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Provide empiric tools to help patients
explore the validity of their thoughts
and the impact of their behaviors

DEMYSTIFYING CBT

Effective, easy-to-use
treatment for depression
and anxiety

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Whether you are in training or an experienced practitioner, you need more than a rudimentary understanding of cognitive-behavioral therapy (CBT). This easy-to-use psychotherapy has broad empiric support, is a first-choice treatment,¹ and can help patients cope with depression, anxiety, and other psychological problems.

Psychiatrists who learn CBT well can alleviate many patients' distress by creatively applying its tools and techniques. For example, the cognitive approach to panic disorder compares favorably to medication¹⁻³ (*Box, page 28*).

AARON BECK'S CBT

Negative thoughts, biased processing. The greater your fidelity to CBT's guiding principles (*Table 1, page 31*), the more effective the therapy becomes.⁴ Beck designed CBT to address his observation that depressed persons hold unrealistically negative views about themselves, the world, and the future.^{5,6} A distorted information-processing system prevents them from correcting underlying negative beliefs. Negative thoughts predominate



Box

Treating anxiety disorders with CBT: A first-line therapy

Using CBT to experientially disconfirm catastrophic cognitions is the psychotherapy of choice for anxiety disorders.¹ Cognitive models exist for each anxiety disorder² and include psychoeducation, self-monitoring, evaluation of anxious cognitions, and testing cognitions interoceptively (within the body) and in vivo (within the environment). The therapist strategically uses adjunctive measures such as relaxation training, controlled breathing, visualization, and distraction.

Panic disorder is characterized by catastrophic misinterpretations of benign bodily sensations

that accompany a fear response.^{2,3} Disability occurs when patients avoid situations or activities they believe will activate bodily sensations such as dizziness or breathlessness. Using environmental manipulations—spinning, hyperventilation, or straw-breathing, to name a few—CBT aims to disconfirm patients' catastrophic thoughts by deliberately exposing them to feared somatic sensations.²

When used to treat panic disorder, CBT is associated with remission rates similar to those achieved by medication and much lower relapse rates.¹

their cognitions and seem to arise spontaneously, reflexively, and unremittingly. These “automatic thoughts,” as he called them, reflect underlying themes about the self that can be identified as:

- intermediate beliefs (conditional assumptions, attitudes, and rules)
- core beliefs (fundamental, often global, and absolute rules).⁷

Activating schema content. Cognitive content and biased processing are elements of the individual's schema, an integrated knowledge structure that influences what he/she remembers and how he/she processes and stores new experiences. Negative schema content remains latent during periods of normal mood, according to Beck, but can be activated by:

- external (environmental) stress that carries symbolic value
- internal (physiologic) stress that activates the affective valence of the underlying schema.^{1,5,6}

For example, a man becomes despondent when a girlfriend cancels a date (external stress) because this activates his pre-existing beliefs of

worthlessness and memories of childhood abandonment. Premenstrual dysphoria caused by hormonal changes—an internal stress—can trigger negative beliefs associated with that mood state.

CBT's scientific method. CBT teaches a person the skills to identify this cognitive material and to recognize biases that affect how he or she processes information. You can help patients understand:

- the bidirectional relationship between thoughts, feelings, and behaviors
- that they can influence their emotions by changing their thoughts and behavior.

Using behavioral experiments, you collaboratively teach patients to examine their thoughts as “hypotheses to be tested” rather than self-evident “truths.”⁶ You encourage them to think like scientists who are observing and evaluating their idiosyncratic internal experience.

You provide empiric tools to help them explore the validity or usefulness of their thoughts and the impact of their behaviors. When patients disprove a negative cognition through this process of “experiential disconfirmation,” you help them to change

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that cognition. Homework is a key ingredient (*Table 2, page 32*); patients who do their CBT homework are more likely to improve than those who don't.⁸

EVALUATING COGNITIVE DISTORTIONS

Thought records. Automatic thoughts are the cognitive content that runs through our minds moment to moment and that we can access by asking ourselves, “What was going through my mind when I felt (emotion)?” Automatic thoughts can exist as:

- verbal messages (“I can’t believe this!” or “I’m such a loser”)
- or images (“I picture my boss screaming at me”).

Teach patients to monitor their experiences by writing down their thoughts and feelings as well as the corresponding situations when they feel strong negative emotions. Compared with psychodynamic therapies—which emphasize retrospective reconstruction of childhood experience—self-monitoring with a “thought record” focuses on the present, is more accessible, and is less prone to recall bias.

Self-monitoring also facilitates “decentering,” or viewing one’s emotional experience from a distance, which may be crucial to therapeutic success.⁹ By using Socratic questioning (*Table 3, page 33*) and guided discovery, you teach patients to evaluate their automatic thoughts as hypotheses to be tested.

If patients discover that their automatic thoughts are inaccurate, you can help them construct more-balanced or alternate appraisals. Conversely, hypothesis testing may help generate new solutions if the process validates the patient’s initial interpretation of a situation.¹⁰

Labels for distorted thoughts. Help patients identify and label their cognitive distortions. These distortions are systematic biases in information processing that reinforce negativistic thinking in

Table 1

Guiding principles of cognitive-behavioral therapy

- **Therapist uses** the cognitive model to create meaning
- **Sessions are structured** and goal-oriented
- **Therapist works actively**, using a problem-solving approach, to help the patient change and develop
- **Patient acquires** new skills through homework, practicing, and experiencing
- **Sessions are time-limited** and focus on collaborative empiricism
- **Patient learns skills** for self-change and becomes empowered

depression or catastrophic thinking in anxiety. Examples include:

- overgeneralization (“Nothing ever works out for me”)
- all-or-nothing thinking (“I failed again” [after getting 95% on an exam])
- mind-reading (“My boss thinks I’m incompetent”)
- catastrophization (“My heart is racing; I think I’m having a heart attack!”).¹¹

Intermediate beliefs may emerge as automatic thoughts or be identified in thought records as consistent themes. These underlying beliefs represent idiosyncratic vulnerabilities that make a person susceptible to distress or decompensation in a given stressful situation. They take the form of:

- attitudes (“Weakness is contemptible”)
- rules (“I will not let others take advantage of me”)
- conditional assumptions (“If I let others take advantage of me, I’m a thoroughly weak person”).

continued



Table 2

Structure of a typical CBT session

Step	What therapist may say or do to introduce this step
Collaborate in setting the agenda	'What would you like to put on the agenda for today's session? If we could address one or two items, what would they be?'
Link to previous session via feedback	Review and comment on the patient's feedback form
Check target symptoms	'How would you rate your level of (depression, anxiety, etc.) this week on a scale of 0 to 100?' Therapist also can review standardized rating scales, such as Beck Depression Inventory II
Check medication	'Are there any concerns this week about your medication?'
Review week/scheduling	'Could you update me about your week?' 'What would be important to focus on this coming week?'
Review homework	'Let's have a look at the homework/self-help work you did this week'
Set new agenda items	'This issue sounds important; would you like to add it to today's agenda?'
Collaborate in developing new homework	'I'd like to work on this further next week; let's decide together what would be doable'
Feedback	'How did you feel about today's session?' 'Is there anything you would like to be sure to remember after you leave today?' 'Anything you want to put on the agenda for next session?'

A man with the above intermediate beliefs who acquiesces to a friend's request for a loan might perceive that the friend has taken advantage of him. Emotionally, he may react with sadness, despair, or anger. You and he can evaluate these beliefs through a thought record and view them as hypotheses to be tested with behavioral experiments.

Framing the intermediate belief as a conditional assumption can help accomplish this goal. Presumably, the patient was distressed about loaning money to his friend. When pressed, he says he didn't want to lend the money but felt he couldn't say no. He identifies his automatic thought as "I've been taken advantage of." Asked what this means if it is true, he replies, "If I get taken advantage of, it means I'm weak."

Core beliefs are deeper cognitive structures that are not always immediately accessible, although they may occasionally emerge spontaneously as automatic thoughts. They are overgeneralized, absolute statements that fall into one of two categories:

- affiliation ("I am bad," "I am unlovable")
- competence/vulnerability ("I am weak," "I am helpless").

Core beliefs can be identified by using the downward arrow technique (Table 4, page 34). After an automatic thought is identified, repeatedly ask the patient, "If that were true, what would that say about you/others/the world?" or, "What would be the worst thing about that if it were true?"

Very often, intermediate and core beliefs must be defined in a measurable

way before you can help the patient test them. For the man feeling distressed about loaning money, for example, you might ask him to define “being taken advantage of” or define “weak” by listing all the characteristics he associates with this label.

RESTRUCTURING NEGATIVE BELIEFS

A variety of techniques can be used to restructure negative beliefs.

- **Cost-benefit** analysis involves exploring advantages and disadvantages of maintaining a negative belief or a more-balanced alternate belief.
- **Core belief logs**¹² can track day-to-day evidence that suggests a core belief is not 100% true. You and the patient can scrutinize evidence that supports the core belief and reframe the evidence in a more-rational manner.
- **Life review**^{10,12} involves asking the patient to re-evaluate a core belief’s historical underpinnings and to reframe these events from an adult perspective.

Automatic thoughts might be restructured quickly, but core beliefs may take months to begin to change. Techniques focused on rational reappraisal are usually not sufficient by themselves. Supplemental approaches that focus on activating and amplifying emotion can be integral to this process. Examples include:

- rational-emotional role play
- empty chair or two-chair dialogue
- restructuring early memories with directed imagery.

ADJUNCTIVE BEHAVIORAL STRATEGIES

Behavioral interventions are used in CBT to combat anergia, increase socialization, diminish avoidance, and accumulate data to challenge negative beliefs. Common strategies are designed to enhance self-esteem and confidence and build therapeutic momentum as patients gain energy, feel better, and disconfirm negative beliefs.

Activity monitoring and scheduling. Instruct anergic

Table 3

Examples of Socratic and non-Socratic questioning

Socratic questioning

- What evidence do you have to support this idea?
- How strongly do you believe this now?
- On a scale of 0 to 100%, where does your belief fall? Where do other people’s fall?
- How does this thought affect how you feel and act?
- Can you describe experiences when this thought was not completely true?
- If a close friend thought this way, what would you tell him or her?
- If you told a close friend about this thought, what would he or she say?
- When you have felt differently in the past, what would you have said about this thought?
- Are any distortions present in the thought you identified?

Non-Socratic questioning

- Why are you being so hard on yourself?
- You say you are a total loser. Would a total loser have accomplished all the things you did this week?
- I’m sure that others don’t see you this way.

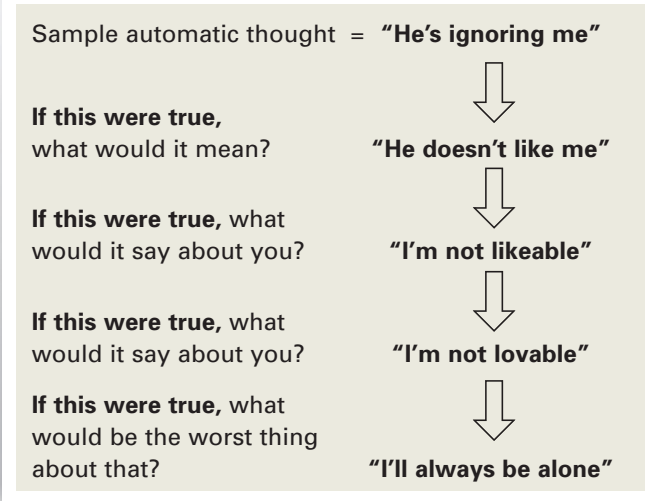
or avoidant patients to monitor daily activities for the week and to rate the degree of pleasure and accomplishment each activity yields on a scale of 1 to 10. As patients become aware of how much time they spend on low-yield activities, help them gradually replace low-yield with higher-yield activities.

Schedule into the week behavioral goals



Table 4

Identifying core beliefs: The downward arrow technique



patients identified at the beginning of therapy. Schedule avoided tasks such as household chores, and link them to pleasurable activities as rewards. To evaluate the accuracy of their thinking, ask patients to predict how much pleasure or mastery they will achieve with scheduled activities, then compare their predictions with actual results.

Also ask patients to anticipate obstacles to achieving their goals, to challenge those obstacles, and to develop contingency plans. Reviewing the patient's week and its bright spots can disconfirm negatively biased recall such as, "My week was

terrible," or, "I don't have the energy to do anything anymore."¹³

Graded task assignments. When scheduling activities, improve success rates by helping patients break down large, unrealistic goals into smaller, more manageable pieces. Ask them to consider realistically what they can accomplish now, not what they could have accomplished before they became ill.

Exposure. Anxious patients avoid feared situations because of catastrophic beliefs that experiencing those situations will harm them. A man with panic disorder may avoid exercise, for example, because he perceives lightheadedness and rapid heart rate as signs of imminent heart attack. Avoiding exercise to prevent the feared symptoms perpetuates his catastrophic beliefs.

Exposure to feared symptoms—while initially arousing high anxiety—allows the patient to experientially disconfirm his beliefs. As he remains well after lightheadedness and rapid heart rate are induced interoceptively (by climbing several flights of stairs, for example), he comes to recognize the situational symptoms as manifestations of anxiety rather than evidence of life-threatening illness.^{2,3}

In vivo exposure entails confronting the patient with the avoided object or situation. For example, you may show a woman with needle phobia pictures of needles, followed by actual needles themselves, then ask her to touch a needle, hold a needle, etc., until her anxiety gradually diminishes.

Imaginal exposure involves asking the patient to imagine himself in a feared situation and manipulating the images to build his sense of mastery. If he stops the image at the moment of highest arousal, instruct him to "continue to play the film forward" by asking, "What happens next?" This approach shows him that he can cope with difficult situations.

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Cognitive-behavioral therapy is easy to use and has broad empirical backing. It can help patients with depression, anxiety, and other psychological conditions. With practice, you can learn its structure and creatively apply its techniques to alleviate patient distress.

BottomLine

Dose Dependency of Adverse Events in Short-Term, Placebo-Controlled Trials—Extrapyramidal Symptoms—In an acute-phase controlled clinical trial in schizophrenia, there was no significant difference in ratings scales incidence between any dose of oral olanzapine (5+2.5, 10+2.5, or 15+2.5 mg/d) and placebo for parkinsonism (Simpson-Angus Scale total score >3) or akathisia (Barnes Akathisia global score ≥2). In the same trial, only akathisia events (spontaneously reported COSTART terms akathisia and hyperkinesia) showed a statistically significantly greater adverse events incidence with the 2 higher doses of olanzapine than with placebo. The incidence of patients reporting any extrapyramidal event was significantly greater than placebo only with the highest dose of oral olanzapine (15+2.5 mg/d). In controlled clinical trials of intramuscular olanzapine for injection, there were no statistically significant differences from placebo in occurrence of any treatment-emergent extrapyramidal symptoms, assessed by either rating scales incidence or spontaneously reported adverse events.

Other Adverse Events—Dose-relatedness of adverse events was assessed using data from a clinical trial involving 3 fixed oral dosage ranges compared with placebo. The following treatment-emergent events showed a statistically significant trend: asthenia, dry mouth, nausea, somnolence, tremor.

Vital Sign Changes—Oral olanzapine was associated with orthostatic hypotension and tachycardia in clinical trials. Intramuscular olanzapine for injection was associated with bradycardia, hypotension, and tachycardia in clinical trials (see PRECAUTIONS).

Weight Gain—In placebo-controlled 6-week schizophrenia studies, weight gain was reported in 5.6% of oral olanzapine patients (average 2.8-kg gain) compared to 0.8% of placebo patients (average 0.4-kg loss); 29% of olanzapine patients gained >7% of their baseline weight, compared to 3% of placebo patients. During continuation therapy (238 median days of exposure), 56% of patients met the criterion for having gained >7% of their baseline weight. Average gain during long-term therapy was 5.4 kg.

Laboratory Changes—Olanzapine is associated with asymptomatic increases in SGPT, SGOT, and GGT and with increases in serum prolactin and CPK (see PRECAUTIONS). Asymptomatic elevation of eosinophils was reported in 0.3% of olanzapine patients in premarketing trials. There was no indication of a risk of clinically significant neutropenia associated with olanzapine in the premarketing database.

In clinical trials among olanzapine-treated patients with baseline random triglyceride levels of <150 mg/dL (N=659), 0.5% experienced triglyceride levels of ≥500 mg/dL anytime during the trials. In these same trials, olanzapine-treated patients (N=1185) had a mean triglyceride increase of 20 mg/dL from a mean baseline of 175 mg/dL. In placebo-controlled trials, olanzapine-treated patients with baseline random cholesterol levels of <200 mg/dL (N=1034) experienced cholesterol levels of ≥240 mg/dL anytime during the trials more often than placebo-treated patients (N=602; 3.6% vs 2.2% respectively). In these same trials, olanzapine-treated patients (N=2528) had a mean increase of 0.4 mg/dL in cholesterol from a mean baseline of 203 mg/dL, which was significantly different compared to placebo-treated patients (N=1415) with a mean decrease of 4.6 mg/dL from a mean baseline of 203 mg/dL.

ECG Changes—Analyses of pooled placebo-controlled trials revealed no statistically significant olanzapine/placebo differences in incidence of potentially important changes in ECG parameters, including QT, QTc, and PR intervals. Olanzapine was associated with a mean increase in heart rate of 2.4 BPM compared to no change among placebo patients.

Other Adverse Events Observed During Clinical Trials—The following treatment-emergent events were reported with oral olanzapine at multiple doses ≥1 mg/d in clinical trials (8661 patients, 4165 patient-years of exposure). This list may not include events previously listed elsewhere in labeling, those events for which a drug cause was remote, those terms which were so general as to be uninformative, and those events reported only once or twice which did not have a substantial probability of being acutely life-threatening. **Frequent** events occurred in ≥1/100 patients; **infrequent** events occurred in 1/100 to 1/1000 patients; **rare** events occurred in <1/1000 patients. **Body as a Whole**—

Frequent: dental pain, flu syndrome; **Infrequent:** abdomen enlarged, chills, face edema, intentional injury, malaise, moniliasis, neck pain, neck rigidity, pelvic pain, photosensitivity reaction, suicide attempt; **Rare:** chills and fever, hangover effect, sudden death. **Cardiovascular**—

Frequent: hypotension; **Infrequent:** atrial fibrillation, bradycardia, cerebrovascular accident, congestive heart failure, heart arrest, hemorrhage, migraine, pallor, palpitation, vasodilatation, ventricular extrasystoles; **Rare:** arteritis, heart failure, pulmonary embolus. **Digestive**—

Frequent: flatulence, increased salivation, thirst; **Infrequent:** dysphagia, esophagitis, fecal impaction, fecal incontinence, gastritis, gastroenteritis, gingivitis, hepatitis, melena, mouth ulceration, nausea and vomiting, oral moniliasis, periodontal abscess, rectal hemorrhage, stomatitis, tongue edema, tooth caries; **Rare:** aphthous stomatitis, enteritis, eructation, esophageal ulcer, glossitis, ileus, intestinal obstruction, liver fatty deposit, tongue discoloration. **Endocrine**—

Infrequent: diabetes mellitus; **Rare:** diabetic acidosis, goiter. **Hemic and Lymphatic**—

Infrequent: anemia, cyanosis, leukocytosis, leukopenia, lymphadenopathy, thrombocytopenia; **Rare:** normocytic anemia, thrombocytopenia. **Metabolic and Nutritional**—

Infrequent: acidosis, alkaline phosphatase increased, bilirubinemia, dehydration, hypercholesterolemia, hyperglycemia, hyperlipemia, hyperuricemia, hypoglycemia, hypokalemia, hyponatremia, lower extremity edema, upper extremity edema; **Rare:** gout, hyperkalemia, hypernatremia, hypoproteinemia, ketosis, water intoxication. **Musculoskeletal**—

Frequent: joint stiffness, twitching; **Infrequent:** arthritis, arthrosis, leg cramps, myasthenia; **Rare:** bone pain, bursitis, myopathy, osteoporosis, rheumatoid arthritis. **Nervous System**—

Frequent: abnormal dreams, amnesia, delusions, emotional lability, euphoria, manic reaction, paresthesia, schizophrenic reaction; **Infrequent:** akinesia, alcohol misuse, antisocial reaction, ataxia, CNS stimulation, cogwheel rigidity, delirium, dementia, depersonalization, dysarthria, facial paralysis, hypesthesia, hypokinesia, hypotonia, incoordination, libido decreased, libido increased, obsessive compulsive symptoms, phobias, somatization, stimulant misuse, stupor, stuttering, tardive dyskinesia, vertigo, withdrawal syndrome; **Rare:** circumoral paresthesia, coma, encephalopathy, neuralgia, neuropathy, nystagmus, paralysis, subarachnoid hemorrhage, tobacco misuse. **Respiratory**—

Frequent: dyspnea; **Infrequent:** apnea, asthma, epistaxis, hemoptysis, hyperventilation, hypoxia, laryngitis, voice alteration; **Rare:** atelectasis, hiccup, hypoventilation, lung edema, stridor. **Skin and Appendages**—

Frequent: sweating; **Infrequent:** alopecia, contact dermatitis, dry skin, eczema, maculopapular rash, pruritus, seborrhea, skin discoloration, skin ulcer, urticaria, vesiculobullous rash; **Rare:** hirsutism, pustular rash. **Special Senses**—

Frequent: conjunctivitis; **Infrequent:** abnormality of accommodation, blepharitis, cataract, deafness, diplopia, dry eyes, ear pain, eye hemorrhage, eye inflammation, eye pain, ocular muscle abnormality, taste perversion, tinnitus; **Rare:** corneal lesion, glaucoma, keratoconjunctivitis, macular hypopigmentation, miosis, mydriasis, pigment deposits lens. **Urogenital**—

Frequent: vaginitis; **Infrequent:** abnormal ejaculation, amenorrhea, breast pain, cystitis, decreased menstruation, dysuria, female lactation, glycosuria, gynecostasia, hematuria, impotence, increased menstruation, menorrhagia, metrorrhagia, polyuria, premenstrual syndrome, pyuria, urinary frequency, urinary retention, urinary urgency, urination impaired, uterine fibroids enlarged, vaginal hemorrhage; **Rare:** albuminuria, breast enlargement, mastitis, oliguria. (*Adjusted for gender.)

The following treatment-emergent events were reported with intramuscular olanzapine for injection at one or more doses ≥2.5 mg/injection in clinical trials (722 patients). This list may not include events previously listed elsewhere in labeling, those events for which a drug cause was remote, those terms which were so general as to be uninformative, and those events reported only once or twice which did not have a substantial probability of being acutely life-threatening. **Body as a Whole**—

Frequent: injection site pain; **Infrequent:** abdominal pain, fever. **Cardiovascular**—

Infrequent: AV block, heart block, syncope. **Digestive**—

Infrequent: diarrhea, nausea. **Hemic and Lymphatic**—

Infrequent: anemia. **Metabolic and Nutritional**—

Infrequent: creatine phosphokinase increased, dehydration, hyperkalemia. **Musculoskeletal**—

Infrequent: twitching. **Nervous System**—

Infrequent: abnormal gait, akathisia, articulation impairment, confusion, emotional lability. **Skin and Appendages**—

Infrequent: sweating. **Postintroduction Reports**—Reported since market introduction and temporally (not necessarily causally) related to olanzapine therapy: allergic reaction (eg, anaphylactoid reaction, angioedema, pruritus or urticaria), diabetic coma, jaundice, pancreatitis, priapism, rhabdomyolysis, and venous thromboembolic events (including pulmonary embolism and deep venous thrombosis). Random cholesterol levels of ≥240 mg/dL and random triglyceride levels of ≥1000 mg/dL have been rarely reported.

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Related resources

For clinicians

- ▶ Persons JB. *Cognitive therapy in practice: a case formulation approach*. New York: WW Norton and Co.; 1989.
- ▶ Academy of Cognitive Therapy. Training, certification as a cognitive therapist. www.academyofct.org.
- ▶ Behavior Online. Gathering site for mental health professionals. www.behavior.net.
- ▶ MySelfHelp.com. Interactive programs and moderated discussion designed as treatment adjuncts for patients with depression, stress, eating disorders, etc. Funded by the National Institute of Mental Health (\$20/month program access fee). www.MySelfHelp.com.

For patients

- ▶ Antony M, Swinson R. *When perfect isn't good enough*. Oakland, CA: New Harbinger Press; 1998.
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