturation. A second limitation is the use of an attributional measure with yet undocumented psychometric properties. Although these results should be interpreted cautiously, the growing acknowledgement of the potential impact of the negative attributional effect of medication justifies its use in an exploratory study.

In conclusion, this study is the first to investigate the sequential combination of drug and behavioural therapies for insomnia. The results open the door to a promising new way of treating insomnia. The present sample represents a fairly homogenous insomniac population, precluding generalisation to milder forms of insomnia or to secondary insomnia. Therefore, additional clinical trials should be conducted with larger samples and with control groups. The Combined → CBT sequence should be contrasted against a control group, a CBT alone, and a concurrent combination of treatments. The Medication → Combined sequence could be modified and tested with a condition involving continuation of CBT after the medication withdrawal phase. In addition, the attribution effect should be further investigated in order to develop the best sequential combination of behaviour and drug therapies. Longer follow-up assessments should also be incorporated in future studies, even though the present results

suggest that treatment gains are stable over time. The long-term impact of booster follow-up additional treatment sessions in the months following the end of treatment should also be evaluated.

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Table 1

Predictors and direction of change between treatment phases for each sleep variable and participant in the Medication → Combined sequence (n = 5)

	Baseline vs. Phase 1			Phase 1 vs. Phase 2			Phase 2 vs. Posttreatment			Posttreatment vs. FU-3		
	Predictor of change	R^2	Direction of change	Predictor of change	R^2	Direction of change	Predictor of change	R^2	Direction of change	Predictor of change	R^2	Direction of change
Sleep Efficiency												
P1	Level	60.6^{***}	+	Quadratic	26.5^{*}	+	Time	23.2^{*}	0	N/A	N/A	N/A
P2	Time	36.5^{***}	0	Time	18.4^{*}	0	None		0	Level	56.4^{**}	+
P3	None		0	Level	66.5^{**}	+	Time	70.9^{*}	0	Fraction	29.5^{*}	-
P4	Quadratic	19.2^{**}	+	Time	35.7^{***}	0	Fraction	10.4^{***}	-	None		0
P5	None		0	Level	31.6^{***}	+	Level	26.6^{*}	+	N/A	N/A	N/A
Total Sleep Time												
P1	Time	47.1^{***}	0	Level	39.2^{***}	-	None		0	N/A	N/A	N/A
P2	None		0	Level	22.6^{**}	-	Time	18.2^{**}	0	Level	38.7^{***}	+
P3	None		0	Time	22.9^{*}	0	Time	26.9^{***}	0	None		0
P4	Level	14.4^{**}	+	Level	10.6^{*}	-	Logarithm	31.0^{***}	-	None		0
P5	None		0	Logarithm	25.7^{*}	-	None		0	N/A	N/A	N/A
Total Wake Time												
P1	Level	44.2^{***}	+	Quadratic	35.4^{*}	+	Time	29.2^{*}	0	N/A	N/A	N/A
P2	Time	26.4^{***}	0	Time	13.0^{*}	0	None		0	No		0
P3	None		0	Level	71.4^{*}	+	Time	81.4^{*}	0	Level	30.9^{*}	-
P4	Quadratic	22.3^{*}	+	Time	44.7^{***}	0	Level	17.8^{**}	-	No		0
P5	None		0	Level	41.8^{***}	+	Time	28.9^{***}	0	N/A	N/A	N/A

Note. P1 to P6 = Participant1 to participant 6; + = improved; 0 = stable; - = worsened; N/A = not available; Phase 1 = first five weeks of treatment; Phase 2 = second five weeks of treatment.

$^{**}p < .01$. $^{***}p < .001$. $^{***}p < .0001$.

Table 2

Predictors and direction of change between treatment phases for each sleep variable and participant in the Combined → CBT sequence (n = 5)

	Baseline vs. Phase 1			Phase 1 vs. Phase 2			Phase 2 vs. Posttreatment			Posttreatment vs. FU-3		
	Predictor of change	R^2	Direction of change	Predictor of change	R^2	Direction of change	Predictor of change	R^2	Direction of change	Predictor of change	R^2	Direction of change
Sleep Efficiency												
P6	Level	19.6^{**}	+	None		0	Level	36.6^{*}	-	None		0
P7	Level	61.2^{***}	+	Quadratic	45.9^{*}	-	Time	54.9^{***}	0	Level	90.6^{***}	+
P8	Level	73.7^{***}	+	None		0	Time	9.5^{*}	0	None		0
P9	Linear	52.5^{***}	+	Time	38.1^{**}	0	Time	41.1^{**}	+	None		0
P10	Linear	10.9^{*}	+	Time	10.6^{*}	0	Fraction	23.8^{**}	+	Level	65.8^{***}	-
Total Sleep Time												
P6	Logarithm	11.7^{*}	-	None		0	None		0	Time	30.9^{**}	0
P7	Time	34.4^{***}	0	Quadratic	51.0^{***}	-	Time	46.9^{***}	0	Level	89.1^{***}	+
P8	Time	46.1^{***}	0	None		0	None		0	Time	18.3^{*}	0
P9	None		0	None		0	Level	64.6^{***}	+	None		0
P10	None		0	None		0	Level	45.8^{***}	-	Level	82.1^{*}	+
Total Wake Time												
P6	Level	29.2^{***}	+	None		0	Level	23.0^{*}	-	Time	21.7^{*}	0
P7	None		0	Linear	37.0^{*}	-	Level	44.7^{**}	-	Level	63.1^{***}	+
P8	Level	77.5^{**}	+	None		0	None		0	None		0
P9	Root	67.9^{***}	+	Time	53.7^{*}	0	Time	38.4^{*}	0	Level	21.7^{*}	-
P10	Root	19.1^{*}	+	Time	13.0^{*}	0	Level	27.6^{**}	+	Level	68.4^{***}	-

Note. P1 to P6 = Participant1 to participant 6; + = improved; 0 = stable; - = worsened; Phase 1 = first five weeks of treatment; Phase 2 = second five weeks of treatment.

$^{*}p < .01.$ $^{**}p < .001.$ $^{***}p < .0001.$

Table 3

Predictors and direction of change between treatment phases for each sleep variable and participant in CBT alone (n= 6)

	Baseline vs. Phase 1			Phase 1 vs. Phase 2			Phase 2 vs. Posttreatment			Posttreatment vs. FU-3		
	Predictor of change	R^2	Direction of change	Predictor of change	R^2	Direction of change	Predictor of change	R^2	Direction of change	Predictor of change	R^2	Direction of change
Sleep Efficiency												
P11	Root	22.6***	+	Fraction	34.8***	+	None		0	Logarithm	63.1***	+
P12	Quadratic	7.3*	+	Time	42.8***	0	Level	8.3*	-	None		0
P13	Linear	24.8***	+	Time	52.36***	0	Time	20.6*	0	N/A	N/A	N/A
P14	Level	39.0***	+	Time	19.8***	0	Level	21.1*	-	Linear	19.3*	-
P15	Level	45.4***	+	Quadratic	61.4**	-	Time	54.2*	0	Level	74.2***	+
P16	Fraction	10.0*	+	Time	10.7*	0	Linear	30.1*	-	None		0
Total Sleep Time												
P11	Time	25.3*	0	Fraction	45.5**	+	Time	64.2***	0	Quadratic	43.0*	+
P12	Level	25.0***	-	Time	16.3*	0	None		0	Level	19.2**	+
P13	None		0	Time	49.2***	0	None		0	N/A	N/A	N/A
P14	Fraction	13.5*	-	Time	61.9***	0	Time	17.2*	0	Root	38.3**	+
P15	Level	25.7**	-	None		0	None		0	Level	82.5***	+
P16	None		0	None		0	None		0	Level	40.2***	+
Total Wake Time												
P11	Time	29.0*	0	Time	43.9***	0	None		0	Logarithm	64.0***	+
P12	Quadratic	26.5*	+	Time	49.6***	0	Level	11.3*	-	Fraction	21.0***	-
P13	Time	49.7**	0	Time	53.4***	0	Time	22.8**	0	N/A	N/A	N/A
P14	Level	50.2***	+	Time	27.1***	0	Root	17.2*	-	Root	38.3**	-
P15	Level	53.7***	+	Quadratic	65.1**	+	Time	57.8*	0	Level	68.0***	+
P16	Fraction	22.9*	+	Time	16.8**	0	Quadratic	25.1**	-	None		0

Note. P1 to P6 = Participant1 to participant 6; + = improved; 0 = stable; - = worsened; N/A = not available; phase 1 = first five weeks of treatment; phase 2 = second five weeks of treatment.

* $p < .01$. ** $p < .001$. *** $p < .0001$.

Table 4

Summary of the number of participants improved on sleep efficiency for each treatment sequence

Comparisons	Medication → Combined (n = 5)			Combined → CBT (n = 5)			CBT alone (n = 6)		
	Participants			Participants			Participants		
	improved	stable	worsened	improved	stable	worsened	Improved	stable	worsened
Baseline vs. Phase 1	2/5	3/5	0/5	5/5	0/5	0/5	6/6	0/6	0/6
Phase 1 vs. Phase 2	3/5	2/5	0/5	0/5	4/5	1/5	1/6	4/6	1/6
Phase 2 vs. Posttreatment	1/5	3/5	1/5	2/5	1/5	2/5	0/6	3/6	3/6
Posttreatment vs. FU-3	1/3	1/3	1/3	1/5	1/5	3/5	2/6	2/6	1/6

Note. Phase 1 = first five weeks of treatment; Phase 2 = second five weeks of treatment; FU-3 = 3-month follow-up assessment.

Table 5
Means (M) and standard deviations (SD) for polysomnographic data for each treatment sequence

Sleep Parameters	Treatment sequences		
	Medication → Combined	Combined → CBT	CBT
	M (*SD*) n	*M* (*SD*) n	*M* (*SD*) n
Sleep onset latency			
Baseline	14.18 (9.47) 5	13.84[1] (4.27) 5	26.07 (19.61) 6
Middle of treatment	18.92[a] (14.04) 5	6.18[b,2] (1.84) 5	9.48[a] (3.36) 4
Posttreatment	11.93 (5.17) 4	6.78[2] (2.55) 4	6.37 (3.17) 3
Total wake time			
Baseline	67.24 (30.19) 5	67.50[1] (31.21) 5	99.23[1] (57.24) 6
Middle of treatment	55.95[a] (14.24) 5	24.02[b,2] (13.16) 5	50.60[a,2] (22.38) 4
Posttreatment	40.60 (30.74) 4	20.48[2] (4.54) 4	34.17[3] (31.42) 3
Total sleep time			
Baseline	389.66 (34.96) 5	387.26 (51.02) 5	332.83 (105.97) 6
Middle of treatment	336.84 (68.26) 5	372.32 (40.33) 5	293.13 (34.87) 4
Posttreatment	332.78 (84.30) 4	366.58 (26.57) 4	335.77 (77.80) 3
Sleep efficiency (%)			
Baseline	86.18 (6.56) 5	85.84[1] (7.18) 5	76.35[1] (18.54) 6
Middle of treatment	85.95[a] (4.87) 5	94.46[b,2] (3.19) 5	85.65[a,2] (7.34) 4
Posttreatment	88.70 (11.59) 4	95.45[2] (1.40) 4	90.97[3] (9.90) 3
% Stage 1			
Baseline	7.20 (2.08) 5	6.72 (1.31) 5	9.22 (2.86) 6
Middle of treatment	6.73(2.96) 5	3.42 (0.85) 5	7.53 (3.01) 4
Posttreatment	8.95 (4.00) 4	3.43 (1.06) 4	5.97 (3.81) 2
% Stage 2			
Baseline	60.90(3.53) 5	59.47 (6.58) 5	64.30 (7.51) 6
Middle of treatment	65.29 (5.65) 5	62.76 (4.31) 5	52.38 (11.10) 4
Posttreatment	63.40 (10.97) 4	61.75 (8.18) 4	59.53 (7.31) 2
% Stage 3-4			
Baseline	6.70 (7.20) 5	10.00 (5.07) 5	7.52 (8.66) 6
Middle of treatment	7.20 (9.02) 5	10.80 (6.87) 5	10.15 (10.56) 4
Posttreatment	3.90 (5.16) 4	9.18 (6.14) 4	10.07 (13.42) 3
% Stage REM			
Baseline	25.30 (2.87) 5	24.64 (2.81) 5	19.03 (6.34) 6
Middle of treatment	21.90 (3.29) 5	24.16 (2.69) 5	22.68 (2.67) 4
Posttreatment	23.75 (8.35) 4	25.75 (15.37) 4	24.60 (3.45) 3

Note. CBT = Cognitive Behavioural Treatment. Means in the same row or column that do not share the same subscripts (a and b or 1 and 2) differ at $p < .05$.

Table 6

Means (M) and standard deviations (SD) of self-report measures for each treatment sequence

Sleep Parameters	Treatment sequences		
	Medication → Combined	Combined → CBT	CBT
	M (*SD*) n	*M* (*SD*) n	*M* (*SD*) n
ISI participant			
Baseline	21.33[1] (2.58)6	22.40[1] (4.56) 5	19.17[1] (3.43) 6
Middle of treatment	15.60[2] (2.79) 5	10.00[2] (6.12) 5	11.83[2] (4.07) 6
Posttreatment	11.20[2] (6.22) 5	11.00[2] (3.08) 5	8.50[2] (4.32) 6
Fu-3	7.00 (4.58) 3	10.40 (4.04) 5	9.17 (5.04) 6
ISI clinician			
Baseline	14.83[1] (3.54) 6	16.20[1] (3.11) 5	13.17[1] (3.19) 6
Middle of treatment	10.80[1](5.81) 5	10.80[2] (3.27) 5	9.83[1] (3.19) 6
Posttreatment	6.20[2] (4.44) 5	9.40[2] (5.86) 5	5.67[2] (3.67) 6
Fu-3	N/A	N/A	N/A
ISI other			
Baseline	20.00 (2.45) 5	20.40[1] (4.88) 5	15.50 (3.32) 4
Middle of treatment	16.13 (2.66) 4	12.00[2] (3.74) 4	14.10 (5.79) 5
Posttreatment	11.33 (6.66) 3	11.00[2] (1.83) 4	10.10 (3.91) 5
Fu-3	3.00 (.) 1	10.25 (4.99) 4	10.40 (6.47) 5
DBAS			
Baseline	39.10 (12.52) 6	47.80[1] (13.59) 5	50.85[1] (8.07) 6
Middle of treatment	41.56 (9.32) 5	32.20[2] (8.56) 5	38.78[2] (6.61) 6
Posttreatment	26.82 (11.52) 5	22.80[2] (8.76) 5	24.34[2] (8.88) 5
Fu-3	17.50 (14.85) 2	24.50 (11.70) 4	27.00 (9.31) 4
BDI			
Baseline	10.17 (4.92) 6	9.00 (2.12) 5	10.83[1] (6.05) 6
Middle of treatment	7.40 (4.92) 5	5.60 (3.97) 5	6.17[2] (5.19) 6
Posttreatment	6.20 (2.77) 5	5.00 (5.29) 5	6.33[2] (3.27) 6
Fu-3	5.67 (3.06) 3	4.00 (3.32) 5	6.33 (3.56) 6
PSWQ			
Baseline	45.83 (10.72) 6	42.50 (10.34) 4	48.83[1] (8.23) 6
Middle of treatment	44.00 (5.22) 5	36.80 (4.87) 5	44.67[2] (7.58) 6
Posttreatment	39.20[a] (5.22) 5	36.00[a] (4.42) 5	47.00[b,1] (7.13) 6
Fu-3	43.00 (10.82) 3	36.40 (6.11) 5	42.00 (9.32) 6

Note. CBT = Cognitive Behavioral Treatment; ISI = Insomnia Severity Index; DBAS = Dysfunctional Beliefs and Attitudes about Sleep Scale; PSWQ = Penn State Worry Questionnaire; BDI = Beck Depression Inventory. N/A = does not apply. Means in the same row or column that do not share the same subscripts (a and b or 1 and 2) differ at $p < .05$.

Figure Captions

Figure 1. Daily change of sleep efficiency for participants in the Medication → Combined sequence.

Figure 2. Daily change of sleep efficiency for participants in the Combined → CBT sequence.

Figure 3. Daily change of sleep efficiency for participants in CBT alone.

Figure 4. Daily change of the weekly attribution of therapeutic gains to medication, behaviour and belief modification.

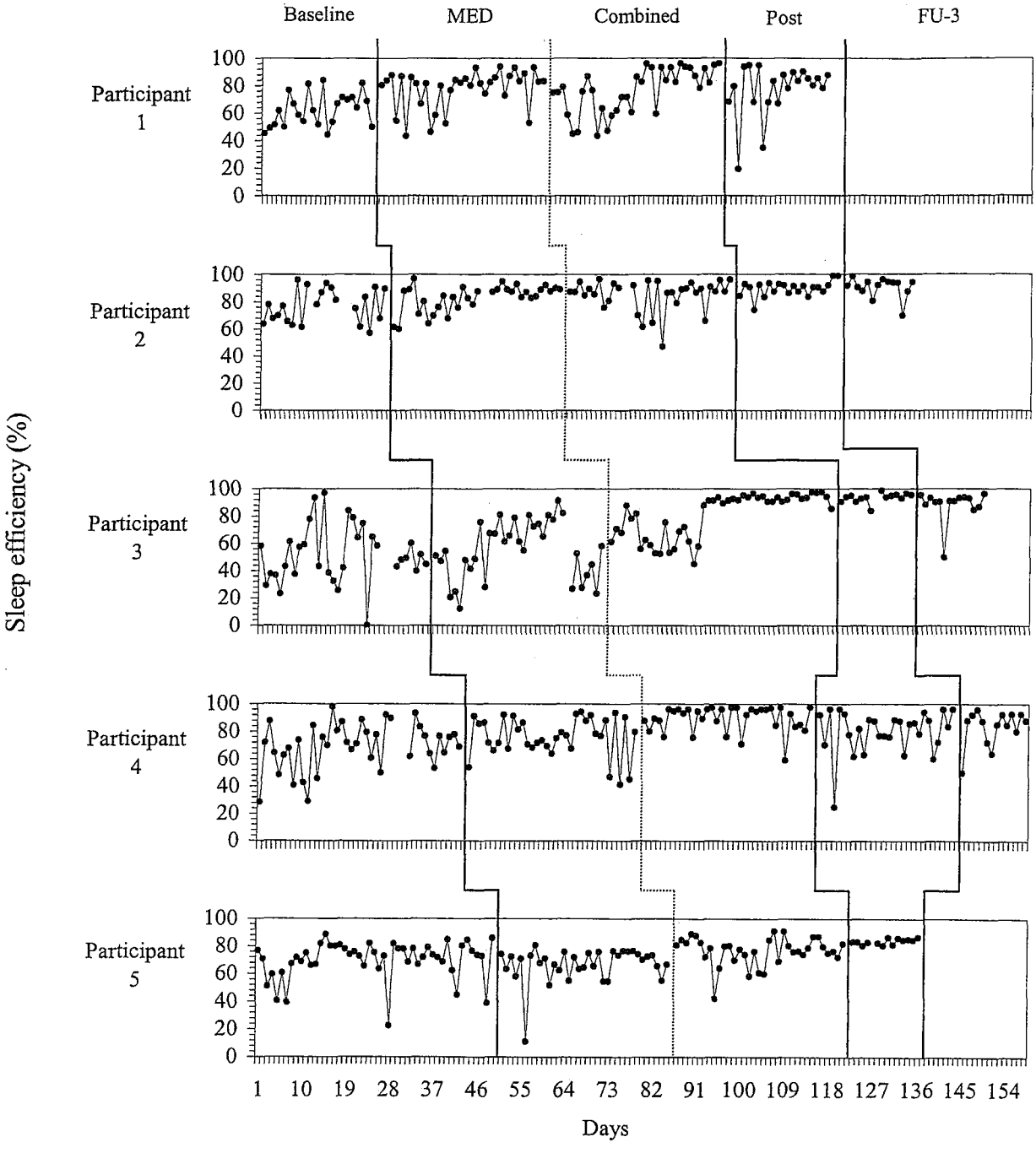

Baseline
MED
Combined
Post
FU-3
Participant 1
Participant 2
Participant 3
Participant 4
Participant 5
Sleep efficiency (%)
100
80
60
40
20
0
1 10 19 28 37 46 55 64 73 82 91 100 109 118 127 136 145 154
Days

Figure 1.

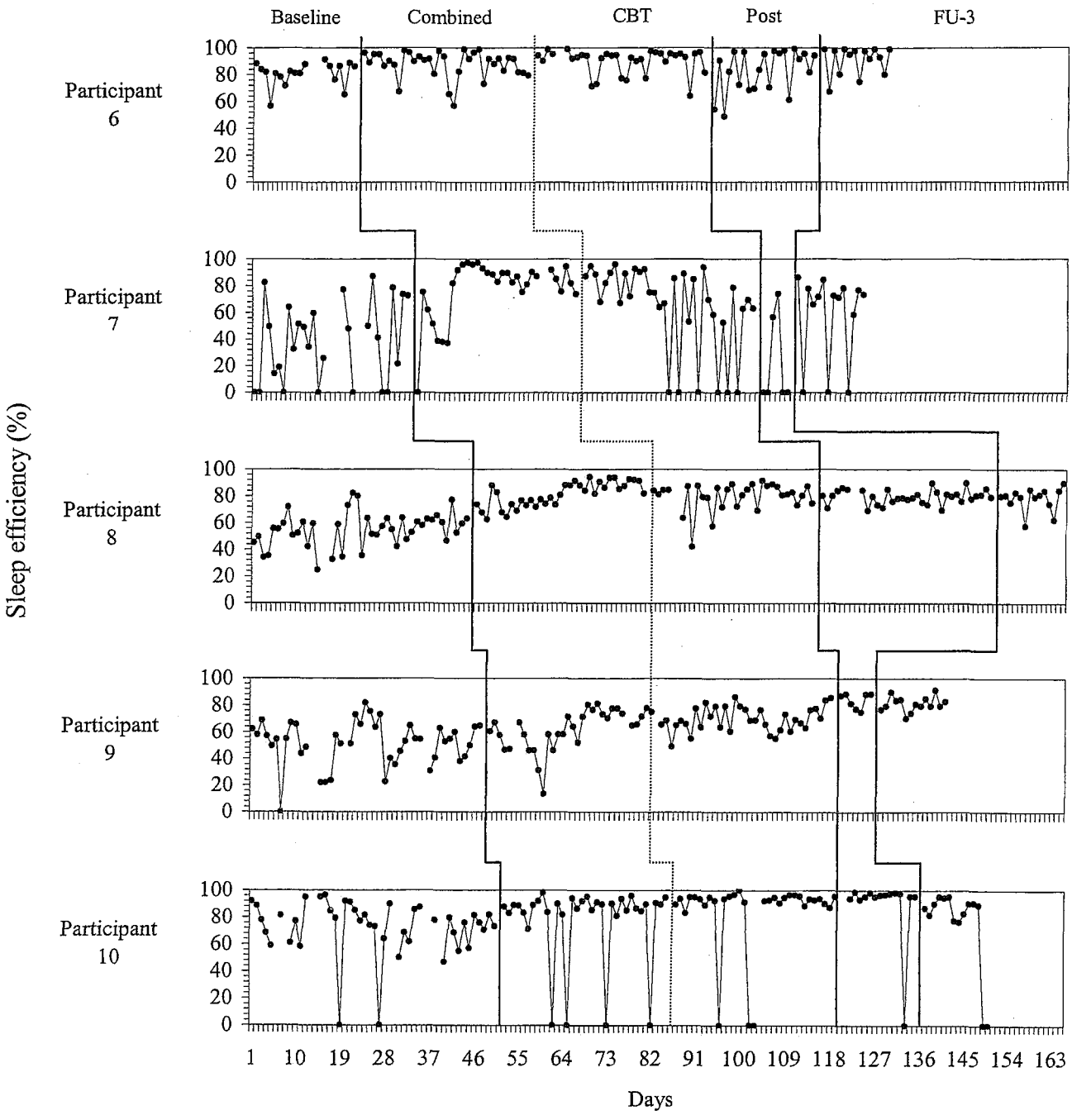

Baseline
Combined
CBT
Post
FU-3
Participant 6
Participant 7
Participant 8
Participant 9
Participant 10
Sleep efficiency (%)
0
20
40
60
80
100
1 10 19 28 37 46 55 64 73 82 91 100 109 118 127 136 145 154 163
Days

Figure 2.

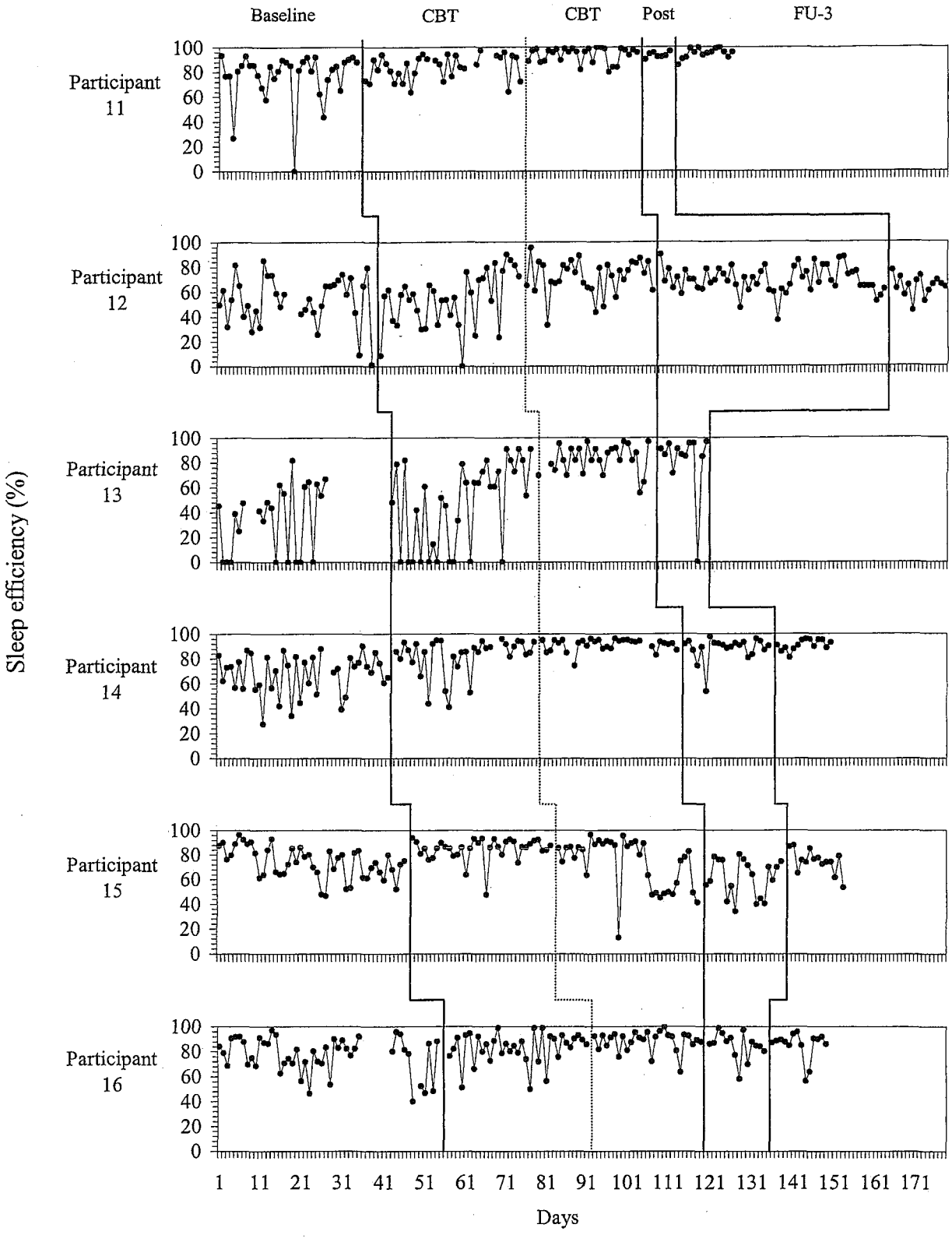
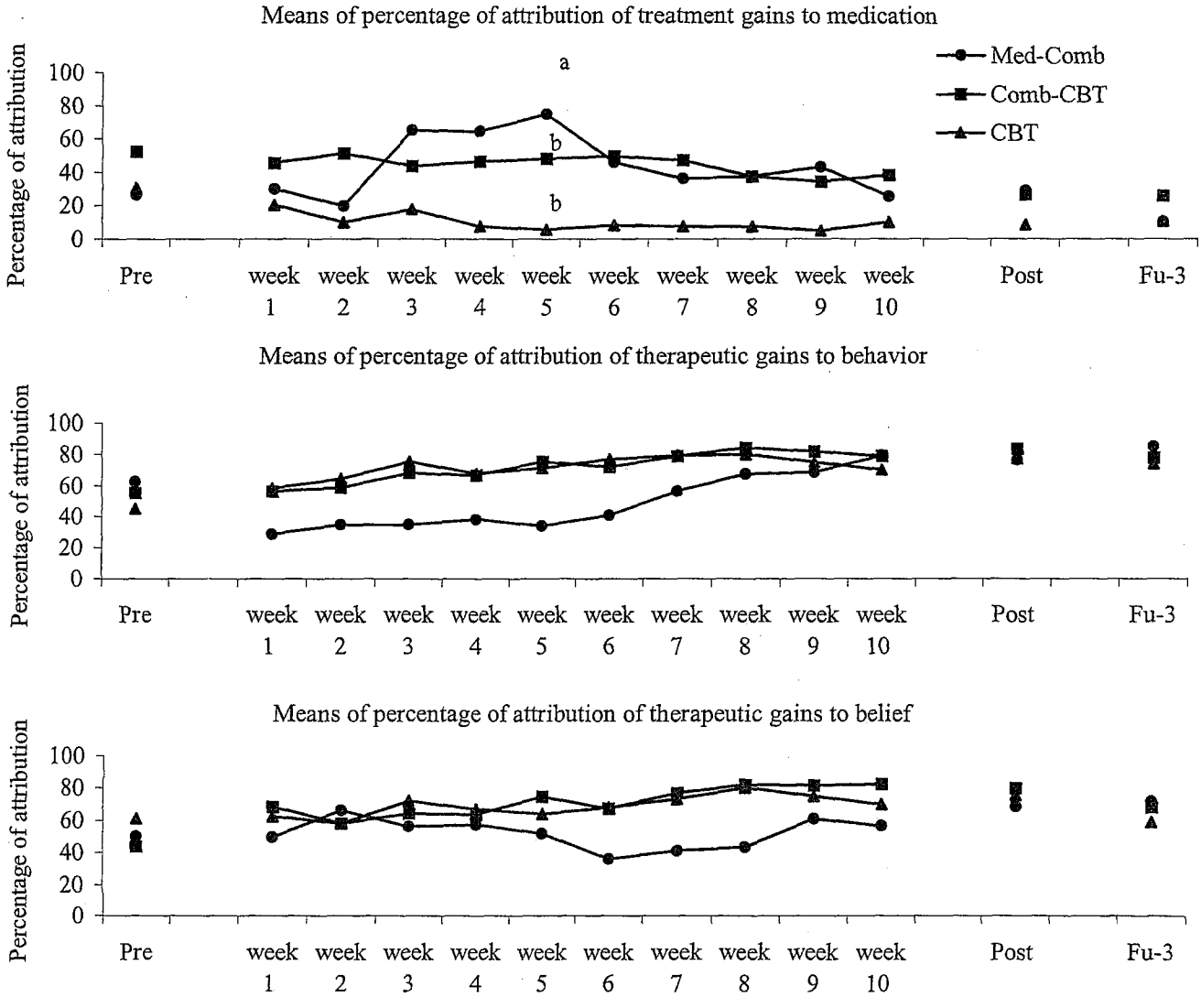

Baseline
CBT
CBT
Post
FU-3
Participant 11
Participant 12
Participant 13
Participant 14
Participant 15
Participant 16
Sleep efficiency (%)
0
20
40
60
80
100
1 11 21 31 41 51 61 71 81 91 101 111 121 131 141 151 161 171
Days

Figure 3.



Note. Non parametric tests were performed to compare means of percentage of attribution of gains to medication, behaviour, and belief among treatment sequences. Means in the same graph that do not share the same subscript differ at $p < .05$.

Figure 4.

CHAPITRE 3

L'ACTIGRAPHIE COMME OUTIL D'ÉVALUATION DE L'INSOMNIE CHRONIQUE

Résumé

Objectif: Cette étude explore l'utilité clinique et la sensibilité de l'actigraphie pour évaluer la réponse au traitement.

Méthode: Dix-sept participants souffrant d'insomnie chronique primaire ont été enrôlés dans un protocole de traitement étudiant différents traitements séquentiels pour l'insomnie (ces résultats sont reportés précédemment). Lors de leur évaluation, les participants dormaient trois nuits au laboratoire avant le traitement et deux nuits après le traitement. Durant ces nuits, les données provenant de la polysomnographie (PSG), de l'actigraphie ainsi que de l'agenda du sommeil ont été recueillies.

Résultats: L'actigraphie et l'agenda du sommeil sous-évaluent le temps total de sommeil et l'efficacité du sommeil par rapport à la PSG et surévaluent le temps total d'éveil. De plus, l'actigraphie sous-estime le délai d'endormissement alors que l'agenda du sommeil le surestime comparé à la PSG. Les résultats révèlent que les données de l'actigraphie sont plus précises que celles obtenues par l'agenda du sommeil et que l'actigraphie est sensible aux changements produits par le traitement.

Conclusions: Ces résultats suggèrent que l'actigraphie est un appareil efficace pour mesurer la réponse au traitement et qu'il devrait être utilisé en complément à l'agenda du sommeil.

Running head: ACTIGRAPHY IN THE ASSESSMENT

Actigraphy in the assessment of insomnia

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Abstract

Objective: The present study explores the clinical utility and sensitivity of actigraphy as an outcome measure in the treatment of chronic insomnia.

Method: Seventeen participants with primary chronic insomnia took part in a treatment protocol investigating different sequential treatments for insomnia (these results are reported elsewhere). As part of their assessment, polysomnography (PSG), actigraphy and sleep diary data were collected for three nights in the laboratory at baseline and for two nights at posttreatment.

Results: Compared to PSG both actigraphy and sleep diary instruments underestimated Total Sleep Time and Sleep Efficiency, and overestimated Total Wake Time. Also, actigraphy underestimated Sleep Onset Latency while sleep diary overestimated it as compared to PSG. Actigraphy data were found to be more accurate than sleep diary data when compared to PSG. Finally, actigraphy was sensitive to treatment effect.

Conclusions: These results suggest that actigraphy is a useful device for measuring treatment response and that it should be used as a complement to sleep diary evaluation.

Actigraphy in the assessment of insomnia

The understanding of the nature of insomnia requires the evaluation of many fundamental components such as nighttime sleep, daytime functioning, and variability in sleep-wake patterns (Sateia, Doghramji, Hauri, & Morin, 2000). Several assessment tools have been developed over the years to assess these components. Polysomnography (PSG) is considered the gold standard for the objective assessment of sleep whereas the sleep diary is the standard procedure for the subjective assessment of sleep (Sateia et al., 2000). PSG provides an accurate description of wake and sleep time as well as of sleep stage. However, it is an expensive approach and its ecological validity is sometimes questionable. The use of a daily sleep diary is recommended for evaluating sleep in the patient's home environment and can be used for a longer period of time (Sateia et al., 2000). Also, the sleep diary has the advantage of measuring night-to-night variability in sleep-wake patterns. However, the accuracy of the sleep diary is dependant on the patient's perception (Edinger & Fins, 1995). Therefore, objective assessment tools that are accurate, more accessible and more economical than PSG for evaluating sleep in a natural environment are needed.

Wrist actigraphy has been designed as an alternative objective evaluation tool [see Sadeh, Hauri, Kripke, & Lavie (1995) for more details]. The actigraph is a small, watch-like device, that records movements. It is worn all night on the wrist of the nondominant hand. The presence of movement is interpreted as time awake and the absence of movement as time asleep. The actigraph has the advantage of being a non-intrusive tool that can assess sleep in a natural environment. In spite of this advantage, its clinical usefulness in assessing insomnia is equivocal (Sadeh et al., 1995). Evaluation of actigraphy should be based on comparisons to same-night readings of PSG and sleep diary. To be useful, actigraphy data should correspond to PSG measures and be more accurate than sleep diary (Verbeek, Klip, & Declerck, 2001). Of the studies that have investigated the accuracy of actigraphy, five have compared PSG, actigraphy, and sleep diary recordings without comparing the same night of sleep (Jean-Louis et al., 1997; Hauri & Wisbey, 1992; Monk et al., 1994; Mullaney et al., 1980; Schmidt-Nowara, Beck, & Jessop, 1992). Only two studies (Friedman et al., 2000; Verbeek et al.,

1994) have reported results of these three measures based on the same night of sleep recording. Five studies compared actigraphy to sleep diary alone (Beck, Schmidt-Nowara, & Jessop, 1992; Brooks, Friedman, Bliwise, & Yesavage, 1993; Krahn et al., 1997; Monk et al., 1994; Wicklow & Espie, 2000; Wilson et al., 1998), and three studies (Brooks et al., 1993; Friedman et al., 2000; Schmidt-Nowara et al., 1992) evaluated the actigraphy's sensitivity to treatment.

These studies indicate that, depending on the participants' insomnia diagnosis types (e.g., psychophysiological or sleep-state misperception), actigraphy underestimates (Brooks et al., 1993) or overestimates total sleep time (Cole et al., 1992; Friedman et al., 2000; Hauri & Wisbey, 1992) compared to PSG. The agreement coefficients for total sleep time, as measured by PSG and actigraphy, varies from .81 (Mullaney et al., 1980) to .88 (Cole et al., 1992) and is lower than for good sleepers (above 90% see Sadeh et al., 1995). The magnitude of the discrepancies between those measures vary from 25 minutes (Jean-Louis et al., 1997) to 49 minutes (Hauri & Wisbey, 1992). Regardless of the direction of the discrepancies, actigraphy has been found to be sensitive to treatment effect (Brooks et al., 1993; Friedman et al., 2000; Schmidt-Nowara et al., 1992). Moreover, data on time spent in bed reveal that actigraphy is a reliable measure of adherence to behavioural treatment procedures (Brooks et al., 1993; Schmidt-Nowara et al., 1992).

Despite technological improvements and innovations in recent years (e.g., Jean-Louis et al., 1996; Sadeh et al., 1994; Verbeek et al., 1994), the accuracy and relevance of using actigraphy in the assessment of insomnia is still controversial. Discrepancies may result from the inclusion of subjects with sleep-state misperception, a factor which can contribute to the underestimation of sleep time (e.g., Hauri & Wisbey, 1992) or the use of heterogeneous samples composed of subjects with other sleep or psychological disorders (e.g., Cole et al., 1992). The paucity of studies using repeated-night PSG, actigraphy and sleep diary recordings of the same night of sleep makes it difficult to understand these discrepancies. There is, therefore, a need to compare PSG, actigraphy, and sleep diary data collected for the same night of sleep on more than one night using a homogeneous insomniac sample both before and after treatment. The present study explores the clinical utility and sensitivity of actigraphy in

documenting objective treatment response in chronic insomnia. PSG, actigraphy, and sleep diary are used for a total of four nights, including two at baseline and two after treatment.

Method

Participants

Participants were recruited through newspaper advertisements or by physician referrals. Inclusion criteria were: (a) being between 30- and 50-years old; (b) reporting insomnia, defined as sleep onset latency, wake after sleep onset, or early morning awakening equal or superior to 60 minutes at least 4 nights a week for the past 6 months; (c) reporting significant distress or daytime impairment as evaluated by the *Insomnia Severity Index* (score of 2 or higher on a 0 [*not at all*] to 4 [*very much*] Likert scale); and (d) cessation, at least one month prior to treatment, of any sleep or other psychotropic medication that could interfere with sleep. Exclusion criteria were: (a) presence of another sleep disorder such as sleep apnea or circadian rhythm disorder; (b) evidence that insomnia was related to a medical condition; (c) presence of major depression, anxiety disorder, alcohol/substance abuse or any other severe psychopathology as diagnosed with the Structured Clinical Interview for DSM-IV (SCID-IV; First, Spitzer, Gibbon, & Williams, 1997); (d) currently in psychotherapy; and (e) regular use of a medication interfering with sleep. Although more stringent, these criteria are consistent with those of the *International Classification of Sleep Disorders* (American Sleep Disorder Association [ASDA], 1990) and the DSM-IV (American Psychiatric Association [APA], 1994) for chronic primary insomnia.

The sample included 17 participants (7 men and 10 women) with a mean age of 41.6 years (SD = 5.7; range from 34-50). The average education level was 15.2 years (SD = 3.0; range from 10-19 years). Fourteen were working and three were unemployed. The average insomnia duration was 11.8 years (SD = 6.2) and the mean age of insomnia onset was 29.8 years (SD = 7.7). One participant presented sleep-onset insomnia, nine sleep-maintenance insomnia and seven mixed insomnia.

Measures

Initial screening and clinical evaluation. The initial screening included a 20-minute telephone questionnaire administered to evaluate the presence of insomnia. A subsequent multi-measure pre-treatment evaluation was composed of a semi-structured sleep history interview to diagnose insomnia, the SCID-IV (First et al., 1997) to evaluate the presence of psychological disorders, and a physical examination. Participants were enrolled in a 10-week treatment that comprised medication (zopiclone) and cognitive behavior therapy (CBT). Additional information about the treatment protocol and treatment outcome is reported elsewhere (Vallières, Morin, & Guay, 2002).

Polysomnography. The polysomnographic montage included electroencephalographic, electromyographic, and electro-oculographic monitoring. Sleep stages, respiratory disturbance and limb movements were scored by an experienced clinician according to standard criteria (Rechtschaffen & Kales, 1968). Respiration (air flow, tidal volume, and oxygen saturation) and anterior tibialis electromyographic readings were recorded during the first night to detect sleep apnea or periodic limb movements. Data were scored for Total Wake Time (TWT), Total Sleep Time (TST), Sleep Onset Latency (SOL), Sleep Efficiency (SE: ratio of TST to Time in Bed), and Time in Bed (TIB).

Actigraphy. When participants slept in the laboratory they also wore an actigraph from the IM System Inc. Company. Data were processed and scored for the following variables: TST, TWT, SOL, SE, and TIB, with the IM System Inc. Company software and algorithm. The variable "Wake After Sleep Onset" was not used in the study because it was not computed by IM System's software.

Sleep diaries. Following each night spent in the laboratory, participants completed their sleep diaries. From these diaries, an estimate was computed for a nightly average of TWT, TST, SOL, SE, and TIB.

Procedures

Participants presented at the laboratory for PSG recordings on three consecutive nights at baseline and two consecutive nights at posttreatment. On those same nights, they wore an actigraph and the next morning, sleep diary measures were also completed. Same-night data from diaries, actigraphy, and PSG were compared. Data from the first baseline night were not used to allow for an adaptation to the laboratory. Thus, the two nights using the three devices (PSG, actigraphy, and sleep diary) before treatment and the two nights after treatment were used in the analysis. Therefore, pretreatment readings were obtained from 17 participants for a total of 34 nights. At posttreatment, the sample was smaller as one participant dropped out of treatment, five participants did not return for their laboratory evaluation, one came for only one night, one did not accept to wear the actigraph because it was uncomfortable to sleep with it during the night, and the data for one subject were unavailable due to a broken device. Thus, a total of 16 nights were obtained for eight participants at posttreatment. The sleep variables compared across the three assessment tools were TST, TWT, SOL, SE, TIB.

Data analysis plan

Data analysis included three steps. The first step was to compute descriptive statistics for all dependent variables at baseline and posttreatment in order to examine the sensitivity of the three devices to treatment effect. A repeated measure analysis of variance with three within-subject effects (2 times, 2 nights, and 3 devices) was used to control the inter-night variability of each subject (intra-subject effect). The next step involved computing relative and absolute discrepancies between actigraphy and PSG, and between sleep diary and PSG. Relative discrepancies were computed with negative and positive values of actigraphy minus PSG. Absolute discrepancies were computed based only on absolute values of actigraphy minus PSG (i.e., |actigraphy – PSG|). For example if actigraphy recorded 350 minutes of sleep time and PSG 375 minutes then the relative discrepancy would be equal to – 25 and the absolute discrepancy equal to 25. Means of relative and absolute values of discrepancies were then computed. A repeated measures analysis of variance with two within-subject effects (2

nights and 2 discrepancies) was used to evaluate differences in the magnitude of the discrepancies (actigraphy minus PSG and sleep diary minus PSG). In addition, Cohen's *d* effect sizes were computed for each dependant variable. This statistic expresses the difference between two means in standard deviation units (Cohen, 1988). Thus, Cohen's *d* provides a standardised magnitude of difference between means of discrepancies. The third and final step was to assess the accuracy of measurement of each device with the objective sleep time estimate (OSE) formula proposed by Edinger and Finns (1995): OSE = (MSE / MSA) x 100. In this formula, MSE represents minutes of sleep estimated by the sleep diary or actigraphy and MSA represents the minutes of actual sleep time obtained by PSG. Therefore, an OSE value of 100% indicates a perfect concordance between either actigraphy or sleep diary and PSG assessment. This formula was adapted to compute an objective estimate of each sleep variable for actigraphy and sleep diary. Thus, MSE becomes the estimated value and MSA the actual value obtained by PSG. Descriptive statistics of the sample distribution were computed for OSE for each variable.

Results

Sensitivity of the three devices to treatment effect

Table 1 presents means and standard deviations for the dependent sleep variables (TST, TWT, SE, SOL, and TIB) as measured with the three devices (PSG, actigraphy, and sleep diary) for two nights at baseline and two nights at posttreatment. Repeated measures ANOVAs with three within-subject effects (2 times, 2 nights, and 3 devices) revealed significant time effects for TWT, SE , TIB, TST, and SOL, Fs(1,6) = 45.83, 33.41, 31.80, 5.71, and 6.30, respectively, ps < .001 and .05. For each device, pairwise comparisons revealed significant reductions from baseline to posttreatment for TST, TWT and TIB (ps = .04, respectively), as well as a significant increase in SE (p = .04).

In addition, significant device effects were obtained for TWT, SE, and SOL, $Fs(2,12) = 4.39$, 4.57, and 8.16 respectively, $ps = .04$ and .006. Pairwise comparisons revealed that sleep diary data differed significantly from PSG data ($ps = .03$) for these three sleep variables. No night effect or interaction such as night by device or device by time effects was significant.

Relative and absolute discrepancies between devices

Means and standards deviations of relative and absolute discrepancies between actigraphy and PSG, as well as between sleep diary and PSG, for all sleep variables are presented in Table 2. Repeated measures ANOVAs with two within-subject effects (2 nights and 2 discrepancies) indicate significant differences for relative discrepancies between actigraphy and PSG for SOL, $F(1,14) = 26.21$, $p < .0001$. There was no other significant difference for absolute or relative discrepancies for any of the remaining variables. However, relative and absolute discrepancies between actigraphy and PSG were generally smaller, as suggested by a large effect size on night 1 ($d = 1.06$) for relative discrepancies and a moderate effect size on nights 1 and 2 ($ds = - 0.96$ and $- 0.36$, respectively) for absolute discrepancies. The magnitude of the discrepancies between sleep diary-PSG and actigraphy-PSG for TIB were much smaller and non-significant.

Objective estimates

The distribution of objective estimates of total sleep time (OSE; sleep diary/PSG and actigraphy/PSG) for each night are illustrated in Figure 1. Visual inspection of the data reveals that the majority of the participants had an OSE slightly lower than or close to 100, indicating that sleep diary and actigraphy estimates of total sleep time were slightly lower than PSG measures. In addition, the range of actigraph OSE scores is narrower than the range of sleep diary OSE scores. Objective estimates of TST from actigraphy generally closer to PSG measures than diary estimates. Descriptive statistics (medians, minimum, and maximum) of actigraph and sleep diary OSE for all sleep variables are presented in Table 3. Again, the data reveal that for all sleep variables the range of OSE scores was smaller for actigraphy than for

sleep diary. Medians for TST and SE, were near 100 and were similar for both devices. However, the range of objective estimate scores is more restricted for actigraphy than for sleep diary. TWT medians indicated that both devices overestimated TWT relative to PSG. For SOL, objective estimates revealed that actigraphy underestimated SOL and sleep diary overestimated it.

Discussion

The present findings demonstrate the clinical utility and sensitivity of actigraphy in objectively documenting treatment response in chronic insomnia. Indeed, actigraphy detected changes on all sleep variables after treatment. Furthermore, discrepancies between actigraphy and PSG were smaller than those obtained between sleep diary and PSG, suggesting that actigraphy was more accurate than sleep diary. Data also showed that objective estimates of TST and SE were generally slightly lower than or close to 100% for actigraphy. Furthermore, the range of scores for actigraphy objective estimates was smaller than the range of those for sleep diary objective estimates.

Taken together, these results suggest that actigraphy is a reliable assessment method for monitoring insomnia treatment response. First, the results show that actigraphy is as sensitive to treatment change as PSG and sleep diary. Also, actigraphy provides a reliable method for assessing time in bed, an important treatment target when using sleep restriction procedures. These results concur with those of previous studies that have used actigraphy as an outcome measure (Brooks et al., 1993; Friedman et al., 2000; Schmidt-Nowara et al., 1992) and support the use of actigraphy as a reliable behavioural measure of wakefulness and sleep in the natural environment (Sadeh et al., 1995; Verbeek et al., 2001). Moreover, actigraphy might promote treatment compliance when used at home. Indeed, if the patient knows that the actigraph monitors his or her movement, he may feel more inclined to observe prescribed behavioural treatment procedures. Therefore, actigraphy is not only an assessment tool to monitor outcome, but it can also be an active ingredient in promoting treatment compliance at home.

Finally, the present findings showed that SOL is underestimated by actigraphy and overestimated by sleep diary. Thus, these discrepancies need to be taken into account when interpreting SOL data without the use of PSG.

The present findings also suggest that actigraphy underestimates TST compared to PSG. Means of relative discrepancies based on actigraphy-PSG comparisons had negative values (-8.56 and -38.29 minutes) and the means of the objective sleep estimates based on actigraphy/PSG were under 100%. Previous studies (e.g., Hauri & Wisbey, 1992; Verbeek et al., 1994) examining absolute discrepancies found that actigraphy overevaluated sleep time relative to PSG, a finding that is opposite to our results. One possible explanation for the difference is that other investigators based their conclusions on absolute values while ours are based on relative values and ratios (OSE). Means of absolute discrepancies are computed based only on absolute values of actigraphy minus PSG (i.e., |actigraphy – PSG|). On the other hand, means of relative discrepancies are computed with negative and positive values of actigraphy minus PSG. Then, absolute discrepancies indicate the presence and the magnitude of a gap between devices but do not reveal in which direction (over or under). Means of relative discrepancies indicate the possible direction of this gap without revealing the magnitude of the gap between devices. Therefore, it appears that previous studies (e.g., Hauri & Wisbey, 1992; Verbeek et al., 1994) found the magnitude of a gap between devices rather than an overestimation or an underestimation of actigraphy. In the present study, conclusions are drawn from relative discrepancies and supported by the OSE results. Consequently, it seems that both means of absolute and relative discrepancies should be computed to better understand discrepancies between actigraphy and PSG. In addition, although the sample of the present study is more homogeneous than those of previous studies (e.g., Hauri & Wisbey, 1992), means of absolute discrepancies were similar. Thus, these observed discrepancies might be inherent to the actigraph. Further investigations should be conducted to clarify this point.

The study poses some methodological limitations. First, results should be interpreted cautiously given the small sample size. Second, missing data due to technical problems

related to the use of the actigraph and missing data due to participants reluctance to sleep in the laboratory for PSG evaluation during this study led to a reduced sample size at posttreatment and hence reduced statistical power. The fact that the actigraph could be damaged during treatment underlines the importance of using more than one type of measure to assess sleep and wakefulness. Furthermore, since insomnia is a complex syndrome including physiological and psychological components (Sateia et al., 2001), it is necessary to use multiple measures to capture all of its different components (Kazdin, 1998).

In conclusion, this study demonstrates the clinical utility of actigraphy as an objective and a non-intrusive tool to assess sleep and wakefulness in a natural environment. Additional studies should be conducted with larger samples and using a greater number of assessment nights. The potential impact of actigraphy on promoting adherence to behavioural treatment could also be investigated in other studies. Until then, it is essential to use the actigraph only as an adjunct to PSG or sleep diary.

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Table 1

Means (M) and standard deviations (SD) for polysomnography, actigraphy and sleep diary data for each night and each sleep variable (n = 8).

	Polysomnography		Actigraphy		Sleep Diary	
	M	*(SD)*	*M*	*(SD)*	*M*	*(SD)*
Total sleep time (minutes)						
night 1	391.63[a]	(52.64)	362.57[a]	(49.95)	327.14[a]	(94.11)
night 2	388.03[a]	(56.07)	349.00[a]	(50.13)	309.43[a]	(53.41)
night 3	349.00[b]	(52.85)	316.29[b]	(30.74)	307.14[b]	(67.69)
night 4	362.76[b]	(49.53)	333.43[b]	(34.65)	322.86[b]	(81.64)
Total	372.85	(17.78)	340.32	(10.16)	316.64	(24.21)
Total wake time (minutes)						
night 1	75.56[a]	(51.57)	104.29[a]	(50.37)	136.43[a]	(92.63)
night 2	71.20[a]	(30.49)	109.29[a]	(55.42)	165.71[a]	(78.82)
night 3	36.29[b]	(24.40)	72.00[b]	(19.33)	80.00[b]	(49.24)
night 4	24.51[b]	(12.23)	50.29[b]	(15.48)	61.43[b]	(45.53)
Total	51.89[y]	(8.58)	83.96[yz]	(12.49)	110.89[z]	(17.07)
Sleep efficiency (%)						
night 1	84.49[a]	(10.99)	77.86[a]	(10.43)	70.60[a]	(19.74)
night 2	85.00[a]	(7.65)	76.43[a]	(10.66)	67.53[a]	(12.17)
night 3	91.06[b]	(7.64)	81.43[b]	(4.16)	78.96[b]	(13.20)
night 4	94.09[b]	(4.17)	85.86[b]	(3.98)	83.16[b]	(13.67)
Total	88.66[y]	(2.35)	80.39[yz]	(2.41)	75.06[z]	(4.09)
Sleep Onset Latency (minutes)						
night 1	16.40[a]	(8.87)	6.57[a]	(2.88)	30.00[a]	(20.41)
night 2	17.70[a]	(7.83)	4.43[a]	(5.00)	40.00[a]	(27.84)
night 3	9.73[b]	(7.12)	4.00[b]	(3.27)	30.71[b]	(40.04)
night 4	6.86[b]	(1.82)	3.29[b]	(3.45)	17.86[b]	(13.18)
Total	12.67[y]	(2.21)	4.57[yz]	(0.73)	29.64[z]	(8.15)
Time in bed (minutes)						
night 1	463.87[a]	(22.54)	466.86[a]	(22.77)	463.57[a]	(24.28)
night 2	455.23[a]	(34.89)	458.29[a]	(40.07)	460.86[a]	(38.99)
night 3	381.73[b]	(33.66)	388.29[b]	(35.41)	387.14[b]	(34.14)
night 4	384.31[b]	(39.91)	383.86[b]	(41.01)	385.71[b]	(41.07)
Total	421.29[y]	(10.46)	424.32[z]	(10.03)	424.32[y]	(10.59)

Note. For each variable, means in the same column and in the same row that do not share the same subscripts (a and b or 1 and 2) differ at $p < .05$.

Table 2

Means (M) and standard deviations (SD) of relative and absolute discrepancies between actigraphy and PSG and between sleep diary and PSG (N = 17).

	Relative discrepancies				Absolute discrepancies			
	night 1		night 2		night 1		night 2	
	M (*SD*)	*d*	*M* (*SD*)	*d*	*M* (*SD*)	*d*	*M* (*SD*)	*d*
Total sleep time								
ACT-PSG	-8.56 (59.97)	1.06	-38.29 (45.29)	0.19	44.57 (39.32)	-0.96	45.59 (37.36)	-0.36
SD-PSG	-72.23 (100.30)		-46.89 (72.41)		82.40 (91.52)		59.17 (62.02)	
Total wake time								
ACT-PSG	6.66 (60.12)	-0.99	35.36 (46.43)	-0.39	45.21 (38.37)	-0.90	43.31 (38.55)	-0.63
SD-PSG	66.39 (95.60)		53.49 (75.52)		79.65 (84.06)		67.67 (62.15)	
Sleep efficiency (%)								
ACT-PSG	-2.74 (12.46)	1.08	-8.23 (9.81)	0.23	9.21 (8.51)	-1.02	9.93 (7.93)	-0.41
SD-PSG	-16.23 (22.24)		-10.48 (14.79)		17.93 (20.80)		13.16 (12.29)	
Sleep onset latency								
ACT-PSG	-14.16 (21.59)	-1.79	-7.01 (11.77)	-2.50	14.16 (21.59)	-0.60	11.27 (7.41)	-1.63
SD-PSG	24.51 (32.13)		22.39 (20.63)		27.05 (29.86)		23.34 (19.47)	
Time in bed								
ACT-PSG	4.00 (8.13)	0.48	0.39 (7.58)	-0.35	6.28 (6.40)	-0.31	6.02 (4.33)	-0.39
SD-PSG	0.07 (11.24)		3.06 (10.97)		8.29 (7.25)		7.71 (8.16)	

Note. ACT-PSG = discrepancy between actigraphy and polysomnography; SD-PSG = discrepancy between sleep diary and polysomnography. *d* = Cohen's *d* effect size (Cohen, 1988).

Table 3

*Medians (*Mdn*), minimum (*Min*) and maximum (*Max*) actigraphic and sleep diary objective estimates for each night and sleep variable*

	Actigraphy/PSG			Sleep Diary/PSG		
	Mdn	*Min*	*Max*	*Mdn*	*Min*	*Max*
Total sleep time						
night 1	103.71	62.70	119.10	89.92	0.00	113.55
night 2	90.93	63.99	117.49	90.56	51.18	122.58
night 3	89.36	80.30	107.4	91.17	69.59	121.11
night 4	92.73	84.10	102.54	91.17	57.68	104.03
Total wake time						
night 1	83.56	30.47	550.56	141.64	64.82	562.50
night 2	163.66	63.19	563.64	194.15	24.83	727.27
night 3	239.05	86.17	458.33	268.61	45.35	519.48
night 4	218.42	75.85	432.00	161.24	80.00	652.17
Sleep efficiency (%)						
night 1	100.69	63.37	119.43	88.39	0.00	114.07
night 2	89.34	63.60	115.61	90.88	51.24	122.47
night 3	88.51	78.53	105.12	90.79	67.78	118.92
night 4	91.96	83.77	103.53	91.88	60.47	101.00
Sleep Onset Latency						
night 1	53.33	6.23	93.33	218.45	0.00	629
night 2	33.33	3.38	243.48	235.60	58.48	1090.91
night 3	57.14	0.00	88.89	162.34	66.67	857.14
night 4	34.76	0.00	95.24	233.33	71.43	500.00
Time in bed						
night 1	100.95	96.97	105.33	99.85	94.95	104.48
night 2	99.53	97.49	103.52	100.04	97.11	106.36
night 3	101.41	99.38	103.16	101.83	100.32	103.42
night 4	99.92	97.27	102.55	100.34	98.26	103.04

Note. For nights 1 and 2 N = 17 and for nights 3 and 4 N = 8. Actigraphy/PSG = ratio of actigraphy data on polysomnography data X 100; Sleep diary/PSG = ratio of sleep diary data to polysomnography data X 100. Both ratios were adapted from Edinger and Finns (1995) OSE formula.

Figure Caption

Figure 1. Ranges score of actigraph and sleep diary objective estimates for total sleep time.

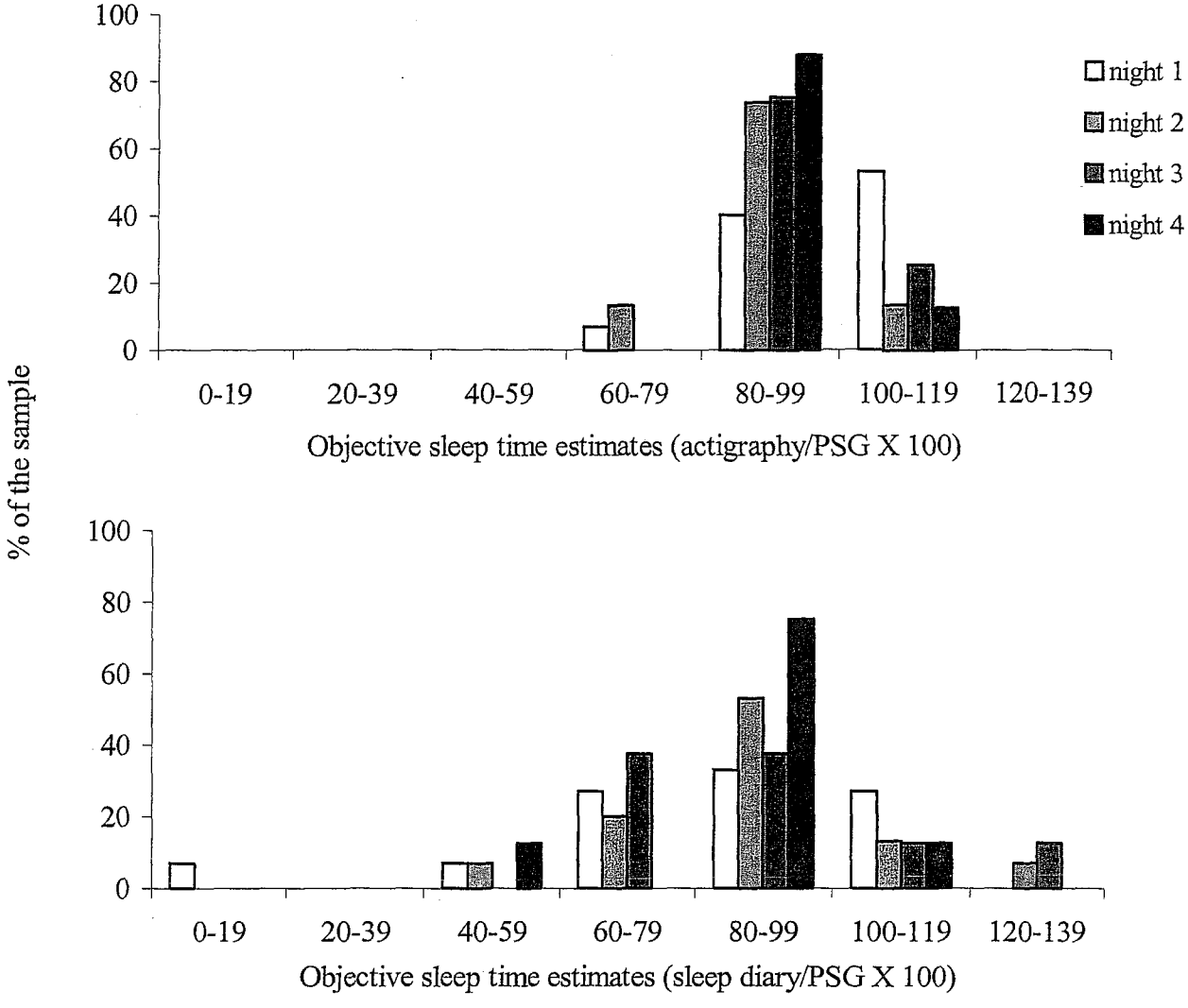
100
80
60
40
20
0
night 1
night 2
night 3
night 4
0-19
20-39
40-59
60-79
80-99
100-119
120-139
Objective sleep time estimates (actigraphy/PSG X 100)
% of the sample
100
80
60
40
20
0
0-19
20-39
40-59
60-79
80-99
100-119
120-139
Objective sleep time estimates (sleep diary/PSG X 100)

Figure 1.

CONCLUSION

Cette thèse de doctorat comprend trois études portant sur les combinaisons séquentielles de traitements pharmacologiques et psychologiques pour l'insomnie chronique et l'évaluation du sommeil. La première étude est une exploration de trois séquences de traitements possibles, permettant ainsi d'établir le protocole et les séquences de traitement ayant un plus grand intérêt. La deuxième étude, basée sur les résultats de la première, est le corps de cette thèse et comprend deux volets : le premier est l'évaluation de l'efficacité de combinaisons séquentielles de traitements pour l'insomnie et le deuxième est l'évaluation de l'utilité clinique d'un outil de mesure de l'insomnie. Un total de 23 participants souffrant d'insomnie chronique ont été enrôlés soit six participants dans l'étude exploratoire et 17 dans la seconde.

Les résultats de cette thèse démontrent que le traitement séquentiel est efficace à court et moyen terme pour améliorer le sommeil. De plus, il semble que le TBC soit essentiel au traitement de l'insomnie chronique. En effet, 87% des participants (soit 20/23 participants) ont une amélioration du sommeil survenant seulement à la suite de l'introduction du TBC. Par ailleurs, il semble que l'ajout de la médication en début de traitement diminue les effets secondaires reliés au début de la restriction du temps passé au lit. De plus, la médication favoriserait ainsi, le maintien ou l'augmentation du temps total de sommeil obtenu durant le traitement et à la fin de celui-ci. En effet, le temps total de sommeil est demeuré stable ou a augmenté plus rapidement pour six des sept participants ayant reçu soit la séquence de traitement comprenant deux semaines de chevauchement (Médication → Combiné → TBC) dans l'étude exploratoire, soit la séquence Combiné → TBC dans la deuxième étude. Enfin, les résultats suggèrent qu'une séquence de traitement débutant par une médication seule pendant cinq semaines (Médication → TBC ou Médication → Combiné) ne serait pas la meilleure option. En effet, seulement deux des sept participants ayant reçu en premier une médication seule ont obtenus des améliorations durant cette première phase de traitement alors que cinq sur sept participants ont vu leur sommeil s'améliorer lorsque le TBC a été introduit. Finalement, il semble que la préférence au traitement et l'attribution des gains thérapeutiques à la médication sont des éléments déterminants de la réponse thérapeutique lors d'un traitement séquentiel et nécessitent une étude plus approfondie. Par conséquent, les résultats de cette

thèse soutiennent l'hypothèse avancée dans la littérature (Edinger & Wholgemuth, 1999; Morin, Hauri, et al., 1999) voulant que la combinaison séquentielle des traitements pharmacologiques et psychologiques optimisent l'efficacité du traitement de l'insomnie.

Les trois études de cette thèse de doctorat ont plusieurs implications au niveau de la recherche sur le traitement de l'insomnie. Premièrement, cette thèse ouvre la porte à l'étude des différentes façons d'introduire et de combiner les traitements pour l'insomnie. En effet, ayant démontré que ces combinaisons séquentielles de traitements sont efficaces, il apparaît maintenant possible de les tester à une plus grande échelle et également d'en développer de nouvelles en fonction de la réalité clinique (comme celles de cette thèse), des attributions ou de la personnalité du patient, ou encore en fonction de la réponse au traitement. Deuxièmement, cette thèse démontre l'importance d'évaluer ainsi que de comprendre les mécanismes de traitement et, par le fait même, rejoint le développement conceptuel récent au niveau de l'insomnie (Espie, 2002). Le rôle d'une attribution élevée des gains thérapeutiques à la médication examiné dans cette thèse est un exemple de mécanisme de traitement qui nécessite plus d'attention. Il est donc envisageable que les limites connues des ingrédients actifs du traitement soient associées à un mécanisme de traitement inconnu à ce jour. Troisièmement, cette thèse démontre que lorsque le but premier de la recherche n'est pas de faire des comparaisons avant et après le traitement mais plutôt de comprendre l'évolution des patients durant le traitement ou de cibler un mécanisme, il importe de modifier l'approche d'évaluation de la réponse au traitement. Ce nouveau sens que prend l'étude du traitement de l'insomnie nécessite une souplesse dans l'utilisation traditionnelle d'outils et de techniques permettant de répondre aux questions d'évolution et de mécanismes de traitement. Ainsi, de nouvelles variables peuvent être étudiées, les temps et période d'évaluation peuvent être modifiées et les analyses statistiques utilisées alors, doivent être adaptées à ces changements. Enfin, au niveau de l'évaluation du sommeil, cette thèse démontre qu'il est important de continuer le développement de nouveaux outils de mesure et des techniques permettant leur comparaison.

Plusieurs impacts cliniques découlent également de cette thèse. D'abord, il semble que

le TBC soit efficace comme il a été démontré dans les études empiriques précédentes (Hauri, 1997; McClusky et al., 1991; Milby et al., 1993; Morin, Colecchi, et al., 1999, Rosen et al., 2000) mais, qu'en plus, il soit essentiel au traitement de l'insomnie. Il semble également bénéfique d'espacer les sessions de traitements et de laisser le temps aux patients d'intégrer et d'appliquer les ingrédients actifs du TBC. Toutefois, afin que le TBC soit efficace même lorsque les séances sont éloignées, il est important de vérifier que le patient comprenne le rationnel du traitement et développe ses habiletés de gestion du sommeil. Ensuite, il semble que la médication en début de traitement peut être favorable en autant que le TBC est introduit tôt dans le processus de traitement et que les attributions des gains thérapeutiques exclusivement accordées à la médication soient restructurées en cours de traitement. Par conséquent, l'ensemble de cette thèse souligne encore une fois, l'importance d'informer les cliniciens et de rendre le TBC plus accessible à la population. Finalement, il apparaît qu'un thérapeute ne devant se fier qu'à l'évaluation subjective du sommeil de ses patients, pourrait enrichir son évaluation à l'aide de données objectives fournies par l'actigraphie. Il devient donc important de promouvoir l'utilisation de cet outil de mesure afin qu'il ne serve pas qu'à la recherche mais qu'il soit aussi une aide pour le thérapeute et, par conséquent, pour le patient.

Cette thèse de doctorat possède néanmoins quelques limites. Tout d'abord, le protocole à niveau de bases multiples en fonction des sujets utilisé ne permet pas de tirer des conclusions sans équivoque sur l'efficacité du traitement relativement à un groupe contrôle (sans traitement). Par conséquent, il ne permet pas la comparaison directe des séquences de traitements. Toutefois, le protocole utilisé est conçu dans le but de démontrer que les changements observés surviennent uniquement à la suite de l'introduction du traitement. Ce protocole se caractérise également par la présence de niveaux de base de durées différentes qui décalent dans le temps le début du traitement. Par conséquent, lorsque les changements surviennent uniquement à la suite de l'introduction du traitement, la probabilité qu'ils soient dus au traitement plutôt qu'à tout autre facteur, augmente. Une deuxième limite est reliée à l'homogénéité et à la grandeur de l'échantillon. En effet, ce protocole ne peut être utilisé qu'avec un petit nombre de participants et nécessitent un échantillon le plus homogène

possible afin de faciliter l'observation de changements thérapeutiques. Ainsi, cette étude met l'accent sur la validité interne de celle-ci et, par le fait même, limite la généralisation externe des résultats. Finalement, une autre limite est l'absence de qualités psychométriques pour la mesure d'attribution des gains thérapeutiques. Toutefois, l'intérêt croissant accordé à l'impact négatif possible des gains attribués à la médication (Edinger & Wolhgemuth, 1999) justifie l'utilisation de cette mesure dans une étude exploratoire.

En conclusion, cette thèse démontre l'efficacité de combinaisons séquentielles de traitements pharmacologiques et psychologiques pour l'insomnie chronique à court et à moyen terme. Il apparaît maintenant évident que les séquences de traitements méritent d'être évaluées dans une étude plus vaste avec un groupe contrôle. L'accent devra être mis à la fois sur les changements qui surviennent au milieu de la séquence et sur le maintien à plus long terme des gains thérapeutiques. Finalement, dans une réalité clinique où les médecins sont souvent les premiers consultés pour un problème d'insomnie, il devient important de diffuser et d'informer ces derniers quant à l'efficacité des traitements séquentiels et de rendre le traitement béhavioral-cognitif plus accessible à la population.

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Annexe A

Les instruments de mesure du sommeil et le questionnaire d'attribution des changements thérapeutiques

AS 2.0 28-11-02

AGENDA DU SOMMEIL

NOM:_____ Semaine du:_____ au _____

Le matin je complète ma nuit du	**Exemple**	**(nuit) Date**						
	Mardi 25/03	_____ /	_____ /	_____ /	_____ /	_____ /	_____ /	_____ /
1. Hier, j'ai fait la sieste entre _____ et _____ (Notez l'heure de toutes siestes).	**13h50 à 14h30**							
2. Hier, j'ai pris _____ mg de médicament et/ou ___ oz d'alcool pour dormir.	**Halcion 0,125 mg**							
3. Je me suis couché(e) à ____ hres J'ai éteint les lumières à ____ hres.	**22h45 23h15**							
4. Après avoir éteint les lumières, je me suis endormi(e) en ____ min.	**40 min.**							
5. Mon sommeil a été interrompu _____ fois. (Spécifiez le nombre de fois).	**3**							
6. Mon sommeil a été interrompu _____ min. (Spécifiez la durée en minute de chaque période d'éveil).	**10 5 45**							
7. Cette nuit, je me suis levé _____ (Indiquez le nombre de fois où vous avez quitté le lit)	**3**							
8. Ce matin, je me suis réveillé(e) à _____ hres (Notez l'heure du dernier réveil).	**6h15**							
9. Ce matin, je me suis levé(e) à _____ hres (Spécifiez l'heure).	**6h40**							
10. Au lever ce matin, je me sentais _____ (1 = épuisé(e), 5 = reposé(e)).	**2**							
11. Dans l'ensemble, mon sommeil de la nuit dernière a été _____ (1 = très agité, 5 = très profond).	**3**							

Index de Sévérité de l'Insomnie-Révisé (ISI-R)

Nom: _____ **Date:** _____

Pour chacune des questions, veuillez encercler le chiffre qui correspond le plus fidèlement possible à votre sommeil **au cours du dernier mois.**

Pour les 3 premières questions, veuillez estimer la **SÉVÉRITÉ** de vos difficultés de sommeil.

1. Difficultés à s'endormir:

Aucune	Légère	Moyenne	Très	Extrêmement
0	**1**	**2**	**3**	**4**

2. Réveils nocturnes fréquents et/ou prolongés:

Aucune	Légère	Moyenne	Très	Extrêmement
0	**1**	**2**	**3**	**4**

3. Problèmes de réveils trop tôt le matin:

Aucune	Légère	Moyenne	Très	Extrêmement
0	**1**	**2**	**3**	**4**

4. Jusqu'à quel point êtes-vous **SATISFAIT(E)/INSATISFAIT(E)** de votre sommeil actuel?

Très Satisfait	Satisfait	Plutôt Neutre	Insatisfait	Très Insatisfait
0	**1**	**2**	**3**	**4**

5. Jusqu'à quel point considérez-vous que vos difficultés de sommeil **PERTURBENT** votre fonctionnement quotidien (p. ex., fatigue, concentration, mémoire, humeur)?

Aucunement	Légèrement	Moyennement	Beaucoup	Extrêmement
0	**1**	**2**	**3**	**4**

6. À quel point considérez-vous que vos difficultés de sommeil sont **APPARENTES** pour les autres en termes de détérioration de la qualité de votre vie?

Aucunement	Légèrement	Moyennement	Très	Extrêmement
0	**1**	**2**	**3**	**4**

7. Jusqu'à quel point êtes-vous **INQUIET(ÈTE)**/préoccupé(e) à propos de vos difficultés de sommeil?

Aucunement	Légèrement	Moyennement	Très	Extrêmement
0	**1**	**2**	**3**	**4**

Croyances et Attitudes concernant le Sommeil (CAS)

No du participant _____ **Date:** _____

Plusieurs énoncés reflétant les croyances et les attitudes des gens concernant le sommeil sont énumérés ci-dessous. S'il-vous-plaît, veuillez indiquer jusqu'à quel point vous êtes personnellement en accord ou en désaccord avec chaque énoncé. Il n'y a pas de bonne ou de mauvaise réponse. Pour chaque phrase, encerclez le chiffre correspondant à votre estimation personnelle. Essayez d'utiliser l'échelle entière plutôt que d'utiliser uniquement ses extrémités. S'il-vous-plaît, veuillez répondre à toutes les questions même si vous n'avez pas de difficulté de sommeil. Pour chacune des questions, veuillez vous référer à l'échelle ci-dessous.

Fortement en désaccord — Fortement en accord

0 1 2 3 4 5 6 7 8 9 10

1. J'ai besoin de huit heures de sommeil pour me sentir reposé(e) et bien fonctionner pendant la journée.

0 1 2 3 4 5 6 7 8 9 10

2. Lorsque je ne dors pas suffisamment durant la nuit, j'ai besoin de récupérer le jour suivant en faisant une sieste, ou la nuit suivante, en dormant plus longtemps.

0 1 2 3 4 5 6 7 8 9 10

3. Parce que je vieillis, j'ai besoin de moins de sommeil.

0 1 2 3 4 5 6 7 8 9 10

4. Je m'inquiète lorsque je passe une nuit ou deux sans dormir. Je pourrais faire une crise de nerfs.

0 1 2 3 4 5 6 7 8 9 10

5. Je crains que l'insomnie chronique puisse avoir des conséquences sérieuses sur ma santé physique.

0 1 2 3 4 5 6 7 8 9 10

6. En passant plus de temps au lit, je dors habituellement plus longtemps et je me sens mieux le lendemain.

0 1 2 3 4 5 6 7 8 9 10

7. Lorsque j'ai de la difficulté à m'endormir ou à me rendormir après un réveil nocturne, je devrais rester au lit et essayer davantage.

0 1 2 3 4 5 6 7 8 9 10

8. Je suis inquiet(ète) de perdre le contrôle sur mes habiletés à dormir.

0 1 2 3 4 5 6 7 8 9 10

9. Parce que je vieillis, je dois aller au lit plus tôt dans la soirée.

0 1 2 3 4 5 6 7 8 9 10

10. Après une mauvaise nuit de sommeil, je sais que cela va nuire à mes activités quotidiennes le lendemain.

0 1 2 3 4 5 6 7 8 9 10

11. Afin d'être éveillé et de bien fonctionner le jour, je crois qu'il serait mieux de prendre une pilule pour dormir plutôt que d'avoir une mauvaise nuit de sommeil.

0 1 2 3 4 5 6 7 8 9 10

12. Lorsque je me sens irritable, déprimé(e) ou anxieux(se) pendant la journée, c'est surtout parce que j'ai mal dormi la nuit précédente.

0 1 2 3 4 5 6 7 8 9 10

13. Parce que mon/ma conjoint(e) s'endort aussitôt qu'il/elle se couche et reste endormi(e) toute la nuit, je devrais être capable d'en faire autant.

0 1 2 3 4 5 6 7 8 9 10

14. Je pense que l'insomnie est essentiellement le résultat du vieillissement et peu de choses peuvent être faites pour ce problème.

0 1 2 3 4 5 6 7 8 9 10

15. J'ai parfois peur de mourir pendant mon sommeil.

0 1 2 3 4 5 6 7 8 9 10

16. Quand j'ai une bonne nuit de sommeil, je sais que j'en payerai le prix la nuit suivante.

0 1 2 3 4 5 6 7 8 9 10

17. Quand je dors mal une nuit, je sais que cela dérangera mon horaire de sommeil pour toute la semaine.

0 1 2 3 4 5 6 7 8 9 10

18. Sans une nuit de sommeil adéquate, je peux à peine fonctionner le lendemain.

0 1 2 3 4 5 6 7 8 9 10

19. Je ne peux jamais prédire si j'aurai une bonne ou une mauvaise nuit de sommeil.

0 1 2 3 4 5 6 7 8 9 10

20. J'ai peu d'habiletés pour faire face aux conséquences négatives d'un sommeil troublé.

0 1 2 3 4 5 6 7 8 9 10

21. Quand je me sens fatigué(e), sans énergie ou simplement incapable de bien fonctionner, c'est généralement parce que j'ai mal dormi la nuit précédente.

0 1 2 3 4 5 6 7 8 9 10

22. Je deviens envahi(e) par mes pensées la nuit et souvent je sens que je n'ai pas le contrôle sur ces pensées.

0 1 2 3 4 5 6 7 8 9 10

23. Je crois que je peux encore mener une vie satisfaisante malgré des difficultés de sommeil.

0 1 2 3 4 5 6 7 8 9 10

24. Je crois que l'insomnie est principalement le résultat d'un déséquilibre hormonal.

0 1 2 3 4 5 6 7 8 9 10

25. Je crois que l'insomnie est en train de ruiner ma capacité à jouir de la vie et m'empêche de faire ce que je veux.

0 1 2 3 4 5 6 7 8 9 10

26. Un verre d'alcool avant l'heure du coucher est une bonne solution aux difficultés de sommeil.

0 1 2 3 4 5 6 7 8 9 10

27. La médication est probablement la seule solution à l'insomnie.

0 1 2 3 4 5 6 7 8 9 10

28. Mon sommeil se détériore continuellement et je ne crois pas que quelqu'un puisse m'aider.

0 1 2 3 4 5 6 7 8 9 10

29. Habituellement, lorsque je n'ai pas bien dormi, ça affecte mon apparence physique.

0 1 2 3 4 5 6 7 8 9 10

30. J'évite ou j'annule mes engagements (sociaux, familiaux) après une mauvaise nuit de sommeil.

0 1 2 3 4 5 6 7 8 9 10

PRÉ

QUESTIONNAIRE D'ATTRIBUTION DES CHANGEMENTS THÉRAPEUTIQUES

No du participant: _____ Date: _____

Veuillez lire et répondre à chaque question concernant l'amélioration de votre sommeil. Pour chaque phrase, inscrivez une marque (/) correspondant à votre estimation personnelle.

1. Jusqu'à quel point pensez-vous que l'amélioration de votre sommeil sera due au médecin?

0 10 20 30 40 50 60 70 80 90 100

0 50 100

Pas du tout Modérément Totalement

2. Jusqu'à quel point pensez-vous que l'amélioration de votre sommeil sera due au psychologue?

0 10 20 30 40 50 60 70 80 90 100

0 50 100

Pas du tout Modérément Totalement

3. Jusqu'à quel point pensez-vous que l'amélioration de votre sommeil sera due à la médication?

0 10 20 30 40 50 60 70 80 90 100

0 50 100

Pas du tout Modérément Totalement

4. Jusqu'à quel point pensez-vous que l'amélioration de votre sommeil sera due à la restriction du sommeil et aux procédures comportementales?

0 10 20 30 40 50 60 70 80 90 100

0 50 100

Pas du tout Modérément Totalement

5. Jusqu'à quel point pensez-vous que l'amélioration de votre sommeil sera due aux changements de vos croyances et attitudes?

0 10 20 30 40 50 60 70 80 90 100

0 50 100

Pas du tout Modérément Totalement

6. Jusqu'à quel point pensez-vous que l'amélioration de votre sommeil sera due à l'effort que vous avez mis?

0 10 20 30 40 50 60 70 80 90 100

0 50 100

Pas du tout Modérément Totalement

7. Jusqu'à quel point pensez-vous que l'amélioration de votre sommeil sera due à la chance?

0 10 20 30 40 50 60 70 80 90 100

0 50 100

Pas du tout Modérément Totalement

8. Jusqu'à quel point avez-vous confiance d'être capable de contrôler votre sommeil actuellement?

0 10 20 30 40 50 60 70 80 90 100

0 50 100

Pas du tout Modérément Totalement

Traitement ____ POST ____ FU-3 ____

QUESTIONNAIRE D'ATTRIBUTION DES CHANGEMENTS THÉRAPEUTIQUES

Nom: _____ Date: _____

Veuillez lire et répondre à chaque question concernant l'amélioration de votre sommeil. Pour chaque phrase, inscrivez une marque (/) le long de la ligne correspondant à votre estimation personnelle.

1. Jusqu'à quel point attribuez-vous l'amélioration de votre sommeil au médecin?

0 10 20 30 40 50 60 70 80 90 100

0 50 100

Pas du tout Modérément Totalement

2. Jusqu'à quel point attribuez-vous l'amélioration de votre sommeil au psychologue?

0 10 20 30 40 50 60 70 80 90 100

0 50 100

Pas du tout Modérément Totalement

3. Jusqu'à quel point attribuez-vous l'amélioration de votre sommeil à la médication?

0 10 20 30 40 50 60 70 80 90 100

0 50 100

Pas du tout Modérément Totalement

4. Jusqu'à quel point attribuez-vous l'amélioration de votre sommeil à la restriction du sommeil et aux procédures comportementales?

0 10 20 30 40 50 60 70 80 90 100

0 50 100

Pas du tout Modérément Totalement

5. Jusqu'à quel point attribuez-vous l'amélioration de votre sommeil aux changements de vos croyances et attitudes?

0 10 20 30 40 50 60 70 80 90 100

0 50 100

Pas du tout Modérément Totalement

6. Jusqu'à quel point attribuez-vous l'amélioration de votre sommeil à l'effort que vous avez mis?

0 10 20 30 40 50 60 70 80 90 100

0 50 100

Pas du tout Modérément Totalement

7. Jusqu'à quel point attribuez-vous l'amélioration de votre sommeil à la chance?

0 10 20 30 40 50 60 70 80 90 100

0 50 100

Pas du tout Modérément Totalement

8. Jusqu'à quel point avez-vous confiance d'être capable de contrôler votre sommeil après le traitement?

0 10 20 30 40 50 60 70 80 90 100

0 50 100

Pas du tout Modérément Totalement