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Is a full psychotherapy program necessary to reduce benzodiazepine dependence for insomnia? A Randomized Controlled Trial Comparing Drug Tapering with Single-Session CBT vs. Full Acceptance and Commitment Therapy

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ABSTRACT

Objective: Benzodiazepines and Z-drugs (BZs) are known to present a high-risk for chronic dependence when patients do not adhere to duration of use guidelines. Cognitive Behavioral Therapy for Insomnia (CBT-I) has shown effectiveness to reduce dependence, but is not frequently sought by consumers due to being costly and time intensive. A single session of CBT-I may show promise as a compromise. The efficacy of these two approaches is herein compared in the context of a drug tapering program.

Method: In this randomized controlled trial, the latest generation of CBT-I, Acceptance and Commitment Therapy for Insomnia (ACT-I), was compared to a single session of CBT-I in the context of a tapering program assessing two tapering speeds (rapid-6 weeks vs. long-18 weeks), and short vs. long BZs drug half-lives. The taper program consisted of 10-min phone guided sessions with a psychologist. Participants were evaluated pre-treatment, and at 1, 3, and 12 months post-treatment.

Results: Across all experimental groups, by 12 months, 58.6 % of the 87 participants achieved successful abstinence, and 29.9 % reduced their daily dosage by more than 50 %. No significant difference was observed between ACT-I and the single session of CBT-I. The rapid taper condition led to cumulative withdrawal symptoms in long BZ half-life consumers.

Conclusion: Both ACT-I and the single session of CBT-I are associated with similar success rates when combined with a drug tapering program consisting of periodic telephone consultations. Clinical practice may be reevaluated to propose slow tapering for long BZ half-lives. The data show this program to be effective to a broad population.

1. Introduction

Benzodiazepines and related Z-drugs (BZs) constitute a widely-used

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Abbreviations

ACT	Acceptance and Commitment Therapy
ACT-I	Acceptance and Commitment Therapy for Insomnia
BI	Brief Intervention
BZs	Benzodiazepines and Z-drugs
CBT	Cognitive Behavioral Therapy
CBT-I	Cognitive Behavioral Therapy for Insomnia
SS CBT-I	"Single Shot" of Cognitive Behavioral Therapy for Insomnia
RCT	Randomized Controlled Trial
TP	Taper Program
WL	Waiting List

set of medications with hypnotic and anxiolytic effects that are frequently prescribed to treat symptoms of acute insomnia [1]. While BZs offer rapid symptom relief, this can foster patient dependence, and health professionals generally advise against their long-term use due to associated risks. These risks include not only psychophysical dependence, but also several potential side effects such as impairments in memory, attention, and disruptions in physiological sleep structure [2,3]. For these reasons, health authorities recommended not exceeding four weeks of use. However, this limit is rarely adhered to by patients, and so chronic consumption is in-fact common, as a psychophysical dependence is typically developed when used beyond 4 weeks, in turn making them even more difficult to quit [4]. As a result, BZs have become stigmatized [5,6], a stigma that might lessen if more effective strategies for discontinuation were available. In this regard, the current challenge in this clinical field is the development of new treatment plans for BZs dependence that are widely and easily accessible to the general population.

It is well known that after BZs dependence has developed, it is very challenging for (chronic) consumers to achieve abstinence. For example, the literature reports abstinence success rates of only 7 % for those without any accompaniment, 33 % with brief interventions (BI), and 80 % for those who followed a Cognitive Behavioral Therapy (CBT) program, which notably targets the underlying issue that led to BZs use in the first place [7]. CBT therapies have been contrasted with substitution strategies [8], which lack empirical evidence of their efficacy, and do not address the core problem: namely dependence on an external substance for sleep, and thus the driver, or root cause of consumption-insomnia [7]. While the current literature points to CBT as the preferred treatment modality, several notable challenges remain: namely, *i)* the financial cost and time commitment associated with CBT in comparison with BI, and *ii)* the shortage of trained professionals in the technique (*e.g.*, by geographical area), altogether resulting in limited access and underutilization of CBT. This calls for an examination of how BI and CBT protocols for BZ discontinuation may be optimized to improve their accessibility, patient adherence and therapeutic efficacy. The following three strategies were evaluated.

Firstly, previous research [9], albeit not with the latest CBT methods for Insomnia (CBT-I), invite us to recognize the potential importance that psychotherapy may have when compared to an effective BI drug tapering schedule. Secondly, works have found comparable efficacy of a "Single-Shot" of CBT-I (SS CBT-I) compared to standard CBT-I length [10,11] for treating insomnia (though not yet tested for BZs dependence): the 2015 study by Ellis et al. [10] compared, in a randomised controlled trial, 20 participants suffering from acute insomnia who received a 60-min session with 20 participants on a waiting list; their results show an improvement in the severity of insomnia and sleep parameters in the experimental group. More recently, Amra et al. [11] tested the effectiveness of one-shot CBT on acute insomnia developed in the context of the COVID-19 pandemic in healthcare workers, and showed a decrease in the severity of post-treatment insomnia in patients who received condensed CBT-I compared to controls. Also, a recent meta-analysis has evaluated the effectiveness of behavioral interventions used alone (without cognitive interventions), known as 'BBT-I', which consist of 1–3 sessions aimed at teaching the principles of bedtime restriction and stimulus control [12]. This study has shown an improvement in sleep parameters in initial follow-up (2 months post-treatment). Indeed, bedtime restriction and stimulus control are known to be the most effective components of CBT-I treatment (recommendation B - strong - in the latest European guideline for the treatment of insomnia) [13]. Altogether, these findings suggest the premise that the SS CBT-I may achieve similar results to full-length CBT-I (being more costly and time intensive), when they are each paired with the same drug tapering schedule. To date, this comparison has not been empirically tested in the context of BZ discontinuation. Therefore, we tested whether a SS CBT-I could provide for an equally effective, but less burdensome, treatment alternative to a full-length CBT-I plan in the context of hypnotic withdrawal. Note that the SS CBT-I retains the crucial components of CBT-I, namely, educating patients on sleep restriction and stimulus control [14], while also possessing the attractiveness of a brief intervention.

Secondly, Acceptance and Commitment Therapy for Insomnia (ACT-I) has been described as a promising new generation of CBT for the discontinuation of benzodiazepines prescribed for hypnotic purposes [15]. ACT has been validated for the treatment of a wide range of psychological disorders, including substance dependence, and shows promising results in the treatment of insomnia: with the therapeutic goal being, beyond sleep, an improvement in quality of life, ACT aims to shift insomniacs' focus away from controlling sleep and fatigue, with the paradoxical result of improving sleep quality [16]. Moreover, as the therapeutic target of ACT is experiential avoidance [15,17,18], this approach appears to be conducive to the treatment of hypnotic dependence, the consumption of which aims to extinguish the negative thoughts and emotions associated with difficulties initiating sleep, as well as the search for sleep on command [15]. A pilot study evaluating the effectiveness of ACT for insomnia showed a significant reduction in hypnotic consumption, although it was not the therapeutic objective [15]. The studies comparing CBT-I and ACT-I protocols have not, to date, demonstrated any added benefit of ACT on sleep parameters [19–23]; none of these studies specifically focused on benzodiazepine discontinuation. We may therefore question its added value in the treatment of BZ dependence for the management of withdrawal symptoms, particularly experiential avoidance, which may underlie BZs use, compared to SS CBT-I intervention.

Thirdly, the current study sought to identify the optimal tapering speeds for BZ discontinuation, and as a function of BZ half-life durations, as this is not well-identified in the current literature. Despite that the literature has identified that tapering schedules should not be abrupt, as this can lead to severe withdrawal symptoms [24,25], including a risk of epileptic seizure [26,27], overly wide clinical tapering guidelines are commonly found at state and national levels [28,29]28, 29 (see Table S2), such as from 4 weeks to 1 year. As per studies specifically examining CBT-based methods for BZ cessation, tapering durations have varied between 4 and 16 weeks [2,9,30–39]. Though, drug half-life should be taken into account, as research has shown a clear relationship between half-life duration and the onset of withdrawal symptoms [40]. Specifically, for BZs with short half-lives, tapering too quickly tends to trigger rebound insomnia as early as within one to two days [41–43], suggesting that slow tapering is preferable for these users [44]. For BZs with long half-lives, withdrawal symptoms are generally milder and experienced 2–10 days after tapering [24,45]. Despite these findings, few studies evaluating the efficacy of CBT-based protocols for BZ discontinuation have accounted for BZ half-life durations.

The present study aimed to compare the efficacy of BZ discontinuation programs by examining three variables: psychotherapeutic intervention method, tapering duration, and BZ half-life. Two

psychotherapeutic approaches, both delivered remotely via teletherapy, were compared: a SS CBT-I, incorporating sleep restriction and stimulus control, and an eight-session of ACT-I. Two tapering durations were evaluated: rapid (6 weeks) and extended (18 weeks), alongside two BZ half-life categories: short versus medium/long. This factorial design was tested in a longitudinal randomized controlled trial (RCT) with follow-up assessments conducted over 12 months post-intervention. The primary outcome was maintenance of BZ abstinence or reduction in use over 12 months, while secondary outcomes included monitoring withdrawal symptoms.

2. Method

The CONSORT 2025 procedure was followed (Supplementary Data Table S2 [46]).

2.1. Participants

All participants were native French speakers of French or Swiss nationality. Recruitment took place through public announcements diffused in the press and professional networks between May 2021 and February 2022. The inclusion criteria were controlled for in a two-step process via an initial screening by an online questionnaire, followed by two clinical intake interviews.

The pre-inclusion criteria controlled for via the online questionnaire, were: (1) being between 18 and 70 years old; (2) for the last 12 months, at least 4 nights per week, using at least one BZ to aid sleep; (3) reporting difficulties falling or staying asleep; (4) obtaining a pathological score (>34) on the Benzodiazepine Dependence Questionnaire (BDEP) [47]; and (5) being sufficiently comfortable with internet-based technologies.

Three main inclusion criteria assessed for during two clinical intake interviews. The first (1), was meeting the DSM-5 diagnostic criteria [48] for Insomnia Disorder (307.42) and Sedative, Hypnotic, or Anxiolytic Use Disorder (304.10). Since hypnotic medications can mask an underlying insomnia disorder, participants had to meet these DSM-5 criteria either at inclusion (while still taking the medication) or following previous attempts to discontinue BZs. Next, patients had to (2) obtain authorization to participate in the study from their prescribing physician and (3) meet the BZ deprescription criteria, as defined by the French National Health Authority [49]. The non-inclusion and exclusion criteria are more extensively detailed in the Supplementary Data.

The estimated sample size (based on the formula by Erdfelder et al. [50]) for a repeated-measures ANOVA with a between-subject interaction involving 4 groups and two time points, assuming an effect size of $f = 0.5$ (based on the effect size of ACT on benzodiazepine and related drug use observed in a pilot study [15], an alpha level of 0.05, and a power of 0.95, is 24 participants per group. Accounting for an anticipated 30 % attrition rate at the 24-month follow-up, a total of 32 participants per group is required.

2.2. Procedure

2.2.1. Inclusion screening and random assignment to experimental groups

Out of 917 received initial expressions of interest to participate from individuals, 660 fully completed the online questionnaire and registration forms, and 258 of these full completions satisfied the inclusion criteria of the questionnaire, who proceeded to the two clinical intake interviews. These interviews were spaced two weeks apart, during which candidates completed a sleep diary, reviewed the study information brochure, signed a consent form, and obtained written approval from their BZ-prescribing physician.

The first interview aimed to assess the previously-noted inclusion and exclusion criteria (DSM-5 criteria, physician authorization, BZ deprescription criteria), and address participants' questions. The second interview focused on collecting the signed consent form, presenting and scheduling the study procedures, and randomly assigning participants into one of the four principal experimental groups of a 2 × 2 design (SS CBT-I/ACT-I, Short/Long Taper duration), as shown in Fig. S1. The personnel responsible for enrolling participants and those assigning them to interventions had access to the random allocation sequence. Tapering monitoring sessions (10-min calls) were conducted. The graduate clinical psychology students who conducted the tapering monitoring sessions were blinded to ACT-I intervention. Randomization of participants into experimental groups was planned using the Random Allocation Software. A simple and sequential randomization procedure was used. At the end of the inclusion phase, group analysis revealed an imbalance between the four experimental groups on some of our outcome measures and variables of interest, namely the level of BZ dependence, psychopathology, and half-life of the BZ consumed. The randomization process during the final recruitment wave was therefore intentionally adjusted to promote balance among the groups: participants were included in the four conditions by controlling their characteristics on these variables; for example, the next subject with a high dependence score was automatically assigned to the condition containing a majority of participants with a low level of dependence. We performed t-tests to verify that no significant residual imbalance remained. The random assignments also equally-distributed participants based on the half-life durations (Short/Long) of the BZ they were taking. Note that participants were randomly scheduled either to immediately begin the experiment (“Direct” condition) or a delayed start between 4 and 7 months later ("Waiting List" – WL condition). This waiting list was necessary due to the high number of inclusion interviews to be conducted, within a limited amount of time and human resources.

In summary, of the initial 660 candidates having completed the questionnaire, 532 candidates were excluded due to severe psychiatric or medical conditions, failure to meet BZ use duration or frequency criteria, or lack of interest. From an initial 258 that proceeded to the clinical intake/screening interviews, 128 candidates satisfied the criteria and were randomized into equal-sized groups (N = 32 each) of one of the following four conditions: 1. Group A.1: Short taper with ACT-I (20 Direct start, 12 WL); 2. Group A.2: Short taper with SS CBT-I (22 Direct start, 10 WL); 3. Group B.1: Long taper with ACT-I (16 Direct start, 16 WL); 4. Group B.2: Long taper with SS CBT-I (20 Direct condition, 12 WL). Just after the pre-treatment evaluation, as part of the BZ tapering program, to control for individuals who took different dosage amounts by day, or multiple kinds of sleep medications concomitantly, two medication stabilization sessions were performed over 4 weeks to align them on the same dose per day. As shown in Fig. S1, in total the BZ tapering program consisted of 10 sessions for adherence, which consisted of 10-min telephone calls. The ACT-I and SS CBT-I sessions were performed by videoconference. In total, Groups A.1 and A3 benefitted from 580 min with a psychologist (eight 60-min sessions of ACT-I + ten 10-min drug tapering phone support sessions) while Groups A.2 and A.4 benefitted from 160 min with a psychologist (one 60-min SS CBT-I session + ten 10-min drug-tapering phone support sessions).

The data were collected in a repeated-measures design, in which the participants were assessed prior to treatment, and then post-treatment, at 1, 3, and 12 months in the form of follow-up interviews. Before each follow-up interview, they completed a sleep diary for two weeks and the same battery of questionnaires detailed in the Measures section. All follow-up interviews were conducted individually by videoconference.

2.3. Measures

Six primary outcome variables of this study were computed. The first three correspond to three classes of outcomes in discontinuing BZ use (Drug-Free, Reduction more than 50 % of BZs, and Failed Reduction), the fourth as the average daily BZ dose taken, and the latter two questionnaire measures of perceived dependence and mental health. In greater detail, these measures are defined as follows:

1) The proportion of patients who successfully discontinued BZ use (Drug-free). Successful discontinuation was defined as completing the tapering plan without significant deviations and abstaining from consumption, except for, under highly exceptional circumstances, a *pro re nata* use within the two weeks following the zero-dose date (ensuring that a minimal dose taken).
2) The proportion of participants who successfully reduced their BZ consumption by 50 % or more in the two weeks preceding the 1, 3, and 12 months post-treatment follow-ups.
3) The proportion of participants who failed to reduce their BZ consumption by more than 50 % in the two weeks preceding the 1, 3, and 12 months post-treatment follow-ups (Failed reduction).
4) The average daily BZ consumption dose, expressed in diazepam equivalents.
5) The severity of benzodiazepine dependence, measured using the Benzodiazepine Dependence Questionnaire (BDEPQ) [51].
6) Overall deterioration of mental health, assessed using the Symptom Checklist-90 (SCL-90) [52,53], was considered a primary outcome to monitor potential adverse effects of BZ withdrawal.

The secondary outcomes variables focused on the following dimensions (see the Supplementary Data for details):

Sleep Variables: Participants maintained a sleep and medication tracking diary for the 14 days preceding each evaluation interview, allowing for the collection of the following parameters: Total Sleep Time (TST), Time in Bed (TIB), Sleep Onset Latency (SOL), Number of Nighttime Awakenings (NWAK), total Wake time After Sleep Onset (WASO), Time Awake prior to rising (TWAK), Sleep Efficiency (SE), Subjective Sleep Quality (SQ), doses and number of medications taken for sleep, and the diazepam-equivalent of BZ consumption (total dosage over the recorded period and daily average). A minimum compliance of 10 out of the 14 requested days was required for the sleep diary to be considered complete.

Participants also completed the Insomnia Severity Index (ISI), the Epworth Sleepiness Scale assessing daytime sleepiness [54], the Pichot Fatigue Scale [55], and the short version of the Morningness-Eveningness Questionnaire [56].

For BZ use, they completed the Cognitive Attachment to Benzodiazepines Scale (ECAB) [57].

Withdrawal Symptoms and Confidence in Discontinuation Variables: The severity of withdrawal symptoms was measured using the Clinical Institute Withdrawal Assessment-B (CIWA-B) [58]. Levels of stress (anxiety), rumination, and withdrawal symptoms before or during bedtime were assessed as a percentage from 0 to 100 %.

Self-efficacy was assessed using two questions: 1) "How confident are you in your ability to stop taking benzodiazepines?" rated on a scale from 0 % ("I won't succeed") to 100 % ("I feel fully capable of discontinuing benzodiazepines"), and 2) "How confident are you in your ability to sleep without benzodiazepines?" also rated from 0 % ("I won't succeed") to 100 % ("I feel fully capable of sleeping without benzodiazepines").

These variables were collected at the same intervals as the 10 tapering sessions (10-min calls), including twice during the stabilization phase, six times during the BZ tapering program, and twice after, referred to as booster sessions (between the last tapering step and the 1-month follow-up).

Acceptance and Commitment Therapy (ACT)-related Variables: Psychological flexibility was measured using the Acceptance and Action Questionnaire (AAQ-II) [59], the Mindful Attention Awareness Scale (MAAS) [60], and the Comprehensive Assessment of Acceptance and Commitment Therapy Processes (CompACT) [61].

Psychopathology Variables: Other complementary psychiatric dimensions were measured using the Short Version of Pichot's Depression and Anxiety Scale (Q2DA) [62], (Pichot, 1986), personality traits with the Big Five Inventory (BFI-10) [63] and the Dependent Personality Questionnaire (DPQ) [64]. (Loas et al., 2010). Thought suppression tendencies were assessed using the White Bear Suppression Inventory (WBSI) [65].

Control Variables: Quality of life with the WHOQOL-26 (WHOQOL-BREF) [66,67], and hyperventilation syndrome (HVS) with the Nijmegen Questionnaire [68]. Social desirability, measured using the short version of the Marlow-Crowne Social Desirability Scale (MC-SD) [69], was assessed pre-treatment. Therapeutic alliance, evaluated with the Working Alliance Inventory-Short Revised (WAI-SR) [70], and treatment satisfaction, assessed using the Client/Patient Satisfaction Questionnaire (CSQ-8) [71] were also completed at the 3-month follow-up.

Except for monitoring and control measures, all questionnaires were completed once before treatment (for participants in the waiting-list condition, an additional assessment was conducted after the waiting period), at 1 month post-treatment, and at 3-month and 12-month follow-ups. The 3-month post-treatment assessment in the short-taper experimental condition corresponded in duration to the 1-month post-treatment assessment in the long-taper experimental condition. Therefore, comparative analyses between these experimental conditions focused solely on these follow-up measures.

2.4. Taper program and psychotherapy

2.4.1. Taper program

The BZ reduction program towards discontinuation, common to all four experimental arms, included several phases. First, a 4-week *stabilization phase* took place during which participants were guided to take the same dosage amount per day, or with poly-substance use, reduced their consumption to only one kind of BZ, and discontinued any other sleep aids (e.g., homeopathy, herbal medicine, melatonin). Participants who consumed BZs in a fragmented manner (e.g., ½ tablet of zolpidem at bedtime and ½ in case of nighttime awakening) were encouraged to take their BZ every night, solely at bedtime. Second, a *withdrawal session* at the end of the stabilization phase, corresponding to the 4th session of ACT-I for groups A.1 and B.1, and to SS CBT-I for groups A.2 and B.2 (described below). This 60-min session involved information about BZs, the creation of a personalized tapering plan for the participant, and strategies for managing possible rebound insomnia related to withdrawal. These sleep management strategies were approached either from an ACT perspective for the ACT-I groups, or according to the traditional CBT-I approach for the SS CBT-I groups. Then, the *taper program* included six reduction steps of 25 % of the dose, that occurred at 1-week intervals for the Rapid condition, and 3 weeks for the long condition (see Fig. S1 in the Supplementary data). Two interviews took place during the stabilization phase, six interviews during the tapering phase (one per reduction step), and the final two booster sessions were held between the last tapering step and the 1-month post-treatment follow-up. A monitoring and psychological support module, consisting of 10 sessions of 10-min each, was designed to assess withdrawal symptoms and encourage participants to adhere to both the tapering program and the behavioral techniques.

2.4.2 Psychotherapy

Single-Shot CBT-I (SS CBT-I): in line with [10], participants that were assigned to this experimental condition received a single, 60-min CBT-I session focused on the CBT-I elements of sleep restriction and stimulus control, which participants were encouraged to implement throughout the taper process to manage any potential rebound insomnia.

ACT-I: Following standards of the latest generation of CBT-I, participants assigned to this condition received 8 weekly 1-h sessions of Acceptance and Commitment Therapy (ACT). Therefore, this intervention followed Steven Hayes' 8-session protocol [72] adapted for insomnia [15], with exercises tailored to the issues related to withdrawal from BZs. Between each session, participants were encouraged to engage in psychoeducational readings from a bibliotherapy book on managing insomnia through ACT, focusing on the functioning of sleep or deepening the ACT-I processes worked on during the sessions.

Further information about therapists and data analysis can be found in the supplementary data.

3. Results

3.1. Participant sample

Out of the 128 subjects randomized in the study, 107 completed the full protocol. The account of these 21 attrition participants are as follows: 12 at post-treatment assessment (7 medical reasons, 1 non-compliance, 4 drop out); 3 at the 3-month follow-up (1 non-compliance, 1 drop out, 1 incomplete data); 7 at the 12-month follow-up (1 medical reasons, 2 non-compliance, and 4 drop out) (Fig. S2).

While sample size equivalence was verified before treatment, participant attrition led to imbalanced sample sizes on key variables. Given that the initial sample size calculations accounted for a 30 % attrition rate but the actual attrition was lower (15 %), randomized down-sampling could be performed to appropriately rebalance the groups, at 87 participants total (21–22 participants per each of 4 experimental groups, as in Table S1). Nonetheless, in respect across-condition pre-baseline tests, such as intention-to-treat (ITT) analyses, biases could be raised; therefore, only the per-protocol analyses are hereafter presented.

The groups, formed after the randomized down-sampling, were equivalent at baseline across all primary variables (Table S1) and secondary variables (Supplementary Data Table S3) (all p-values >0.05). Therapeutic alliance and treatment satisfaction were also comparable between groups.

3.2. Effect size of the program: analysis of the WL subsample

Twenty-eight participants were on the waiting list for an average duration of 5.8 months (equivalent to the time between the start of the program and the one-month follow-up for the long taper or the three-month follow-up for the short taper). Among them, 60.7 % followed the ACT-I + Taper Program protocol. After the waiting period, prior to starting the experiment, no participant independently succeeded in either abstaining or significantly reducing their consumption (Table 1). Then, at the one-month follow-up (Fu-1mo), 54 % were abstinent, and 46 % had reduced their BZ consumption by more than 50 %. The average diazepam-equivalent dose significantly decreased from 6.06 mg (SD = 2.89) at pre-baseline to 0.50 mg (SD = 0.83) at Fu-1mo, with a large effect size ($d = 1.37$, $p < 0.001$). The level of dependence also significantly decreased from 42.79 (SD = 11.76) at pre-baseline to 20.39 (SD = 13.31) at Fu-1mo, with a moderate effect size ($d = 0.59$, p = 0.004). At the 12-month follow-up (Fu-12mo), a relapse was observed in 10 % of participants.

During the waiting period (without intervention), the 28 participants significantly and slightly reduced their global psychopathology score (SCL-90, 38.2 from 50.2, $t = -3.91$, $df = 27$, $p < 0.001$), their mental rumination (WBSI) ($t = -3.70$, $df = 27$, $p = 0.001$), and the severity of their insomnia (ISI) ($t = -4.147$, $df = 27$, $p = 0.0003$), while slightly improving their mental health-related quality of life (WHOQOL mental health) ($t = 3.45$, $df = 27$, $p = 0.002$) (Supplementary Data Table S4).

Comparing the non-intervention period to the intervention period at the 1-month follow-up, undergoing the tapering program (regardless of ACT status = with or without ACT) resulted in a significant reduction in diazepam-equivalent doses ($d = -1.37$, $t = -6.37$, $df = 21$, $p < 10^{-5}$) and additional benefits with the following effect sizes: large reduction in benzodiazepine-related attachment cognitions (ECAB) ($d = -1.0$, $t = -5.28$, $df = 27$, $p < 10^{-4}$); moderate reduction in benzodiazepine dependence (QDEPB) and time spent in bed (TIB) (all $|d| > 0.5$ and <1.0); and small reduction in global psychopathology (SCL-90), mental rumination (WBSI), insomnia severity (ISI), and improvements in acceptance and commitment processes (COMPACT) (all $|d| < 0.5$).

3.3. BZ discontinuation and reduction of use: full participant sample

Across all experimental groups, at 12 months, 58.6 % of participants were abstinent, 29.9 % had maintained a dose reduction of more than 50 %, and 11.5 % had not managed to reduce their consumption by more than half (failure) (Table 2). On average, we observed a significant 90 % reduction in diazepam-equivalent doses one month after the intervention ($d = -1.50$) and one year later ($F(3,258) = 200.156$, $p < 0.0001$). Dependence levels (QDEP) and mental health disorders (SCL-90) significantly decreased, and these improvements were maintained over time (respectively: $F(3,258) = 173.8$, $p < 0.0001$, $F(3,258) = 58.53$, $p < 0.0001$).

Across all experimental groups, at 12 months, participants significantly improved on all secondary outcomes (Supplementary Data Table S5). Only daytime sleepiness, total sleep time, and Wake Time After Sleep Onset did not improve.

3.4. Evaluation of the added benefit of the full 8-sessions ACT-I program vs. the single-shot CBT-I

Overall, the addition of 8 sessions of ACT-I did not provide added value on the main outcomes (see Table 2). Benefits of ACT-I are observed at post-treatment 1 or 3 months for certain variables, but do not persist in the long term. Indeed, all p-values related to the interaction of time and ACT group are >0.05 (column P4 row ACT-I, Supplementary Data Table S6).

Table 1
Evolution over time of BZ Consumption, Dependence (QDEP), and Psychopathology Levels in Participants on the Waiting List (n = 28) between Pre-Baseline, Pre-Treatment, and the 1, 3, and 12 month follow-ups (FU-1mo/FU-3mo/FU-12mo).

	Pre-baseline (PreB)	Pre-treatment (PreT)	1 month follow-up (T1)	3 month follow-up (T3)	12 month follow-up (T12)	Effect size[a]	p-value
Drug-free - n (%)	0 (0)	0(0)	15(53.6)	15(54)	14(50.0)		*<0.001*
Reduction more than 50 % of BZs - n (%)	0 (0)	0(0)	13(46.4)	12(43)	11(39.3)		*<0.001*
Failed reduction - n (%)	28(100)	28(100)	0(0)	1(4)	3(10.7)		*<0.001*
Hypnotic dosage (daily lorazepam equivalent in mg) - mean (sd)	6.06 ± 2.89	5.49 ± 2.74[22]	0.50 ± 0.83	0.48 ± 0.62	1.05 ± 2.05	−1.37 [22]	*<0.001*
QDEP - mean (sd)	42.8 ± 11.8	38.9 ± 12.3	20.4 ± 13.3	17.4 ± 16.6	19.0 ± 20.0	−0.59	*0.004*
SCL-90 (total) - mean (sd)	50.2 ± 30.8	38.2 ± 23.9	28.6 ± 22.56	25.2 ± 20.7	29.5 ± 25.8	0.10	0.59

n(%) and mean ± SD for binary and quantitative variable, respectively.
p-value: for binary variable, binomial test comparing T1 observed proportion to 0 or 28; for quantitative variables: paired Student test between (Pre-Treatment – Pre-Baseline) and (T1 - Pre-Treatment).
[n] in case of missing data.
[a] Effect size: Cohen'd for paired data between (Pre-Treatment – Pre-Baseline) versus (1-month FU - Pre-Treatment).

Table 2
Evolution of main outcomes based on therapeutical condition and time (pre-treatment, one-month follow-up (FU-1mo), three-month follow-up (FU-3mo), and twelve-month follow-up (FU-12mo)), $N = 87$.

	Pre-treatment	1-month FU T1	P1	3-month FU T3	P2	12-month FU T12	P3	P4
Drug-free - n (%)								
All	**0(0)**	**53(60.9)**	0.66	**49(56.3)**	0.39	**51(58.6)**	1.00	
ACT-I	**0(0)**	**28(63.6)**	<0.001	**27(61.4)**	<0.001	**26(59.1)**	<0.001	
SS CBT-I	**0(0)**	**25(58.1)**	<0.001	**22(51.2)**	<0.001	**25(58.1)**	<0.001	
Reduction ore than 50 % of BZs - n (%)								
All	**0(0)**	**29(33.3)**	0.82	**31(35.6)**	1.00	**26(29.9)**	0.64	
ACT-I	**0(0)**	**14(31.8)**	<0.001	**16(36.4)**	<0.001	**12(27.3)**	<0.001	
SS CBT-I	**0(0)**	**15(34.9)**	<0.001	**15(34.9)**	<0.001	**14(32.6)**	<0.001	
Failed reduction - n (%)								
All	**87(100)**	**5(5.7)**	0.68	**7(8)**	0.06	**10(11.5)**	0.74	
ACT-I	**44(100)**	**2(4.5)**	<0.001	**1(2.3)**	<0.001	**6(13.6)**	<0.001	
SS CBT-I	**43(100)**	**3(7)**	<0.001	**6(14)**	<0.001	**4(9.3)**	<0.001	
Hypnotic dosage (daily lorazepam equivalent in mg) - mean (sd)								
All	**6.02 ± 3.49**	**0.6 ± 1.19**	0.48	**0.74 ± 1.24**	0.22	**1.06 ± 2.05**	0.85	<0.001
ACT-I	**6.17 ± 3.73**	**0.67 ± 1.47**	<0.001	**0.57 ± 1.05**	<0.001	**1.10 ± 2.17**	<0.001	0.57
SS CBT-I	**5.87 ± 3.27**	**0.53 ± 0.83**	<0.001	**0.91 ± 1.4**	<0.001	**1.02 ± 1.94**	<0.001	
QDEP - mean (sd)								
All	**43 ± 13.2**	**19.5 ± 12.9**	0,25	**16.1 ± 15.3**	0.20	**16.7 ± 16.7**	0.74	<0.001
ACT-I	**41.9 ± 13.8**	**17.9 ± 12.2**	<0.001	**14 ± 14.1**	<0.001	**16.2 ± 17.6**	<0.001	0.72
SS CBT-I	**44.2 ± 12.7**	**21.1 ± 13.5**	<0.001	**18.3 ± 16.3**	<0.001	**17.3 ± 16.0**	<0.001	
SCL-90 (total) - mean (sd)								
All	**55.9 ± 31.6**	**34.5 ± 30.1**	0.94	**31.8 ± 30.2**	0.74	**30.1 ± 24.9**	0.10	<0.001
ACT-I	**51.5 ± 27.1**	**34.3 ± 30.8**	<0.001	**30.7 ± 28.9**	<0.001	**25.8 ± 17.9**	<0.001	0.65
SS CBT-I	**60.4 ± 35.3**	**34.8 ± 29.6**	<0.001	**33 ± 31.7**	<0.001	**34.6 ± 30.0**	<0.001	

FU: Follow-Up.
P-value for all data: P1, P2, P3: intersubject comparison between both groups on each occasion (1, 3, or 12 months).
P-value for each group (ACT, SS CBT-I), P1, P2, P3: intrasubject comparison between each occasion and pre-treatment.
For binary variable, Fisher exact test (for All) and unilateral binomial test (for each group).
For quantitative variable, P1, P2 P3, Student, Welch or Wilcoxon test as appropriate.
P4: univariate (with only time for All data) or complete (with time and group on "ACT-I" lines) mixed model with a random intercept (due to the subject); P4: p-value for time effect (for lines All) or p-value for the interaction time and group effect (for ACT-I lines).

Table 3
Evolution of main outcomes based on tapering speed condition (rapid = 6 weeks, long = 18 weeks) and time (pre-treatment, follow-up at one month (FU-1mo), three months (FU-3mo), and twelve months (FU-12mo)), $N = 87$.

	Pre-treatment	1-month FU T1	P1	3-month FU T3	P2	12-month FU T12	P3	P4
Drug-free - n (%)								
All	**0(0)**	**53(60.9)**	0.51	**49(56.3)**	0.83	**51(58.6)**	0.83	
Short Taper	**0(0)**	**28(65.1)**	<0.001	**25(58.1)**	<0.001	**26(60.5)**	<0.001	
Long Taper	**0(0)**	**25(56.8)**	<0.001	**24(54.5)**	<0.001	**25(56.8)**	<0.001	
Reduction ore than 50 % of BZs - n (%)								
All	**0(0)**	**29(33.3)**	0.36	**31(35.6)**	1.00	**26(29.9)**	0.48	
Short Taper	**0(0)**	**12(27.9)**	<0.001	**15(34.9)**	<0.001	**11(25.6)**	<0.001	
Long Taper	**0(0)**	**17(38.6)**	<0.001	**16(36.4)**	<0.001	**15(34.1)**	<0.001	
Failed reduction - n (%)								
All	**87(100)**	**5(5.7)**	0.68	**7(8)**	1.00	**10(11.5)**	0.52	
Short Taper	**44(100)**	**3(7)**	<0.001	**3(7)**	<0.001	**6(14)**	<0.001	
Long Taper	**43(100)**	**2(4.5)**	<0.001	**4(9.1)**	<0.001	**4(9.1)**	<0.001	
Hypnotic dosage (daily lorazepam equivalent in mg) - mean (sd)								
All	**6.02 ± 3.49**	**0.6 ± 1.19**	0.38	**0.74 ± 1.24**	0.98	**1.06 ± 2.05**	0.40	<0.001
Short Taper	**5.4 ± 2.59**	**0.44 ± 0.94**	<0.001	**0.82 ± 1.35**	<0.001	**0.93 ± 1.78**	<0.001	0.73
Long Taper	**6.63 ± 4.13**	**0.76 ± 1.39**	<0.001	**0.66 ± 1.14**	<0.001	**1.18 ± 2.29**	<0.001	
QDEP - mean (sd)								
All	**43 ± 13.2**	**19.5 ± 12.9**	0.46	**16.1 ± 15.3**	0.40	**16.7 ± 16.7**	0.79	<0.001
Short Taper	**43 ± 13.4**	**18.4 ± 12.5**	<0.001	**14.7 ± 11.8**	<0.001	**16.3 ± 15.2**	<0.001	0.80
Long Taper	**43.1 ± 13.3**	**20.5 ± 13.4**	<0.001	**17.5 ± 18.1**	<0.001	**17.2 ± 18.3**	<0.001	
All								
SCL-90 (total) - mean (sd)	**55.9 ± 31.6**	**34.5 ± 30.1**	0.53	**31.8 ± 30.2**	0.34	**30.1 ± 24.9**	0.87	<0.001
Short Taper	**55.9 ± 34.3**	**35.9 ± 32.5**	<0.001	**35.3 ± 34**	<0.001	**30.6 ± 25.9**	<0.001	0.34
Long Taper	**55.8 ± 29**	**33.2 ± 27.8**	<0.001	**28.5 ± 25.9**	<0.001	**29.7 ± 24.1**	<0.001	

FU: Follow-Up.
p-value for all data: P1, P2, P3: intersubject comparison between both groups at each occasion (1, 3, or 12 months).
p-value for each group (short taper, long taper), P1, P2, P3: intrasubject comparison between each occasion and pre-treatment.
For binary variable, Fisher exact test (for All) and unilateral binomial test (for each group).
For quantitative variable, P1, P2 P3, Student, Welch or Wilcoxon test as appropriate.
P4: univariate (with only time for All data) or complete (with time and group on "Taper Duration" lines) mixed model with a random intercept (due to the subject); P4: p-value for time effect (for lines All) or p-value for the interaction time and group effect (for Taper Duration).

3.5. Evaluation of the optimal tapering schedule between 6 weeks and 18 weeks

We observed no significant difference between the short tapering protocol (6 weeks) and the long tapering protocol (18 weeks) either on the evolution of the main outcomes (Table 3) or on the secondary outcomes (Supplementary data Table S6).

In general, we observed a progressive reduction in withdrawal symptoms (CIWA-B) at each stage of reduction, except when participants transitioned from 5 nights of consumption to 3 nights (Fig. 1). However, when comparing the half-lives of BZs with the duration of withdrawal (short versus long), we observed a cumulative and progressive effect of withdrawal symptoms for intermediate to long half-lives when the withdrawal was rapid (6 weeks) (Fixed time and 4 subgroups interaction effects; $F(3,431) = 2.50$, $p = 0.059$). Slopes were estimated at 0.38 for ShortTaper:LongHalf-Life, −0.57 for LongTaper:LongHalf-Life, −0.70 for ShortTaper:ShortHalf-Life, and −1.02 for LongTaper:ShortHalf-Life subgroups.

4. Discussion

This study evaluated the effectiveness of a remotely-delivered Benzodiazepine and Z-drug (BZ) abstinence or reduction of use program, comparing the outcomes associated with full vs. shortened treatment plans (8 sessions of ACT-I vs. single-shot CBT-I), rapid vs. long tapering schedules (6 vs. 18 weeks), and BZ half-life durations (short vs. long). The principal outcomes measured were abstinence success or maintained BZ-dose reduction, and withdrawal symptoms as a function of tapering rates. Notable secondary variables assessed were those linked to mental wellness and psychopathology.

The results of the study demonstrate a remarkable effectiveness of this BZ reduction program, irrespective of modality, based on an observed abstinence success rate of 58.6 % and otherwise 29.9 % of consumers who maintained a BZ dose reduction equivalent to at least one diazepam dose, in total 88.5 % of consumers. Corroborating this result, the waiting list (WL) subgroup within each experimental condition allowed for verifying that this result is not due to a placebo effect, through comparing their non-intervention period to treatment beginnings and follow-ups. These analyses also highlighted the secondary benefits directly observed one month after, which remained stable over time: less insomnia, less time spent in bed, fewer mental ruminations, fewer mental health issues, and greater mental flexibility.

The effectiveness of the program herein observed (88.5 % success rate) is comparable to those observed in previous studies exploring the efficacy of CBT for BZ cessation [2,30,33,36,38,73–75]. While one may postulate that discontinuing a medication aimed to assist sleep health would negatively impact clinical parameters, we herein observed the opposite: those who followed the program improved sleep, quality of life, mental flexibility, and reduced insomnia, mental ruminations, and other mental health issues. These data are consistent with the known effects of BZs on sleep quality, which show a reduction in slow-wave sleep and REM sleep [35,76,77].

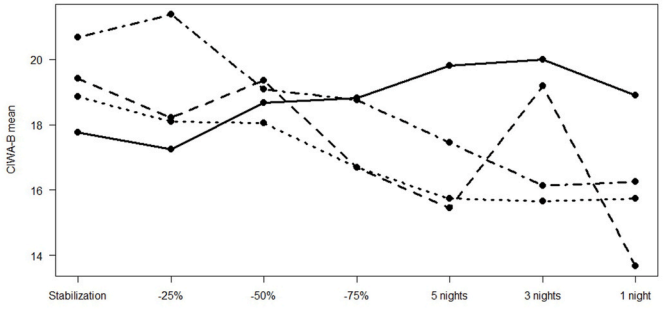

Fig. 1. Evolution of benzodiazepine (BZ) withdrawal symptoms (CIWA-B mean scores) across different tapering schedules and BZ half-lives: Dashed line with long dashes (– –): Short taper with a short half-life BZ. Solid line (—): Short taper with a long half-life BZ. Dash-dotted line (–·–·): Long taper with a short half-life BZ. Dotted line (···): Long taper with a long half-life BZ.

In respect to the analysis of the therapeutical conditions: no added benefit of the full-length ACT-I protocol was observed over the single-shot (SS) CBT-I that focused on sleep restriction and stimulus control. This may potentially be explained by having reached therapeutic ceiling effect in the BZ Tapering program, which proved quite effective, to which we added CBT methods (ACT-I and SS CBT-I) to be compared. Therefore, while ACT-I may potentially provide an added benefit, this study does not allow us to confirm it. Furthermore, it may be speculated that, as ACT-I is a psychotherapeutic approach targeting both general and complex processes, it is possible that this approach does not align with a certain percentage of participants. A similar result was obtained in Morin et al., 's 2005 study, where it was observed that although the TCC + Taper program group had a higher rate of abstinence at the end of treatment, at 3 months and 12 months post-treatment, the abstinence rate remained higher in the group that underwent the Taper program alone. The authors hypothesized that patients who managed to stop their medication without assistance might have developed better self-efficacy compared to those who stopped with the help of psychotherapy. On the other hand, in our study, all participants received psychological support throughout the tapering process, and the majority of them reported that being regularly supported and encouraged was helpful, even indispensable, to the success of tapering or discontinuing their BZ use. Another factor to mention regarding comparative effectiveness is the confounding factor of therapist-contact time: participants in the ACT-I groups received substantially more therapist-contact time (580 min) compared to those in the single-session CBT-I groups (160 min), which may have increased the effectiveness of the ACT condition through greater therapeutic support, independent of ACT processes. However, given the lack of evidence demonstrating the superiority of ACT over SS-CBT, the additional time spent in this condition may also have reduced its effectiveness, based on the aforementioned hypothesis of a possible lack of motivation to engage in introspective work among a number of participants. Similarly, it is possible that more therapist-contact time in the SS-CBT condition could have increased the effectiveness of this intervention.

As for withdrawal symptoms which could complicate the BZ abstinence success, the only difference we observed was for rapid (6 week) tapering in individuals that took long half-life BZs, in which a cumulative trend of withdrawal symptoms followed. Indeed, the withdrawal symptoms for long half-life BZs are delayed in time between 2 and 10 days and can last for several weeks, unlike those with short half-lives, which occur within 24 h and disappear within a few days [24,45]. This means that reducing the dose every week could progressively intensify the withdrawal symptoms. Cumulative withdrawal symptoms during rapid tapering of long half-life benzodiazepines are primarily driven by neuroadaptations in the GABAergic system. Chronic benzodiazepine exposure leads to downregulation and desensitization of GABA(A) receptors, as well as compensatory upregulation of excitatory neurotransmission (e.g., glutamatergic pathways). When benzodiazepines are rapidly reduced, the abrupt decrease in GABAergic tone unmasks this hyperexcitable state, resulting in withdrawal symptoms such as anxiety, insomnia, autonomic instability, perceptual disturbances, and - in severe cases -seizures and delirium [78,79]. Long half-life benzodiazepines (e.g., diazepam) have slower elimination, so withdrawal symptoms may be delayed, typically emerging several days to a week after dose reduction, peaking in the second week, and resolving over several weeks. However, rapid tapering can still precipitate cumulative withdrawal due to insufficient time for neuroreceptor adaptation, leading to a build-up of symptoms with each dose reduction [79]. Physiologically, cumulative withdrawal reflects the mismatch between the rate of benzodiazepine clearance and the slower pace of neuroadaptation. Rapid tapering increases the risk of severe withdrawal,

including protracted symptoms that may persist for months due to persistent neurochemical dysregulation [80,81]. Adjusting the taper pace and using hyperbolic (nonlinear) reductions can help maintain more consistent receptor occupancy and reduce symptom burden [81].

This observation is a new, important finding to explore in greater depth, as it contrasts with current clinical practice, that typically promotes long tapering schedules for BZs with short half-lives rather than for long half-lives drugs [82]. Furthermore, a commonly used strategy in clinical practice is to substitute a BZ with a longer half-life in users of short half-life drugs before withdrawal, as reducing long half-lives would result in fewer withdrawal symptoms. In its recommendations on BZ withdrawal, the NICE committee suggests that we can, "if the person is withdrawing from a benzodiazepine with a short half-life, consider switching to a benzodiazepine with a longer half-life », but also states in its rationale section that « Despite being common practice, there is a lack of evidence to support conversion to a preparation with a longer half-life" [83]. Our study presents initial results indicating that switching to a BZ with a longer half-life would be of little benefit, in the sense that the half-life of the BZ consumed is less decisive than the duration of withdrawal: consuming a long half-life would not exempt users from a very gradual withdrawal, similar to withdrawal from short half-life BZs, in order to minimize potential withdrawal symptoms. The implications of cumulative withdrawal symptoms during rapid tapering of long half-life benzodiazepines are well addressed in current clinical guidelines. The National Institute for Health and Care Excellence (NICE) and the British Association for Psychopharmacology (BAP) both emphasize that rapid tapering, even with long half-life agents, can lead to significant withdrawal symptoms due to delayed onset and accumulation of neuroadaptation deficits. This is because long half-life benzodiazepines are eliminated slowly, so withdrawal symptoms may not manifest immediately after dose reduction but can accumulate and intensify with successive reductions, reflecting delayed receptor adaptation and the slow reversal of GABA(A) receptor downregulation and compensatory glutamatergic upregulation. The American Society of Addiction Medicine and the American College of Medical Toxicology, in their joint guideline, explicitly recommend gradual, individualized tapering—typically 5–10 % dose reductions every 2–4 weeks, with even slower reductions at lower doses—to minimize cumulative withdrawal and allow for neuroreceptor homeostasis. They caution that rapid tapers increase the risk of both acute and protracted withdrawal, including symptoms that may persist for months due to incomplete receptor adaptation. The guidelines also note that withdrawal symptoms may be delayed for several days to weeks after each reduction, and recommend close monitoring and flexible adjustment of the taper pace in response to emerging symptoms, including pausing or slowing the taper if significant symptoms develop. This approach is designed to mitigate the risk of cumulative withdrawal and to support safe discontinuation, especially in patients with high physical dependence or psychiatric comorbidity [80,81]. In summary, current clinical guidelines from the American Society of Addiction Medicine and the American College of Medical Toxicology, as well as consensus from NICE and BAP, highlight that rapid tapering of long half-life benzodiazepines can result in cumulative and delayed withdrawal symptoms due to slow receptor adaptation, and recommend gradual, individualized tapering strategies to minimize these risks.

4.1. Strengths

A noteworthy angle of this work involved the assessment of a more readily-accessible therapeutic approach to patients, with lower financial cost and time commitment, which could also be more often sought; and secondly, examining a more ecologically-valid sample, representing a population which often involves a larger age range than those assessed in previous studies, and naturally, individuals who take multiple medications or present comorbidities.

Contrasting with a number of other studies in the field, our sample was comparatively large (n = 87), heterogeneous, and ecological: it involved long-term users (1st quartile = 9 years, 3rd quartile = 22 years), 63 % that used multiple sleep medications, who had tried to stop using BZs multiple times, suffering from various comorbidities (40 % with mental disorders, 57 % with organic disorders), with a sizeable age range of 39–69 years old. All participants presented insomnia symptoms despite regular BZ use. This study therefore allowed for a wider generalization of the effectiveness of this type of intervention, which has mainly been previously demonstrated in narrower samples: i.e., seniors with few comorbidities [2,30,32,34,35].

This study demonstrated that short interventions, including CBT methods targeting the root cause of BZ use (here, insomnia) may be as effective as a full psychotherapy program when paired with a tapering program. Thus, a single-shot of CBT (SS CBT-I) could be sufficient to improve sleep and abstinence rates. The current results add to those of previous studies suggesting that a full CBT program may not be essential for successful BZ discontinuation [2,9]. This finding is important because short interventions for BZ abstinence, which focus on BZ dependence without providing methods to manage the insomnia that lead to their use, or rebound insomnia, only achieve a 33 % abstinence rate, compared to 80 % in our program [7]. In this regard, it could easily be implemented by primary care physicians, nurses, or psychologists trained in basic sleep concepts, sleep restriction, and stimulus control methods.

4.2. Limitations

This study did not allow us to draw definitive conclusions about potential different effects from different treatment options. That is, we observed too few differences between the experimental groups, likely due to three reasons: 1) *ceiling effect*: the SS CBT-I paired with the Tapering program was already very effective, 2) *tapering contrast*: the 6-week tapering schedule is already very gradual (compared to studies that compare gradual tapering to abrupt withdrawal), 3) *sample size*: to ensure that the differences observed after treatment were indeed attributable to the experimental conditions, the need to make the experimental groups equivalent post hoc forced us to reduce the sample size (from N = 106 to N = 87); this reduction in statistical power may have limited the observation of inter-group differences.

Inherent to this type of experimental design, selection bias is likely: of the all individuals initially interested (who potentially required tapering), only 87 completed the full program. Our study aimed to optimize benzodiazepine (BZD) tapering strategies in light of findings showing that (1) brief interventions are low-cost but only modestly effective, and that the integration of CBT-I components could enhance their effectiveness, and (2) ACT-I represents a promising but costly approach for BZD discontinuation. The protocol was not designed to specifically compare the therapeutic components of the two approaches, but rather to estimate the effectiveness of each intervention. We might have expected better long-term clinical outcomes with ACT-I; however, this was not observed. Finally, the results of this BZ discontinuation program cannot be generalized to individuals using BZs for anxiolytic purposes or to those with substance abuse issues whose diazepam-equivalent doses far exceed those in our sample.

4.3. Perspectives

It would be relevant to replicate our base protocol with users that take BZs for anxiolytic purposes, especially among individuals with panic disorder. Instead of following Barlow's 12-session therapeutic program [84], a "single shot" CBT program could focus solely on teaching panic attack management techniques and gradual *in vivo* exposure to anxiety-inducing stimuli.

It would also be interesting to conduct a Mediation Model to identify relevant moderators and mediators in the success or failure of BZ cessation. Some psychological factors are known to predict the severity

of withdrawal symptoms or the success of BZ cessation, such as dependent personality traits [45,85,86], neuroticism [44,45,87], or self-efficacy related to withdrawal [88–91]. Analyzing the moderators would allow us to establish patient profiles for which a simple clinical intervention would be sufficient, and those who would require the addition of psychotherapy.

5. Conclusion

Let's not put the cart before the horse: or, *let's not demand individuals to cut their BZ use before teaching them the effective (or necessary) psychotherapeutic methods to manage their underlying problem*. By offering consumers tools to resolve their chronic or rebound insomnia, while providing light but regular psychological support, we propose an intervention that is low in cost financially and temporally, with very satisfactory abstinence success and reduction of use rates. Furthermore, it is worthwhile to consider a flexibility of extending the minimal tapering duration of 6 weeks herein proposed, based on a patient's extenuating circumstances and their sense of self-efficacy. Though, tapering duration should not exceed 18 weeks. Special attention should be given to consumers of BZs with medium/long half-lives, for whom a 6-week tapering duration could induce excessively intense withdrawal symptoms.

CRediT authorship contribution statement

Mélinée Chapoutot: Writing – review & editing, Writing – original draft, Methodology, Investigation, Funding acquisition, Conceptualization. **Marie-Paule Gustin:** Writing – review & editing, Writing – original draft, Formal analysis, Data curation. **Royce Anders:** Writing – review & editing, Formal analysis, Data curation. **Laure Peter-Derex:** Writing – review & editing, Writing – original draft, Investigation. **Hélène Bastuji:** Writing – review & editing, Writing – original draft. **Yasser Khazaal:** Writing – review & editing, Writing – original draft, Conceptualization. **Benjamin Putois:** Writing – review & editing, Writing – original draft, Validation, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Conceptualization.

Transparency and openness

The study's methods, aims, and inclusion/exclusion criteria were preregistered in 2021 on ClinicalTrials.gov under the title "Telepsychology for Benzodiazepine Withdrawal in Adults Suffering From Hypnotic-Dependent Insomnia" (NCT04751851) and received ethical approvals from both French (ID RCB 2021-A00196-35 – 05/06/2021) and Swiss (ID 2021-01626 – 11/25/2021) regulatory bodies the same year. Data collection was declared to the French Data Protection Authority (CNIL, 1612473vO). The REDCap and Symptosite software platforms were used to ensure data storage security.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2025.108744.

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