TEVA PHARMACEUTICAL INDUSTRIES LTD
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Report of Foreign Private Issuer

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Teva Pharmaceutical Industries Limited

(Translation of registrant's name into English)

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NEW DATA EVALUATING LAQUINIMOD FOR THE TREATMENT OF MULTIPLE SCLEROSIS DEMONSTRATE NEUROPROTECTIVE EFFECTS

Data presented at the 26th Congress of the European Committee for Treatment and Research in Multiple Sclerosis (ECTRIMS)

Jerusalem, Israel and Lund, Sweden, October 15, 2010 - Teva Pharmaceutical Industries Ltd. (NASDAQ: TEVA) and Active Biotech (NASDAQ OMX NORDIC: ACTI) announced today that data providing further evidence of the neuroprotective properties of laquinimod in animal studies were presented at the 26th Congress of the European Committee for Treatment and Research in Multiple Sclerosis (ECTRIMS) in Gothenburg, Sweden. Laquinimod is an investigational, once-daily oral immunomodulator for the treatment of relapsing remitting multiple sclerosis (RRMS).

These results, generated from several pre-clinical studies evaluating the mechanism of action (MOA) of oral laquinimod, demonstrated that:

Laquinimod reverts the disruption of neurogenic processes that can occur with chronic inflammation in the central nervous system (CNS), and is associated with a significant reduction in the percentage of demyelination and axonal damage.

Laquinimod differentially influenced the activity of select immune cells, reducing their pro-inflammatory characteristics while increasing the production of neurotrophic factors known to be involved in neuroprotection and repair mechanisms.

Laquinimod treatment is associated with an increase in brain-derived neurotrophic factor (BDNF), a pivotal factor in the development and maintenance of the CNS.

"These results add to the accumulating body of data establishing the novel MOA of laquinimod. These MOA studies suggest that laquinimod has the potential to prevent demyelination, which is associated with multiple sclerosis, and

therefore may provide neuroprotection in the treatment of RRMS," said Prof. Ralf Gold, Department of Neurology, St. Josef-Spital, Ruhr University Bochum, Germany. "Research is ongoing to further evaluate laquinimod, and we look forward to additional data, including the forthcoming results from the Phase III clinical development program."

Laquinimod received Fast Track designation from the U.S. Food and Drug Administration (FDA) in February 2009. Two global Phase III clinical studies, ALLEGRO and BRAVO, have completed enrollment and are currently ongoing, with results anticipated in 2011.

ABOUT THE STUDIES

[P885] Laquinimod prevents the inflammation-induced derangement of neurogenic niches in experimental autoimmune encephalomyelitis mice.

Poster Session: Neuroprotection 2, October 15, 3:30pm-5:00pm CET *(F. Ruffini, A. Bergamaschi, C. Marinaro, R. De Ceglia, L. Muzio, R. Furlan, L. Hayardeny, G. Comi, G. Martino)*

o To determine if laquinimod is

o capable of reverting non-cell autonomous dysfunction of endogenous neural stem cells following chronic inflammation within the central nervous system (CNS), C57Bl/6 mice were immunized with myelin oligodendrocytes glycoprotein (MOG) peptide 35-55 and treated subcutaneously (s.c.) with increasing doses of laquinimod (1, 5, 10 or 25 mg/kg laquinimod). At the end of the study (day 25 p.i.), CNS of EAE mice was collected for pathological and molecular studies. Pathological and molecular study results in mice with EAE suggest that treatment with 25 mg/kg of laquinimod administered after EAE onset resulted in a significant reduction in the percentage of demyelination and axonal damage as well in the number of inflammatory responses. An in vivo increase in cell proliferation was also observed.

[P881] Laquinimod ameliorates experimental autoimmune encephalomyelitis via BDNF-dependent mechanisms.

Poster Session: Neuroprotection 2, October 15, 3:30pm-5:00pm CET *(J. Thöne, D. Lee, S. Seubert, L. Hayardeny, R. Linker, R. Gold)*

o To further elucidate the mechanism of action of laquinimod and to examine its potential neuroprotective capacity via modulation of BDNF secretion, laquinimod was tested in myelin oligodendrocyte glycoprotein (MOG) induced EAE experiments using mice at 6-8 weeks of age on C57Bl/6J background, and with a conditional deficiency for BDNF in T cells and monocytes (LLF mice). Histological analyses reveal that mice with BDNF deficiency, treated with laquinimod, had more severe experimental autoimmune encephalomyelitis (EAE), or animal model of MS progression, than mice not BDNF deficient, and actually experience an increase in BDNF levels. This suggests that the mechanism of action of laquinimod is BDNF dependent and may contribute to neuroprotection.

[P882] Differential activity of laquinimod on production of inflammatory molecules and neurotrophic factors by human microglia and macrophages.

Poster Session: Neuroprotection 2, October 15th, 3:30pm-5:00pm CET (*C. Silva, J. Wang, M. Mishra, V.W. Yong*)

o To determine whether laquinimod modulates the production of inflammatory molecules and growth factors by microglia and macrophages, the cells were isolated from the peripheral blood of healthy adult human volunteers and cultured from the brains of human fetal samples or from adult human surgical brain resections. Cytokines and matrix metalloproteinase-9 (MMP-9) were measured by ELISA of cell-conditioned media and growth factor transcripts were determined using PCR of cellular extracts. Treatment with laquinimod reduces the expression of inflammatory responses by autoimmune microglia and in healthy human cells and increases production of the neuroprotective BDNF by threefold.

[P251] Laquinimod rescue therapy in mice with experimental autoimmune encephalomyelitis.

Poster Session: Experimental Models 1, October 14th, 2010, 3:30pm-5:00pm CET (*C. Wegner, R. Pförtner, W. Brück)*

o To test whether laquinimod could improve EAE symptoms as a late-stage rescue therapy. The next goal was to assess if this late-stage treatment alters the extent of demyelination and inflammation in MOG induced EAE. Laquinimod rescue therapy given from days 30 to 60 improved clinical disease scores in the majority of animals in comparison to the control group. Findings indicate that late stage therapy with laquinimod is effective in ameliorating the disease severity. Results indicate that laquinimod may have a role in future treatment of MS.

ABOUT MULTIPLE SCLEROSIS

Multiple sclerosis (MS) is the leading cause of neurological disability in young adults. It is estimated that more than 400,000 people in the United States are affected by the disease and that two million people may be affected worldwide. MS is a progressive, demyelinating disease of the central nervous system affecting the brain, spinal cord and optic nerves. Demyelination is the destructive breakdown of the fatty tissue that protects nerve endings.

ABOUT LAQUINIMOD

Laquinimod is a novel once-daily, orally administered immunomodulatory drug being developed as a disease-modifying treatment for RRMS. Active Biotech developed laquinimod and licensed it to Teva Pharmaceutical Industries, Ltd. in June 2004. Results from a Phase IIb study in 306 patients were published in June 2008 in *The Lancet*, and reported that an oral 0.6 mg dose of laquinimod, administered daily, significantly reduced MRI disease activity by a mean of 51 percent ($p<0.0001$) and median of 60 percent ($p=0.01$) versus placebo in RRMS patients. Some transient and dose-dependent increases in liver enzymes were reported, without clinically-evident liver damage.

Two pivotal, global Phase III studies of laquinimod for the treatment of RRMS, ALLEGRO and BRAVO, are nearing completion. ALLEGRO, a 24-month multinational, double-blind, placebo-controlled study, designed to evaluate the efficacy, safety and tolerability of laquinimod versus placebo in the treatment of RRMS, enrolled 1,106 patients and data from the study are expected in Q1 2011. BRAVO, a 24-month multinational, multi-center, randomized, parallel-group study designed to evaluate laquinimod compared to placebo, as well as to provide risk-benefit data for laquinimod compared to a currently available injectable treatment, Avonex&reg, has enrolled 1,332 patients and will be complete in Q3 2011.

In addition to the ongoing RRMS clinical studies, laquinimod is currently in Phase II development for Crohn`s disease and Lupus, and is being studied in other autoimmune diseases.

ABOUT TEVA

Teva Pharmaceutical Industries Ltd. (NASDAQ:TEVA) is a leading global pharmaceutical company, committed to increasing access to high-quality healthcare by developing, producing and marketing affordable generic drugs as well as innovative and specialty pharmaceuticals and active pharmaceutical ingredients. Headquartered in Israel, Teva is the world's largest generic drug maker, with a global product portfolio of more than 1,250 molecules and a direct presence in approximately 60 countries. Teva's branded businesses focus on neurological, respiratory and women's health therapeutic areas as well as biologics. Teva's leading innovative product, Copaxone&reg, is the number one prescribed treatment for multiple sclerosis. Teva employs more than 40,000 people around the world and reached $13.9 billion in net sales in 2009.

ABOUT ACTIVE BIOTECH

Active Biotech AB (NASDAQ OMX NORDIC: ACTI) is a biotechnology company with focus on autoimmune/inflammatory diseases and cancer. Projects in or entering pivotal phase are laquinimod, an orally administered small molecule with unique immunomodulatory properties for the treatment of multiple sclerosis, TASQ for prostate cancer as well as ANYARA for use in cancer targeted therapy, primarily of renal cell cancer. In addition, laquinimod is in Phase II development for Crohn`s and Lupus. Further projects in clinical development comprise the two orally administered compounds, 57-57 for SLE & Systemic Sclerosis and RhuDex(TM) for RA. Please visit www.activebiotech.com for more information.

Teva's Safe Harbor Statement under the U. S. Private Securities Litigation Reform Act of 1995:
This release contains forward-looking statements, which express the current beliefs and expectations of management. Such statements are based on management's current beliefs and expectations and involve a number of known and unknown risks and uncertainties that could cause our future results, performance or achievements to differ significantly from the results, performance or achievements expressed or implied by such forward-looking statements. Important factors that could cause or contribute to such differences include risks relating to: our ability to successfully develop and commercialize additional pharmaceutical products, the introduction of competing generic equivalents, the extent to which we may obtain U.S. market exclusivity for certain of our new generic products and regulatory changes that may prevent us from utilizing exclusivity periods, potential liability for sales of generic products prior to a final resolution of outstanding patent litigation, including that relating to the generic versions of Neurontin&reg, Lotrel&reg, Protonix&reg and Yaz&reg, the extent to which any manufacturing or quality control problems damage our reputation for high quality production, the effects of competition on sales of our innovative products, especially Copaxone&reg (including potential generic and oral competition for Copaxone&reg), the impact of

continuing consolidation of our distributors and customers, our ability to identify, consummate and successfully integrate acquisitions (including the acquisition of ratiopharm), interruptions in our supply chain or problems with our information technology systems that adversely affect our complex manufacturing processes, intense competition in our specialty pharmaceutical businesses, any failures to comply with the complex Medicare and Medicaid reporting and payment obligations, our exposure to currency fluctuations and restrictions as well as credit risks, the effects of reforms in healthcare regulation, adverse effects of political or economical instability, major hostilities or acts of terrorism on our significant worldwide operations, increased government scrutiny in both the U.S. and Europe of our agreements with brand companies, dependence on the effectiveness of our patents and other protections for innovative products, our ability to achieve expected results through our innovative R&D efforts, the difficulty of predicting U.S. Food and Drug Administration, European Medicines Agency and other regulatory authority approvals, uncertainties surrounding the legislative and regulatory pathway for the registration and approval of biotechnology-based products, potentially significant impairments of intangible assets and goodwill, potential increases in tax liabilities resulting from challenges to our intercompany arrangements, our potential exposure to product liability claims to the extent not covered by insurance, the termination or expiration of governmental programs or tax benefits, current economic conditions, any failure to retain key personnel or to attract additional executive and managerial talent, environmental risks and other factors that are discussed in this report and in our other filings with the U.S. Securities and Exchange Commission ("SEC").

Active Biotech's Safe Harbor Statement in Accordance with the Swedish Securities Market Act:
This press release contains certain forward-looking statements. Such forward-looking statements involve known and unknown risks, uncertainties and other important factors that could cause the actual results, performance or achievements of the company, or industry results, to differ materially from any future results, performance or achievement implied by the forward-looking statements. The company does not undertake any obligation to update or publicly release any revisions to forward-looking statements to reflect events, circumstances or changes in expectations after the date of this press release.

Active Biotech is obligated to publish the information contained in this press release in accordance with the Swedish Securities Market Act.

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Teva Pharmaceutical Industries Ltd. Web Site: www.tevapharm.com

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized.

TEVA PHARMACEUTICAL INDUSTRIES LIMITED

(Registrant)

By: /s/ Eyal Desheh

Name: Eyal Desheh
Title: Chief Financial Officer

Date October 15, 2010