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 October 2011

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:

Form 20-F X Form 40-F _____

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(1):_____

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(7):_____

Treatment with COPAXONE® in Multiple Sclerosis Demonstrated Remyelination and Neuroprotective Effects in a Preclinical Setting

Additional Preclinical Data to be Presented at the Fifth Joint Triennial Congress of ECTRIMS and ACTRIMS Further Elucidate the Anti-inflammatory Mechanism of COPAXONE®

JERUSALEM--(BUSINESS WIRE)--October 19, 2011--Teva Pharmaceutical Industries Ltd. (NASDAQ: TEVA) today announced preclinical data demonstrating reparative and neuroprotective effects of treatment with COPAXONE® (glatiramer acetate injection) in experimental autoimmune encephalomyelitis (EAE) models. In the study, researchers compared mice treated with COPAXONE® versus non-treated mice in relapsing-remitting and chronic multiple sclerosis (MS) disease models. Researchers observed both remyelination indicative of repair and a drastic reduction of demyelination and axonal loss in mice treated with COPAXONE®.

“While several previous studies, both clinical and preclinical, pointed to possible neuroprotective properties of COPAXONE® treatment, these data demonstrate a process of remyelination as a consequence of the treatment,” said lead study author, Rina Aharoni, Senior Staff Scientist, Department of Immunology, The Weizmann Institute of Science, Rehovot, Israel. “These data may also help explain why COPAXONE® continues to demonstrate efficacy in the long-term.”

In addition to remyelination and neuronal preservation, the central nervous system of mice treated with COPAXONE® had smaller lesions, increased axonal density and a higher prevalence of normal appearing axons. Measurements were taken both before and after induction of EAE, showing that COPAXONE® prevented new damage and caused reversal of existing neurological degeneration. These data will be published this fall in the *Journal of Autoimmunity*.

A second preclinical study demonstrated that a signaling pathway for COPAXONE® deactivated white blood cells called macrophages that induce inflammation and autoimmune response.

“Data have indicated that activated damaging macrophages may contribute to axonal loss in MS, and that the deactivation of these macrophages may be a therapeutic goal of treatment,” said lead study author, Nicolas Molnarfi, Researcher Neuroimmunologist, Department of Neurology and Program in Immunology, University of California, San Francisco, California. “These data showed that COPAXONE® deactivated specific macrophages, elucidating a potential mechanism for the impact of COPAXONE® treatment.”

The results of both studies will be presented on October 20, 2011 at the Fifth Joint Triennial Congress of the European and Americas Committees for Treatment and Research in Multiple Sclerosis (ECTRIMS and ACTRIMS).

ABOUT THE STUDIES:

In the study evaluating potential neuroprotective effects of COPAXONE®, the in situ pathological manifestations of two different EAE models, the relapsing-remitting PLP-induced and the chronic MOG-induced diseases were analyzed and compared, utilizing both transmission electron microscopy (TEM) and immunohistochemistry. The effect of the MS drug COPAXONE® on myelin damage/repair and on motor neuron loss/preservation was studied in both models.

Quantitative TEM analysis of the relative remyelination extent, compared to demyelination, provides, for the first time, evidence of significant augmentation of remyelination after treatment with COPAXONE® (glatiramer acetate injection). Loss of motor neuron was also reduced in mice treated with COPAXONE®, in comparison to that of EAE untreated mice. These effects were obtained even when COPAXONE® treatment was applied in a therapeutic schedule, after the appearance of clinical symptoms.

In the study evaluating the anti-inflammatory mechanism of COPAXONE®, exposure of murine macrophages to COPAXONE® induces phosphoinositide 3-kinase (PI3K) activation, a central negative regulator in inflammation. The results provide a direct mechanism of inhibition of innate signals by COPAXONE® and delineate a signaling pathway important for deactivation of macrophages in inflammation and autoimmunity.

ABOUT COPAXONE®

COPAXONE® is indicated for the reduction of the frequency of relapses in relapsing-remitting multiple sclerosis, including patients who have experienced a first clinical episode and have MRI features consistent with multiple sclerosis. The most common side effects of COPAXONE® are redness, pain, swelling, itching, or a lump at the site of injection, flushing, rash, shortness of breath, and chest pain. COPAXONE® (glatiramer acetate injection) is now approved in more than 50 countries worldwide, including the United States, Russia, Canada, Mexico, Australia, Israel, and all European countries. In North America,

COPAXONE® is marketed by Teva Neuroscience, Inc., which is a subsidiary of Teva Pharmaceutical Industries Ltd. In Europe, COPAXONE® is marketed by Teva Pharmaceutical Industries Ltd. and sanofi-aventis. COPAXONE® is a registered trademark of Teva Pharmaceutical Industries Ltd.

See additional important information at: http://www.sharedsolutions.com/pdfs/PrescribingInformation.aspx or call 1-800-887-8100 for electronic releases.

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 1,300 molecules and a direct presence in about 60 countries. Teva's branded businesses focus on CNS, oncology, pain, respiratory and women's health therapeutic areas as well as biologics. Teva currently employs approximately 45,000 people around the world and reached $16.1 billion in net sales in 2010.

***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 version of Protonix®, 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® (including potential generic and oral competition for Copaxone®), the impact of continuing consolidation of our distributors and customers, our ability to identify, consummate and successfully integrate acquisitions (including the acquisition of Cephalon), 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 our Annual Report on Form 20-F and other filings with the U.S. Securities and Exchange Commission.

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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 20, 2011