SPECIAL ARTICLE

Insomnia in children and adolescents. A consensus document☆

Gonzalo Pin Arboledas a,∗, Víctor Soto Insuga b, María José Jurado Luque c, Cleofé Ferrández Gomariz d, Inés Hidalgo Vicario e, Amalia Lluch Rosello f, Pedro Javier Rodríguez Hernández g, Juan Antonio Madrid h

a Asociación Española de Pediatría (AEP), Spain
b Sociedad Española de Neuropediatría (SEN), Spain
c Sociedad Española de Sueño (SES), Spain
d Fundación Prandi, Sociedad Española de Pediatría Extrahospitalaria y Atención Primaria (SEPEAP), Spain
e Sociedad Española de Medicina de la Adolescencia (SEMA), Spain
f Sociedad Española de Pediatría Extrahospitalaria y Atención Primaria (SEPEAP), Spain
g Sociedad Española de Psiquiatría Infantil (SPI), Spain
h Laboratorio de Cronobiología, Universidad de Murcia, Murcia, Spain

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KEYWORDS
Insomnia; Cognitive-behavioural; Melatonin; Antihistaminic

Abstract Insomnia is very common during childhood (30% of children under 5), and causes a serious cognitive and emotional consequence in learning, as well as significant medical comorbidity. It also affects the quality of life, not only of the child, but also of the whole family. Paediatrician training in its diagnosis and treatment is usually poor. For this reason a consensus document is presented on the management of insomnia in children and adolescents. This has been developed by members of the Spanish Paediatrics Association, the Spanish Sleep Society, the Spanish Society of Paediatric Outpatient and Primary Care, the Spanish Adolescent Medicine Society, the Spanish Child and Adolescent Society, and the Spanish Paediatric Neurology Society. This group suggests that diagnosis must be clinical and complementary tests will only be required in doubtful cases or when a differential diagnosis is needed. Likewise, treatment should be mainly based on cognitive-behavioural therapy and the modification of sleeping habits. Using medicines and other substances to make the sleep easier is currently quite common, even although there are no clinical guidelines to support this.
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∗ Corresponding author.
E-mail address: pinarboledasgonzalo@gmail.com (G. Pin Arboledas).

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Insomnio en niños y adolescentes. Documento de consenso

Resumen El insomnio es una patología muy frecuente en edad pediátrica (30% en niños menores de 5 años) que ocasiona una grave repercusión cognitiva y emocional en el apren-
dizaje junto con una importante comorbilidad médica y afectación de la calidad de vida del
niño y la familia. La formación de las pediatras en el diagnóstico y el tratamiento del mismo
suele ser deficitaria. Por todo ello, se presenta el documento de consenso sobre el manejo
del insomnio en la infancia y la adolescencia elaborado por representantes de la Asociación
Española de Pediatría, la Sociedad Española de Sueño, la Sociedad Española de Pediatría Extra-
hospitalaria y de Atención Primaria, la Sociedad Española de Medicina de la Adolescencia,
la Sociedad Española de Psiquiatría Infantil y la Sociedad Española de Neurología Pediátrica. Este
grupo recomienda que el diagnóstico debe ser clínico y solo en los casos dudosos o en que sea
necesario un diagnóstico diferencial serán necesarias pruebas complementarias. Asimismo el
tratamiento se debe basar principalmente en terapias cognitivo-conductuales y en una modifi-
cación de los hábitos de sueño. El uso de medicamentos y sustancias para facilitar el sueño es
elevado, aunque no existen guías clínicas que lo apoyen.
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Introduction and justification

Sleep is a developmental and active process that starts before birth as a result of a shifting and dynamic interaction
of biological, psychological and social factors; its evolution depends on the balance between these three domains.1
Insomnia affects 30% of children aged 6 months to 5
years. In Spain, 27% of children aged 5 to 7 years resist
going to sleep, 11% exhibit a prolonged sleep onset latency,
6% experience frequent awakenings, and 17% have diffi-
culty waking up in the morning.2 As for adolescents, 38.5%
report a perceived poor quality of sleep, and 23.1% a sleep
onset latency of more than 30 min.3 Sleep problems have an
impact on children and their environment and lead to an
increase in health care utilisation, with children with prob-
lems making a mean of 8.84 sick visits a year compared to
6.34 in children without.4
Health care professionals have insufficient training on
sleep. Various scientific associations present this consensus
as a contribution towards its improvement.

Consensus methodology

Method modified nominal group technique in which in-
person meetings were replaced using online means.
Systematic reviews of primary, secondary and tertiary
sources (backward search starting from references in the
literature). The search period ended in May 2015.
Language limits: English and Spanish.
The participating scientific associations appointed the
members of the consensus group, who signed a statement
regarding their conflicts of interest.
The group was divided in subgroups: Aetiology-definition;
Clinical Manifestations-Diagnosis; and Treatment. Each sub-
group elaborated their conclusions, which were later
distributed to the entire group for discussion and approval. A
writing team (G. Pin, V. Soto) produced the final manuscript,
which was revised and approved by the rest of the group. The
work was conducted online. Neither the consensus project
nor the authors have received any funding.
Object of the consensus: chronic insomnia in children
with normal development. The authors consider that most
cases of insomnia can be managed in primary care (PC).

Concept and classification

a. Concepts:
1. The mother-infant dyad is a "complex adaptive sys-
tem": a dynamic network of systems that acting in a
parallel yet interconnected and mutually complemen-
tary; one cannot be understood without reference to
the other.5 Maternal stress during the third trimester of
gestation facilitates placental permeability to cort-
icosteroids, which influences:
- An increase in foetal corticosteroid levels.
- A delayed development of diurnal cortisol rhythms.
- Hyperarousal with activation of the hypothalamic-
pituitary-adrenal axis in stressful situations.6
2. Sleeping through the night: It requires three
elements’: i. A period of uninterrupted sleep.
ii. Sleep schedule that coincides with that of the rest
of the family.
iii. Ability to go back to sleep independently follow-
ing nighttime awakenings.
3. Paediatric insomnia: the second edition of the Inter-
national Classification of Sleep Disorders (ICSD-2) of
the American Academy of Sleep Medicine defined it as
a "repeated difficulty with sleep initiation, dura-
tion, consolidation, or quality that occurs despite age
appropriate time and opportunity for sleep, which
results in some form of daytime functional impairment for the child and/or family.17

A sleep onset latency of more than 30 min and/or wake times lasting longer than 20 min can lead to clinically significant distress or impairment in social, familial, academic or other important areas of functioning.

b. Classification: the Diagnostic and Statistical Manual (DSM-5) of the American Psychiatric Association includes insomnia as a single category; however, the International Classification of Sleep Disorders, Third Edition (ICSD-3) includes:

1. Chronic insomnia disorder (CID) (Table 1).
2. Short-term insomnia disorder (Table 2).
3. Other insomnia disorders. Rare cases that do not meet the criteria for short-term insomnia disorder with symptoms significant enough to require medical attention.

c. Conditioning factors: various factors play a role in insomnia:

1. Circadian factor: melatonin and light. Initiation of melatonin secretion at night under dim light conditions (dim-light-melatonin-onset [DLMO]) precedes sleep onset by approximately 2 h.

Children that are forced to go to sleep at a time that is too close to or too far from their DLMO exhibit longer sleep onset latencies and greater resistance to go to bed.9

2. Homeostatic factor: late naps. The more hours of wakefulness that precede bedtime, the easier it is to fall asleep.

3. Environmental factor: light, noise and temperature. Exposure to light during the night, combined with reduced exposure in the morning, favours insomnia. The percentage decrease in melatonin secretion in the presence of light before bedtime reaches 88% in children.10

4. Educational factor: healthy lifestyle habits. Evidence on bedtime routines has shown a dose-dependent direct association between sleep habits and perceived sleep problems.11


Sleep deprivation is associated with elevated cortisol levels in the evening.12

Clinical manifestations

Dissatisfaction with sleep quantity or quality, expressed in terms of bedtime resistance, frequent awakenings and/or difficulty falling asleep without help.

The clinical impact results from sleep deprivation and includes nocturnal as well as diurnal disturbances:

a. Nocturnal: sleep onset latency of more than 20 to 30 min, wake time after sleep onset of more than 60 min, or nighttime awakenings (requiring caregiver intervention).

b. Diurnal: somnolence, attention and memory impairments, mood changes, behavioural problems and poor academic performance.13 It can impact cardiovascular, immune and metabolic systems, including obesity and growth disorders.14 It affects the quality of life of the family.

In many instances, insomnia results from inappropriate associations or inadequate limit-setting:10

- **Insomnia due to inappropriate sleep onset associations.** Results from a dependency on stimulation with specific objects or specific changes to initiate sleep or to fall back asleep after awakening. Falling asleep is associated with some form of stimulation (rocking chair, TV watching, object (bottle) or environment (lit room, parental presence in the room, or falling asleep in the parental bed). Absence of the stimulus leads to difficulties and restoration of the stimulus facilitates sleep.

- **Clinical manifestations:** frequent nighttime awakenings, nighttime fears or anxiety about sleeping alone. It is considered a disorder if: (1) the associations are problematic and very demanding; (2) sleep onset is significantly delayed or sleep is interrupted in the absence of the associated conditions, and (3) frequently requires the intervention of parents or caregivers.

- **Limit-setting insomnia.** Refusal to go to bed reinforced by inappropriate or inconsistent limit setting. It can lead to nighttime awakenings depending on the response of parents or caregivers during the night.

- **Insomnia due to poor sleep hygiene.** The child or the parents/caregivers engage in behaviours incompatible with quality sleep and normal wakefulness during the day, such as late daytime naps, irregular schedules, use of stimulant substances, arousing or disturbing mental, emotional or physical activities close to bedtime, inadequate sleep environment (noise, light stimulation, TV, computer, etc.).15

- **Psychophysiological insomnia.** Older children and adolescents may have sleep disturbances resulting from hyperarousal and/or associations with past experiences.

Clinical manifestations: excessive preoccupation with sleep, fear of sleeping or of being unable to fall asleep, fear of elements close to the bedroom, memories of past negative experiences... These individuals sleep better outside their normal environment.

Diagnosis

The diagnosis is essentially clinical, based on the information provided by the parents/caregivers and the child.14

1. **History-taking.** Must explore potential organic causes and psychomotor development, with observation of the behaviour of the child and the family. Include assessment of16,17:

   - **Onset of sleep disorder**, attempted solutions and their outcomes.
   - **24 hour sleep pattern.** Timing and amounts of sleep, place of sleep, number of awakenings, and interventions.
   - **Environmental component:** light exposure during day and night, television, electronic device screens, physical activity, noise, temperature.
ICSD-3 diagnostic criteria for chronic insomnia disorder (CID)

\[ \text{ICD-9-CM code: 307.42. ICD-10 code: F51.01} \]

Alternative names:
Chronic insomnia, primary insomnia, secondary insomnia, comorbid insomnia, disorder of initiating and maintaining sleep, behavioural insomnia of childhood, sleep-onset association disorder, limit-setting sleep disorder

Diagnostic criteria
Criteria A–F must be met

A. The patient reports, or the patient’s parent or caregiver observes, one or more of the following\(^a\):
1. Difficulty initiating sleep
2. Difficulty maintaining sleep
3. Waking up earlier than desired
4. Resistance to going to bed on appropriate schedule
5. Difficulty sleeping without parent or caregiver intervention

B. The patient reports, or the patient’s parent or caregiver observes, one or more of the following related to the nighttime sleep difficulty:
1. Fatigue/malaise
2. Attention, concentration or memory impairment
3. Impaired social, family, occupational or academic performance
4. Mood disturbance/irritability
5. Daytime sleepiness
6. Behavioural problems (e.g., hyperactivity, impulsivity, aggression)
7. Reduced motivation/energy/initiative
8. Proneness for errors/accidents
9. Concerns about or dissatisfaction with sleep

C. The reported sleep/wake complaints cannot be explained purely by inadequate opportunity (i.e. enough time is allotted for sleep) or inadequate circumstances (i.e. the environment is safe, quiet and comfortable) for sleep

D. The sleep disturbance and associated daytime symptoms occur at least three times per week

E. The sleep disturbance and associated daytime symptoms have been present for at least three months\(^b\)

F. The sleep/wake difficulty is not explained more clearly by another sleep disorder\(^c,d\)

Notes:
\(^a\) Difficulties initiating or maintaining sleep or waking up early can occur in all age groups. Resistance to going to bed at an appropriate time and difficulty falling asleep without the intervention of a parent or caregiver is most common in children and elderly individuals that require monitoring by a caregiver due to significant functional impairment (e.g., seniors suffering from dementia).

\(^b\) Some patients with chronic insomnia may experience recurrent episodes of sleep/wake problems lasting several weeks and for many years, yet not meet the criterion of three months’ duration for each isolated episode. Nevertheless, these patients should be given a diagnosis of CID, given the intermittent difficulties in sleep that persist through time.

\(^c\) Some patients that use hypnotics regularly may sleep well and not meet the criteria of insomnia disorder while they are taking them. However, in the absence of these medications these same patients may meet the diagnostic criteria. This diagnosis applies to these patients if they experience significant concern about their inability to sleep without pharmacological treatment.

\(^d\) Several comorbidities, such as chronic pain disorders or gastroesophageal reflux disorder (GERD) may cause the sleep/wake complaints presented here. When these comorbidities are the only cause of the sleep disturbance, a separate diagnosis of insomnia is not warranted. However, in many patients these are chronic conditions and are not the only source of sleep problems. Their clinical manifestations must be assessed. If there is evidence that the symptoms are not caused by the medical problem alone and they require independent treatment, the diagnosis of CID should be made.

- Educational component. Presence or absence of bedtime routines, schedules, type of parental responses.
- Impact of the sleep disorder on the child and on the family structure.

2. Full clinical evaluation. Indispensable.

3. Sleep log/diary. Graphic representation of the sleep-wake cycle over 15 days. Keeping a log of unscheduled sleep is recommended for the purpose of calculating the DLMO.\(^{18,19}\)

4. Scales and questionnaires. Various questionnaires can be used for a general assessment of sleep. None of them are validated in Spanish or for use in Spanish children and adolescents.\(^{14}\)
   - Brief Infant Sleep Questionnaire (BISQ).
   - BEARS.
   - Sleep Disturbance Scale for Children (SDSC).

5. Polysomnography and actigraphy. Diagnostic tests should be selected based on clinical findings with the purpose of confirming the clinical impression. They should never replace the anamnesis. Polysomnography is indicated when there is a strong clinical suspicion of sleep-related breathing disorder (SRBD), atypical parasomnia, periodic limb movement disorder, clinically unconfirmed restless
Table 2  ICSD-3 diagnostic criteria for short-term insomnia disorder.

<table>
<thead>
<tr>
<th>ICSD-3 diagnostic criteria for short-term insomnia disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICD-9-CM code: 307.41. ICD-10-CM code: F51.02</td>
</tr>
</tbody>
</table>

**Alternative names**
- Acute insomnia, adjustment insomnia

**Diagnostic criteria**

*Criteria A–F must be met*

A. The patient reports, or the patient’s parent or caregiver observes, one or more of the following:
- Difficulty initiating sleep
- Difficulty maintaining sleep
- Waking up earlier than desired
- Resistance to going to bed on appropriate schedule
- Difficulty sleeping without parent or caregiver intervention

B. The patient reports, or the patient’s parent or caregiver observes, one or more of the following related to the nighttime sleep difficulty:
- Fatigue/malaise
- Attention, concentration or memory impairment
- Impaired social, family, occupational or academic performance
- Mood disturbance/irritability
- Daytime sleepiness
- Behavioural problems (e.g., hyperactivity, impulsivity, aggression)
- Reduced motivation/energy/initiative
- Proneness for errors/accidents
- Concerns about or dissatisfaction with sleep

C. The reported sleep/wake complaints cannot be explained purely by inadequate opportunity (i.e. enough time is allotted for sleep) or inadequate circumstances (i.e. the environment is safe, dark, quiet and comfortable) for sleep.

D. The sleep disturbance and associated daytime symptoms have been present for less than three months

E. The sleep/wake difficulty is not explained more clearly by another sleep disorder

Notes:
- Difficulties initiating or maintaining sleep or waking up early can occur in all age groups. Resistance to going to bed at an appropriate time and difficulty falling asleep without the intervention of a parent or caregiver is most common in children and elderly individuals that require monitoring by a caregiver due to significant functional impairment (e.g., seniors suffering from dementia).
- Patients with short-term insomnia disorder may complaint of sleep/wake difficulties occurring less than three times a week on average, yet may experience clinically significant anxiety regarding their symptoms warranting clinical attention.
- Many processes such as grieving, acute pain or other stressors are often associated with sleep deprivation. When these conditions are the sole cause of difficulty sleeping, a separate diagnosis of insomnia does not apply. The main factor in the diagnosis of short-term or adjustment insomnia is the degree to which the sleep disturbance impacts the individual and/or warrants independent clinical attention.

**Differential diagnosis**

Insomnia is frequently secondary to the manifestations of other pathologies and/or circumstances:

1. Normal variations in sleep. Changes in sleep duration and pattern occur throughout the lifespan, with great interindividual variability.
2. Medical disorders:
   - Pain, pruritus.
   - Respiratory disorders.
   - Gastrointestinal disorders. Gastroesophageal reflux, constipation, chronic abdominal pain or inflammatory intestinal disease.
   - Neurodevelopmental disorders. ADHD (25–50%), cognitive disorders (10–86%), autism spectrum disorders (48–56%), cerebral palsy (45%). Insomnia is one of the diagnostic criteria for certain diseases (Rett syndrome, Williams syndrome or Smith-Magenis syndrome).
   - Epilepsy. Frontal lobe epilepsy, requires differential diagnosis including nighttime awakenings or parasomnias.
   - Iron deficiency. Ferritin levels of less than 35 to 50 ng/mL, even in the absence of anaemia, have been associated with insomnia.
   - Other medical conditions. Baby colic, moderate-severe traumatic brain injury (even months after the injury).
   - Primary sleep disorders. RLS, enuresis, SRBDs, circadian rhythm disorders (delayed sleep phase syndrome...
### Treatment

Although pharmacological treatment has been used extensively, behavioural therapy (teaching the patient strategies to promote good habits) is more widely accepted and has longer-lasting effects.

The consensus group proposes a progressive approach to insomnia disorders, as shown in the flowchart (Fig. 1).

(A) Behavioural therapy: various strategies, such as sleep hygiene, environmental and dietary measures, and psychological treatment. A combination of several or all strategies increases effectiveness.

(B) A, 1. Sleep hygiene: establishment bedtime routines, with regular times for going to bed and getting up (Table 4). Sending the child to sleep, to bed, or to the child’s room should not be used as a punishment.

(C) A, 2. Environmental and dietary measures

1. Promote physical activity, avoiding it close to bedtime.
2. Avoid screens within one hour from bedtime.
3. Temperature of 19 to 22 °C.
4. Quiet.
5. Avoid eating within half hour from bedtime, and do not consume caffeinated or energy drinks after midday. Carbohydrates and foods rich in tryptophan promote sleep.

(A), 3. Psychological treatment: intervention at the family level. It should start with a psychoeducational intervention, explaining the theoretical underpinnings of behavioural modification, and resolving questions and concerns.

The treatment plan will be individualised based on the behaviours and expectations of the family (Fig. 2). Different strategies whose efficacy is supported by high-grade scientific evidence should be contemplated. Chief among them are those aiming to reduce arousal and cognitive-behavioural interventions. Relaxation has proven to be very effective, and includes teaching breathing, progressive muscle relaxation or meditation techniques.

The available scientific evidence shows that cognitive-behavioural treatment is the most effective and widely used approach in the management of insomnia. The main strategies are:

1. Extinction. Ignoring the child’s demands from bedtime to waking time. This shortens sleep onset latency and reduces the number of wakeings. If the child goes to the parents’ bed, parents must return the child to bed without additional interaction. At first, there may be a transient increase in the problem behaviour (an “extinction burst”), as the child intensifies intrusions in an attempt to elicit past responses. If parents persist in ignoring the child’s demands, the problematic behaviour will gradually fade. The psychological process at play is the elimination of reinforcements.

2. Graduated extinction. Parents progressively ignore the child, increasing the time intervals between interactions.

3. Positive reinforcement. Positive reinforcers help initiate sleep or prolong its duration.

4. Response cost. Suppression of the positive stimulus that acts as a reinforcer for insomnia. This is achieved by delaying bedtime when the positive reinforcer of insomnia is the positive interaction with the parents at bedtime.

5. Delayed bedtime. Bedtime is temporarily delayed to match the actual timing of sleep initiation.

6. Scheduled awakenings. Pre-emptive awakenings are scheduled prior to the spontaneous awakenings of the child. The parents then provide positive reinforcers such as feeding or tucking in the child, preventing the unpleasant stimuli associated with spontaneous awakenings.

7. “Bedtime pass programme”. Effective in children aged more than 3 years that resist going to bed. The child is given a series of notecards that can be exchanged for behaviours such as getting a story, drinking water, etc., with the agreement that once the child runs out of cards, the child must go to sleep.

(D) Pharmacological treatment: it should not be the first or the only treatment modality, and must be integrated in a broader treatment plan.

Table 3 summarises the most frequently used drugs. They are all used off-label, and there is little scientific evidence on their efficacy and safety in the intermediate and long term.

Certain overall guidelines apply:

1. Pharmacological treatment must be as short as possible (<4 weeks) and administered at the minimum effective dose.
2. The drug should be selected based on the characteristics of the insomnia, the patient and the environment. Paediatricians should become acquainted with the pharmacological profile of hypnotics.
3. The timing of administration is important. Treatment is less effective if administered at the circadian peak in alertness that typically occurs in late afternoon.
## Table 3  Drugs used to treat insomnia.

<table>
<thead>
<tr>
<th>Pharmaceutical</th>
<th>Class</th>
<th>Mechanism of action</th>
<th>Half life (h)</th>
<th>Site of metabolism</th>
<th>Peak concentration (min)</th>
<th>Interactions</th>
<th>Effect on sleep</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diphenhydramine</td>
<td>Antihistaminic</td>
<td>H1 agonist crosses blood–brain barrier</td>
<td>4–6 h</td>
<td>Hepatic</td>
<td>Fast absorption</td>
<td>CNS depressants</td>
<td>Reduces latency</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Fast onset of action</td>
<td></td>
<td>May decrease quality</td>
</tr>
<tr>
<td>Hydroxyzine</td>
<td>Antihistaminic</td>
<td>H1 agonist crosses blood–brain barrier</td>
<td>6–24 h</td>
<td>Hepatic</td>
<td>Fast absorption</td>
<td>CNS depressants</td>
<td>Reduces latency</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Fast onset of action</td>
<td></td>
<td>May decrease quality</td>
</tr>
<tr>
<td>Melatonin</td>
<td>Neurohormone</td>
<td>Hypnotic</td>
<td>90% excreted in 4 h</td>
<td>Hepatic</td>
<td>Peak at 2–4 h</td>
<td>Unknown</td>
<td>Reduces latency</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>30–60 min</td>
<td></td>
<td>Maximum circadian effect</td>
</tr>
<tr>
<td>Clonazepam</td>
<td>Benzodiazepine</td>
<td>Central GABA receptors</td>
<td>30–40 h</td>
<td>CYP 450 3A oxidation</td>
<td>60–240</td>
<td>Fluoxetine</td>
<td>Suppresses slow-wave sleep</td>
</tr>
<tr>
<td>Flurazepam</td>
<td>Benzodiazepine</td>
<td>Central GABA receptors</td>
<td>2–100</td>
<td>CYP 450 3A oxidation</td>
<td>30 min to 13 h</td>
<td>Fluoxetine</td>
<td>Suppresses slow-wave sleep</td>
</tr>
<tr>
<td>Zolpidem</td>
<td>Z-drug</td>
<td>Benzodiazepine-like</td>
<td>2.5–3 h</td>
<td>CYP 450 3A oxidation</td>
<td>90 min</td>
<td></td>
<td>Reduces latency</td>
</tr>
<tr>
<td>Clonidine</td>
<td>Alpha agonist</td>
<td>Inhibits noradrenaline release</td>
<td>6–24 h</td>
<td>50–80% in urine</td>
<td>Fast absorption 100% bioavailability</td>
<td>Reduce REM</td>
<td>Weak effect on sleep architecture</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Onset of action: 1 h</td>
<td></td>
<td>Reduce REM</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Peak effect: 2–4 h</td>
<td></td>
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</tr>
</tbody>
</table>
Child/adolescent complaining of perceived insomnia

Clinical interview

Insomnia confirmed

Sleep diary
Medical evaluation
Pharmacological history
Psychiatric evaluation
Diagnostic tests

Comorbid insomnia
Non-comorbid insomnia

Etiotropic treatment +

Treatment

SLEEP HYGIENE + 3-STEP CONCEPT(1)

2 weeks (sleep diary ± actigraphy)

Favourable response
2 weeks
Discharge

Persistence of insomnia

Reevaluation

Family tolerance questionnaire (**) 

Sleep hygiene + Individualised cognitive behavioural therapy (ICBT)
4–6 weeks (sleep diary ± actigraphy ± salivary melatonin)

Favourable response
Persistence/worsening

ICBT + Sleep hygiene + Pharmacological Tx
Sleep diary ± actigraphy (4–6 weeks)

2–4 weeks
(sleep diary ± actigraphy ± salivary melatonin)

Favourable response
Persistence/worsening

2–4 weeks: sleep diary ± actigraphy

DISCHARGE 

Referral: SU/PN/MH(***)

(*) Zurich group 3-step concept:
First step: introducing a regular sleep-wake rhythm
Second step: adjusting the child’s bedtimes to individual sleep need
Third step: assisting the child to fall asleep on his/her own

(**) Family tolerance questionnaire: See Figure 3

(***): Based on availability. SU: Sleep Unit. PN: Paediatric Neurology. MH: Mental Health

Figure 1 Diagnosis and treatment flowchart.
Disruption tolerance (1) & Mother & Father \\
1. The behaviour is too serious or upsetting to ignore & Yes & No & Yes & No \\
2. It is difficult for you to listen to your child scream/cry for long periods of time & Yes & No & Yes & No \\
3. It is difficult for you to put your child back once he/she gets out of bed & Yes & No & Yes & No \\

Schedule tolerance (2) & Mother & Father \\
4. Is anyone in the family willing to stay up later at night to put a sleep plan into action? & Yes & No & Yes & No \\
5. Is anyone in the family willing to get up earlier in the morning to put a sleep plan into action? & Yes & No & Yes & No \\

Attitudinal barriers (3) & Mother & Father \\
6. Do you feel emotionally unable to deal directly with the problem? & Yes & No & Yes & No \\
7. Do you feel guilty making your child go back to bed? & Yes & No & Yes & No \\
8. Do you think it would be emotionally damaging to your child if you tried to change the situation? & Yes & No & Yes & No \\

1. If one of the answers is "yes", extinction is contraindicated. 
2. If one of the answers is "no", consider gradual extinction in comparison to other interventions 
3. If one of the answers is "yes": prior intervention with the family required 

Figure 2 Family tolerance questionnaire. Source: del Rosso and Hoque.25

4. Close monitoring of side effects, especially during withdrawal.
5. It may exacerbate comorbid sleep disorders (SRBD).
6. Beware of potential interactions with other drugs. 
   Especially care should be taken in adolescents (possibility of pregnancy and substance use).
7. Evaluate the use of over-the-counter medication by the family.

The consensus group considers that, in addition to iron supplementation, two other drug groups are indicated for management at the PC level. If they are not effective, the patient should be referred to a specialised unit:

(A) First-line drug: melatonin.35

Advantages: reduces sleep onset latency. 
Disadvantages: few studies support its safety in the intermediate and long term when it is used for prolonged periods of time. 
Dosage: the initial dose should be 1 to 3 mg in infancy and early childhood, and 2.5 to 5 mg in older children, gradually adjusting the dose based on patient response. 
Adolescents: 1 to 5 mg. 
It should always be administered at the same time, between 30 and 60 min before the usual bedtime. 
There are no data on the use of extended-release melatonin in children with normal development. 
Duration: it should not exceed four weeks of continued treatment. 
In sleep-onset insomnia treated with melatonin alone, treatment discontinuation leads to recurrence of the original problem in 90% of cases. 
(B) Second-line drugs (after 4 weeks of treatment with melatonin with unsatisfactory results). 
First-generation antihistamines (diphenhydramine, hydroxyzine).40 
Diphenhydramine: rapidly absorbed, does not produce gastric irritation. 
Advantages: paediatricians are familiar with its use. Especially useful in acute situations. 
Disadvantages: some studies support its efficacy, while others failed to find a difference between treatment and placebo. 
There is a risk of paradoxical reaction. 
Dosage: 1 mg/kg. 
Hydroxyzine: dosage: 12 months to 6 years, 1 to 2.5 mg/kg at night. Age more than 6 years, 1 to 2 mg/kg at night. 
Duration: 4 weeks. 
The rest of the drugs listed in Table 3 should be used following a thorough evaluation by a specialised unit. 
(C) Phytotherapy. Table 4 summarises the main properties of the most frequently used treatments. 
Its use is based on tradition, with scarce data from evidence-based medicine.

<table>
<thead>
<tr>
<th>Name</th>
<th>Mechanism of action</th>
<th>Dosage</th>
<th>Effect on sleep</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valerian</td>
<td>Binds benzodiazepine receptors</td>
<td>2–3 g × 3/day</td>
<td>Shortens sleep latency</td>
<td>Rare toxicity</td>
</tr>
<tr>
<td>Chamomile</td>
<td>Binds benzodiazepine receptors</td>
<td>1–3 g × 3/day</td>
<td>Increases deep sleep (?)</td>
<td>Hypertensive effect</td>
</tr>
<tr>
<td>Kava</td>
<td>CNS depression</td>
<td>60–120 mg/day</td>
<td>Improves sleep efficiency</td>
<td>Weak anxiolytic Liver toxicity</td>
</tr>
<tr>
<td>Lavender</td>
<td>CNS depression</td>
<td>Inhaling essential oil</td>
<td>Improves sleep efficiency</td>
<td>Potentiates effects of alcohol</td>
</tr>
</tbody>
</table>
few studies have found evidence of the usefulness of specific herbal treatments in the paediatric population, and these remedies have generally not been tested. Parents must be made aware of the lack of studies demonstrating the efficacy and safety of these substances in children and adolescents, as well as the lack of knowledge regarding their dosage, susceptibility and potential contaminants.

Conclusion

This consensus group recommends a holistic approach to chronic insomnia in children and adolescents, with evaluation of predisposing and aetiological factors.

Treatment should always be individualised and start with sleep hygiene and cognitive-behavioural measures. Pharmacological treatment should be as short as possible and be administered at the minimum effective dose.

Conflicts of interest

The authors have no conflict of interests to declare.

References