**DESVENLAFAXINE: FREQUENTLY ASKED QUESTIONS**

**DISCUSSANT**
Lawrence J. Cohen, PharmD, BCPP, FASHP, FCCP

**INTERVIEWER**
Diane M. Sloan, PharmD

---

**FACULTY AFFILIATION AND DISCLOSURES**

Dr. Cohen is professor of pharmacotherapy at Washington State University College of Pharmacy, and assistant director for psychopharmacology research and training at the Washington Institute for Mental Illness Research and Training in Spokane.

**Disclosure:** Dr. Cohen is a consultant to Janssen and Wyeth; is on the speakers bureau of AstraZeneca, Eli Lilly and Forest; and serves on the advisory board of AstraZeneca and Eli Lilly.

---

**INTRODUCTION**

Desvenlafaxine is a newly available serotonin-norepinephrine reuptake inhibitor (SNRI) antidepressant that has been shown to be an effective and safe treatment for major depressive disorder (MDD) in adults. Desvenlafaxine (administered as desvenlafaxine succinate) is the succinate salt of O-desmethylvenlafaxine, which is the major pharmacologically active metabolite of venlafaxine. The metabolic profiles of these antidepressants are distinct: venlafaxine is extensively metabolized by cytochrome P450 (CYP) 2D6 and has several metabolites, whereas desvenlafaxine does not interact with CYP2D6 and is minimally transformed through oxidative metabolism (ie, CYP). Since February 2008 when desvenlafaxine first came to market, clinicians have wondered about the differences between this new antidepressant and venlafaxine extended release (ER).
What is the difference between desvenlafaxine and venlafaxine ER?

Desvenlafaxine is an orally administered ER tablet that contains desvenlafaxine (O-desmethylvenlafaxine), which is the major pharmacologically active metabolite of venlafaxine ER (venlafaxine HCl). Desvenlafaxine is recognized by the United States Food and Drug Administration as a new chemical entity distinct from venlafaxine. Venlafaxine is an SNRI with antidepressant activity that is extensively metabolized by the hepatic microsomal CYP enzyme system to multiple metabolites (Figure 1A), with only 4.7% of an oral dose recovered in the urine as unchanged drug. Venlafaxine is oxidized by CYP2D6 to O-desmethylvenlafaxine; both are pharmacologically active. Over 55% of a single dose of venlafaxine is eliminated in the urine as desvenlafaxine and its glucuronide conjugate. Other nonpharmacologically active metabolites of venlafaxine, N,O-didesmethyl-venlafaxine, N-desmethyl-venlafaxine, and N,N,O-tridesmethyl-venlafaxine, are formed through metabolism by CYP3A4, CYP2C19, and CYP1A2.

In contrast, desvenlafaxine is eliminated primarily as unchanged drug or as the glucuronide conjugate in the urine. Desvenlafaxine delivers the pharmacologically active compound O-desmethylvenlafaxine in its active state directly to the bloodstream. Desvenlafaxine undergoes minimal metabolism to an inactive glucuronide conjugate (19%) and to a much lesser extent (<5%) via CYP3A4 to N,O-didesmethyl-venlafaxine (Figure 1B). Desvenlafaxine is not metabolized by CYP2D6 enzymes.

FIGURE 1A
Metabolism of Venlafaxine to Desvenlafaxine (O-desmethylvenlafaxine) and Minor Metabolites


CYP=cytochrome P450.

In addition, data from a study in Caco-2 monolayers showed that desvenlafaxine is not a substrate of P-glycoprotein; therefore, the pharmacokinetic properties of desvenlafaxine are unlikely to be affected by drugs that inhibit the P-glycoprotein transporter, which mediates transcellular transport of numerous drugs. Similarly, desvenlafaxine minimally inhibited P-glycoprotein in vitro, and therefore would not be expected to alter the pharmacokinetic properties of drugs that are substrates of the P-glycoprotein transporter. Desvenlafaxine is minimally bound to plasma proteins (30%), which should minimize the risk of drug-drug interactions with highly protein-bound drugs. Because desvenlafaxine is less extensively metabolized, its absolute oral bioavailability is ~80%. This is in contrast to oral bioavailability for venlafaxine ER of 45% (with desvenlafaxine, formed via first-pass metabolism, accounting for the remainder of pharmacologic activity).

What is the effect of desvenlafaxine on serotonin and norepinephrine? How does this compare to venlafaxine ER and duloxetine?

Serotonin (5-HT) and norepinephrine (NE) are monoamine neurotransmitters involved in the regulation of mood. The mechanism of action of monoamine reuptake inhibitor antidepressants consists of inhibiting the reuptake of 5-HT and NE by presynaptic transporter proteins, thereby increasing the concentration of these monoamines in the synaptic cleft. Desvenlafaxine is an SNRI that is a more selective inhibitor of 5-HT reuptake than NE reuptake. Desvenlafaxine does not have significant affinity for cholinergic, histaminergic, dopaminergic, or α-adrenergic receptors. Membrane radioligand binding...
bioassays determine the Ki value, which is a measure of binding affinity; lower Ki values indicate more selective binding. In vitro studies demonstrate that the Ki values for desvenlafaxine are 40.2 nM for the human serotonin transporter (hSERT) and 558.4 nM for the human norepinephrine transporter (hNET), for a ratio of 14. In contrast, the Ki values for venlafaxine are 82 nM for hSERT and 2480 nM for hNET, indicating a ratio of 30. The Ki values for duloxetine are 0.8 nM (hSERT) and 7.5 nM (hNET) for a ratio of 9. These data demonstrate that desvenlafaxine, venlafaxine, and duloxetine each inhibit 5-HT and NE reuptake, with more selective effects on 5-HT reuptake. The differences in the relative degree of inhibition of hSERT and hNET could translate to differences in relative efficacy for certain depressed patients; thus it may be speculated that some patients who do not respond to venlafaxine will respond to desvenlafaxine. However, it should be noted that, in the absence of direct head-to-head studies, it is not possible to compare data from in vitro bioassays that are obtained using different methods and from different laboratories.

Is there value in switching a patient who did not respond to venlafaxine ER to therapy with desvenlafaxine?

Patients with MDD who experience intolerable side effects or who do not respond to an adequate initial course of antidepressant therapy often are switched to another agent in an attempt to achieve a more favorable outcome. Finding the best possible treatment for an individual patient may require multiple trials with different antidepressants. Indeed, the Sequenced Treatment Alternatives to Relieve Depression trial found that only one in four patients who failed an initial course of selective serotonin reuptake inhibitor (SSRI) therapy achieved remission after switching to an alternate agent, thus suggesting that more than one trial may be necessary. Switching to an alternate antidepressant should be reserved for patients who have intolerable side effects or who, after receiving an adequate dose for a sufficient period of time, fail to respond to treatment. Patients who are not fully responding to venlafaxine and particularly those who experience adverse effects (AEs) or who are at risk of drug-drug interactions involving the CYP2D6 enzyme system may indeed be candidates for switching to desvenlafaxine. Desvenlafaxine is well-tolerated and, based on its pharmacokinetic profile, has a lower risk of drug-drug interactions compared with venlafaxine. Additionally, as mentioned above, venlafaxine and desvenlafaxine differ in the relative degree of 5-HT and NE reuptake inhibition; these differences may lead to differences in relative efficacy or tolerability, or both, for some patients, although conclusions are limited in the absence of data from head-to-head clinical trials. In general, although there may be similarities between drugs in terms of pharmacological effects on target symptoms (efficacy), there may be differences in terms of the impact on an individual patient (effectiveness). The importance of obtaining an accurate personal (and family, when appropriate) drug history that documents prior antidepressant trials, including treatment outcome (effectiveness), dose, duration of treatment, and AEs, cannot be emphasized strongly enough. Careful review of the patient’s drug history to determine adequacy of prior antidepressant trials and outcome will guide decisions to individualize the treatment strategy.

What dose of desvenlafaxine should be used for patients starting treatment? What dose should be used for patients switching from venlafaxine ER?

The recommended dose of desvenlafaxine for treatment of MDD is 50 mg/day. Findings from a randomized, double-blind, placebo-controlled trial demonstrated the efficacy of desvenlafaxine when administered in daily doses of 50 mg. However, no additional efficacy benefit is obtained with doses higher than 50 mg/day. In addition, higher doses may be associated with an increased rate of some AEs (e.g., nausea, sweating) compared with the 50-mg/day dose. Initial titration of desvenlafaxine is not necessary—desvenlafaxine 50 mg/day was well tolerated in clinical trials and was associated with a low rate of discontinuation due to AEs. Patients can begin treatment with the 50-mg/d dose. Switching from venlafaxine ER to desvenlafaxine (at doses of 200 to 400 mg/day) has been studied in an open-label extension of randomized, controlled short-term clinical trials, but the results have not been published at this time. However, clinicians may consider tapering the dose of venlafaxine ER when switching to desvenlafaxine.

How should the 100-mg dose of desvenlafaxine be used in practice?

In general, an adequate trial of antidepressant therapy for an episode of MDD is often considered to be treatment with a full therapeutic dose for 4 to 6 weeks, depending on clinician judgment. Depressive symptoms may improve in the first week of treatment with an antidepressant for some patients. However, a full clinical response and return to normal functional status may not be achieved for 3 months. Unless the patient is experiencing intolerable AEs, the antidepressant should not be switched to another agent until an adequate therapeutic trial has been completed. With regard to desvenlafaxine, the recommended dose is 50 mg/day. Nevertheless, it may be reasonable, based on clinical judgment, to increase the dose to 100 mg/day for select patients who do not respond to an adequate trial with the 50-mg dose.

Can the desvenlafaxine tablet be cut or crushed?

Desvenlafaxine is an ER formulation for once-daily administration that is designed to release desvenlafaxine over the course of the dosing interval. Desvenlafaxine tablets contain 76 mg or 152 mg of desvenlafaxine in a matrix formulation that is designed to gradually release the
How should treatment with desvenlafaxine be discontinued?

The abrupt cessation of SSRI and SNRI treatment in some patients has been associated with the emergence of a constellation of symptoms referred to as the discontinuation syndrome. Symptoms associated with the discontinuation syndrome vary in type and severity from one patient to another, emerge within 2–5 days after the antidepressant is abruptly stopped, and resolve within 1–2 weeks. In those patients who do experience symptoms upon abrupt discontinuation of antidepressants, dysphoric mood, irritability, agitation, dizziness, sensory disturbances (e.g., paresthesia, such as electric shock sensations), anxiety, confusion, headache, lethargy, emotional lability, insomnia, and tinnitus are among the symptoms that have been reported. Gradual tapering of the dose of antidepressants, either by decreasing the daily dose or by lengthening the dosing interval is recommended to minimize discontinuation symptoms.

Symptoms associated with discontinuation of desvenlafaxine were assessed in two short-term studies (8 weeks in duration) of 50 and 100 mg/day. Upon completion of the studies, treatment was discontinued without an intermediate dose for patients randomized to the 50-mg groups, and for patients in the 100-mg groups, the dose was reduced to 50 mg for 1 week and discontinued thereafter. Although the rate of discontinuation symptoms was significantly higher in the 50-mg groups during the first week (or in the second week for patients being tapered from the 100-mg dose), neither the group of patients whose desvenlafaxine therapy was tapered nor those who were discontinued from the 50-mg dose had a mean score on the 43-item Discontinuation-Emergent Signs and Symptoms checklist that fulfilled criteria for "discontinuation syndrome." Nonetheless, patients should be advised that discontinuation symptoms should be anticipated, though the severity is not predictable. Some patients who experience discontinuation symptoms following cessation of the 50-mg/day dose may benefit from a more gradual dose reduction; because the lowest available dosage form of desvenlafaxine is a 50-mg tablet that may not be broken or crushed, tapering may be achieved by increasing the dosing interval (i.e., desvenlafaxine 50 mg every other day). However, it should be noted that this tapering strategy has not been specifically evaluated in clinical studies.

How do discontinuation symptoms for desvenlafaxine compare with venlafaxine ER?

Direct head-to-head comparative studies of desvenlafaxine and venlafaxine ER to evaluate discontinuation symptoms have not been conducted. In the absence of data from prospectively conducted studies, the relative rates of discontinuation symptoms are not known. However, it may be possible to speculate that a lower rate of discontinuation symptoms could occur with desvenlafaxine, because it has fewer metabolites that do not have antidepressant pharmacological activity (but that may have pharmacological activity potentially associated with AEs), compared with venlafaxine ER, which has additional metabolites (e.g., N-desmethylvenlafaxine) that may contribute to AEs, including discontinuation symptoms. Data from the venlafaxine ER and desvenlafaxine clinical trial programs indicate that abrupt discontinuation of both drugs was associated with dizziness, nausea, headache, insomnia, diarrhea, anxiety, fatigue, abnormal dreams/nightmares, and sweating. In addition, following abrupt discontinuation of venlafaxine ER, agitation, anorexia, confusion, impaired coordination and balance, dry mouth, dysphoric mood, fasciculation, flu-like symptoms, hypomania, nervousness, sensory disturbances (including shock-like electrical sensations), somnolence, tremor, vertigo, and vomiting were noted.

Why do the efficacy data from desvenlafaxine studies appear to be somewhat inconsistent?

This is a very interesting question that is germane to studies of all antidepressants. There are several reasons why the results of antidepressant studies can be inconsistent, despite standard and seemingly similar methodologies. One well-recognized factor is the high placebo response rate inherent to antidepressant trials. Antidepressants fail to separate statistically from placebo in ~50% of randomized, controlled trials. This may be especially true for studies that enroll patients with mild to moderate depression (i.e., 17-item Hamilton Rating Scale for Depression [HAM-D17] total scores <24). Study design also may have an impact. Positive outcomes (i.e., statistical superiority compared with placebo) occur nearly twice as often in flexible-dose antidepressant studies compared with fixed-dose studies. In addition, rates of placebo response may differ from one country to another, which may contribute to variable response rates to placebo particularly in multinational studies. This may be due in part to differences in culture, genomics, ethnicity, gender, beliefs and attitudes, language, and other factors. For example, some languages do not have a term that describes depression. In some cultures, having a family member with a psychiatric condition, such as depression, is seen as a disgrace that could result in ostracism. Also, in some cultures, it is more acceptable to report physical symptoms (e.g., pain, fatigue, weakness) rather than depressive symptoms.

The choice of depression rating scale also may contribute to variable study results. The standard for many years has been the HAM-D which was originally designed to assess symptom changes in patients with depression. Even though the FDA requires the use of the HAM-D in antidepressant efficacy studies, it may not be sufficiently sensitive to detect real differences between placebo and
active drug. In addition, some of the terms used in depression rating scales, such as sedation or fatigue, may reflect AEs from medications and may not be related to an individual’s depressive episode.

Another factor that may contribute to variable study results is a nonlinear dose-response relationship in which higher doses increase rates of AEs (and possibly early treatment discontinuation) without added efficacy compared with lower doses. This may be relevant for desvenlafaxine, because the findings of randomized, controlled trials demonstrated no additional efficacy benefit above a daily dose of 50 mg. Rates of early study discontinuation due to AEs were twice as high in one fixed-dose desvenlafaxine study in the 100-mg group compared with the 50-mg group, which may have contributed to lower overall response rates in the last-observation-carried-forward analysis.

### How does the desvenlafaxine efficacy data compare with other antidepressants?

It is not possible to directly compare the efficacy of antidepressants in the absence of head-to-head comparative studies. However, one means of evaluating differences between one drug and another is to compare the magnitude of symptom reduction for an antidepressant and placebo. Using this method, the antidepressant efficacy of desvenlafaxine compares very favorably with other newer antidepressants (ie, noninferiority). Data from fixed-dose, randomized, double-blind antidepressant trials were assessed to determine differences between drug and placebo in the change from baseline to end point on mean HAM-D total scores. Trials lasting 6 to 12 weeks were included in this analysis. Findings from fixed-dose desvenlafaxine studies demonstrated a 2.0- to 2.9-point difference from placebo for the 50-mg dose (Table), which is similar to duloxetine 40 mg (1.1–3.0 points), venlafaxine 75 mg ER (2.5 points), venlafaxine 150 mg ER (1.5 points), fluoxetine 20 mg (1.2 points), paroxetine 20 mg (1.1 points), citalopram 20 mg (0.5–0.6 points), and selegiline transdermal patch 20 mg (2.2–2.6 points).

### Is desvenlafaxine as effective as venlafaxine ER?

In the absence of head-to-head comparative efficacy studies, the relative efficacy of desvenlafaxine and venlafaxine ER cannot be conclusively stated. In a rigorously conducted meta-analysis of eight studies, Thase and colleagues compared remission rates for venlafaxine and the SSRIs fluoxetine, paroxetine, and fluvoxamine. Remission rates for venlafaxine and the SSRIs were: fluoxetine 20 mg (61%), paroxetine 20 mg (58%), fluvoxamine 100 mg (60%), and venlafaxine ER 75 or 150 mg (67% and 70%, respectively). The SSRI fluvoxamine demonstrated a competitive efficacy for the treatment of major depression compared with venlafaxine ER.

### Table

<table>
<thead>
<tr>
<th>Medication</th>
<th>Drug Dose</th>
<th>Active Drug</th>
<th>Placebo</th>
<th>Difference Between Antidepressant and Placebo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Desvenlafaxine</td>
<td>50 mg</td>
<td>–11.5</td>
<td>–9.5</td>
<td>2.0</td>
</tr>
<tr>
<td></td>
<td>100 mg</td>
<td>–11.0</td>
<td>–9.5</td>
<td>1.5</td>
</tr>
<tr>
<td></td>
<td>50 mg</td>
<td>–14.4</td>
<td>–11.5</td>
<td>2.9</td>
</tr>
<tr>
<td></td>
<td>100 mg</td>
<td>–14.9</td>
<td>–11.5</td>
<td>3.4</td>
</tr>
<tr>
<td>Duloxetine</td>
<td>40 mg</td>
<td>–7.2</td>
<td>–4.2</td>
<td>3.0</td>
</tr>
<tr>
<td></td>
<td>60 mg</td>
<td>–9.3</td>
<td>–5.2</td>
<td>4.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>–6.9</td>
<td>–7.2</td>
<td>1.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>–10.9</td>
<td>–6.1</td>
<td>4.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>–10.5</td>
<td>–8.3</td>
<td>2.2</td>
</tr>
<tr>
<td>Venlafaxine ER</td>
<td>75 mg</td>
<td>–15.6</td>
<td>–13.1</td>
<td>2.5</td>
</tr>
<tr>
<td></td>
<td>150 mg</td>
<td>–14.6</td>
<td>–13.1</td>
<td>1.5</td>
</tr>
<tr>
<td>Fluoxetine</td>
<td>20 mg</td>
<td>–7.8</td>
<td>–6.6</td>
<td>1.2</td>
</tr>
<tr>
<td>Paroxetine</td>
<td>20 mg</td>
<td>–11.9</td>
<td>–10.8</td>
<td>1.1</td>
</tr>
<tr>
<td>Citalopram</td>
<td>20 mg</td>
<td>–11.1</td>
<td>–10.6</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>40 mg</td>
<td>–9.9</td>
<td>–9.3</td>
<td>0.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>–13.3</td>
<td>–10.6</td>
<td>2.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>–12.2</td>
<td>–9.3</td>
<td>2.9</td>
</tr>
<tr>
<td>Selegiline</td>
<td>20 mg</td>
<td>–8.7</td>
<td>–6.1</td>
<td>2.6</td>
</tr>
<tr>
<td>Transdermal</td>
<td></td>
<td>–11.1</td>
<td>–8.9</td>
<td>2.2</td>
</tr>
</tbody>
</table>

HAM-D = 17-item Hamilton Rating Scale for Depression; ER = extended release.
Desvenlafaxine: Frequently Asked Questions

Desvenlafaxine is the major pharmacologically active metabolite of venlafaxine and is a clinically and metabolically distinct entity. Analogous situations of active metabolites and parent compounds include nortriptyline/amitriptyline, desipramine/imipramine, and the antihistamines fexofenadine and terfenadine. Terfenadine was removed from the market because of toxicity when coadministered with a potent CYP3A4 inhibitor, such as ketoconazole.

I believe there are several reasons why clinicians would choose desvenlafaxine for their patients with depression. One reason relates to the question of antidepressant efficacy in patients who are poor metabolizers (PMs) of drugs that are biotransformed by the CYP2D6 hepatic enzyme system. The efficacy of venlafaxine in the treatment of depression in patients who are CYP2D6 extensive metabolizers (EMs) compared with patients who are CYP2D6 PMs has been studied, and when published these data will add to our understanding of the effect of metabolic status on antidepressant efficacy. Thus, one benefit of desvenlafaxine relates to its pharmacokinetic profile, which is not dependent on CYP2D6 status. In addition, desvenlafaxine has a very low potential for drug-drug interactions. Desvenlafaxine is not metabolized by CYP2D6. This is in marked contrast to venlafaxine, duloxetine, and other antidepressants (eg, tricyclic antidepressants, SSRIs, mirtazapine), which are metabolized by CYP2D6. Indeed, 20% to 25% of all drugs are biotransformed by CYP2D6. Further, desvenlafaxine does not inhibit CYP2D6 or other CYP enzymes. Thus, the potential for drug-drug interactions between desvenlafaxine and drugs that are either metabolized by or that inhibit or induce CYP2D6 is very low.

Although the absence of head-to-head comparative studies currently limits conclusions regarding relative efficacy or tolerability, other clinical considerations relate to tolerability and ease of use of desvenlafaxine. Specifically, treatment with desvenlafaxine can be initiated at the recommended therapeutic dose; this is in contrast to other SNRIs (eg, venlafaxine and duloxetine), which may require titration from a lower dose to improve tolerability. Furthermore, desvenlafaxine is associated with a low rate of discontinuations due to AEs. In fact, in an analysis of data from fixed-dose registration studies, the rate of discontinuation due to AEs was the same for the desvenlafaxine 50-mg/day group and the placebo group (both 4%).

How prevalent are slow metabolizers of CYP2D6?

Expression of the hepatic enzyme, CYP2D6, is subject to genetically determined polymorphism. Most individuals have functioning CYP2D6 enzymes and are classified as EMs. However, as many as 10% of the Caucasian population lacks functioning CYP2D6 alleles and are considered to be slow or PMs of drugs that are biotransformed by this enzyme system. Venlafaxine is primarily metabolized by CYP2D6. Administration of venlafaxine to PMs results in increased plasma venlafaxine concentrations compared with EMs; some evidence suggests that PMs may be at risk for an increased rate of AEs, although this has not been consistently documented. A recent study examined plasma concentrations of venlafaxine (VEN), desvenlafaxine (ODV), and N-desmethylvenlafaxine (NDV) in EMs, heterozygous EMs (HEMs; ie, individuals with one functional and one defective allele) and PMs. The results showed that although the sum of ODV + VEN plasma levels did not differ for HEMs or PMs compared with EMs, there was a shift (decrease) in the ODV/VEN ratio for both groups (P < .05). Further, plasma NDV levels were 5-fold higher among HEMs and 22-fold higher among PMs compared with EMs (P < .001). Thus, it is not unreasonable to speculate that the differences in relative ODV and VEN plasma concentrations in conjunction with substantially greater levels of NDV could be one mechanism by which venlafaxine is less well tolerated among PMs compared with EMs.

A pharmacokinetic study in EMs and PMs of CYP2D6 was conducted to determine the effect of this genetic polymorphism on the pharmacokinetic properties of venlafaxine ER and desvenlafaxine. The peak plasma concentrations and area under the plasma concentration-versus-time curve of the active metabolite, desvenlafaxine, were significantly lower following venlafaxine ER administration in PMs compared with EMs (Figure 2). In contrast, there is no difference in the pharmacokinetic parameters of desvenlafaxine for EMs versus PMs (Figure 3). This absence of variability in plasma concentrations may confer a more uniform response to desvenlafaxine treatment that is not dependent on CYP2D6 metabolizer status. Data from studies that evaluate the relative efficacy of desvenlafaxine in EMs versus PMs would undoubtedly help to inform the choice of antidepressant therapy in patients whose metabolic status is not known. Furthermore, the absence of plasma concentration variability with desvenlafaxine may warrant consideration for studies using doses lower than 50 mg/day to assess efficacy in treating depression. Desvenlafaxine is not dependent on active metabolites to achieve efficacy and is highly bioavailable (ie, 80%); as such, lower doses of desvenlafaxine may be sufficient to achieve therapeutic plasma concentrations for many patients, regardless of metabolizer status.
Are there any desvenlafaxine data in the treatment of anxiety?

Desvenlafaxine is indicated for the treatment of MDD in adults and is not approved for treatment of anxiety disorders. However, anxiety symptoms are a common component of MDD, and the presence of anxiety symptoms is associated with a more severe course of depression with increased levels of psychiatric comorbidity. The effect of desvenlafaxine on anxiety symptoms was assessed as a secondary outcome measure in randomized, placebo-controlled trials in patients with depression. Based on changes from baseline Covi Anxiety Scale total scores, treatment with 50 or 100 mg/day resulted in scores that were significantly lower or trended toward significance compared with placebo. The value of desvenlafaxine in treating anxiety should be evaluated further in controlled clinical trials where improvement in standardized anxiety scales is a primary outcome measure.

Will there be other indications for desvenlafaxine?

Pfizer Inc, formerly Wyeth Pharmaceuticals, is currently investigating the efficacy and safety of desvenlafaxine for the treatment of vasomotor symptoms in postmenopausal women and its use in other conditions is being explored. These uses are not FDA-approved at this time. Desvenlafaxine is only approved for the treatment of MDD in adults.

REFERENCES


