ALTEON INC /DE
Form 8-K
October 20, 2004

SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

FORM 8-K

CURRENT REPORT

PURSUANT TO SECTION 13 or 15(d) OF THE

SECURITIES EXCHANGE ACT OF 1934

Date of report (Date of earliest event reported) October 18, 2004

ALTEON INC.
(Exact Name of Registrant as Specified in Charter)

Delaware	001-16043	13-3304550
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(I.R.S. Employer Identification No.)

6 Campus Drive, Parsippany, New Jersey	07054
(Address of Principal Executive Offices)	(Zip Code)

Registrant's telephone number, including area code (201) 934-5000

(Former Name or Former Address, If Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

[ ] Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

[ ] Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

[ ] Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2 (b))

[ ] Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4 (c))

Item 8.01. Other Events

On October 18, 2004, Alteon issued the following press release:

Alteon's Alagebrium Reverses Erectile Dysfunction In Preclinical Study
Leading Investigators in ED Field Note Unique Mechanism of Action

PARSIPPANY, N.J., Oct. 18 /PRNewswire-FirstCall/ -- Alteon Inc. (Amex: ALT) announced today that its lead A.G.E. Crosslink Breaker compound alagebrium (formerly known as ALT-711) has demonstrated an ability to reverse erectile dysfunction (ED) in a preclinical model of diabetes. The preclinical study, entitled "Delayed Administration of ALT-711, but not of Aminoguanidine, Improves Erectile Function in Streptozotocin Diabetic Rats: Curative Versus Preventive Medicine," was presented today at the 11th World Congress of the International Society for Sexual and Impotence Research in Buenos Aires. Independent study authors, Mustafa F. Usta(1,3), Muammer Kendirci(1), Trinity J. Bivalacqua(1,4), Serap Gur(1), Wayne J.G. Hellstrom(1), Neale A. Foxwell(2), and Selim Cellek(2), conclude that alagebrium -- through what appears to be a unique mechanism of action -- offers significant potential for the treatment of diabetic erectile dysfunction.

According to the investigative team, these data are unlike results for existing ED drugs in similar experiments, particularly due to a beneficial effect on the function of the corpus cavernosum. "Alagebrium appears to have significant therapeutic potential for the treatment of diabetic erectile dysfunction, with a unique mechanism of action," said Wayne J.G. Hellstrom, M.D., an author of this study and many of the seminal studies in ED.

An estimated 30-40 percent of diabetic and aged patients with ED do not receive benefit from currently available drugs, and patients with diabetes or severe vascular disease are among the most refractory to such treatment. This occurs, in part, because the corpus cavernosum, the structure that acts as an expandable reservoir for blood, has become significantly glycated and fibrotic, unable to properly dilate due to the accumulation and crosslinking of pathological structures called advanced glycation end-products (A.G.E.s). A.G.E.s have been implicated in the fibrosis and stiffening of tissues and organs throughout the body and have been shown to contribute to many inflammatory processes. A.G.E.s have been demonstrated to impair erectile function in diabetes by affecting the functional capabilities of the corpus cavernosum and by interfering with the production of natural penile vasodilating agents, endothelial and neuronal nitric oxide (NO).

Study authors investigated the effect of delayed administration of alagebrium, a crosslink breaker of A.G.E.s, as compared to aminoguanidine (pimagedine), an inhibitor of A.G.E.s, in a well-established ED animal model. In previous preclinical and clinical testing, alagebrium has demonstrated the ability to cleave advanced glycation end-product crosslinks as well as diminish deleterious inflammatory responses caused by A.G.E.s. Aminoguanidine has previously been shown to prevent ED in diabetic animals if given immediately after the induction of diabetes. In the current study, both aminoguanidine and alagebrium were initiated at the 6th week of diabetes and the two treated groups were compared to a group of age-matched controls and a group of untreated diabetic animals. Twelve weeks after diabetes induction, in vivo intracavernosal pressure measurements were assessed, as well as serum and penile A.G.E. levels. The diabetic

rats had a significant decrease in erectile function as assessed by the level of intracavernosal pressure obtained after cavernosal nerve stimulation and elevated A.G.E. levels when compared to the control rats. The administration of alagebrium resulted in a significant improvement in erectile function, as well as a decrease in serum and tissue A.G.E. levels, while the delayed

administration of aminoguanidine did not correct either A.G.E. levels or erectile dysfunction. In addition, alagebrium normalized other diabetes-induced pathologies associated with ED, an effect unlike any currently marketed therapies used to treat symptoms of the condition. The results of the preclinical study have been submitted for publication in a peer-reviewed medical journal.

In prior clinical and preclinical studies, alagebrium has been shown to have a remodeling effect on the cardiovascular system(5) as well a positive effect on systolic hypertension(6) and vascular compliance(7). The drug is currently in Phase 2 studies in patients with hypertension and heart failure. In addition, it is being studied for its effect in endothelial dysfunction, a condition also linked to erectile dysfunction.

"We are actively evaluating a clinical development pathway for the ED indication," said Kenneth I. Moch, President and CEO. "Because ED is an early indicator of vascular disease, this exciting research by world-renowned ED investigators is fully consistent with, and supportive of, our ongoing clinical development programs for alagebrium in hypertension and heart failure. Should alagebrium's therapeutic effect be seen in human studies in ED, this would not only represent a breakthrough for ED, but would also be indicative of alagebrium's potential to reverse pathologies in a number of other human vascular diseases."

Understanding the Link between Erectile Dysfunction and Vascular Disease

Erectile dysfunction is defined as the persistent inability to attain and maintain an erection sufficient to permit satisfactory sexual intercourse. ED has been reported to affect as many as 20 to 30 million men in the United States and 152 million men worldwide, according to the National Institutes on Health. The risk for ED increases progressively with advancing age, with an estimated 54 percent of men ages 65 to 70 reporting some degree of impotence (Nicolosi, 2003). It is believed that 85-90 percent of ED cases are related to a physical or medical condition, while 10-15 percent is due to psychological causes.

Many studies have identified ED as an early indicator of cardiovascular diseases, including hypertension, heart attack and stroke, and point to the underlying dysfunction of the arteries and vascular system as a principal cause. ED is commonly associated with a number of other conditions frequently occurring in aging men, including prostatic hypertrophy, arterial hypertension, ischemic heart disease, peripheral vascular disease, atherosclerosis, hyperlipidemia, and diabetes mellitus.

About Alteon

Alteon is developing several new classes of drugs that have shown the potential to reverse or slow down diseases of aging and complications of diabetes. These compounds have an impact on a fundamental pathological process caused by the progressive formation of protein-glucose

complexes called Advanced Glycation End-products (A.G.E.s). The formation and crosslinking of A.G.E.s lead to a loss of flexibility and function in body tissues and organs and have been shown to be a causative factor in many age-related diseases and diabetic complications. Alteon has created a library of novel classes of compounds targeting the A.G.E. Pathway.

Alteon's lead compound alagebrium chloride (formerly ALT-711), the only A.G.E. Crosslink Breaker in advanced human testing, has demonstrated safety and efficacy in several Phase 2 trials and is actively being developed for systolic hypertension and heart failure. Over 1200 patients have been involved in alagebrium's human clinical trials to date, of whom approximately 900 have received active compound. Ongoing clinical trials include the phase 2b systolic

hypertension trial, SPECTRA (Systolic Pressure Efficacy and Safety Trial of Alagebrium), and the phase 2a heart failure trial, PEDESTAL (Patients with Impaired Ejection Fraction and Diastolic Dysfunction: Efficacy and Safety Trial of ALagebrium), as well as a third trial exploring mechanism of action in endothelial dysfunction. For more detailed scientific information about alagebrium and a copy of the full abstract, please visit the scientific publications section of the Alteon website, http://www.alteon.com .

Any statements contained in this press release that relate to future plans, events or performance are forward-looking statements that involve risks and uncertainties including, but not limited to, those relating to technology and product development (including the possibility that early clinical trial results may not be predictive of results that will be obtained in large-scale testing or that any clinical trials will not demonstrate sufficient safety and efficacy to obtain requisite approvals or will not result in marketable products), regulatory approval processes, intellectual property rights and litigation, competitive products, ability to obtain financing, and other risks identified in Alteon's filings with the Securities and Exchange Commission. The information contained in this press release is accurate as of the date indicated. Actual results, events or performance may differ materially. Alteon undertakes no obligation to publicly release the result of any revision to these forward-looking statements that may be made to reflect events or circumstances after the date hereof or to reflect the occurrence of unanticipated events.

1. Department of Urology, Tulane Health Sciences Centre, New Orleans, USA

2. Wolfson Institute for Biomedical Research, University College London, London, UK

3. Present address: Urology Department, Akdeniz University, Antalya, Turkey

4. Present address: Department of Surgery, Johns Hopkins Hospital, Baltimore, USA

5. "Effect of ALT-711, a Novel Glucose Cross-link Breaker, in the Treatment of Diastolic Heart Failure." Poster presentation, European Society of Cardiology Congress 2003. Dalane W. Kitzman, Michael Zile, William C. Little, W. Gregory Hundley, Terrence X. O'Brien, Robert C. deGroof.

6. "A Clinical Trial of an A.G.E. Cross-link Breaker, Alagebrium Chloride (ALT-711), in Systolic Hypertension." Abstract presented at the American Society of Hypertension Nineteenth

Annual Scientific Meeting, May 19, 2004. George L. Bakris, Alan Bank, David C. Kass, Joel Neutel, Richard Preston.

7. "Improved Arterial Compliance by a Novel Advanced Glycation End-Product Crosslink Breaker." Circulation: 2001; 104: r8-r14. David A. Kass, Edward P. Shapiro, Miho Kawaguchi, Anne R. Capriotti, Angelo Scuteri, Robert C. deGroof, Edward G. Lakatta.

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 hereunto duly authorized.

Alteon Inc.

By: /s/ ELIZABETH A. O'DELL

Elizabeth A. O'Dell
Vice President, Finance

Dated: October 19, 2004