

POLICY & PRACTICE

Bill Halts 4.4% Cut

Congress' long-awaited passage of the budget reconciliation package (also called the Deficit Reduction Act) put a freeze on a 4.4% cut Medicare physicians experienced in the month of January. Although the congressional action stopped any further reductions to payments, it did not increase Medicare physician pay for 2006. The Centers for Medicare and Medicaid Services will reimburse physicians retroactively for the reductions experienced in January and has instructed its contractors to automatically reprocess claims. But work on this issue is far from over, Dr. J. Edward Hill, president of the American Medical Association, said in a statement. "With 6 years of cuts still scheduled to come as practice costs continue to rise—we fear more physicians will make difficult practice decisions about treating Medicare patients. ... We must build on the momentum and awareness raised in 2005 to make 2006 the year Congress permanently repeals the broken Medicare physician-payment formula." President Bush's fiscal-year 2007 budget request to Congress briefly mentioned the impending cuts, although it expounded on CMS's efforts to expand pay-for-performance initiatives to "achieve better outcomes at a lower overall cost."

And On to the 2007 Budget

The president's 2007 budget request for the Department of Health and Human Services—\$698 billion—is a \$58 billion increase from 2006, but it contains a number of cost-containment measures that would either whittle down or cut certain programs entirely. A series of Medicare initiatives to "encourage efficient and appropriate payment for services; foster competition; and promote beneficiary involvement in their health care decisions" would save nearly \$36 billion from 2007 to 2011, according to a statement from HHS. But Part A hospital payments would incur \$22 billion of these cuts, which is "the wrong policy at the wrong time," given that hospitals have been losing money caring for Medicare beneficiaries since 2003, said Chip Kahn, president of the Federation of American Hospitals. In an attempt to meet President Bush's goal of cutting the federal deficit in half by 2009, the budget request proposes other targeted reductions or the elimination of certain programs whose performance ratings were low or whose purposes are being covered by other HHS programs. These cuts include \$133 million to rural health programs run by the Health Resources and Services Administration, and the elimination of the \$630 million Community Services Block Grant program. The budget request did ask for more money for some programs. For example, to achieve the president's goal for most Americans to have secure personal electronic medical health records by 2014, \$169 million was requested for 2007 (\$59 million more than in 2006) for health information technology. The Food and Drug Administration's 2007 budget request totaled \$1.95 billion, a \$70.8 million or 3.8% increase over 2006.

Arthritis Research Funding

On the flip side, the president's 2007 budget request freezes the overall budget for

the National Institutes of Health at \$28.5 billion and cuts funding for the National Institute of Arthritis and Musculoskeletal and Skin Diseases. Under the proposal, NIAMS funding would fall from about \$508 million in FY 2006 to about \$505 million in FY 2007. The president's budget was met with concern by a coalition of medical research-advocacy groups, including the National Health Council and Research!America. The coalition is calling for a 5% increase in NIH funding above its current level to allow research to keep pace with inflation. "Saving a little mon-

ey today makes no sense when the price we pay tomorrow will surely be much higher in terms of lives that could have been saved and the loss of our competitive edge in medical research," Myrl Weinberg, president of the National Health Council, said in a statement.

2007 Medicare Formulary Guidance

The U.S. Pharmacopeia (USP) last month released its final model guidelines for use in developing Medicare prescription drug formularies in 2007. The model guidelines are used by the Centers for Medicare and Medicaid Services to evaluate the formularies created by private drug plans

that participate in the Medicare Part D program. There are fewer unique categories and classes in the 2007 document—133, compared with 146 in 2006. In addition, the number of formulary key drug types, which are used by CMS to test the comprehensiveness of the formulary, has been increased from 118 to 141. The final model guidelines eliminate the distinction between nonsteroidal anti-inflammatory drugs and cyclooxygenase-2 inhibitors and between SSRIs and serotonin/norepinephrine reuptake inhibitors. The USP model guidelines are available online at www.usp.org.

—Mary Ellen Schneider

IN PAH, TAKE AIM AT ET-1 THROUGH ET_A SELECTIVITY

Circulating levels of ET-1, the most potent subtype of ET, have been associated with disease severity in PAH.¹ The deleterious effects of elevated ET-1 include cellular proliferation, vasoconstriction, and vascular remodeling.²⁻⁴

In pulmonary arterial hypertension (PAH), endothelin (ET-1) exerts its cardiovascular effects through 2 receptors: ET_A and ET_B. When ET-1 activates the ET_A receptor on the vascular smooth muscle, it leads to vasoconstriction and vascular remodeling.^{4,5} Endothelial ET_B receptors mediate the release of vasodilating nitric oxide (NO) and prostacyclin (PGI₂), while inhibiting and clearing ET-1 from circulation.^{5,6} Blockade of ET_B receptors may significantly impair the balance of endothelium-derived vasodilating substances.^{4,7}

Endothelial dysfunction has been shown to improve with selective ET_A blockade.⁸ Hence, preemptive targeting of ET-1 through selective ET_A receptor antagonism can slow the progression of PAH, and may even provide better overall outcomes.^{2,4,8}

TARGETED ET-1 TREATMENTS MAY PROVIDE BETTER OUTCOMES

Imbalances in the key endothelial cell-derived mediators NO, PGI₂, and specifically ET-1 are thought to be central to the pathogenesis of PAH.⁹ NO and PGI₂ are potent vasodilators with antiproliferative activity.¹⁰ ET-1 is a potent vasoconstrictor with proliferative activity.⁵ Chronically elevated levels of ET-1 are associated with pulmonary vascular resistance, excessive scar formation and cardiac remodeling, cellular proliferation, and cardiac hypertrophy.^{1,11-13}

A reduction of excess ET-1 levels may result in positive outcomes for patients with PAH. It has been shown that in patients with congestive heart failure, elevated ET-1 plasma

Figure 1: ET_A receptor pathway

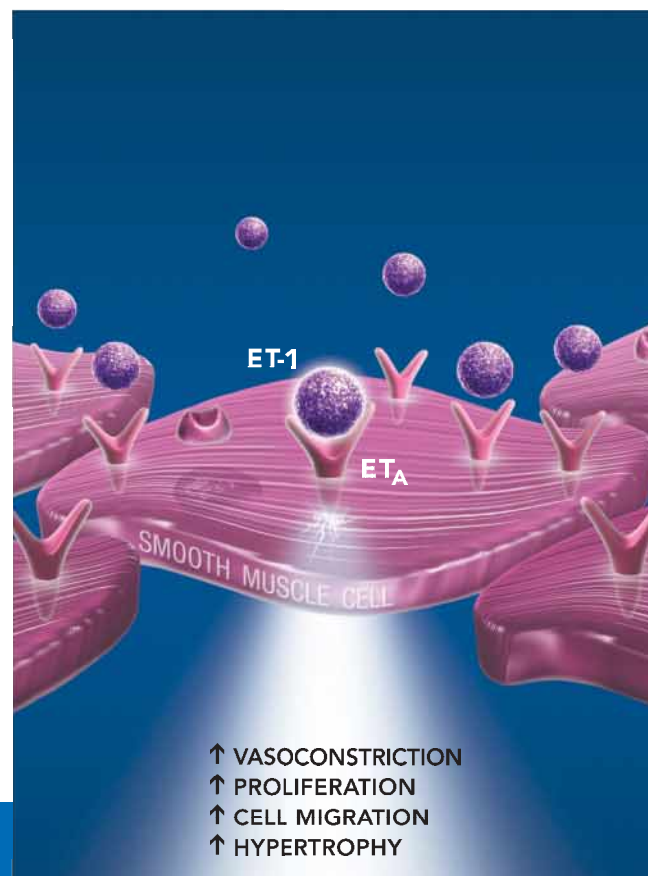


Figure 2: ET_B receptor pathway

