

# Health Reform May Hinge on Public Plan Option

BY MARY ELLEN SCHNEIDER

The chances of passing health reform legislation this year could depend on whether lawmakers can resolve their differences over the public insurance plan option.

The proposal to include a government-sponsored health plan that would compete against private insurance became a major wedge in the health care debate,

and how much to pay physicians under such a plan emerged as a key sticking point, according to observers.

"It could wind up bringing down the whole agenda," said Grace-Marie Turner, president of the Galen Institute, a non-profit research organization that advocates for free market ideas in health care.

Ms. Turner, who opposes the public plan option, said that although Democrats have control of the presidency and

both chambers of Congress, there is disagreement within their own ranks, with many moderate and conservative Democrats opposed to a public plan.

The idea of a public plan was debated extensively at the recent policy-making meeting of the American Medical Association, where the delegates ended up endorsing "health system reform alternatives that are consistent with AMA principles of pluralism, freedom of choice, freedom of practice, and universal access for patients."

The AMA has stated publicly that it does not support any plan that would force physicians to participate in a public plan or that would pay physicians based on Medicare rates. The AMA has said, however, that it will consider some of the variations on a public plan that are being discussed in Congress now, such as a federally chartered co-op health plan.

Officials at the American College of Physicians agree that provider participation in any plan should be voluntary and not tied to current participation in Medicare. The college also advocates for payment rates to be competitive with commercial payers, rather than based on the low rates now offered by Medicare.

But the ACP also sees potential advantages to creating a public plan, according to its president, Dr. Joseph W. Stubbs. A public plan could provide a "nationwide blanket" of fall-back coverage, which would be especially helpful in areas of low penetration by insurance carriers. It could also offer a mechanism for rapidly introducing new models of care and reimbursement, such as the medical home concept. A public plan could also be a way to hold private plans accountable in areas where there is little competition.

"The devil will be in the details as far as whether this is a good idea or not," Dr. Stubbs said.

Meanwhile, other physicians have been disappointed by talk of a public plan for different reasons. Dr. David Himmelstein, an associate professor of medicine at Harvard University in Boston and the cofounder of Physicians for a National Health Program, said what's being discussed in Congress now is really "just a clone of private insurance."

Dr. Himmelstein, who favors a single-payer health system, said a public plan would fall far short of realizing the savings that could be seen with a single-payer system. A public plan wouldn't even be able to achieve the type of low overhead seen with Medicare, he said, which benefits from automatic enrollment and easy premium collection, and has no need to spend money on marketing.

President Obama, who reached out to physicians for support at the AMA meeting last month, said he understands that many physicians are skeptical about how they would fare under a public plan. In his speech to the AMA, President Obama said he intended to change the way physicians get paid, rewarding best practices and good patient care. "The public option is not your enemy," he said. "It is your friend."



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"The public option is not your enemy," President Obama told AMA delegates.

Part of the problem with evaluating the public plan option is that there isn't just one. Among the health care reform proposals circulating in both the House and the Senate, some include a government-run or quasi-government-run option to compete with private insurance.

The purest form of a so-called public plan would be one that is something like Medicare, where federal dollars, not just premiums, are used to support it, said Kathleen Stoll, health policy director at Families USA, which supports the general idea of a public plan but hasn't thrown its support to a particular proposal. But many lawmakers and analysts have said this design would give the public plan an advantage over private insurance products and cause private payers to leave the market, she said.

A proposal put forward by leaders in the House would create a public plan on the same footing as other insurance plans. For example, public and private plans alike would have to adhere to the same benefit requirements and insurance market reforms and would have to be financially self-sustaining based on premiums. This proposal would not require participation by physicians but initially would use payment rates similar to those of Medicare. Rates would be unlinked from Medicare rates over time as other payment mechanisms were developed.

In the Senate, an approach getting a lot of attention is to create not a public plan but rather a federally chartered, nonprofit cooperative plan, Ms. Stoll said. This proposal is seen by many as a compromise between a government-run plan and no public plan at all.

Overall, the discussion on a public plan is heading in a direction that is positive for physicians, said Elizabeth Carpenter, associate policy director for the Health Policy Program at the New America Foundation, a nonpartisan think tank.

At the beginning of discussions on health care reform, the thinking was that a public plan would use Medicare rates in paying physicians and other providers. Now that idea seems to be losing support, Ms. Carpenter said. Instead, in those cases where reform proposals are referencing Medicare rates, those rates are intended only as a starting point, she said.

Combined administration of racemic citalopram (40 mg) and ketoconazole (200 mg), a potent CYP3A4 inhibitor, decreased the  $C_{max}$  and AUC of ketoconazole by 21% and 10%, respectively, and did not significantly affect the pharmacokinetics of citalopram. **Ritonavir**-Controlled administration of a single dose of ritonavir (600 mg), both a CYP3A4 substrate and a potent inhibitor of CYP3A4, and escitalopram (20 mg) did not affect the pharmacokinetics of either ritonavir or escitalopram. **CYP3A4 and -2C19 Inhibitors**-*In vitro* studies indicated that CYP3A4 and -2C19 are the primary enzymes involved in the metabolism of escitalopram. However, coadministration of escitalopram (20 mg) and ritonavir (600 mg), a potent inhibitor of CYP3A4, did not significantly affect the pharmacokinetics of escitalopram. Because escitalopram is metabolized by multiple enzyme systems, inhibition of a single enzyme may not appreciably decrease escitalopram clearance. **Drugs Metabolized by Cytochrome P4502D6**-*In vitro* studies did not reveal an inhibitory effect of escitalopram on CYP2D6. In addition, steady state levels of racemic citalopram were not significantly different in poor metabolizers and extensive CYP2D6 metabolizers after multiple-dose administration of citalopram, suggesting that coadministration, with escitalopram, of a drug that inhibits CYP2D6, is unlikely to have clinically significant effects on escitalopram metabolism. However, there are limited *in vivo* data suggesting a modest CYP2D6 inhibitory effect for escitalopram, i.e., coadministration of escitalopram (20 mg/day for 21 days) with the tricyclic antidepressant desipramine (single dose of 50 mg), a substrate for CYP2D6, resulted in a 40% increase in  $C_{max}$  and a 100% increase in AUC of desipramine. The clinical significance of this finding is unknown. Nevertheless, caution is indicated in the coadministration of escitalopram and drugs metabolized by CYP2D6. **Metoprolol**-Administration of 20 mg/day Lexapro for 21 days in healthy volunteers resulted in a 50% increase in  $C_{max}$  and 82% increase in AUC of the beta-adrenergic blocker metoprolol (given in a single dose of 100 mg). Increased metoprolol plasma levels have been associated with decreased cardioselectivity. Coadministration of Lexapro and metoprolol had no clinically significant effects on blood pressure or heart rate. **Electroconvulsive Therapy (ECT)**-There are no clinical studies of the combined use of ECT and escitalopram.

**USE IN SPECIFIC POPULATIONS: Pregnancy:** Pregnancy Category C-In a rat embryo/fetal development study, oral administration of escitalopram (56, 112, or 150 mg/kg/day) to pregnant animals during the period of organogenesis resulted in decreased fetal body weight and associated delays in ossification at the two higher doses (approximately 56 times the maximum recommended human dose [MRHD] of 20 mg/day on a body surface area [ $mg/m^2$ ] basis). Maternal toxicity (clinical signs and decreased body weight gain and food consumption), mild at 56 mg/kg/day, was present at all dose levels. The developmental no-effect dose of 56 mg/kg/day is approximately 28 times the MRHD on a  $mg/m^2$  basis. No teratogenicity was observed at any of the doses tested (as high as 75 times the MRHD on a  $mg/m^2$  basis). When female rats were treated with escitalopram (6, 12, 24, or 48 mg/kg/day) during pregnancy and through weaning, slightly increased offspring mortality and growth retardation were noted at 48 mg/kg/day which is approximately 24 times the MRHD on a  $mg/m^2$  basis. Slight maternal toxicity (clinical signs and decreased body weight gain and food consumption) was seen at this dose. Slightly increased offspring mortality was also seen at 24 mg/kg/day. The no-effect dose was 12 mg/kg/day which is approximately 6 times the MRHD on a  $mg/m^2$  basis. In animal reproduction studies, racemic citalopram has been shown to have adverse effects on embryofetal and postnatal development, including teratogenic effects, when administered at doses greater than human therapeutic doses. In two rat embryo/fetal development studies, oral administration of racemic citalopram (32, 56, or 112 mg/kg/day) to pregnant animals during the period of organogenesis resulted in decreased embryo/fetal growth and survival and an increased incidence of fetal abnormalities (including cardiovascular and skeletal defects) at the high dose. This dose was also associated with maternal toxicity (clinical signs, decreased body weight gain). The developmental no-effect dose was 56 mg/kg/day. In a rabbit study, no adverse effects on embryo/fetal development were observed at doses of racemic citalopram up to 16 mg/kg/day. Thus, teratogenic effects of racemic citalopram were observed at a maternally toxic dose in the rat and were not observed in the rabbit. When female rats were treated with racemic citalopram (4.8, 12.8, or 32 mg/kg/day) from late gestation through weaning, increased offspring mortality during the first 4 days after birth and persistent offspring growth retardation were observed at the highest dose. The no-effect dose was 12.8 mg/kg/day. Similar effects on offspring mortality and growth were seen when dams were treated throughout gestation and early lactation at doses  $\geq$  24 mg/kg/day. A no-effect dose was not determined in that study. There are no adequate and well-controlled studies in pregnant women; therefore, escitalopram should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. **Pregnancy-Nonteratogenic Effects**-Women exposed to Lexapro and other SSRIs or SNRIs, late in the third trimester, have developed complications requiring prolonged hospitalization, respiratory support, and tube feeding. Such complications can arise immediately upon delivery. Reported clinical findings have included respiratory distress, cyanosis, apnea, seizures, temperature instability, feeding difficulty, vomiting, hypoglycemia, hypotonia, hypertonia, hyperreflexia, tremor, jitteriness, irritability, and constant crying. These features are consistent with either a direct toxic effect of SSRIs and SNRIs or, possibly, a drug discontinuation syndrome. It should be noted that, in some cases, the clinical picture is consistent with serotonin syndrome [see Warnings and Precautions]. Infants exposed to SSRIs in late pregnancy may have an increased risk for persistent pulmonary hypertension of the newborn (PPHN). PPHN occurs in 1-2 per 1000 live births in the general population and is associated with substantial neonatal morbidity and mortality. In a retrospective, case-control study of 377 women whose infants were born with PPHN and 836 women whose infants were born healthy, the risk for developing PPHN was approximately six-fold higher for infants exposed to SSRIs after the 20th week of gestation compared to infants who had not been exposed to antidepressants during pregnancy. There is currently no corroborative evidence regarding the risk for PPHN following exposure to SSRIs in pregnancy; this is the first study that has investigated the potential risk. The study did not include enough cases with exposure to individual SSRIs to determine if all SSRIs pose similar levels of PPHN risk. When treating a pregnant woman with Lexapro during the third trimester, the physician should carefully consider both the potential risks and benefits of treatment [see Dosage and Administration]. Physicians should note that in a prospective longitudinal study of 201 women with a history of major depression who were euthymic at the beginning of pregnancy, women who discontinued antidepressant medication during pregnancy were more likely to experience a relapse of major depression than women who continued antidepressant medication. **Labor and Delivery**-The effect of Lexapro on labor and delivery in humans is unknown. **Nursing Mothers**-Escitalopram is excreted in human breast milk. Limited data from women taking 10-20 mg escitalopram showed that exclusively breast-fed infants receive approximately 3.9% of the maternal weight-adjusted dose of escitalopram and 1.7% of the maternal weight-adjusted dose of desmethylcitalopram. There were two reports of infants experiencing excessive somnolence, decreased feeding, and weight loss in association with breastfeeding from a racemic citalopram-treated mother; in one case, the infant was reported to recover completely upon discontinuation of racemic citalopram by its mother and, in the second case, no follow-up information was available. Caution should be exercised and breast-feeding infants should be observed for adverse reactions when Lexapro is administered to a nursing woman.

**Pediatric Use**-Safety and effectiveness of Lexapro has not been established in pediatric patients (less than 12 years of age) with Major Depressive Disorder. Safety and effectiveness of Lexapro has been established in adolescents (12 to 17 years of age) for the treatment of major depressive disorder [see Clinical Studies]. Although maintenance efficacy in adolescent patients with Major Depressive Disorder has not been systematically evaluated, maintenance efficacy can be extrapolated from adult data along with comparisons of escitalopram pharmacokinetic parameters in adults and adolescent patients. Safety and effectiveness of Lexapro has not been established in pediatric patients less than 18 years of age with Generalized Anxiety Disorder. **Geriatric Use**-Approximately 6% of the 1144 patients receiving escitalopram in controlled trials of Lexapro in major depressive disorder and GAD were 60 years of age or older; elderly patients in these trials received daily doses of Lexapro between 10 and 20 mg. The number of elderly patients in these trials was insufficient to adequately assess for possible differential efficacy and safety measures on the basis of age. Nevertheless, greater sensitivity of some elderly individuals to effects of Lexapro cannot be ruled out. SSRIs and SNRIs, including Lexapro, have been associated with cases of clinically significant hyponatremia in elderly patients, who may be at greater risk for this adverse event [see Hyponatremia]. In two pharmacokinetic studies, escitalopram half-life was increased by approximately 50% in elderly subjects as compared to young subjects and  $C_{max}$  was unchanged [see Clinical Pharmacology]. 10 mg/day is the recommended dose for elderly patients [see Dosage and Administration]. Of 442 patients in clinical studies of racemic citalopram, 137 were 60 and over, 1034 were 65 and over, and 457 were 75 and over. No overall differences in safety or effectiveness were observed between these subjects and younger subjects, and other reported clinical experience has not identified differences in responses between the elderly and younger patients, but again, greater sensitivity of some elderly individuals cannot be ruled out.

**DRUG ABUSE AND DEPENDENCE: Abuse and Dependence:** Physical and Psychological Dependence-Animal studies suggest that the abuse liability of racemic citalopram is low. Lexapro has not been systematically studied in humans for its potential for abuse, tolerance, or physical dependence. The premarketing clinical experience with Lexapro did not reveal any drug-seeking behavior. However, these observations were not systematic and it is not possible to predict on the basis of this limited experience the extent to which a CNS-active drug will be misused, diverted, and/or abused once marketed. Consequently, physicians should carefully evaluate Lexapro patients for history of drug abuse and follow such patients closely, observing them for signs of misuse or abuse (e.g., development of tolerance, increments of dose, drug-seeking behavior).

**OVERDOSAGE: Human Experience**-In clinical trials of escitalopram, there were reports of escitalopram overdose, including overdoses of up to 600 mg, with no associated fatalities. During the postmarketing evaluation of escitalopram, Lexapro overdoses involving overdoses of over 1000 mg have been reported. As with other SSRIs, a fatal outcome in a patient who has taken an overdose of escitalopram has been rarely reported. Symptoms most often accompanying escitalopram overdose, alone or in combination with other drugs and/or alcohol, included convulsions, coma, dizziness, hypotension, insomnia, nausea, vomiting, sinus tachycardia, somnolence, and ECG changes (including QT prolongation and very rare cases of torsade de pointes). Acute renal failure has been very rarely reported accompanying overdose. **Management of Overdose**-Establish and maintain an airway to ensure adequate ventilation and oxygenation. Gastric evacuation by lavage and use of activated charcoal should be considered. Careful observation and cardiac and vital sign monitoring are recommended, along with general symptomatic and supportive care. Due to the large volume of distribution of escitalopram, forced diuresis, dialysis, hemoperfusion, and exchange transfusion are unlikely to be of benefit. There are no specific antidotes for Lexapro. In managing overdose, consider the possibility of multiple-drug involvement. The physician should consider contacting a poison control center for additional information on the treatment of any overdose.

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