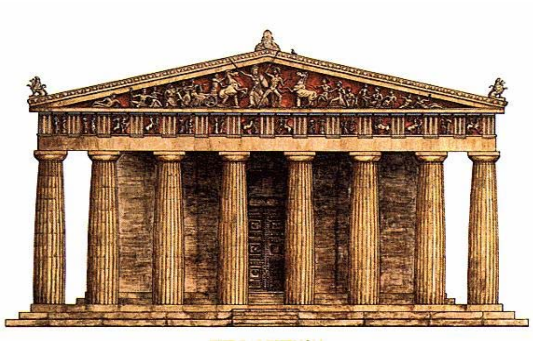

Parthenon Research

September 7, 2010

CURAXIS PHARMACEUTICAL

CURX.OB $1.30

Company Description Curaxis Pharmaceutical Corporation is a clinical-stage biotechnology company developing treatments for Alzheimer's' disease and cancer. Its lead product, Memryte or VP4896, is based on a novel theory of how Alzheimer's disease develops. Memryte is in Phase IIb clinical development for Alzheimer's disease, which is expected to begin patient enrollment in fall 2010.

- Curaxis is now a public company, trading under the symbol CURX.
- Curaxis' lead product, Memryte, is based on a novel theory of how Alzheimer's disease develops and has a unique mechanism of action. The current Alzheimer's disease patient population is estimated at 5.3 million patients in the US alone. There are no treatments that stop the disease or slow its progression, making it one of the greatest unmet medical needs today.
- Numerous drugs based on widely accepted, established theories of Alzheimer's disease have failed clinical trials. Recent discoveries have made scientists reconsider the established theories of the pathway of disease and look for new ideas. We believe Curaxis is still relatively undiscovered, and expect the academic and investor communities to begin recognizing its progress.
- Clinical authorities in the field are starting to emphasize early diagnosis and treatment, rather than just symptom management. A proposed update to the current diagnostic criteria would include new preclinical and mild stages that would greatly enlarge the market.
- We believe these developments in the field of Alzheimer's research are all positive for Curaxis.

Investment Thesis

Curaxis' has completed its reverse merger and is now a publicly traded company. It trades under the symbol CURX, previously ASCH, the symbol of the shell company.

We believe the company has secured adequate funds to complete its Phase IIb clinical trial for Memryte, its lead product for Alzheimer's disease. Unlike many reverse mergers that include a simultaneous closing of a private placement, no securities offering was made at that time. This should allow the company to raise additional capital when valuation and timing are right, without the financial pressure of running out of funds. This should also eliminate the fear of dilution for new investors.

Numerous Milestones Ahead The Company's Phase IIb trial is scheduled to begin enrolling patients with mild-to-moderate Alzheimer's disease around year end 2010/early 2011. Since the company became publicly traded, its market capitalization reflects its unknown, underappreciated status. We expect medical publications and industry developments in the Alzheimer's field to attract investment to novel, disease-modifying therapies like Curaxis' Memryte.

We believe recent events in the Alzheimer's disease field are positive for Curaxis. At a medical conference in July, Alzheimer's specialists presented research on the basic science and treatments for Alzheimer's. New studies detailed scientific advances in understanding the disease and its treatment. Presentations included studies on cost effectiveness of early diagnosis and intervention, genetics of the disease, risk factors, and a proposal for updating the diagnostic stages of Alzheimer's disease. There were several studies that we see as supportive of Curaxis Pharmaceuticals' theories and our expectations.

In August, data was published by a leading neurology journal in which a diagnostic test was able to accurately detect the disease up to 5 years before symptoms were clinically diagnosable. This breakthrough was on the front page of many newspapers and headline news on television. Curaxis' lead drug acts early in the disease process, and could potentially be used before clinical symptoms of the disease are detectable. Thus, earlier diagnosis could enlarge the patient population and market potential for the drug.

Clinical Trial Failures Leave No Frontrunners In Clinical Trials

The prevailing hypotheses about Alzheimer's are based on accumulation of two proteins in the brain, beta amyloid and tau, as causes of the disease. Preventing these proteins from forming or removing them has been the basis of drug development for over 25 years.

Two late stage clinical studies based on these ideas were recently discontinued due to lack of efficacy and high adverse events. Both drugs were based on the Beta Amyloid hypothesis. Numerous drug failures and new data have resulted in increasing doubts about the validity of the Beta Amyloid theory.

In contrast, the Curaxis drug development program is based on a relatively new theory in which hormonal changes due to normal aging are an initiating event. These changes occur earlier in the disease process than the formation of beta amyloid. The Curaxis theory is consistent with the emerging data suggesting that the disease process begins many years before clinical symptoms are detectable.

Early Intervention Is Consistent With The Treatment We Expect For Memryte

An update to the current diagnostic criteria has been proposed that would allow earlier diagnosis and treatment of Alzheimer's disease. Memryte, Curaxis' lead product, is aimed at the cause of the disease, rather than eliminating the unique structures associated with the clinical stages of disease. This mechanism could potentially be used with early stage patients to prevent progression to moderate or severe stages of the disease.

July, a study presented at the Alzheimer's research meeting that examined the cost-effectiveness of screening and early diagnosis found it to be beneficial both medically and economically. This would also support the idea of early treatment.

The Memryte Phase IIb clinical trial is scheduled to begin in late 2010/early2011. The trial is expected to enroll 200 to 250 patients who will be treated with the Memryte implant and concurrent acetylcholine inhibitor therapy. Primary endpoints are standard measures for Alzheimer's clinical trials.

Several Important Industry Developments During Summer 2010

During the summer of 2010, there were several important developments that impacted the Alzheimer's research and patient treatment. The positive news included scientific data that supports early diagnosis and treatment, which we see as enlarging the market for Alzheimer's products. A new diagnostic test was able to identify protein markers that could identify several stages of Alzheimer's and predict progression of the disease.

New scientific studies on the disease process continue to show that the two most widely accepted theories of Alzheimer's disease are at least partially incorrect. In August 2010, another two drugs based on these theories failed clinical development. Earlier this year, completion of enrollment for a widely followed drug in Phase III trials was delayed by two years to the increase number of patients. We now see no drugs on

the horizon that we would consider leading candidates to be the next successful therapy for Alzheimer's disease.

AAICAD Meeting Supports Our Expectations

The Alzheimer's Association's 2010 International Conference on Alzheimer's Disease (AAICAD 2010) was held from July 10 to 15th in Honolulu, Hawaii. This is the largest annual medical meeting for research on Alzheimer's disease. Presentations included many new studies on the basic science of the disease process, new theories, and clinical trial data. In addition to the scientific advances in defining and understanding the disease, several studies were presented that we believe have relevance to Curaxis Pharmaceuticals. Some of the presentations included:

- Data that disagrees with the older, established theories on the causes of Alzheimer's disease. In 2008, academic leaders were beginning to state publicly that the beta amyloid and tau lesions may not be the right targets for drug development. New studies have shown that amyloid formation may have beneficial and protective effects, as an antioxidant, metal chelator, and oligomer detoxifier.

- The National Institute on Aging (NIA) and the Alzheimer's Association presented a proposal to revise the diagnostic criteria for Alzheimer's. The current criteria were established in 1984 and have never been updated to include new discoveries and biomarkers. The proposal would allow earlier diagnosis of the disease and enlarge the potential patient population for Memryte.

- Additional studies covered genetic markers, risk factors, and treatment strategies. These are consistent with Curaxis hypothesis of the disease process and clinical trials.

- A study analyzing the cost effectiveness of early diagnosis and treatment showed a reduction in outpatient costs by almost 30 percent. Earlier detection in patients diagnosed with cognitive impairment and dementia affected the management of other patients' other medical conditions and lifestyle, reducing the overall cost of care as soon as the first year. These findings and recommendations are consistent with the proposed Memryte treatment plans, and could help market adoption.

Testing Spinal Fluid Could Mean Earlier Diagnosis Of Alzheimer's

In August 2010, a study published in the *Archives of Neurology* showed that testing spinal fluid for proteins associated with Alzheimer's disease can provide accurate, early diagnosis, and predict the progression of disease. This study is the first to identify a

specific protein "fingerprint" for diagnosis, as well detection of the disease process several years before diagnosis could be made by clinical symptoms. The test also showed 100 percent accuracy in identifying patients with significant memory loss (mild cognitive impairment or early stage disease) that progressed to full Alzheimer's disease five years later.

Currently, diagnosis is made by clinical symptom assessment and brain imaging with about 95% accuracy, but only after the symptoms have started. Previous research has established that the disease process may start at much as 10 years before symptoms are noticeable, and identification of proteins related to Alzheimer's in cerebrospinal fluid (CSF) had been demonstrated before. This test was able determine the ratios of proteins that could accurately diagnose the disease process by laboratory measures as well as making the diagnosis years earlier than currently possible.

Furthermore, there are other types of dementia with similar symptoms to Alzheimer's that can be misdiagnosed. The disease is not classified as "confirmed Alzheimer's disease" until autopsy. The high accuracy of the test would avoid that problem, as well as to allow laboratory measures to objectively diagnose the disease without clinical testing or clinician bias.

Study Data: The study enrolled 316 patients over the age of 70, of whom 114 had normal memory, 200 had some memory problems, and 102 had been diagnosed with Alzheimer's disease. The study measured concentrations of three proteins previously identified as potential biological indicators, or biomarkers, for Alzheimer's and mild cognitive impairment (MCI). Low levels of the amyloid protein CSF $A\beta_{1\text{-}42}$, along with high levels of total CSF tau protein and elevated CSF phosphorylated tau_{181} (P-tau_{181}), were used to identify Alzheimer's disease in the three patient groups.

The protein ratios determined to be diagnostic for the disease were present in the spinal fluid of 90% of the patients with Alzheimer's disease, 72% of patients with mild cognitive impairment, and 36% of the cognitively normal patients.

To confirm the results, 68 cases of autopsy-confirmed Alzheimer's disease were compared with the protein profile used in the diagnostic test. There was a 94% match (64 out of 68 cases) between patients with confirmed disease and the protein profile that indicated a positive test result. In another set, 57 patients diagnosed with MCI were followed for 5 years. In this group, 100% of the MCI patients went on to develop Alzheimer's dementia.

Another interesting finding was that 36% of the study patients with normal memories had CSF fluid profiles indicating Alzheimer's disease. The study authors suspect that those people have low levels of disease activity and will develop memory problems in the future.

Interpretation: This is the first study to determine a protein profile that can be used as a marker of disease. This "fingerprint" can be used for diagnosis and predicting the future course of disease for the patients.

We also believe this study is also consistent with the theory that the disease begins many years before the first symptoms appear, and that it is an active and ongoing earlier than previously documented. This suggests that many people who developing the disease can be diagnosed some time before disability accumulates and symptoms develop.

While the test is a breakthrough, it is still a research tool. Once the test is verified with additional studies, it could be used to diagnose the disease in early stage patients. There are currently no approved drugs to give such patients, so that diagnosis would have limited therapeutic value. (A separate study reported at AAICAD, described below, reported cost reductions in first year cost of care through early diagnosis and improved patient management.) It also requires a spinal tap to collect CSF for analysis, which patients avoid due to its reputation for pain and side effects.

Once a drug that can delay the progression of disease becomes available, this type of diagnostic could get widespread use for early diagnosis. Its results could factor into a decision to start therapy before symptoms start and perhaps delay the progression of disease. We believe this type of diagnostic complements Memryte's mechanism of action and could enable slowing or stopping early stage disease.

Established Theories Are Losing Supporters

For the past 25 years, there have been two widely accepted theories of how Alzheimer's disease develops. Both theories are based on two unique proteins found in the brains of Alzheimer's patients. The two proteins, beta amyloid and tau, have been believed to have a cause-effect relationship with the disease. Most drug development has involved removing or preventing formation of these proteins as a way to stop the disease.

The Beta Amyloid Theory is based on findings that beta amyloid is toxic to brain neurons, and is a major contributor to cell death and dementia in Alzheimer's disease. The Tau theory is based on internal chemistry changes within the brain cells causing changes to the tau protein, which then result in cell death. Despite extensive research, the exact cause and details of the process that leads to disease has never been fully determined.

Many drugs have been developed based on these theories and every one has failed clinical trials. Recent studies have produced data that undermines the theorized roles of beta amyloid and tau proteins. Drugs developed against beta amyloid have success-

fully bound their targets and removed amyloid, yet the patients still develop dementia and do not benefit. Recently, studies have shown little to no correlation between the amount of beta amyloid and the severity of an Alzheimer's patient's symptoms.

Other studies have shown that beta amyloid may be part of the brain's immune defense system with an anti-infective function, as well as an antioxidant. New data has shown that it has beneficial effects as a metal chelator (binder), and oligomer detoxifier. These new data has caused a reevaluation of these theories, and an acknowledgement that these proteins may not be the right target for intervention.

Curaxis' Theory The Curaxis drug development theory ties together the normal aging process, hormone levels in the brain, and pathological responses seen in Alzheimer's disease and cancer. These ideas were developed by the Company and external scientists within the past ten years.

The theory attributes the start of the Alzheimer's disease process to an age-related dysregulation of hormones in the brain, leading to several processes that start the Alzheimer's disease pathway. These processes include inappropriate cell division, beta amyloid formation, neurofibrillary tangle formation, oxidative stress, and inflammation.

The theory was developed after data showed elevated hormone levels in the brain could lead to inappropriate division, followed by cell death. The data support the idea that this may be the initiating factor that leads to other processes in the disease pathway. Importantly, this theory does not contradict established mechanisms but could explain how other theories fit together to cause Alzheimer's symptoms and clinical findings. For a more detailed discussion of the mechanisms of Alzheimer's disease and Memryte's mechanism of action, please see our Curaxis Company Report dated May 11, 2010.

Lilly Drops Its First Shoe: In August 2010, Lilly stopped development of semagacestat, an experimental Alzheimer's disease drug. The action came after an interim evaluation of two late-stage clinical trials in which patients on the drug were deteriorating faster than the placebo patients and had a higher risk of skin cancer. Semagacestat was one of two drugs in Lilly's pipeline in late-stage studies for Alzheimer's disease.

- Semagacestat was a gamma secretase inhibitor. It method of action was to block the action of gamma secretase, an enzyme that processes amyloid precursor protein (APP). This is a key step in the formation of beta amyloid protein and beta amyloid plaques on the brain.

- Lilly's second drug, solanezumab, is a humanized monoclonal antibody directed against beta amyloid. Solanezumab is intended to remove plaques after they have formed. The solanezumab Phase III trials are continuing.

Both drugs were designed to prevent the accumulation of beta amyloid and thus stop the pathological process. Semagacestat is the latest addition to in a long list of failed drugs that have targeted beta amyloid by removing it or preventing its formation.

Clinical Trial Findings: The two phase III clinical trials of semagacestat enrolled more than 2,600 patients with mild to moderate Alzheimer's, randomizing them to treatment with semagacestat or placebo. The interim analysis found that semagacestat-treated patients were actually performing worse on tests of cognition and ability to complete daily living tasks than the control group treated with placebo. In addition, semagacestat patients were at a higher risk for developing skin cancer.

The drugs in the gamma secretase inhibitor category have all failed clinical trials. In March 2008, Myriad Genetics reported that its Phase III trial with its gamma secretase inhibitor, Flurizan (tarenflurbil), showed that the drug was able to bind to its target to successfully block amyloid formation. Yet the patients still showed progression of disease and developed dementia.

Furthermore, patients in Flurizan trial showed high rates of brain infections such as encephalitis (an inflammation of the brain). In March 2010, new research published in Public Library of Science demonstrated that beta amyloid was genetically similar to a protein that is part of the brain's innate immune system. When tested for immune activity, beta amyloid was able to protect against 8 out of 12 pathogens that cause brain infections.

Earlier in 2010, Johnson & Johnson delayed the completion of its Phase III study with bapinezumab in order to enlarge its patient enrollment. Bapinezumab is a monoclonal antibody intended to remove beta amyloid. We believe the Phase II data presented in July 2008 showed little to no effect of the drug, and that the enrollment is being increased to help show statistical difference for a lower-than-expected benefit.

Interpretation: The failure of another gamma secretase inhibitor with high rates of side effects undermines the strategy of preventing beta amyloid formation and supports the theory that beta amyloid may have a beneficial function. We expect Lilly's solanezumab to fail clinical trials as well. If J&J's bapinezumab trial is ever completed, we expect it to fail to achieve its endpoints.

These trials add to the growing consensus that beta amyloid and tau protein are not the right targets for Alzheimer's drug therapy. We expect the Alzheimer's research community to be increasingly receptive to new drugs based on novel theories, such as Curaxis' Memryte.

Early Alzheimer's Diagnosis May Reduce Cost of Care for Patients

A study presented at the AAICAD found that additional screening for earlier diagnosis of Alzheimer's disease could give a 29% reduction in the cost of care during the first year after diagnosis. The study, known as the Dementia Demonstration Project (DDP), was conducted by the Minneapolis Veterans (VA) Medical Center at seven sites with the intention of increasing diagnosis of dementia in primary care. After diagnosis, the DPP provided guidance, support, and care coordination for newly diagnosed dementia patients.

A significant point about the study is that it reduced costs in the first year. The initial up-front expense for the testing is the main obstacle to performing the tests. The study data show the program is cost-effective, and had a meaningful cost reduction in the first year.

The study attributed cost savings to reduced hospitalizations and visits to urgent-care centers. Patients are typically diagnosed by clinical symptoms that occur after the disease has affected their ability to function. Many miss doses of medications for chronic conditions like diabetes, or have accidents leading to costly hospitalizations. By diagnosing the disease early, patients' families and caregivers were taught how to care for the condition. The data showed that patients visit medical facilities less frequently, and that the reduced costs may be related to having a caregiver help the patient better manage chronic health conditions.

We believe this study shows the importance of early diagnosis in formulating a cost effective treatment plan for Alzheimer's patients. Early diagnosis and treatment would fit well with the range of disease Curaxis is planning its first indications for Memryte. The cost effectiveness studies are additional support for the benefit of diagnosis for the patients' outcome and for the health care system. The cost of caring for an Alzheimer's patient is borne by the families, Medicare and Medicaid, and private insurers – all of whom would benefit from better patient management.

Updated Alzheimer's Disease Classifications Presented at AAICAD

The current criteria for diagnosing Alzheimer's is through memory tests, clinical symptoms of progressing dementia, and inability to carry out day-to-day activities like

dressing or bathing. The diagnosis is confirmed by autopsy findings of amyloid plaques and neurofibrillary tangles in the brain. These diagnostic criteria have not been changed since 1984.

The National Institute on Aging (NIA) and the Alzheimer's Association convened panels of Alzheimer's specialists to recommend changes the current criteria. Work began about a year ago, and the first draft was presented at AAICAD.

The recommendations were intended to include advances in scientific knowledge and diagnostic technologies. The new updated criteria reflect evidence that the disease process begins many years before the symptoms of dementia. The clinical symptoms of dementia that determine diagnosis today are now considered a late stage in the disease process.

The version presented at the AAICAD meeting includes two new stages of the disease and includes the use of biomarkers. Classifications would be preclinical AD, mild cognitive impairment, and Alzheimer's disease dementia. These new classifications are intended to allow for early detection and treatment.

The NIA/Alzheimer's Association divided the disease into three stages:

- **Preclinical:** The new classifications are designed to include assessments that can identify and predict risk for developing the disease. Classification could be made by biomarkers and other clinical assessment tools to identify early cognitive decline. This would establish the presence of Alzheimer's brain changes in people with no observable symptoms and to identify those who may eventually develop the disease.

- **Mild cognitive impairment (MCI):** New criteria will help to indicate cognitive change before dementia and better differentiate MCI from full Alzheimer's.

- **Alzheimer's dementia:** Revisions to the existing criteria for diagnosing Alzheimer's many include possible biomarkers and other assessments that may aid in diagnosis.

The use of biomarkers shifts the focus to the disease process itself rather than its effects and symptoms. Diagnoses will aim to identify the disease as it is developing by using results from biomarkers like genetic analysis, PET Scans (positron emission tomogramphy), MRI (magnetic resonance imaging) scans, and spinal taps that detect changes in the brain.

The new NINDS/ADRDA diagnostic criteria would include biomarkers for Alzheimer's. These fall into several categories:

- Biomarkers of beta amyloid pathology, including amyloid PET imaging and levels of beta amyloid in cerebrospinal fluid (CSF).
- Biomarkers of neuronal injury, including levels of CSF tau and phosphorylated tau.
- Biomarkers of neuronal dysfunction, including decreased uptake of FDG (a tracer used to measure activity) on PET scans.
- Biomarkers of neurodegeneration, including brain atrophy on structural MRI scans.
- The e4 allele of the APOE gene, a well accepted major genetic risk factor for late onset Alzheimer's disease, defined as onset at 65 or older.

The guidelines may be adopted as early as fall 2010. At this time the population of Alzheimer's patients is estimated at 5.3 million in the US alone. We would expect these patients to be classified as having Alzheimer's dementia. The Mild Cognitive Impairment and Preclinical Disease categories are likely to include many people who are not currently considered Alzheimer's patients, which could greatly enlarge the patient population.

Conclusion: Developments Support Curaxis and May Enlarge The Market

The scientific developments in favor of early diagnosis and treatment are positive for Curaxis. The new diagnostics and proposed classifications would greatly increase the number of patients diagnosed.

Curaxis has a developed novel theory and first-in-class therapeutic. Memryte's mechanism of action is early in the disease process, which could potentially be used for early treatment to prevent the progression of the disease. Now that clinical trial failures have nearly exhausted the pipeline of late-stage drugs based on Beta Amyloid and Tau Protein theories, we believe the field is open to new theories and drugs based upon them.

We believe the numerous failures in Alzheimer's have made investors avoid the entire field. As a result, the companies in the field reflect high discount rates and little technology value relative to their fair value. This represents an opportunity to build positions at low prices while the stocks are out of favor.

Company Contacts

Mr. Patrick Smith

Chief Executive Officer

Mr. David Corcoran

Chief Financial Officer and

Executive Vice President

Curaxis Pharmaceutical Corporation

4819 Emperor Boulevard, Suite 400
Durham, NC 27703
Toll Free: 1-888-919-CURE (2873)

www.curaxispharma.com

Risk Factors

Risks Inherent to the Drug Development Business in General – Drug development is based upon scientific and technological discovery. It is experimental by nature and has a high failure rate relative to other business ventures. Products depend on successful medical theories, clinical development, regulatory approval, and commercial acceptance. It is a highly competitive business, which can be affected by scientific or clinical discoveries that could make products obsolete or less commercially viable. Products can fail at any point in the development cycle.

Curaxis Pharmaceutical Corporation may face challenges with competition from larger and better capitalized firms, potential for increasing financing costs, government regulation, ability to retain key personnel, technological change, and changes in the marketplace for its products.

Dependence on Other Companies – Curaxis depends on third party vendors for its supply of raw drug and manufacturing the finished product. These vendors are subject to business risks outside the control of the company, and may not perform as expected. Conflicts, problems, or need to replace the clinical research organization or manufacturers could adversely affect the company.

Intellectual Property — Curaxis depends on its patented technologies and those of its third party vendors. At this time, the company holds a method of use patent for its lead product, and relies on trade secrets, industry know-how, and confidentiality agreements. The company's intellectual property protection may be circumvented by other companies or challenged, resulting in a loss of uniqueness.

Patents in the drug development business involve complex legal and scientific issues, and litigation could affect company resources and future prospects. Litigation has produced conflicting verdicts with regard to patent validity and enforceability, with no reliable precedents. Patent protection also varies widely between countries. The ability to enforce patent rights is a risk inherent to the drug development business.

Government Regulation — In the US, drug development is regulated by the US Food and Drug Administration (FDA). FDA regulations are subject to change while the

company is developing its products, which could adversely affect the Company's programs. If the company does not receive regulatory approvals, it will not be able to commercialize its products. Foreign countries have their own requirements and government agencies, which may or may not approve drugs for commercialization.

Products for the Alzheimer's target market in the US could be subject to reimbursement payments by Medicare, a government program run by the Centers for Medicare and Medicaid (part of the Department of Health and Human Services). Medicare reimbursement policies for medical products are subject to change, and market acceptance could be affected.

Advancing drugs through stages of development depends on approval of the FDA, institutional review boards, and other patient advocacy committees. The outcome of such approvals cannot be predicted in advance. Requirements may be subject to changes that may make the Company's products and services less competitive or less viable in the target markets. The company's novel theory and drug development plans may not receive the anticipated level of acceptance, which could slow development or affect commercial success.

Uncertain Profitability – Curaxis is a development stage company with no marketable products. It expects to have financial losses and insignificant revenues until products reach the market. Before it can market its products, it must run clinical trials to prove efficacy and safety then submit its data to the US Food and Drug Administration for approval. This process carries scientific, technical, and regulatory risks. Drug development is a high-risk business and products can fail at any point. It the products do not have sufficient efficacy and safety to be sold in the US, the Company may be able to achieve any product revenues from them. Profitability would depend upon successful clinical development and commercialization, neither of which can be assured.

Key Personnel – The Company may lose certain key members of its management team or production staff, which could affect the ability to deliver its goods and services.

Need To Raise Capital – Curaxis Pharmaceutical Corporation is a growing company that may need to raise capital to fund its strategic plan or operations. The ability to raise new funds is highly dependent on the capital markets, over which the company has no control. The company may choose to finance its operations with the sale of equity, debt, licensing product rights, or corporate collaborations. There can be no assurance that, if necessary, the company will be able to raise additional capital. Investors may encounter dilution if the company raises funds, and if the company needs to raise new capital and cannot do so, operations may be affected.

Management Ownership - An estimated 25.6% of the common stock is held by officers and directors. This may or may not be in the best interests of all shareholders.

Small Capitalization Market – At this time, Curaxis stock has just begun trading on the Over The Counter bulletin board market. Volatility may be high, trading volume could be irregular, and liquidity may be limited. There can be no assurance that the public market for the shares will develop.

For a full description of the risks and uncertainties related to an investment in this company, please refer to the company's filings with the Securities and Exchange Commission.

Disclaimer This report has been prepared by Parthenon Associates with information and public documents from Curaxis Pharmaceutical Corporation. Parthenon has not independently verified all of the information provided, and does not purport to be a complete statement or summary of the available data.
Some of the information in this report relates to future events of future business and financial performance. Such statements constitute forward-looking information within the meaning of the Private Securities Litigation Act of 1995. Such statements can only be predictions and actual events or results may differ from those discussed. This report is published solely for information purposes and is not to be construed as an offer to sell or the solicitation of an offer to buy any security in any state. Past performance does not guarantee future performance.

Analyst Certification

I, Robert LeBoyer, certify that all views expressed in this report accurately reflect my personal views about the Company. The views expressed in this report are my own. Parthenon Associates has been compensated for producing this report.

The views expressed reflect the views of the author only through the end of the period of the report as stated on the cover and so not necessarily represent the view of Parthenon Associates or any other person affiliated with the firm. Any such views are subject to change at any time based on market conditions, new scientific developments by the company or others, developments at the company or its competitors and Parthenon Associates disclaims any responsibility to update such views. These views may not be relied upon as investment advice and are provided for information purposes only. Any investment decisions should be made in consideration of the investor's time frames, objectives, risk tolerance, and ability to sustain loss of investment.

Investors should make their own determinations whether an investment in any particular security is consistent with their investment objectives, risk tolerance, financial situation, and evaluation of the investment opinion. Parthenon is not recommending any investments in this presentation. This report is provided for educational purposes only.