Insmed Presents Positive IPLEX(TM) Data At ENDO 2006

BOSTON, Jun 27, 2006 (BUSINESS WIRE) -- Insmed, Inc. (NASDAQ: INSM):

Results from 3 IPLEX studies demonstrate:

-- IPLEX increases growth and improves blood sugar control in patients with severe insulin-resistance syndromes, such as Leprechaunism and Type A syndrome (1)

-- IPLEX improves growth rate in children with severe primary IGF-I deficiency(2)

-- IPLEX safely increases IGF-I levels in healthy adults without causing abnormal increases in free IGF-I levels.(3)

Insmed, Inc. (NASDAQ: INSM) today announced new study results that show that IPLEX(TM) (mecasermin rinfabate (rDNA origin) injection) is effective in increasing growth and improving glycemic control in patients with severe insulin-resistance syndromes, specifically Leprechaunism and Type A syndrome. In another clinical trial, once-daily treatment with IPLEX significantly improved height velocity in children with severe primary insulin growth factor-I (IGF-I) deficiencies. A third study of the pharmacokinetics of IPLEX in normal adults showed positive results, with simultaneous increases in IGF-I and IGFBP-3 and without undue increases in "free" IGF-I. Findings of all three studies were presented this week at the annual meeting of the Endocrine Society, ENDO 2006.

In a study showing the impact of IPLEX treatment on infants diagnosed with Leprechaunism (or Donohue syndrome) (Abstract OR40-2), preliminary results indicate IPLEX improved growth and glycemic (glucose) control, as well as potentially prolonged the life of at least one patient. Leprechaunism, the rarest and most severe insulin resistance syndrome, is diagnosed in infancy and in some patients can result in death in the first year of life. In this study, two patients with Leprechaunism were treated with IPLEX. Results showed an improved height standard deviation score in both patients (one from -3.3 pre-treatment to -2.4 with treatment and the second from -2.8 pre-treatment to -1.8 with treatment). Additionally the first patient achieved a reduction in HbA1c (7.6 percent pre-treatment to 6.7 percent with treatment), as well as a decrease in mean daily glucose levels (9.4 mmol/L pre-treatment to 6.6 mmol/L with treatment), indicating IPLEX produced a significant physiological benefit in this patient. This patient has been on therapy for three years and both patients currently remain on IPLEX treatment.

In the severe insulin resistance study, three adolescents with Type A syndrome, a disease in which the patient exhibits poor glycemic control despite conventional therapies, were evaluated on IPLEX treatment. Each patient entered the trial with elevated glucose levels and inadequate response to treatment with traditional therapeutic agents. Results showed that these patients, when treated with IPLEX, demonstrated approximately a 20% average decrease in HbA1c and daily glucose levels. Patients receiving IPLEX experienced decreased glucose excursions, reduced insulin usage, and noticed less prominent acanthosis nigricans (dark, velvety skin patches), which are commonly associated with severe insulin resistance. In further evidence of drug-dependent changes, all of these improvements worsened in each patient once IPLEX treatment was terminated.

"The preliminary results of this study suggest that IPLEX can be more effective than conventional diabetes therapies in patients with severe insulin resistance syndromes," said Kenneth M. Attie, M.D., Vice President, Medical Affairs, Insmed. "We look forward to the completion of these studies with IPLEX and continuing to evaluate its utility in diabetes-related disorders, particularly in those with extreme insulin resistance."

Safety and Efficacy in Children with Severe Primary IGF-I Deficiency

A second study (Abstract OR 40-1) presented at ENDO demonstrates IPLEX's safety and efficacy in children with severe primary IGF-I deficiency. A prospective, multicenter clinical trial of IPLEX administered once daily showed that treatment resulted in statistically significant, dose-dependent increases in height velocity (growth rate) with a favorable safety profile. Average height velocity in one of the treatment groups, treated with a dose of up to 2 mg/kg/day, increased from 2.0 cm/year pre-treatment to 8.3 cm/year during the first year of treatment. Children with genetic and acquired forms of growth hormone (GH) insensitivity appeared to respond equally well to treatment in this study.

IPLEX was generally well tolerated in these patients. Adverse events included injection site reactions (including erythema, lipohypertropy, and hair growth), hypoglycemia (generally rated as mild and asymptomatic), headache, and tonsillar/adenoid hypertrophy.

IPLEX Pharmacokinetics in Normal Adults Presented

Results from a pharmacokinetic study of IPLEX, presented during 2 poster presentations, demonstrate that IPLEX has unique, positive properties. The first poster (P1-191) presented the pharmacokinetic results of single-dose administration of the drug to 28 healthy adult volunteers. The investigators concluded that due to the gradual absorption and elimination of IPLEX in the bloodstream, sustained increases in levels of IGF-I and IGFBP-3 are achieved lasting more than 24 hours. These data indicate that the drug can be administered once-daily or, in certain patient populations, possibly less frequently, such as every other day. IPLEX was well tolerated during the study.

A second poster from this study (P1-192) examined the levels of free IGF-I in healthy adult volunteers after administration of IPLEX. Under normal circumstances, less than 2 percent of IGF-I circulates as the free biologically active form because of a complex physiology that keeps it bound to other proteins, principally IGFBP-3 and ALS. The IPLEX complex was designed to limit the amount of free IGF-I associated with replacement therapy, thus maintaining a physiological balance of IGF-I with IGFBP-3. Results of this study conclude the mean percent of free IGF-I did not exceed 1 percent at any time during the 72-hour sampling period, thus avoiding supra-physiologic levels of free IGF-I.

"IPLEX was designed to provide IGF-I in a stable complex with its natural binding protein, and thus mimic what occurs naturally in human circulation," said Attie. "In the pharmacokinetic study presented at ENDO, we demonstrated that the drug limits the excursions of free IGF-I levels by providing a gradual absorption of the apparently intact complex. The pharmacokinetics of this drug make it an ideal treatment for children with severe primary IGFD and, in the future, other patient populations that might benefit from once daily IGF-I replacement."

About IPLEX

IPLEX is approved in the United States as the only once daily treatment for children with short stature associated with severe primary IGF-I deficiency (Primary IGFD). IPLEX, a complex of recombinant human IGF-I and its binding protein IGFBP-3 (rhIGF-I/rhIGFBP-3), is the only FDA-approved IGF-I replacement therapy that also replaces deficient IGFBP-3 in these patients. The drug, which was launched in the second quarter of 2006, is also being investigated for various other indications with unmet medical needs, including severe insulin resistance, myotonic muscular dystrophy and HIV Associated Adipose Redistribution Syndrome (HARS). For more information about IPLEX please go to www.go-IPLEX.com.

About Insmed Incorporated

Insmed is a biopharmaceutical company focused on the development and commercialization of drug candidates for the treatment of metabolic diseases and endocrine disorders with unmet medical needs. For more information, please visit www.insmed.com. The Company's leading product, IPLEX was approved as an orphan drug by the United States Food and Drug Administration in December 2005 for the treatment of growth failure in children with severe primary IGF-I deficiency (Primary IGFD) or with growth hormone (GH) gene deletion who have developed neutralizing antibodies to GH.

Statements included within this press release, which are not historical in nature, may constitute forward-looking statements for purposes of the safe harbor provided by the Private Securities Litigation Reform Act of 1995. Forward-looking statements in this press release include, but are not limited to, statements regarding our IPLEX utilization program, regulatory and business strategies, manufacturing capabilities, product costs, plans and objectives of management and growth opportunities for existing or proposed products. Such forward-looking statements are subject to numerous risks and uncertainties, including risks that product candidates may fail in the clinic or may not be successfully launched, marketed, manufactured or reimbursed, we may lack financial resources to complete development of product candidates, the FDA may interpret the results of our studies differently than we have, competing products may be more successful, demand for new pharmaceutical products may decrease, the biopharmaceutical industry may experience negative market trends and other risks detailed from time to time in our filings with the Securities and Exchange Commission. As a result of these and other risks and uncertainties, actual results may differ materially from those described in this press release. For further information with respect to factors that could cause actual results to differ from expectations, reference is made to our reports filed by the Company with the Securities and Exchange Commission under the Securities Exchange Act of 1934, as amended. The forward-looking statements made in this release are made only as of the date hereof and Insmed disclaims any intention or responsibility for updating predictions or financial guidance contained in this release.

(1)ENDO 2006 Abstract OR40-2 - rhIGF-1/rhIGFBP-3 Treatment of Patients with Severe Insulin Resistance Syndromes: Preliminary Data.

(2)ENDO 2006 Abstract OR40-1 - Once Daily rhIGF-1/rhIGFBP-3 Treatment Improves Growth in Children with Severe Primary IGF-I Deficiency: Results of a Multicenter Clinical Trial

(3)ENDO 2006 Abstract P1-192 - Subcutaneous Administration of rhIGF-1/rhIGFBP-3 in Healthy Adult Volunteers Does Not Result in Supraphysiological Concentrations of Free IGF-I.