TEVA PHARMACEUTICAL INDUSTRIES LTD
Form 6-K
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FORM 6-K

SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

Report of Foreign Private Issuer

Pursuant to Rule 13a 16 or 15d 16

under the Securities Exchange Act of 1934

For the month of April 2010

Commission File Number 0-16174

Teva Pharmaceutical Industries Limited

(Translation of registrant's name into English)

5 Basel Street, P.O. Box 3190

Petach Tikva 49131 Israel

(Address of principal executive offices)

Indicate by check mark whether the registrant files or will file annual reports under cover of Form 20-F or Form 40-F:

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For immediate release

NEW DATA suggest oral laquinimod may confer neuroprotection in addition to immunomodulation in THE treatment of MULTIPLE sclerosis

Enhanced levels of brain derived neurotrophic factor, possibly contributing to neuroprotection, were shown in laquinimod treated multiple sclerosis (MS) patients

Laquinimod significantly reduced demyelination and axonal damage as shown in animal models

Two ongoing pivotal, global Phase III clinical trials are fully enrolled and results are anticipated next year

Jerusalem, Israel and Lund, Sweden, April 15, 2010 - Teva Pharmaceutical Industries Ltd. (NASDAQ: TEVA) and Active Biotech (NASDAQ OMX NORDIC: ACTI) today announced results from several studies demonstrating that laquinimod, a novel, investigational once-daily oral immunomodulator for relapsing remitting multiple sclerosis (RRMS) may have neuroprotective properties in addition to its anti-inflammatory effects. These studies were presented at the 62nd Annual Meeting of the American Academy of Neurology (AAN).

New data from studies in RRMS patients demonstrate that treatment with laquinimod results in a significant increase in brain derived neurotrophic factor, a key protein responsible for the maintenance of mature neurons.

Additionally, data from new animal models show that following treatment with laquinimod there were significant reductions in the extent of demyelination, and more axonal preservation within spinal cord lesions. Furthermore, treatment with laquinimod inhibited the infiltration of inflammatory cells into the spinal cord and brain, and caused a positive shift in specific white blood cells involved in multiple sclerosis (MS) pathology.

These findings suggest that laquinimod may have neuroprotective properties in addition to anti-inflammatory effects. Coupled with the Phase IIb study results, which demonstrated oral laquinimod to be effective and safe in RRMS patients, these data provide further insight into the contributing factors surrounding the favorable benefit-risk profile associated with this compound to date.

"As MS research progresses, emphasis will not only be on delaying disease progression, but on preventing permanent nerve damage," said Ralf Linker, M.D., Department of Neurology, St. Josef-Hospital/Ruhr-University Bochum. "These data are encouraging and suggest that the mechanism of action of laquinimod may contribute to neuroprotection in RRMS patients, in addition to providing therapeutic benefit through immunomodulation."

Laquinimod received Fast Track designation from the U.S. Food and Drug Administration (FDA) in February 2009. Two pivotal global Phase III clinical trials, BRAVO and ALLEGRO, are fully enrolled and results are anticipated next year.

ABOUT THE STUDIES

The studies evaluating the mechanism of action of laquinimod being presented at AAN include:

Laquinimod Induces Up-Regulation of BDNF in Serum of Patients with Relapsing-Remitting Multiple Sclerosis (PD5.004, Poster Discussion Session V: Thursday, April 15th from 7:30 AM-12:00 PM)
Jan Thöne, Silvia Seubert, Rebecca Conrad, Bochum, NRW, Germany, Giancarlo Comi, Milan, Italy, Stefan Wiese, Bochum, NRW, Germany, Liat Hayardeny, Netanya, Israel, Ralf Gold, Ralf Andreas Linker, Bochum, NRW, Germany

o Treatment with 0.6 mg of laquinimod for three months resulted in a significant and specific increase (up to 11-fold) in brain derived neurotrophic factor (BDNF) serum levels as compared to baseline or placebo treatment (Total amount of mean +/- SEM: 16088 +/- 760 pg/ml BDNF in the laquinimod-group vs. 12882 +/- 625 pg/ml in the placebo-group, $p < 0.05$ and vs. 14436 +/- 778 pg/ml at baseline, $p < 0.05$). This effect on BDNF was maintained at 9 months and when analyzing the relapse-free subgroup. The increased levels of the neurotrophic factor BDNF in vivo in laquinimod treatment groups possibly contributes to neuroprotection in MS patients.

Effect of Laquinimod on Monocyte Subsets (S41.005, Scientific Sessions: Multiple Sclerosis: Clinical Immunology: Thursday, April 15, 2010 at 2:15 PM)

Tal Birnberg, Steffen Jung, Rehovot, Israel, Israel

o Results indicate a profound effect of laquinimod on the steady state distribution of murine blood and bone-marrow (BM) monocytes. Short-term treatment resulted in a significant shift of the monocyte subset balance towards the Ly6C^{hi} subset, established precursors of CNS dendritic cells (DCs) and macrophages in EAE lesions, which are accumulating in the blood and CNS immediately prior to EAE clinical episodes. Furthermore, the laquinimod-mediated effect was significantly increased in EAE induced mice. Study authors conclude the effect of laquinimod on autoimmune deviation might be due in part to its impact on monocyte subsets.

Axonal Protection Effect of Laquinimod Appears Partially Independent of Its Inhibitory Effect on Inflammation and Demyelination in Experimental Autoimmune Encephalomyelitis (P 06.208, Poster Session VI: Thursday, April 15, 2010 from 3:00 PM-7:30 PM)

Christiane Wegner, Christine Stadelmann, Goettingen, Germany, Emanuel Nathan Raymond, Bracha Timan, Liat Hayardeny, Netanya, Israel, Wolfgang Brück, Goettingen, Germany

o Histological analysis revealed a marked reduction of macrophage and T-cell infiltration in the total white matter of mice with MOG-induced experimental autoimmune encephalomyelitis (EAE) treated with laquinimod compared to control mice. Pro-inflammatory cytokines were down-regulated and the extent of demyelination was significantly reduced in laquinimod treated mice. In addition after therapeutic treatment, laquinimod treated mice displayed significantly less axonal damage (measured by APP immunohistochemistry) within spinal cord lesions in comparison to control mice (697 +/-185/mm2 vs. 892 +/- 205/mm2, p=0.014). Treatment with laquinimod is effective in ameliorating the extent of macrophage and T cell infiltration, demyelination and axonal damage. These findings indicate that laquinimod might have axon-protection properties in addition to its anti-inflammatory effect and may therefore play 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 compound that is 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 median of 60 percent (51 percent mean reduction) versus placebo in RRMS patients. In addition, the study showed a favorable trend toward reducing annual relapse rates and in the number of relapse-free patients compared with placebo. Treatment was well tolerated, with some transient and dose-dependent increases in liver enzymes reported, without clinically-evident liver damage.

In addition to the efficacy that laquinimod has shown in Phase IIb RRMS studies, laquinimod has demonstrated potent therapeutic efficacy in preclinical models of other autoimmune diseases such as rheumatoid arthritis, insulin-dependent diabetes mellitus, Guillain Barré Syndrome, lupus and Inflammatory Bowel Disease. The broad profile of efficacy in animal models of inflammatory diseases suggests that laquinimod affects a pivotal pathway of inflammation and autoimmunity. Laquinimod is currently in Phase II development for Crohn`s disease and Teva will initiate the clinical development of the compound for Lupus Nephritis in the near future.

ABOUT TEVA

Teva Pharmaceutical Industries Ltd., headquartered in Israel, is among the top 15 pharmaceutical companies in the world and is the leading generic pharmaceutical company. The company develops, manufactures and markets generic and innovative pharmaceuticals and active pharmaceutical ingredients. Over 80 percent of Teva's sales are in North America and Western Europe.

ABOUT ACTIVE BIOTECH

Active Biotech AB (OMX NORDIC: ACTI), headquartered in Sweden, is a biotechnology company with R&D focus on autoimmune/inflammatory diseases and cancer. Projects in pivotal phase are laquinimod, an orally administered small molecule with unique immunomodulatory properties for the treatment of multiple sclerosis, as well as ANYARA for use in cancer targeted therapy, primarily renal cancer. Further key projects in clinical development comprise the three orally administered compounds TASQ for prostate cancer, 57-57 for SLE and RhuDex™ 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 and Protonix&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, 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 April 15, 2010