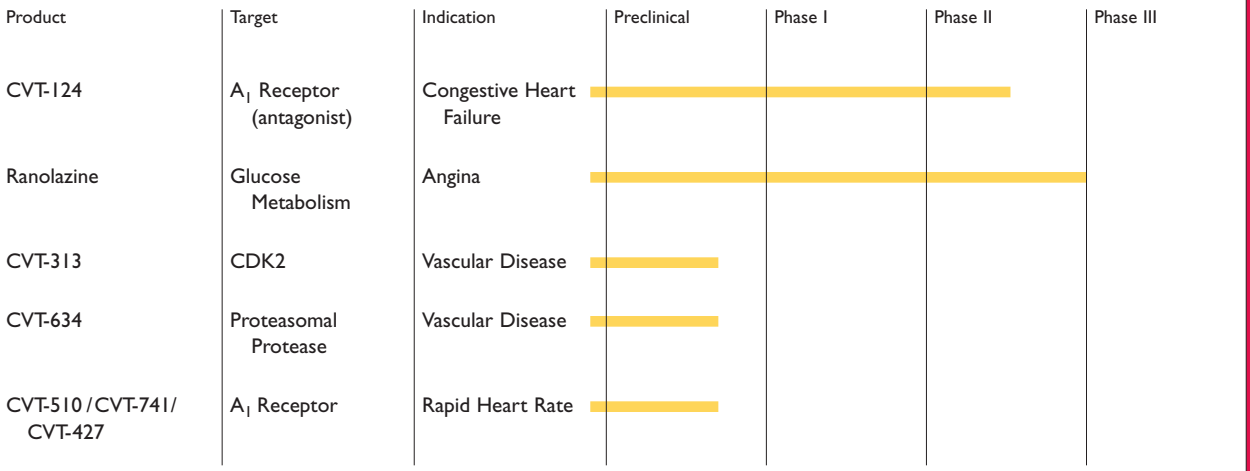
1996 ANNUAL REPORT

CV THERAPEUTICS

Developing treatments for chronic cardiovascular disease

Product Pipeline



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The Company

CV Therapeutics is engaged in the discovery and development of novel, small molecule drugs for the treatment of chronic cardiovascular diseases. The Company is pioneering a new biomedical discipline, molecular cardiology, which applies advances in molecular biology and genetics to identify and delineate new mechanisms of cardiovascular disease and hence new targets for drug discovery.

Building on the experience and expertise of the Company's Scientific Advisory Board and scientific staff, the Company is using molecular modeling, combinatorial chemistry and advanced assay techniques to build a library of new, highly targeted chemical compounds that are designed to modify these newly discovered mechanisms of cardiovascular disease. The Company's two late stage drug candidates, CVT-124 and ranolazine, illustrate this approach and are designed to modify molecular functions that are implicated in congestive heart failure-related edema and angina, respectively.

The following annual report – our first as a public company – is intended to give you an overview of both CVT's business strategy and our scientific/product development programs. We are very proud of the progress the Company has made in the past and continues to make today, and we are delighted to share that pride with our new shareholders. We hope you will take a moment to read more about CVT, and we welcome any questions or comments you may have about the Company.

To Our Shareholders

Growing Momentum on All Fronts in 1997

I am happy to report the progress CVT has made over the last year and especially pleased that substantial progress has been made since our Initial Public Offering in November 1996.

1996 was a year marked by:

- *Moving our lead program (CVT-124) forward in the clinic and showing proof of mechanism in a Phase I study;*
- *Acquiring the rights to an exciting late stage angina drug, ranolazine, that could provide a new mechanism of therapy for patients suffering from angina;*
- *Identifying several preclinical candidates from our internal research, one of which will be targeted for an IND in the next twelve months;*
- *Completing our Initial Public Offering, generating approximately $13 million in net proceeds.*

In March of this year, we signed an agreement to collaborate with Biogen, Inc. on the development of CVT-124, our novel therapeutic for the treatment of Congestive Heart Failure (CHF). CVT received $16 million upon closing, and Biogen received exclusive worldwide rights to develop and sell CVT-124. In addition to the upfront payments, CVT may receive significant milestone payments on clinical progress, significant royalties on product sales and access to a credit facility. CVT will continue to participate in developing the drug, but Biogen will pay all costs of commercializing the product. Biogen is an excellent collaborator for us because they have successfully developed and recently marketed another high value added product, AVONEX®, and now they are committing significant resources to accelerate as rapidly as possible the commercialization of CVT-124.

In the year ahead, we intend to make significant progress on the clinical front. In partnership with Biogen, we have begun a Phase II trial with CVT-124, our adenosine A_1 receptor antagonist for CHF. We will also begin the first of our planned multi-center Phase III trials of ranolazine later in the year. Our preclinical compounds will move forward toward our goal of filing an Investigational New Drug (IND) application for one of our promising candidates.

This is CVT's first annual report as a public company, and I would like to take this opportunity to elaborate on the significance of our mission to become a leader in the development of novel, cost-effective treatments for chronic cardiovascular diseases by leveraging our expertise in molecular cardiology. This rapidly emerging field has enhanced the search for innovative cardiovascular drugs by providing an increasing number of new molecular targets for drug discovery and yielded insights into the molecular mechanisms of cardiovascular disease. CVT's drug candidates are based on these insights. Our products represent a new generation of cardiovascular therapeutics with the potential for better treatment and possibly prevention of disease.

We should always be mindful of the significant numbers of patients suffering from cardiovascular diseases such as CHF and angina. Approximately 4.7 million people in the United States suffer from CHF, with an estimated 400,000 new diagnoses each year. These patients typically seek medical help in part because of edema, the accumulation of fluid in the lungs and extremities. Older therapies to treat this condition can have limited therapeutic impact, and an estimated 25% of the 875,000 CHF patients hospitalized in the United States each year are resistant to or even harmed by current treatments. There are also approximately 6.7 million patients currently diagnosed and suffering from angina. It is estimated that over half of these patients are currently being treated with multiple medications without adequate responses. We believe that such underserved patients represent the initial but not the only market for our drug candidates.

Although our products under development serve large patient populations, the scope of our planned clinical trials is not correspondingly large. This is because the cardiovascular conditions we are currently targeting are severe, clear-cut, widely suffered and without adequate therapy. Accordingly, we believe that therapeutic efficacy can be shown statistically in fewer numbers of patients and in shorter time frames than many other clinical trials that have been recently publicized.

In closing, it is appropriate to comment on our philosophy of implementing our mission of leveraging molecular cardiology to target new mechanisms of clinical significance in large markets. Our approach is informed by a strong scientific base and by our clinical experience as well as a focused adherence to short term productivity within the broader vision of molecular cardiology. We will continue to establish proof of new, mechanistically based small molecule compounds in clinical trials, but we have found no way to defy the laws of drug development success/failure rates. We believe that our shareholders are best served by aggressively pursuing these innovative drug development candidates, while actively managing risk to avoid concentrating the company's assets on a single product or approach. Strategic advantages for us include a diversified pipeline, partnerships with proven drug developers that will focus on our compounds, rapid incorporation of new technologies, and a cash position to allow meaningful investment in our programs that have large upside potential.

Thank you for your support of CVT. We look forward to reporting our progress in the year ahead.

Louis G. Lange, M.D., Ph.D.
Chairman of the Board and Chief Executive Officer

April 21, 1997


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ROB
RACHEL ENGL
MICHELLE VON ROEDELBRONN
CATHERINE FERANDIN
KATH
DORIS PON
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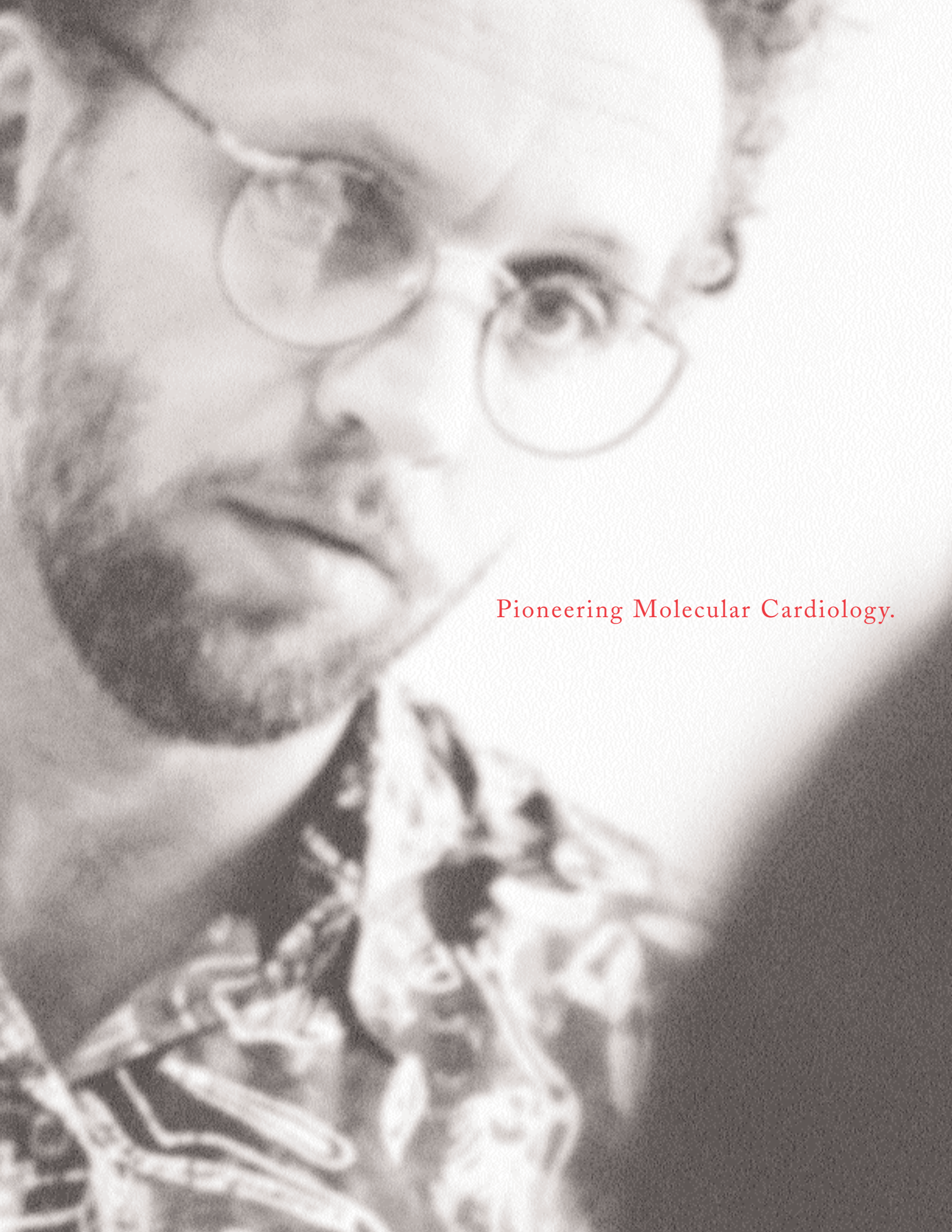


Pioneering Molecular Cardiology.

CVT-124

Targeting Cardiovascular-Related Edema at its Source

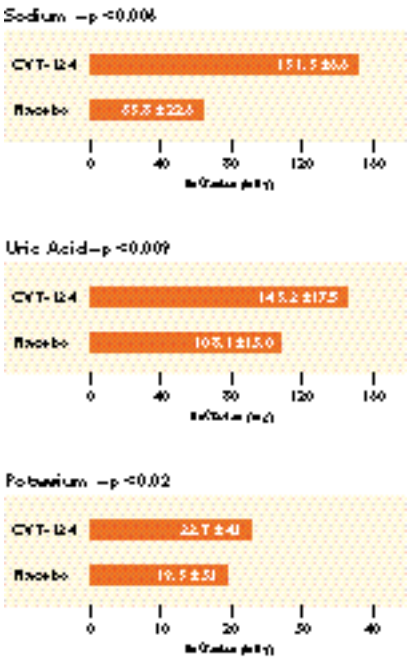
CONTROL *of Fluid Retention…*

Almost all patients suffering from congestive heart failure also experience edema, an accumulation of fluid in the lungs and extremities. As the heart's pumping function weakens, the kidneys respond to the slowing blood flow by increasing retention of salt and water to maintain blood pressure. The resulting build-up leads to lung congestion, decreased oxygen to the heart causing a further decline in pump function and, finally, death. To help prevent this dangerous spiral of events, CVT-124 has been designed to interfere with the chemical signals telling the so-called proximal tubular kidney cells to reabsorb excess salt and water.

…Without Problematic Side Effects

Because existing edema drugs do not act on the proximal tubular kidney cells that are most responsible for this excess reabsorption, they cannot block all of the problematic fluid retention. In an attempt to continue to achieve the desired diuretic effect, dosages of these drugs must be increased, sometimes causing toxic side effects, including excessive retention of uric acid (which can cause gout) and over-excretion of potassium, an electrolyte critical to maintaining the heart's normal rhythm. Data from a Phase I clinical trial have shown that CVT-124 avoids these drawbacks. It selectively binds to those particular kidney cells that carry out most of the salt/fluid reabsorption – mitigating the disease state at its cellular source – and it causes no retention of uric acid or meaningful loss of potassium.

Promising Clinical Results



Unlike existing edema drugs, CVT-124 promotes desired excretion of sodium and uric acid without dangerously depleting potassium.

Biogen Partnership: Maximizing the Potential of CVT-124

Our development partnership with Biogen is an important step toward maximizing the therapeutic and market potential of CVT-124. Biogen's experience in completing clinical trials and executing a successful market launch of its AVONEX® product will be valuable as CVT-124 moves toward approval for treating edema. As the next most clinically advanced product in the Biogen pipeline after AVONEX®, CVT-124 will receive a high degree of support and focus as it moves steadily through its pivotal clinical trials.

CVT-124	Development Status
	Phase II

Ranolazine	Development Status
	Phase II/III

Ranolazine

A New Therapeutic Approach to Angina

DECREASING *the Heart's Need for Oxygen*

Angina is chest pain caused by blocked coronary arteries unable to supply sufficient blood flow – and oxygen – to the heart. Because the heart is a muscle, anginal pain often occurs during exertion when there is an increase in energy demand by the muscle tissue. Normally, the primary source of this energy is the fatty acids in the heart. To release the energy from the fatty acids, the heart literally "burns" them – it breaks them down by combining them with oxygen. The Company's novel, small molecule, ranolazine, modifies the cellular mechanism governing this energy release, shifting the energy source in part from fatty acid to glucose. Because glucose requires less oxygen to produce an equal amount of energy, ranolazine reduces the heart's oxygen requirements, so that attacks of angina are less likely during exertion.

Angina Treatment/Better Side Effect Profile

Current drug treatments for angina act either by reducing the heart's work load through slowing the rate at which it beats, by reducing the force of the muscle's pump action or by widening the arteries to lower blood pressure (see table, right). These modes of action make these drugs inappropriate for patients whose pump function or blood pressure is already compromised by their heart condition. Other side effects of current therapies include depression, impotence and headaches. Compounding the problem, many patients take more than one medication to control their angina. Ranolazine, with its unique mechanism of action, is designed to provide effective relief from angina without these unacceptable side effects.

Drawbacks of Current Angina Treatments

DRUG	MECHANISM	LOWERS HEART RATE	LOWERS BLOOD PRESSURE
Nitrates	Dilate Arteries and Veins	No	Yes
Beta Blockers	Reduce Pump Function	Yes	Yes
Calcium Channel Blockers	Dilate Arteries/ Reduce Pump Function	Yes	Yes
Ranolazine	Modifies O_2 Metabolism	**NO**	**NO**

Ranolazine's unique mode of action prevents angina without dangerous declines in heart pumping function, heart rate or blood pressure.

Significant Market Need

Approximately 6.7 million people in the United States suffer from angina, accounting for $3.0 billion in annual sales of anti-anginal drugs. It is estimated that 20-25% of these patients also have congestive heart failure – which makes them ill-suited for many current angina therapies – or are resistant to the available treatments. Ranolazine's mechanism of action and improved side effect profile in Phase II clinical trials make it a promising therapy for these 20-25% of angina patients. We will begin the first of our planned multi-center Phase III trials of ranolazine later in the year.

Preclinical Products

Leveraging Our Expertise in Molecular Cardiology

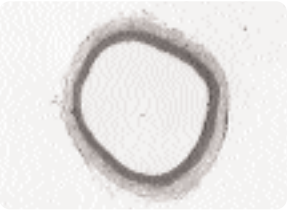
ADENOSINE *Receptor Program*

The hormone adenosine is involved in a wide array of cellular functions throughout the body. Its mechanism of action is governed by specialized receptors on the surfaces of cells. The specific structure of the adenosine receptor varies among different tissue types. In our adenosine program, the Company has developed a series of promising preclinical compounds which are designed to slow the conduction of electrical impulses in the heart by stimulating an adenosine receptor found on certain heart cells. We are testing ways to use these compounds to treat rapid heartbeat conditions, such as atrial fibrillation and other arrhythmias. These conditions are the primary reason for more than 300,000 hospital admissions in the U.S. each year, and complicate the course of several hundred thousand more.

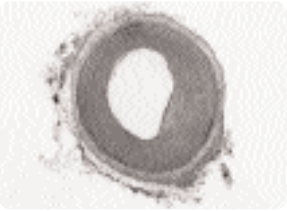
CELL CYCLE *Program*

A second area in which we are applying our deep understanding of the molecular biology and genetics of cardiovascular disease is the intracellular "clock" that controls cell division. Abnormal proliferation of connective tissue and vascular smooth muscle cells is a problem in many cardiovascular diseases, causing arterial blockages, scarring and loss of normal organ function. One of our preclinical compounds, CVT-313, is designed to inhibit the cell cycle regulatory protein CDK2. Another compound, CVT-634, is designed to inhibit proteasomal protease, another regulatory enzyme. We are now assessing the potential of CVT-313 and CVT-634 as treatments for restenosis (or recurrent obstruction) after angioplasty procedures to clear blocked arteries and other vascular injury disease states.

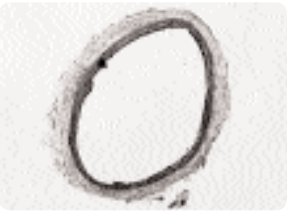
CVT-313 Effective in Preclinical Model of Restenosis



Healthy Rat Cortoid Artery *(no injury)*



Injured Rat Cortoid Artery with No Treatment
Showing thickening of arterial wall and restricted opening



Injured Rat Cortoid Artery
Treated with CVT-313
Tissue thickening is absent and opening is comparable to normal, healthy artery

CVT Research	Development Status
	Preclinical

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