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Form 10-K
March 12, 2004

UNITED STATES SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 10-K

ANNUAL REPORT PURSUANT TO SECTION 13 OR 15(d) OF
THE SECURITIES EXCHANGE ACT OF 1934

FOR THE FISCAL YEAR ENDED DECEMBER 31, 2003, OR

TRANSITION REPORT PURSUANT TO SECTION 13 OR 15(d)
OF THE SECURITIES EXCHANGE ACT OF 1934

FOR THE TRANSITION PERIOD FROM _____ TO _____

Commission file number 001-16043

ALTEON INC.

(Exact name of registrant as specified in its charter)

DELAWARE

13-3304550

(State or other jurisdiction of
incorporation or organization)

(I.R.S. Employer Identification No.)

6 CAMPUS DRIVE, PARSIPPANY, NEW JERSEY 07054

(Address of principal executive offices)
(Zip Code)

(201) 934-5000

(Registrant's telephone number, including area code)

Securities registered pursuant to Section 12(b) of the Act:

Title of Each Class -----	Name of Each Exchange On Which Registered -----
Common Stock, Par Value \$.01 per share	American Stock Exchange

Securities registered pursuant to Section 12(g) of the Act:

NONE

Indicate by check mark whether the registrant (1) has filed all reports required to be filed by Section 13 or 15(d) of the Securities Exchange Act of 1934 during the preceding 12 months (or for such shorter period that the registrant was required to file such reports), and (2) has been subject to such filing requirements for the past 90 days.

Yes No

Indicate by check mark if disclosure of delinquent filers pursuant to Item 405 of Regulation S-K is not contained herein, and will not be contained, to the best of registrant's knowledge, in definitive proxy or information statements

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incorporated by reference in Part III of this Form 10-K or any amendment to this Form 10-K. [X]

Indicate by check mark whether the registrant is an accelerated filer (as defined by Rule 12b-2 of the Act). Yes [X] No []

The aggregate market value of the voting and non-voting common equity held by non-affiliates of the registrant, based on the American Stock Exchange closing price of the common stock (\$4.85 per share), as of June 30, 2003, was \$173,745,192

At March 5, 2004, 40,472,898 shares of the registrant's common stock, par value \$.01 per share, were outstanding.

PART I

ITEM 1. BUSINESS.

OVERVIEW

We are a product-based biopharmaceutical company engaged in the discovery and development of small molecule drugs to reverse or slow down diseases of aging and complications of diabetes. Our product candidates represent novel approaches to some of the largest pharmaceutical markets. Our lead compound is in clinical development; several others are in earlier development stages. These pharmaceutical candidates were developed as a result of our research on the Advanced Glycation End-Products ("A.G.E.") pathway, a fundamental pathological process and inevitable consequence of aging that causes or contributes to many medical disorders, including cardiovascular, kidney and eye diseases.

A.G.E.s are glucose/protein complexes that form as a result of circulating blood glucose reacting with proteins. These A.G.E. complexes subsequently interact and bond (crosslink) with other proteins, resulting in "hardened" (stiffened) arteries, toughened tissues and impaired flexibility and function of many body organs. In healthy individuals, this pathological A.G.E.-formation process occurs slowly as the body ages. In diabetic patients, the rate of A.G.E. accumulation and the extent of protein crosslinking are accelerated because of high glucose levels.

Our research and drug development activities targeting the A.G.E. pathway have taken three directions: the breaking of A.G.E. crosslinks between proteins in order to reverse damage ("A.G.E. Crosslink Breakers"); the prevention or inhibition of A.G.E. formation ("A.G.E.-Formation Inhibitors") and the reduction of the A.G.E. burden through a novel class of anti-hyperglycemic agents, Glucose Lowering Agents ("GLA"). We believe that we were the first company to focus on the development of compounds to treat diseases caused by A.G.E. formation and crosslinking. Since our inception, we have created an extensive library of novel compounds targeting the A.G.E. pathway, and have actively pursued patent protection for these discoveries.

The primary focus of our research and development activities is alagebrium chloride (formerly ALT-711), which is our lead product candidate and we believe the only A.G.E. Crosslink Breaker in advanced clinical development. In February, the United States Adopted Name (USAN) Council approved alagebrium chloride as the generic name of the chemical compound formerly known as ALT-711. Alagebrium offers the possibility of the first therapeutic approach to "breaking" A.G.E. crosslinks, the benefit of which may be to reverse tissue damage caused by aging and diabetes, thereby restoring flexibility and function to tissues, blood vessels and organs of the body. Alagebrium has demonstrated

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safety and efficacy in three Phase 2 trials and several Phase 1 studies in which over 800 patients received alagebrium in clinical trials. We are actively developing the compound for the treatment of cardiovascular diseases including systolic hypertension and heart failure. In July 2003, we announced initial results from the Phase 2b SAPPHIRE (Systolic And Pulse Pressure Hemodynamic Improvement by Restoring Elasticity) and SILVER (Systolic Hypertension Interaction with Left VEntricular Remodeling) trial that focused on patients with systolic hypertension. Alagebrium was safe and well tolerated at all doses tested. Results from this 768 patient, six-month, placebo-controlled, dose-ranging study showed that although the pre-specified primary endpoint of reduction of systolic blood pressure by office cuff pressure measurement did not demonstrate statistical significance, as compared to placebo, pre-specified secondary analyses of ambulatory blood pressure measurements ("ABPM") in all patients who completed the study demonstrated a blood pressure lowering effect at lower doses of approximately 4 mm Hg net of placebo. In February 2004, we announced the partial results of a post hoc analysis which showed that alagebrium treatment resulted in significant lowering of systolic blood pressures in patients with a baseline systolic ABPM of > or = 140 mm Hg, with little concurrent effect on diastolic blood pressure readings. The treatment effects were greatest in patients with higher starting systolic blood pressure readings.

The DIAMOND (Distensibility Improvement And ReMOdeliNg in Diastolic Heart Failure) open-label, single dose trial of alagebrium was conducted in 23 patients with diastolic heart failure ("DHF"). Treatment with alagebrium over 16 weeks demonstrated a statistically significant reduction in left ventricular mass and a marked improvement in left ventricular diastolic filling. The trial also showed statistically significant improvements in multiple quality of life measurements. Pre-specified primary endpoint data was not evaluable. Patients with Class III heart failure at baseline, the sickest patients in the study, appeared to benefit the most from alagebrium treatment. Side effects were as expected for a similar patient population of this size and severity. In 2001, we conducted a Phase 2a clinical trial, in which 93 patients received alagebrium or placebo tablets once daily for eight weeks. Study results showed that alagebrium patients experienced a statistically significant and clinically meaningful reduction in pulse pressure (p