



Annual Information Form

Fiscal Year Ended April 30, 2015

July 27, 2015

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Note Regarding Forward-Looking Information

All statements, other than statements of historical facts, included in this Annual Information Form regarding our strategy, future operations, financial position, future revenues, projected costs, prospects, plans and objectives of management are forward-looking statements that contain forward looking information within the meaning of Canadian securities legislation. Forward looking statements and forward looking information are referred to collectively herein as “forward looking statements”. The words “believe”, “anticipate”, “estimate”, “plan”, “expect”, “intend”, “may”, “project”, “will”, “would” and similar expressions and the negative of such expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. We cannot guarantee that we actually will achieve the plans, intentions or expectations disclosed in our forward-looking statements and you should not place undue reliance on our forward-looking statements.

Our statements of “belief” in respect of our drug candidate(s) are based primarily upon our results derived to date from our pre-clinical and clinical research and development and our research and development program. We also use the term “demonstrated” in this Annual Information Form to describe certain findings that we make arising from our research and development including any pre-clinical and clinical studies that we have conducted to date.

We believe that we have a reasonable scientific basis upon which we have made such statements of “belief” or arrived at such findings. It is not possible, however, to predict, based upon in vitro, animal and/or human studies whether a therapeutic agent will be proved to be safe and/or effective in humans and no conclusions should be drawn in that regard from what we state has been demonstrated by us to date. We cannot assure you that the particular results expected by us will occur.

Any forward-looking statements and statements of “belief” represent our estimates only as of the date of this Annual Information Form and should not be relied upon as representing our estimates as of any subsequent date. The forward-looking statements contained in this Annual Information Form include, but are not limited to, statements regarding our:

- aim to commercialize or license to a pharmaceutical partner our products for the treatment of unmet medical needs related to major adverse cardiovascular events in patients with higher risk such as acute coronary syndrome, peripheral arterial disease, diabetes mellitus and chronic kidney disease;
- aim to carry out trials on our products for the treatment of unmet medical needs related to major adverse cardiovascular events in patients with higher risk such as acute coronary syndrome, peripheral arterial disease, diabetes mellitus and chronic kidney disease, and the timing of such trials;
- plans to undertake a Phase 3 MACE-related secondary prevention study;
- expectations relating to the timing of significant clinical trial milestones;
- the function and effectiveness of RVX-208;
- the development of new compounds and the potential impact of these compounds on multiple diseases;
- aim to obtain regulatory approval for our products;
- expectations with respect to the cost of the testing and commercialization of our products;
- anticipated sources of revenue;
- expectations regarding the protection of our intellectual property;
- business strategy;
- intentions with respect to dividends; and
- potential milestone payments and royalties pursuant to the license agreement with Shenzhen Hepalink Pharmaceutical Co., Ltd.

Such forward-looking statements are based on a number of assumptions which may prove to be incorrect, including, but not limited to, assumptions about:

- general business and economic conditions;
- interest rates;

- the timing of the receipt of regulatory and governmental approvals for research and development projects;
- the availability of financing for research and development projects, or the availability of financing on reasonable terms;
- the cost of clinical trials;
- our ability to attract and retain skilled staff;
- the impact of changes in Canadian dollar-US dollar and other foreign exchange rates on our costs and results;
- market competition;
- tax benefits and tax rates; and
- ongoing relations with employees and with business partners.

Such forward-looking statements involve known and unknown risks and uncertainties, including those referred to in this Annual Information Form, which may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by such forward-looking statements. These risks include, but are not limited to:

- risks related to the early stage of our products and the Company;
- uncertainties related to clinical trials and product development;
- uncertainties relating to current economic conditions;
- rapid technological change;
- uncertainties relating to forecasts and timing of clinical trials and regulatory approval;
- competition in the market for therapeutic products to treat cardiovascular disease, neurodegenerative diseases, diabetes mellitus and other high risk vascular diseases;
- risks relating to potential product liability claims;
- availability of additional financing and access to capital for research and development, clinical trials and regulatory approval;
- market acceptance and commercialization of our products;
- the availability and supply of raw materials, including supplies of sufficient active pharmaceutical ingredients for larger clinical trials and future commercial production;
- risks relating to the effective management of our growth;
- potential reliance on partnering agreements to provide support for discovery and development efforts, and on corporate sponsors, pharmaceutical companies, universities, research groups and others to successfully develop and commercialize the Company's technology;
- the willingness of health care insurers and other organizations to pay for our products;
- risks relating to our reliance on key personnel;
- risks relating to the regulatory approval process for the manufacture and sale of non-therapeutic and human therapeutic products; and
- our ability to secure and protect our intellectual property, and to operate without infringing on the proprietary rights of others or having third parties circumvent the rights owned or licensed by the Company.

The foregoing list of important factors and assumptions is not exhaustive. Events or circumstances could cause the Company's actual results to differ materially from those estimated or projected and expressed in, or implied by, these forward-looking statements. You should also carefully consider the matters discussed under "Risk Factors" in this Annual Information Form. We undertake no obligation to update publicly or otherwise revise any forward-looking statements or the foregoing list of factors, whether as a result of new information or future events or otherwise, except as required by securities legislation.

Currency

In this Annual Information Form, unless otherwise noted, all dollar amounts are expressed in Canadian dollars.

Corporate Structure

Name and Incorporation

Resverlogix Corp. was incorporated under the ABCA on August 17, 2000 as Apsley Management Group Inc. and changed its name to Resverlogix Corp. on April 25, 2003. The Company amalgamated with Resverlogix Inc. to form the consolidated entity Resverlogix Corp. on February 7, 2005.

In connection with the spin-out of the Company's former subsidiary, RVX Therapeutics Inc., to Zenith Epigenetics Corp. pursuant to a Plan of Arrangement under the ABCA completed on June 3, 2013, the Company amended its articles to authorize the issuance of royalty preferred shares which were issued to Zenith. On July 2, 2015, the articles were amended to make certain changes to the dividend entitlement of holders of royalty preferred shares.

Our head office is located at Suite 300, 4820 Richard Road SW, Calgary, Alberta, T3E 6L1. The registered and records office is located at Suite 600, 815 - 8th Avenue S.W., Calgary, Alberta, T2P 3P2.

Inter-Corporate Relationships

The Company owns all of the voting securities of Resverlogix Inc., a corporation incorporated under the laws of the state of Delaware on July 18, 2008.

Prior to June 3, 2013, the Company owned all of the voting securities of RVX Therapeutics Inc. ("RVX Therapeutics"), a corporation incorporated under the ABCA. On June 3, 2013, the Company sold all of the voting securities of RVX Therapeutics to Zenith Epigenetics Corp. ("Zenith") as described below under "Spin Out of Epigenetics Drug Discovery Platform".

After giving effect to the foregoing transactions, the only subsidiary of the Company is Resverlogix Inc.

Description of Business

General

We are a clinical stage cardiovascular company developing small molecules that selectively inhibit Bromodomain and ExtraTerminal domain ("BET") proteins, a new and emerging target for high risk vascular diseases. Our lead drug compound, RVX-208 ("apabetalone"), targets BET proteins to impact several important biological processes that drive risk in vascular disease patients, namely: (i) therapeutic increases in apolipoprotein A-I ("ApoA-I"), a key protein in reverse cholesterol transport ("RCT"), (ii) reduced C-Reactive Protein ("CRP") and IL-6, key vascular inflammation markers, (iii) reduction of glucose and alkaline phosphatase ("ALP"), two key markers of metabolic risk, (iv) modulation of complement, coagulation and acute phase response cascades, known drivers in cardiovascular disease ("CVD") and acute cardiac events. RVX-208 is a first-in-class small molecule targeted for the treatment of atherosclerosis via the reduction of major adverse cardiovascular events ("MACE") and other chronic diseases such as diabetes mellitus ("DM"), chronic kidney disease ("CKD") and Alzheimer's disease ("AD"). RVX-208 is the first BET bromodomain inhibitor in clinical trials for high risk vascular disease.

Epigenetics

The selective production of the proteins encoded by human genes is what leads to differences between cells, and the alteration of their levels can contribute to disease. Epigenetics, a mechanism for regulating gene activity to affect protein production, has recently emerged as a promising new field in biotechnology research and drug development. It encompasses mechanisms for regulating the production of proteins from genes without altering the genetic code. In cells, DNA is surrounded by proteins to form chromatin and ultimately human chromosomes. Epigenetics is the study of secondary modifications to DNA (without affecting the sequence) or its associated proteins which alters their relative disposition, resulting in changes in gene transcription, the first step in producing the proteins that each gene encodes. Such modifications to the DNA or the proteins associated with DNA, such as histones, in turn determine whether a gene is on or off or whether its activity is high or low. Examples of these modifications include acetylation, methylation and phosphorylation. These modifications are added to histones by enzymes called "writers" and removed by enzymes called "erasers". Other proteins, called "readers", recognize a specific pattern of modifications by binding to them and recruiting additional proteins to regulate gene activity. In contrast to "writers" and "erasers" that add or remove post translational modifications to histones, "readers" detect the presence or absence of these modifications and serve as a scaffold for the transcriptional machinery in regulating gene expression. RVX-208 targets one group of reader proteins called the BET proteins. Differences in the pattern of modifications occur between different cells and organs of the body or in response to different physiological stimuli. There is now substantial evidence that alterations in these patterns underlie multiple diseases. Epigenetics

represents an important new area of drug development and is now a hallmark of several complex pathologies, including metabolic disorders, cardiovascular and neurological diseases. Epigenetic protein and enzyme molecular targets are positioned as promising targets for therapeutic intervention. Epigenetic drug research and development is designed to explore the promise of therapeutic advances in important diseases of high unmet medical need.

RVX-208 (apabetalone)

RVX-208 (apabetalone) is a first-in-class, small molecule, selective inhibitor of the BET family of proteins. Bromodomains (“BRDs”) are a family of evolutionary conserved protein-interaction modules that play key functions in chromatin organisation and regulation of gene transcription. A hallmark of many diseases such as cancer, inflammation and more recently also cardiovascular disease, is aberrant transcription. Thus, proteins that contain BRDs have been implicated in the development of a large variety of diseases. One recognised family of bromodomain containing proteins is the BET family. BET inhibition represents a novel, epigenetic approach to treat CVD. RVX-208 is the first oral agent in the BET inhibitor class that preferentially targets bromodomain 2 (“BD2”) of BET protein 4 (“BRD4”) thereby regulating gene activities. Its primary mode of action appears to result in the modulation of vascular risk pathways and markers that drive MACE in high risk CVD patients. Some of the pathways that are modulated are: the transcriptional up-regulation of apoA-I producing an increase in plasma ApoA-I protein, the reduction of ALP, the modulation of complement, coagulation and acute phase reactant cascades and also a decrease in inflammatory cytokines. Analysis of recent clinical trials data showed that RVX-208 significantly reduces MACE in patients with CVD who have a low level of HDL and elevated CRP, and other select populations with unmet medical need, especially those with diabetes mellitus co-morbidity. Select BET inhibition may also exert beneficial effects in AD and CKD. RVX-208 has anti-inflammatory effects including effects on Interleukin-6 inhibition, vascular cell adhesion-1 and monocyte chemoattractant protein-1, complement, factors known to be involved in atherosclerosis and vascular risk. These combined effects on multiple biological pathways such as RCT, vascular inflammation, vascular calcification and metabolism provide RVX-208 a highly differentiated product position for reduction of CVD risk in patients with high vascular risk burden.

The Regulatory Process for Drug Development

In the United States, it takes approximately 12 to 15 years for a typical experimental drug to go from concept to approval. The production, manufacture, research and development activities are subject to regulation for safety and efficacy by various governmental authorities around the world. In the United States, drugs and biological products are subject to regulation by the FDA. There are other comparable agencies in Canada, Europe and other parts of the world. Applicable legislation requires licensing of manufacturing and contract research facilities, carefully controlled research and testing of products, governmental review and approval of results prior to marketing therapeutic products. Additionally, adherence to good laboratory practices, good clinical practices during clinical testing and good manufacturing practices during production is required. The system of new drug approval in the United States is generally considered to be one of the most rigorous in the world. In Canada, these activities are regulated by the Food and Drugs Act and the rules and regulations promulgated thereunder, which are enforced by the TPD.

Briefly, the steps required for drug approval in the United States, Europe and Canada post-Phase 1 are similar and follow the procedures laid out by the International Conference on Harmonisation of Technical Requirements for Registration of Pharmaceuticals for Human Use (“ICH”):

Phase 2 Clinical Trials: Phase 2 clinical trials have the main objective to establish optimal treatment regimen for phase 3 studies. They take approximately one to three years to complete and are carried out on a relatively small to moderate number of patients (compared to Phase 3) suffering from the targeted condition or disease to determine the drug’s efficacy, optimal doses, treatment regimens, pharmacokinetics, pharmacodynamics and dose response relationships. This phase also provides additional safety data and serves to identify possible common short-term side effects and risks in a larger group of patients. These trials often include randomization of patients as well as a placebo or comparator arm.

Phase 3 Clinical Trials: Phase 3 clinical trials, if successful, provide the supporting clinical evidence to register a drug and make a drug available to patients. Phase 3 clinical trials in CVD typically take two to five years to complete and involve tests on a much larger population of patients (several thousand patients) suffering from the targeted condition or disease. This type of study is usually double-blind using the dose and treatment regimen determined in phase 2.

New Drug Application (“NDA”) or Marketing Authorisation Application (“MAA”): Upon completion of Phase 3 Clinical Trials, the company sponsoring the new drug then assembles all the preclinical and clinical data and submits it to the TPD and/or the FDA as part of a NDA in the United States, an MAA in Europe or a New Drug Submission (“NDS”) in Canada. The NDA, MAA or NDS is then reviewed by the regulatory body for approval to market the product. This process usually takes between one and two years to complete with the exception of evaluations of breakthrough products in only 6 months.

Resverlogix's Drug Development Strategy

Given the high cost, long development times and high attrition rates associated with drug development, many biotechnology companies seek the assistance of a pharmaceutical partner to advance their products through clinical trials. We will seek to partner or license our drug candidate at the stage that will provide our shareholders with the optimal value for their investment. Should such partnering or licensing be successful, a pharmaceutical company will provide some or all of the funding and expertise to complete the latter stages of drug development and commercialization.

Cardiovascular Disease and Residual Risk

Of all the clinical endpoints that are analyzed for providing future predictability for CVD risk, MACE is by far the most important. Health systems and payer groups look at MACE carefully when looking at potential reimbursement of a new CVD therapeutic agent. MACE includes a variety of key markers of cardiovascular risk such as worsening angina, worsening of peripheral artery pain and ischemia, prevention of percutaneous stent procedures, hospitalization for cardiac-related incidents, stroke, myocardial infarction and death. According to the 2015 American Heart Association Statistics report, based on 2011 death rate data, over 2,150 Americans die of CVD each day, an average of 1 death every 40 seconds (Mozaffarian, Benjamin et al. 2015). Many of these CVD patients will have some form of MACE during or after they have been diagnosed with CVD.

Although CVD has many current therapeutic agents that are utilized including lipid lowering drugs such as statins, heart rate lowering agents such as beta blockers, blood pressure lowering drugs such as ACE inhibitors, there still remains a large residual risk of MACE in patients that take all of these current medicines. CVD and MACE remain a major cause of mortality and morbidity in North America. According to the 2015 American Heart Association Statistics report, more than 85 million Americans have one or more vascular diseases (Mozaffarian, Benjamin et al. 2015). By the year 2030 the total projected economic burden and direct costs of CVD in the United States is estimated at \$918 billion annually (Mozaffarian, Benjamin et al. 2015). These conditions include angina, heart attack, stroke, aortic aneurysms, kidney failure and severe limb ischemia; all are contributed to by the increasing prevalence of obesity, hypertension, diabetes and dyslipidemia.

Research in the field of CVD residual risk is focusing on additional vascular risk pathways and targets in addition to atherosclerosis, the key underlying cause of coronary artery disease ("CAD") and CVD. Vascular inflammation, coagulation and vascular calcification are other important areas of CVD research that are being carefully examined in high residual risk patients.

Residual risk in high risk CVD patients represents a large market opportunity. Several pharmaceutical companies have tried to develop therapeutics that exhibit efficacy over current standard of care therapies for this critically important therapeutic segment. According to the 2012 Deutsche Bank CVD Industry Update report, the potential of this market segment was estimated to range from \$4 billion to \$90 billion USD (Parkes, Race et al. 2012). At the time of publication of the report, the lead products in the CVD pipeline, many of which failed large scale phase 3 clinical trials in recent years, included the cholesteryl ester transfer protein ("CETP") inhibitors; Torcetrapib (Pfizer) and Dalcetrapib (Roche), and other approaches such as Lp-PLA2 inhibitors; Darapladib (GSK). At present time, the proprotein convertase subtilisin/kexin type 9 ("PCSK9") inhibitor programs; Alirocumab (Sanofi/Regeneron), Evolocumab (Amgen) and Bococizumab (Pfizer) are currently targeting the CVD residual risk market.

Statin-induced reduction of low-density lipoprotein ("LDL") has been proven to be beneficial for people with CAD, stroke, and DM as demonstrated by a 10% and 21% reduction in all-cause mortality and major vascular events, respectively, following 1.0 mmol/L decrease in LDL levels. However this population remains at substantial risk for cardiovascular events. Despite adequate control of LDL, statin-treated patients with clinically evident CVD have remained at 5-year elevated risk for MACE of 45%, and as high as 69.3% overall (Barter, Gotto et al. 2007).

The risk factors for CVD are also associated with insulin resistance and DM such that patients with DM have a two to four times greater risk for death or serious cardiovascular morbidity compared to individuals without DM (Wu and Parhofer 2014). This increased risk is due to consequences of insulin resistance, where hypertriglyceridaemia leads to low levels of HDL and high levels of predominantly small, dense, LDL (Wu and Parhofer 2014). The majority (75%) of deaths in patients with DM are due to CVD. In the United Kingdom Prospective Diabetes Study, after 9 years of follow-up, fatal cardiovascular disease events were 70 times more frequent than fatal microvascular complications (Turner, Cull et al. 1996).

Unfortunately, therapeutic interventions for improved glycaemic control (including glitazones, DPP-4 inhibitors, insulin and sulphonylureas) have not been shown to reduce the risk for MACE or mortality, nor have therapies designed to raise serum HDL levels (such as niacin and the CETP inhibitors) (Maru, Koch et al. 2005; Barter, Caulfield et al. 2007; Gerstein, Miller et al. 2008; Patel, MacMahon et al. 2008; Duckworth, Abraira et al. 2009; Boden, Probstfield et al. 2011). CETP inhibition does not increase ApoA-I production but rather increases cholesterol in HDL by preventing its transport to LDL (Kingwell, Chapman et al. 2014).

Following CETP inhibition, HDL cholesterol residence time is extended making it susceptible to oxidation and modification both of which are implicated in the development of atherosclerosis. These CETP inhibition features could explain why CETP inhibition treatment is associated with CRP increase and lack of effects on atherosclerosis and MACE in human studies on top of standard of care inclusive of statins.

High residual risk of MACE, even in patients with controlled LDL, is further evident in a recent study of patients with DM receiving statin therapy. In a cohort of 5,380 patients with a history of acute coronary syndrome (“ACS”), there was no difference in incidence of MACE between alogliptin-treated and placebo-treated groups which remained high (both >11% during 18 months therapy) despite all patients receiving the standard of care for type 2 diabetes and ACS (White, Cannon et al. 2013). The findings in this recent trial add to many other preceding studies; (UKPDS, ACCORD, ADVANCE AND VADT) to further underscore the challenge that despite intensive glucose control and standard of care for lowering LDL, the incidence of MACE remains significant in the setting of DM. Additionally, revascularisation of patients with DM remains subject to increased risk of adverse outcomes. Diabetic patients are predisposed to more aggressive atherosclerosis and a higher risk for restenosis. Following percutaneous stent procedures, the incidence of MACE in DM was 27% and 19% respectively over the subsequent 5 years (Farkouh, Domanski et al. 2012). There is an urgent need for new approaches to reduce MACE in patients with CVD and especially for DM patients with CVD.

Protective Role of BET Inhibition Target Pathways and Markers

Numerous epidemiological and interventional studies have demonstrated that the pathways and biomarkers that RVX-208 modulates and improves via select BET inhibition provide biological plausibility and rationale why this novel molecule has the potential to be disruptive in the improvement of residual risk in patients. Below are several key studies that highlight the potential roles of these pathways and biomarkers that RVX-208 have been studied to date that may play a role in the early signal of MACE reduction.

The Framingham Heart Study illustrated the importance of HDL enhancement for CVD risk reduction. For every mg/dL increase in HDL, the 10-year risk of a heart attack fell by 2-3% (Gordon, Probstfield et al. 1989). The Veterans’ Affairs Cooperative Studies Program showed that men who took a lipid regulating drug for five years had a 6% increase in HDL levels, resulting in a 22% risk reduction in death due to CAD, heart attack, or stroke (Rubins, Robins et al. 1999).

The Framingham Offspring Study (FOS), illustrated that certain types of HDL particles, specifically large HDL or Alpha 1 HDL particles were even more predictive in calculating future CAD events in CVD patients (Asztalos, Cupples et al. 2004). For every mg/dl increase in Alpha1 HDL, patients in the FOS cohort had a reduction of future coronary events by 26%. It was suggested that Alpha 1 particles were significantly better predictors of risk than HDL values.

Landmark trials such as INTERHEART (2004) and AMORIS (2005) clinically validate ApoA-I as an important target for the reduction of CVD risk. The INTERHEART trial, a landmark study of 30,000 patients, demonstrated that the ratio of ApoB to ApoA-I was the strongest risk predictor of acute myocardial infarction (heart attack) (Yusuf, Hawken et al. 2004). In the AMORIS trial, more than 175,000 patients with cardiovascular risk factors were studied for the incidence of cardiac and stroke events. AMORIS clearly illustrated that reducing the ratio of ApoB to ApoA-I was associated with a dramatic reduction of stroke in this population (Walldius and Jungner 2005). The key findings of this study indicate that improvement of ‘cholesterol balance’, or the ApoB to ApoA-I ratio, is a robust and specific marker of virtually all ischemic events.

Based on multiple epidemiological and intervention studies (MRFIT, PHS, CHS/RHPP, WHS) minor CRP elevations are known to be associated with future major CVD risk (Ridker 2001). It is recommended by the American Heart Association that patients at intermediate or high risk of coronary heart disease may benefit from measurement of CRP with regard to their individual risk prediction (CRP<1 mg/L=low risk; 1-3 mg/L=intermediate risk; 3-10 mg/L=high risk). In addition, elevation of CRP is associated with increased risk of DM development (Ridker 2001). An analysis of the Women’s Health Study illustrated that CRP is a stronger predictor of future cardiovascular events than LDL (Ridker, Rifai et al. 2002).

Analysis of the CARE study and NHANES III data provided insight into the epidemiological aspect that ALP contributes to all-cause mortality and CVD mortality. Data from the CARE study participants illustrated that patients in the highest tertile of ALP levels (>99 U/L) had increased risk of coronary heart disease death, nonfatal myocardial infarction, symptomatic heart failure or stroke of 21% and an increased risk of all-cause mortality of 43%. Data from the NHANES III participants revealed that in the highest tertile of ALP levels (>87 U/L) had increased risk of all-cause and CVD mortality of 27% (Tonelli, Curhan et al. 2009). It is speculated that this epidemiological link may be a result of the role that ALP plays in the regulation of vascular calcification or that higher ALP levels represents an inflammatory state (Tonelli, Curhan et al. 2009).

Analysis of the complement, coagulation and acute phase reactant cascades illustrate their role in CVD risk. Overactivation of the complement pathway has been implicated in plaque development and destabilization (Seifert and Kazatchkine 1988; Hertle, Stehouwer et al. 2014). Complement activation also influences thrombosis through activation of platelets, promotion of fibrin formation, and impairment of fibrinolysis. Fibrin clotting is fundamental in the formation of thrombi and emboli. Fibrin and fibrinogen degradation products have been associated with CVD development and severity as well as cardiac events and death (Kannel, Wolf et al. 1987; Tataru, Heinrich et al. 1999; Zacharowski, Zacharowski et al. 2006; Wannamethee, Whincup et al. 2009). Components of the acute phase response cascade are pro-inflammatory, pro-atherogenic and markers of CVD risk (Lowenstein and Matsushita 2004).

Based on the findings from these studies, there has been considerable interest and effort within the pharmaceutical industry to identify, develop or acquire therapies that modulate the multiple risk pathways associated with the pathogenesis of CVD and MACE. With a number of pending patent applications, we believe we have broad intellectual property in the area. If shown efficacious in clinical trials, we believe that our novel small molecule, RVX-208, will be well-positioned to participate in the critically important global residual risk CVD market.

Atheroma Volume & Composition as Predictive Factors in CVD Risk

In CVD intervention trials, gathering data to assess the effects of a therapeutic on atheroma volume, also known as atheromatous plaque represents an indicator of efficacy. Imaging technology is used to make such visual assessments. Data from early imaging studies using coronary angiography and carotid ultrasound established a strong link between atheroma burden, its progression and CVD outcome. Therapies that slow the progression of atheroma burden have repeatedly been shown to reduce CVD events in randomized clinical trials. Thus, many medical institutions have set stabilization or slowing of atheroma burden progression as a goal of medical therapy.

Intravascular Ultrasound (“IVUS”) is an invasive procedure, performed along with cardiac catheterization; a miniature sound probe (transducer) on the tip of a coronary catheter is threaded through the coronary arteries and, using high-frequency sound waves, produces detailed images of the wall of the artery. Where angiography shows a two-dimensional silhouette of the interior of the coronary arteries, IVUS shows a cross-section of both the interior, and the layers of the artery wall itself. Several key markers of atheroma volume are used when using this type of technology including PAV (Percent Atheroma Volume), Total Atheroma Volume (TAV) and 10mm Worst Occluded segment (10mmTAV). Although RVX-208 did not meet its primary endpoint of PAV in ASSURE statistical significance was observed in TAV and 10mm TAV indicating potential effects on atheroma volume in high risk patients.

Advancements in atheroma dynamics and visualization now include virtual histology IVUS (“VH-IVUS”), an emerging technology that is useful for assessing tissue characteristics of an atherosclerotic plaque. VH-IVUS is analyzed to provide insight into vulnerability of an atherosclerotic plaque to rupture and its relationship to future cardiovascular risk. The ratio of necrotic core to dense calcium (“NC/DC”) details the ratio of vulnerable plaque prone to rupture (necrotic core) to the dense stabilized plaque (dense calcium) (Missel, Mintz et al. 2008). The NC/DC can be used to assess whether a therapeutic is lessening the vulnerability of the atherosclerotic plaque for rupture.

Commercial Rationale

We believe that our approach may be considered therapeutically and commercially attractive for the following reasons:

- BET proteins play a critical role in the epigenetic regulation of transcription of particular genes;
- BET proteins all contain highly conserved bromodomains that play a key role in their epigenetic control of gene expression;
- Our small molecule functions via inhibition of BET bromodomains and, therefore, specifically modulates transcription of particular targets;
- RVX-208 is highly differentiated from other therapies that focus only on increasing HDL or decreasing LDL in plasma. Our small molecule has been shown to enhance multiple pathways and biomarkers that function together to reduce CVD risk; and
- RVX-208 works via a physiological approach of activating the body’s own health-promoting genes (such as ApoA-I) to fight diseases or repressing disease-causing genes. This approach minimizes the likelihood of immunologic complications associated with peptide, recombinant or monoclonal antibody therapies currently in development.

For many of the above reasons as well as current clinical data, RVX-208 has illustrated the early potential to become an important and differentiated therapeutic for high risk patients with CVD, DM and CKD.

Diabetes Mellitus

Diabetes mellitus (“DM”) is the most common endocrine disease in the world. A primary defect in DM is the inability of the pancreas to provide enough insulin for the body, thus leading to increased blood glucose. Our interest in exploring a potential therapy for DM stems from our technology that enhances ApoA-I/HDL production and function.

HDL can directly modulate glucose metabolism through multiple mechanisms. The key clinically relevant observation is that both acute and chronic HDL elevations reduce blood glucose in patients with type 2 diabetes mellitus (“T2DM”). This is the most common form of DM affecting roughly 90% of those with the disease. Studies have shown that HDL can directly stimulate pancreatic insulin secretion. Additionally, HDL may also improve insulin action in those with T2DM. These actions of HDL help to increase levels of insulin and its action in the body underlies the thinking that ApoA-I/HDL-raising strategies may have benefits beyond vascular disease to not only treat but possibly prevent T2DM. Therefore, we have conducted an exploratory study examining the effects of using RVX-208 to chronically elevate ApoA-I /HDL levels. This exploratory Phase 2 clinical trial examined insulin secretion, insulin sensitivity and whole-body glucose and lipid metabolism in individuals with pre-diabetes. These studies are an important prerequisite to potentially expand the indications for RVX-208 to type 2 diabetes mellitus.

On July 23, 2014, we announced the preliminary results of the exploratory trial. The investigators postulated that the RVX-208 induced rise in ApoA-I/HDL-C may impact pancreatic insulin secretion and thereby lower blood glucose (detected using an oral glucose tolerance test). Patients (n=23) with pre-diabetes mellitus (also called metabolic syndrome) were given 200 mg/day RVX-208 for a short duration of only 4 weeks. The preliminary results were not consistent with their hypothesis. However, this finding was useful in understanding the ASSURE data because for RVX-208 to reduce blood glucose in patients with diabetes mellitus required at least 12 weeks of treatment. Analysis of data from the trial beyond preliminary results reported here will include; HDL abundance, lipidomics, platelet aggregation, monocyte activation and neutrophil adhesion. We are planning to submit the above important findings and other new data to scientific journals for peer review prior to publication and presentation at leading medical conferences.

Chronic Kidney Disease

Chronic Kidney Disease (“CKD”) results from the long term effects of DM on blood vessels and the filtration apparatus (nephrons) of the kidneys. It is often referred to as a silent killer because it is insidious in onset, progressing slowly over many years and sometimes decades. According to the National Institute of Diabetes and Digestive and Kidney Diseases, more than 31 million people in the US (or 10% of the adult population) currently suffer from CKD. Healthy kidneys filter metabolic by-products from the blood in order for these unwanted materials to be components in the urine and thus eliminated from the body. One important measure of kidney function is estimated glomerular filtration rate (“eGFR”) which assesses the amount of fluid the kidney can filter over a period of time. In patients with CKD, as kidney function declines, the eGFR decreases. Unfortunately for many patients, CKD can progress to a point when the kidneys fail completely. This is called end-stage renal disease (“ESRD”) and these patients require hemodialysis, often multiple times per week, in which a machine removes the metabolic waste products from the blood. The cost of ESRD and hemodialysis to the healthcare system is enormous, exceeding \$34 billion each year in the U.S. The cost for one patient to be on dialysis for one year is almost \$90,000.

On June 1, 2015, we announced results of an analysis examining the effects of RVX-208 on key renal parameters. In the pooled analysis from the SUSTAIN and ASSURE trials, assessment of the metabolic biomarker alkaline phosphatase, ALP, revealed a significant reduction of -10.98% in all patients treated with RVX-208 (n=331) compared to a reduction of -3.23% in placebo treated patients (n=168) (p<0.0001) at the combined time points of 24 and 26 weeks. In addition, several subgroup analysis were performed. In patients with a history of diabetes, a significant reduction in ALP of -13.9% was observed in the group treated with RVX-208 (n=127) compared to -4.49% in the placebo treated group (n=65) (p<0.0001). Further analysis was performed on patients with Chronic Kidney Disease (CKD), defined by an estimated glomerular filtration rate (eGFR) of below 60 mL/min/1.73 m². In this group, patients treated with RVX-208 (n=35) had reduced ALP levels of -13.9% versus -6.28% in placebo (n=13) (p=0.008). In addition, following 6 months of RVX-208 treatment, an increase in eGFR of +3.4% (p=0.04 vs. baseline) in the group treated with RVX-208 was observed compared to a decrease of -5.9% in the placebo group.

We plan to further evaluate CKD biomarkers to determine the potential of RVX-208 could have in this important disease area. A subgroup analysis is planned in the Phase 3 RVX-208 BETonMACE clinical study in this therapeutic area.

Neurodegenerative

Epidemiological and mechanistic evidence indicate a link between CVD and neurodegenerative diseases such as dementia and Alzheimer's disease ("AD"). A growing body of evidence now supports a strong and possibly causal relationship between the two. Both diseases are most prevalent in aged individuals and they share many of the same risk factors including, but not limited to, smoking, hypertension, altered glucose metabolism, obesity, genetic susceptibility (i.e., ApoE allele status), inflammation, and abnormal blood lipids. Multiple studies have now demonstrated that factors affecting CVD such as moderate-to-high mid-life cholesterol values, diabetes, obesity, and smoking approximately double the risk of AD. There are also links to cognition demonstrating an association between risk of cognitive decline (and dementia) with a history of stroke, myocardial infarction, peripheral artery disease, and carotid plaques. Other findings demonstrate similar relationships between AD and coronary artery disease, myocardial infarction, cardiac arrest, carotid atherosclerosis, and hypercholesterolemia.

Based on its epigenetic mechanism, RVX-208 has been shown to affect expression of numerous targets important for both CVD and AD such as ApoA-I/HDL, inflammatory mediators, components of the complement cascade and others. Moreover, RVX-208 has been shown to repress multiple biological processes including pro-inflammatory, pro-atherosclerotic and pro-thrombotic pathways that can contribute to CVD and AD risk. As such, RVX-208 is a target for a more physiologically relevant approach of treating the multiple biologies that contribute to a neurodegenerative disease state.

To further evaluate the potential of BET inhibition in neurodegenerative disease, such as AD, a variety of studies are underway to investigate the role of epigenetic modification in this disease. Moreover, ongoing preclinical studies are exploring the role of RVX-208 mediated modulation of neuroinflammation and innate immunity in this important disease area. With leading experts on our neurodegenerative clinical and scientific advisory board providing input and guidance, a Phase 2 clinical study in this therapeutic area is planned.

Competitive Environment

The current landscape of drugs in development for CVD risk management is presented in the table below.

The LDL lowering hypothesis is being further tested in CVD risk with aggressive reduction of LDL lowering to unprecedented levels such as 40-50 mg/dl. Newly reported trials such ODYSSEY LONG TERM (Robinson, Farnier et al. 2015), PCSK9 LDL-lowering approach via Alirocumab, OSLER-1/OSELR-2 (Sabatine, Giugliano et al. 2015), PCSK9 LDL-lowering via Evolocumab and IMPROVE IT (Cannon, Blazing et al. 2015), and cholesterol absorption LDL-lowering approach via Ezetimibe have provided new information on the potential for very aggressive LDL lowering and its effects on efficient MACE reduction in CVD patients, on top of standard of care therapy. Although both ODYSSEY LONG TERM, OSLER-1/OSELR-2 and IMPROVE-IT showed significance in lowering MACE, with a reported annual treatment cost per patient of US \$7,000-12,000 (Staton 2015), the cost to prevent an event could be argued to be inefficient.

In addition, there are several other approaches to CVD risk; however most of them have been unsuccessful in the clinical stages of development. Nicotinic acid had been widely used as a HDL raising strategy but due to findings in the AIM-HIGH study (Boden, Probstfield et al. 2011), this approach to HDL enhancement is now facing increased scrutiny. CETP inhibition is another approach that is being currently investigated by several large pharma organizations. CETP inhibition, although highly effective in raising HDL in plasma, has little target validation on clinical outcomes. The largest epidemiology study to study CETP, COPENHAGEN, studied approximately 9,000 subjects and clearly illustrated that CETP is important for RCT biology. The study focused on subjects who naturally inhibited CETP. In the study subjects had high HDL levels but also had significantly higher CVD events, specifically in women (Agerholm-Larsen, Tybjaerg-Hansen et al. 2000). These findings, along with additional data from the meta-analysis of CETP, (Thompson, Di Angelantonio et al. 2008), suggest that HDL functionality and RCT biology are potentially more important than standard HDL plasma measurements when looking for biomarker impact on MACE measurements. A third analysis of CETP in the PREVENT study group also came to similar conclusions (Borggreve, Hillege et al. 2006). In addition, specific to patients with DM, therapeutic interventions for improved glycaemic control (including glitazones, DPP-4 inhibitors, insulin and sulphonylureas) have not been shown to reduce the risk for MACE or mortality (Gerstein, Miller et al. 2008; Patel, MacMahon et al. 2008; Duckworth, Abaira et al. 2009).

Efficient numbers needed to treat ("NNT") are also an important assessment in the accretive value added of these technologies by reimbursement and payer groups. There are several advantages to small molecule products and research platform over potential antibody competitors such as Pfizer and Amgen's PCSK9 programs. The key issue that has to be addressed is not only safety and efficacy but also cost efficiency and valid pharmacoeconomic modeling. Payers are now demanding cost efficiency for new

therapeutics to be approved on health plans. The PCSK9 programs require intravenous or subcutaneous routes of administration which represent pricing hurdles. Estimates for these types of therapeutics are in the range of US\$7,000-12,000 annually (Staton 2015). As a result, payers will demand clear efficacy and price value model for risk reduction. If RVX-208 can illustrate a greater than 20-25% relative risk reduction over standard of care medicines, pricing will be a major competitive advantage over large molecule agents such as the PCSK9 programs.

Key Products in Development (Internal and Competitors)

Product	Indication	Mechanism of Action	Market Status	Threat Assessment
Resverlogix RVX-208/ apabetalone (Resverlogix)	Secondary MACE Reduction	BET Inhibition	Phase 3	N/A
Alirocumab (Sanofi/ Regeneron)	CVD	PCSK9 Inhibition	Phase 3	Moderate / High Cost
Evolocumab (Amgen)	CVD	PCKS9 Inhibition	Phase 3	Moderate / High Cost
Ezetimibe (Merck)	CVD	Cholesterol Absorption	Phase 3	Moderate / Inefficient
Losmapimod (GSK)	CVD	Inflammation Mediators	Phase 3	Moderate / Proof of Concept
Darapladib (GSK)	CVD	Inflammation	Phase 3	Low / Trial Failed
A-002 (Anthera)	CVD	Inflammation	Phase 3	Low / Trial Failed
Anacetrapib (Merck)	CVD	CETP Inhibition	Phase 3	Low / Long Half Life
Dalcetrapib (Roche)	CVD	CETP Inhibition	Phase 3	Low / Trial Failed
Evacetrapib (Lilly)	CVD	CETP Inhibition	Phase 3	Low / Pending

General Development of the Business

Epigenetic BET Database

As described below under “General Development of the Business – Clinical Development”, we conducted a Phase 2b program in high risk CVD patients with low high-density lipoprotein (“HDL”). These two trials, SUSTAIN and ASSURE (N=499), provided us with a large and growing proprietary database of critically important biomarkers targeted with our novel epigenetic small molecule RVX-208. This is the first and largest integrated dataset of multiple vascular risk markers with an epigenetic drug treatment on these specific high risk vascular target patients. A pooled analysis of these markers of vascular risk was performed as well as correlation analysis on markers and MACE which occurred during the studies. Detailed additional analysis on markers of RCT, ApoA-I, HDL-C, HDL particle number, large HDL, HDL particle size; vascular inflammation markers such as CRP; and metabolic markers such as glucose, HB1AC, ALP and others of interest in relation to epigenetic drug development were also studied and captured in the database. To date our BET database is made up of over 650,000 data points which help us understand in much greater detail how epigenetics and select BET inhibition affect target risk markers for vascular disease. We will continue to add to this important information tool to further elucidate the role of epigenetics in biology and its role in vascular disease. A pooled analysis is a formal quantitative method for evaluating potential health effects of a drug across a body of clinical trials. In pooled analysis, pharmaceutical and biotechnology organizations assess heterogeneity across studies, examine subgroups of studies to determine if selected subsets of the research data provide similar or different results, and calculate summary relative risk estimates. In many cases pooled analysis provides a more statistically precise risk estimate, as well as a better understanding of the consistency of findings (or lack of) of a drug based on the fact that there is more power in the data as more patients are involved in the analysis. A pooled analysis is similar to a traditional meta-analysis, except that exposure and outcome data are combined (or pooled) from multiple studies and are analyzed as a single dataset. We continue to develop our proprietary database with a specific focus on

building a more comprehensive understanding of epigenetics biology and how to best target RVX-208 in patient groups that will be the best responders to treatment.

Pooled MACE Analysis

As described below under “General Development of the Business – Clinical Development”, we have reported several important findings over the past two years. One of the most important finding was that of pooled MACE results reported on January 15, 2014. This analysis, performed by an independent statistician, focused on the potential benefit of RVX-208 and select BET inhibition, to reduce MACE over a short treatment time period of six months. When MACE data (n=499) from both SUSTAIN and ASSURE trials were combined, it demonstrated that treatment with RVX-208 led to a significant reduction in MACE. Patients treated with RVX-208 (n=331) had less cumulative events of 6.74% vs. 15.09% (p=0.02) in the placebo treated group (n=168). Furthermore, in patients who had elevated CRP > 2.0mg/dL (n=283) the benefit of RVX-208 treatment of patients (n=179) appeared more striking with a cumulative event rate of 6.42% vs. 20.53% (p=0.007) in the placebo group (n=104). Results from an additional high risk patient population, those with diabetes mellitus were reported September 2, 2014. In this analysis, patients treated with RVX-208 (n=127) had less cumulative events of 4.71% vs. 20.31% (p=0.008) in the placebo treated group (n=65). These early pooled analyses on MACE are also being examined in other high risk populations within both SUSTAIN and ASSURE patients such as CKD patients. A similar trend was observed in these patients as well. We will continue to utilize this important information to further determine the best path moving forward for future clinical development of RVX-208.

Recent Clinical Development

The following principal events have influenced the general development of our business in the last three fiscal years. Detailed findings and highlights from the clinical development of RVX-208 are highlighted below.

Phase 3 BETonMACE – Secondary Prevention Trial – Planned to Commence in 2015

Our intention is to reconfirm in a larger prospective setting with patients that have modifiable vascular disease (i.e. low HDL-C and diabetes) positive effects on markers of vascular risk and reduction of MACE. The proposed study is a large multi-center, double-blind, randomized, parallel group, placebo-controlled clinical trial in high-risk Type II Diabetes patients with CAD to determine whether treatment with RVX-208 in combination with rosuvastatin and atorvastatin increases the time to major adverse cardiovascular events compared to treatment with rosuvastatin and atorvastatin alone. The study is an event-based trial and will continue until a minimum of 250 primary endpoint events have occurred. We anticipate that no fewer than 2,400 patients will be enrolled.

Pooled ASSURE and SUSTAIN Analysis – Ongoing

On January 15, 2014, we announced new information arising from ongoing analysis of data from both the SUSTAIN and ASSURE trials in atherosclerotic patients with high risk for recurrent events. This analysis, performed by an independent firm, focused on the potential benefit of RVX-208 to impact MACE over a short time period of six months.

When data from both SUSTAIN and ASSURE trials (n=499) were combined, it demonstrated that treatment with RVX-208 led to a significant reduction in MACE. Patients treated with RVX-208 (n=331) had less cumulative events of 6.74% vs. 15.09% (p=0.02) in the placebo treated group (n=168). Furthermore, in the case of patients who had elevated CRP > 2.0mg/dL (n=283) the benefit of RVX-208 treatment of patients (n=179) appeared more striking with a cumulative event rate of 6.42% vs. 20.53% (p=0.007) in the placebo group (n=104).

On September 2, 2014, we announced new information from the analysis of data pooled from the ASSURE and SUSTAIN clinical trials. The findings were presented in an oral presentation at the European Society of Cardiology (“ESC”) Congress entitled, “Effects of RVX-208 on MACE, ApoA-I and HDLs; a post-hoc analysis from the pooled SUSTAIN and ASSURE clinical trials”. Patients with CVD arising from atherosclerosis when given RVX-208 had a 55% (p=0.02) relative risk reduction (“RRR”) in MACE. More importantly, this beneficial effect of RVX-208 on patients with diabetes mellitus was accentuated with a RRR in MACE of 77% (p=0.01). These observed reductions in MACE may stem from the ability of RVX-208 to significantly improve specific markers of CVD risk measured in the SUSTAIN and ASSURE trials, including: lower levels of ALP (p<0.0001), higher levels of ApoA-I protein (p<0.01), an increase in HDL-C (p<0.001), more large HDL-particles (p<0.05) and growth in HDL particle size (p<0.05). The significant improvement in these markers was in treated patients vs. placebo, which may point to the RVX-208 BET inhibition effects on reverse cholesterol transport and other emerging risk pathways such as vascular inflammation and calcification.

On March 16, 2015, we announced additional findings from the analysis of both the SUSTAIN and ASSURE clinical trials. In a poster entitled, “RVX-208 the first selective bromodomain extra-terminal (BET) protein inhibitor being developed for patients with high residual risks of cardiovascular disease”, clinical biomarker analysis was presented and revealed significant changes between RVX-

208 vs. placebo (change, p value) in: serum alkaline phosphatase (-6 U/L, <0.0001), HDL-C (+3 mg/dL, <0.001), ApoA-I (+7.5 mg/dL, <0.01), large HDL (+0.7 umol/L, <0.05), HDL size (+0.1, <0.05), and total HDL particles (+1.8 umol/L, <0.1). While these findings were evident in all patients, two groups appeared to benefit more from RVX-208 were those with diabetes mellitus ("DM") or CKD. In those with DM (n=192) given RVX-208, glucose was unchanged vs. a non-significant (p<0.1) rise of +0.7 mmol/L in placebo patients. In those with DM (n=119) and low HDL <40 mg/dL, RVX-208 reduced glucose significantly (p<0.01) by -0.3 mmol/L while in placebo the glucose increased +0.9 mmol/L. In CKD subjects (n=48) with mild to moderate renal failure (estimated glomerular filtration rate ("eGFR") <60 mL/min/1.73m²) given RVX-208 vs placebo there was a +3.4% vs. -5.9% in eGFR, respectively. In addition, microarray studies were performed using primary human liver cells exposed to RVX-208. This treatment demonstrated significant changes in cellular pathways or networks characterized by: an attenuation in inflammation, coagulation, complement and cholesterol synthesis. These clinical findings illustrate the effects of RVX-208 on ALP, HDL profile, glucose and eGFR and provide a better understanding of how BET inhibition may lower MACE. The microarray data provides, at a cellular level, novel insights that detail the multiple activities of RVX-208 beyond its ApoA-I effects in lowering MACE.

On June 1, 2015, we announced new information from the analysis of data pooled from the ASSURE and SUSTAIN clinical trials relating to specific biomarkers relevant to CKD. In a poster entitled, "Effects of RVX-208, a First-in-Class Epigenetic BET-Inhibitor, on Key Renal Parameters in Subjects with a History of CVD, and Chronic Kidney Disease (CKD); a Post-hoc Analysis of Patients from the ASSURE, SUSTAIN and ASSURE Clinical Trials," presented at the European Renal Association-European Dialysis and Transplant Association (ERA-EDTA) Congress, an assessment of the metabolic biomarker, ALP, revealed a significant reduction of -10.98% in all RVX-208 treated patients (n=331) compared to a reduction of -3.23% in placebo treated patients (n=168) (p<0.0001) at the combined time points of 24 and 26 weeks. In addition several subgroup analyses were performed. In patients with a history of diabetes, a significant reduction in ALP of -13.9% was observed in the RVX-208 treated group (n=127) compared to -4.49% in the placebo treated group (n=65) (p<0.0001). Further analysis was performed on patients with CKD defined by an eGFR of below 60 mL/min/1.73 m². In this group, patients treated with RVX-208 (n=35) had reduced alkaline phosphatase levels of -13.9% versus -6.28% in placebo (n=13) (p=0.008). In addition, following 6 months of treatment with RVX-208, an increase in eGFR of +3.4% (p=0.04 vs. baseline) in the RVX-208 treated group was observed compared to a decrease of -5.9% in the placebo group. Dr. Kam Kalantar-Zadeh, Professor and Chief, Division of Nephrology and Hypertension at University of California in Irvine and Los Angeles examined these findings and has also contributed to additional abstracts that have been submitted for peer review presentation. Together these new findings warrant additional clinical trials for target responder CKD and/or dialysis populations who have a high burden of cardiovascular disease and risk.

Continued analysis of the Phase 2b program pooled data will not only broaden our understanding but also provide a more targeted pathway for our future clinical trials of RVX-208. We plan to perform further detailed analysis on potential new biomarkers and biological pathways that RVX-208 may affect through its select BET inhibition mechanism. New findings in these analyses will seek out potential additional indications that can be applied to broadening the scope of diseases that BET inhibition can benefit. Appropriate intellectual property will be developed in concert with any novel findings.

Phase 2b ASSURE Clinical Trial – Completed

ASSURE (ApoA-I Synthesis Stimulation in Acute Coronary Syndrome patients) was a 26-week randomized double-blind placebo-controlled multi-center study that examined the early effects of RVX-208 induced ApoA-I production on atherosclerotic plaque regression in the setting of patients with coronary artery disease who have a low level of high density cholesterol ("HDL-C") using IVUS. Secondary objectives for ASSURE were safety and tolerability of RVX-208 as reflected by adverse events, and effects of RVX-208 on HDL, ApoA-I, Large HDL particles and non-HDL lipid parameters such as CRP and other markers of interest in cardiovascular disease risk reduction.

Enrollment in ASSURE was completed in September 2012. A total of 323 patients participated in ASSURE, of which 243 were given RVX-208 and 80 received placebo. Top line ASSURE data was announced on June 27, 2013; ASSURE did not meet its primary endpoint of a -0.6% change in percent atheroma volume as determined by IVUS. The RVX-208 treated group had -0.4% plaque regression (p= 0.08) v.s. baseline. The patient group receiving active treatment met the secondary endpoints of regression of total (coronary) atheroma volume ("TAV") and increases in ApoA-I and HDL-C.

On September 3, 2013, we announced the Full Analysis Set (FAS) data from 281 treated patients in ASSURE. Findings showed that the below median HDL (<39 mg/dL) baseline population consisted of 92 patients who were taking either rosuvastatin (Crestor®) or atorvastatin (Lipitor®) together with RVX-208. Those patients taking rosuvastatin and RVX-208 had a highly statistically significant Percent Atheroma Volume ("PAV") plaque regression of -1.43% with probability value of p<0.002, v.s. baseline. This PAV regression exceeded the trial's pre-specified PAV endpoint (-0.6%) by more than two-fold. Those patients taking atorvastatin (Lipitor®) together with RVX-208 had a PAV plaque progression of +0.19% with a non-significant probability value v.s. baseline.

A subgroup analysis revealed a responder population (those with HDL <39 mg/dL taking rosuvastatin and RVX-208) exceeded the primary endpoint and also surpassed secondary endpoints reflecting regression in coronary atherosclerosis. These measures included TAV and changes in the 10 mm most diseased segment of the coronary arteries; we noted marked regression versus baseline of -12.3 mm³ (p<0.0001) and -4.3 mm³ (p<0.0001), respectively, v.s. baseline. Other secondary endpoints assessed in this population were biomarkers of reverse cholesterol transport ("RCT"), including: HDL-C, ApoA-I and large HDL particles which increased by 18.2% (p<0.0001), 16.4% (p<0.0001) and 74.7% (p<0.0001), respectively, vs. baseline.

On November 4, 2013, we announced two additional results from ASSURE. First, the data showed statistically significant improvements in coronary IVUS atheroma measurements and MACE in patients with a high (>2.0 mg/dL) serum high sensitivity C-Reactive Protein ("hsCRP"). Serum levels of this biomarker when >2.0 mg/dL reflect a heightened state of inflammation that is a well-known and major component of high CVD risk. Patients with hsCRP>2.0 mg/dL at time of entry into ASSURE totaled n=184 of which n=54 were given placebo while n=130 received RVX-208. In patients treated with RVX-208, there was a 60% reduction (p<0.0001) in hsCRP vs. baseline and (p=0.054) vs. placebo. Furthermore, atheroma regression was observed in patients treated with RVX-208 as measured by PAV, TAV regressed, and the worst 10mm TAV segment by -0.75% (p<0.03), -6.3mm³ (p<0.001) and -2.63mm³ (p<0.001), respectively vs. baseline. Equally intriguing and perhaps more important is that in RVX-208 treated patients with hsCRP>2.0 mg/dL the incidence of MACE was lower by 63% (p=0.023) vs. placebo. These findings were detailed at the American College of Cardiology conference on March 31, 2014 in a poster titled, "Effects of an Apolipoprotein A-1 Inducer on Progression of Coronary Atherosclerosis and Cardiovascular Events in Patients with Elevated Inflammatory Markers" and presented by Rishi Puri, Medical Director of Cleveland Clinic Atherosclerosis Imaging Core Laboratory. The preceding observation is of value in that hsCRP of >2.0 mg/dL is well known to be clinically important in predicting CVD risk.

The second observation arose from a pre-specified exploratory endpoint in ASSURE gathered using a new catheter (Volcano Revolution 45mghz) designed for radiofrequency analysis of the IVUS signal. Data from this catheter reveals so called virtual histology IVUS ("VH-IVUS"), an emerging technology that is useful for assessing tissue characteristics of an atherosclerotic plaque. VH-IVUS data was analyzed to provide insight into vulnerability of an atherosclerotic plaque to rupture and its relationship to future cardiovascular risk. In ASSURE, while all (n=323) patients were studied using IVUS, 87 of these were examined using the Volcano Revolution catheter to gather VH-IVUS information. This information was used to reflect plaque vulnerability by calculating the ratio of necrotic core to dense calcium ("NC/DC"). The NC/DC ratio in RVX-208 treated patients (n=61) was significantly lower by -7.5% (p<0.03) vs. baseline while those (n=24) given placebo had a non-significant reduction of -3.8% (p=0.47) vs. baseline. The initial VH-IVUS findings show that the actions of RVX-208 improved the NC/CS ratio pointing to less vulnerability of the atherosclerotic plaque for rupture.

The addition of these findings to the previously announced impact of RVX-208 to regress PAV (-1.43%, p=0.001) in ASSURE patients with low HDL-C given rosuvastatin further define a large high risk population where RVX-208 illustrates profound effects to reduce atheroma volume and plaque vulnerability. Together these findings help explain the observed reduction in MACE.

Phase 2b SUSTAIN Clinical Trial – Completed

SUSTAIN was a 24-week, multi-center, double-blind, randomized, parallel group, placebo controlled clinical trial conducted in South Africa. 176 subjects with established CVD who continue to have a high-risk for recurrent CVD events were enrolled. All subjects in SUSTAIN had a low level of HDL-C and were receiving standard of care therapy that included up to 40 mg Atorvastatin (Lipitor®) or 20 mg Rosuvastatin (Crestor®). Subjects received 200 mg/day of RVX-208 or placebo in order to assess lipid trends and safety. In addition, other biomarkers of reverse cholesterol transport were examined. The primary endpoint of SUSTAIN was the change in HDL-C from baseline after receiving RVX-208 for 24 weeks vs. placebo. Secondary endpoints included change in ApoA-I, LDL-C, non-HDL-C, apoB, triglycerides and HDL subclasses.

In August 2012, we announced that SUSTAIN met its primary endpoint. RVX-208 significantly increased HDL-C (statistical significance of p=0.001), the primary endpoint of SUSTAIN. SUSTAIN also successfully met secondary endpoints, showing increases in levels of ApoA-I (statistical significance of p=0.002) and large HDL particles (statistical significance of p=0.002). ApoA-I and HDL are both believed to be important factors in enhancing RCT, the process whereby cholesterol is packaged and transported by special particles in the plasma called lipoproteins for movement from peripheral tissues through the blood and back to the liver for excretion from the body. The SUSTAIN trial also showed that increases in alanine aminotransferase ("ALT") liver signals similar to those which were reported in previous trials were infrequent and, when allowed according to the trial's protocol, returned to normal either as a result of continued dosing or short-term interruption, with no new increases observed beyond week 12 of the 24-week trial. Management and the Clinical Steering Committee have chosen to submit the remaining data for a peer reviewed publication.

The SUSTAIN trial provided us with important data regarding improvement in the functionality of the HDL produced by RVX-208. Safety data from SUSTAIN reconfirmed our belief that early liver signals witnessed in this and previous trials were of a transient nature.

Findings drawn from SUSTAIN included:

- low baseline HDL and low baseline ApoA-I were the best responders; and
- there was 1 MACE event in treated subjects vs. 6 in placebo.

Pre-Diabetes Mellitus – Completed

In October 2012, we initiated an exploratory Phase 2 clinical trial in patients with pre-diabetes mellitus to examine the effects of RVX-208 and ApoA-I production on glucose metabolism. The foundation of this trial builds upon the actions of RVX-208 and the knowledge that RVX-208 triggers a key epigenetic pathway which results in enhanced ApoA-I protein production to raise the body's level of functional HDL particles. This trial was also built upon the belief that the effect of newly created ApoA-I/HDL may help to preserve pancreatic cells that make and secrete insulin, and that increased abundance of insulin in subjects with pre-diabetes mellitus may prevent or substantially delay the progression towards diabetes mellitus. The trial was conducted in collaboration with Baker IDI Heart & Diabetes Institute in Melbourne, Australia. Enrollment of this trial was completed in December 2013.

On July 23, 2014, we announced that the preliminary results of this exploratory trial. The investigators postulated that the RVX-208 induced rise in ApoA-I/HDL-C may impact pancreatic insulin secretion and thereby lower blood glucose (detected using an oral glucose tolerance test). Patients (n=23) with pre-diabetes mellitus (also called metabolic syndrome) were given 200 mg/day RVX-208 for a short duration of only 4 weeks. The preliminary results were not consistent with their hypothesis. However, this finding was useful in understanding the ASSURE data because for RVX-208 to reduce blood glucose in patients with diabetes mellitus required at least 12 weeks of treatment. Analysis of data from the trial beyond preliminary results reported here will include: HDL abundance, lipidomics, platelet aggregation, monocyte activation and neutrophil adhesion. We are planning to submit the above important findings and other new data to scientific journals for peer review prior to publication.

On June 8, 2015, we announced findings from a study performed by the Baker IDI Heart and Diabetes Institute, Melbourne, Australia and our scientists which were reported at the American Diabetes Association Scientific Sessions. The presentation was entitled "Effects of the ApoA-I Inducer, RVX-208 on Glucose Metabolism in Individuals with pre-diabetes Mellitus." The data in the presentation was based on patients with pre-diabetes mellitus who already had abnormal blood glucose levels. Treatment with RVX-208 (200 mg/day) for 29-33 days led to not only a reduction in glucose absorption but also suppression of endogenous glucose production. The significance of these findings are as follows: (1) short duration of RVX-208 treatment had effects on glucose metabolism, and (2) both the reduction in glucose absorption and production are expected to be of benefit in patients with pre-diabetes mellitus. The above findings are intriguing when viewed in the light of additional new data arising from the same study reported at the recent International Society of Atherosclerosis (ISA) meeting, May 23-26, 2015 in Amsterdam, NL. At the ISA meeting, a presentation entitled, "The effects of a novel apoA-I transcriptional regulator (RVX-208) on whole plasma and HDL lipidomes," the same team of investigators detailed the ability of RVX-208 to change the lipid profile within the HDL favouring normalization of the composition towards that observed in healthy individuals. Together, the data contained in the two presentations support the proposition that RVX-208 has the ability to affect glucose and lipid metabolism in ways that will be of benefit to patients with CVD risks.

Summary of Additional Phase 2 Clinical Trials

Phase 2 ASSERT Clinical Trial - Completed

ASSERT was a 13-week randomized, double-blind, placebo-controlled, multi-center US study with 299 patients enrolled with stable coronary artery disease. The primary endpoint of the study was increased plasma ApoA-I levels compared to placebo group after three months of dosing of RVX-208. Other objectives were to examine the safety and tolerability of RVX-208 and to compare the dose and time response relationship for ApoA-I as well as to examine key reverse cholesterol markers involved with HDL functionality.

In November 2010, we announced top line results of the ASSERT Phase 2 clinical trial. The ASSERT trial data demonstrated that the three key biomarkers in the reverse cholesterol transport process showed dose dependant and consistent improvement. The trial showed dose dependent increases, though not statistically significant, in ApoA-I, the trial's primary endpoint. The trial also showed statistically significant increases in HDL cholesterol including alpha1 particles or functional HDL, and highly statistically significant increases in large HDL particles. RCT is a pathway by which accumulated cholesterol is transported from the arterial wall to the liver for excretion, thus reducing and/or preventing atherosclerosis. In the high dose, ApoA-I achieved a 5.6% increase with a statistical value of $p=0.06$ versus placebo. Across all subjects, ApoA-I showed a trend to increase in higher doses with statistical significance of $p=0.035$. ApoA-I and other HDL particles continued to be increasing at the end of the 12 week study. Both the 8.3% HDL cholesterol increase and the 21.1% large particle HDL increase were highly statistically significant, $p<0.01$ and $p<0.001$ respectively. These pronounced HDL related increases via ApoA-I production are important as they take place later in the reverse cholesterol transport chain of events and strongly indicate the potential for plaque regression.

In the high risk sub population, patients who had low HDL 45mg/dl, the middle dose of 200 mg saw the most pronounced increases of 12% in ApoA-I (p.<0.02), 21% in HDL (p<0.015) and large HDL of 32% (p.<0.018) versus baseline. These findings helped us better target the patient population as well as dosing of RVX-208 for the ASSURE Phase 2b trial.

Across all patients, incidents of elevated alanine aminotransferase liver enzymes (“ALTs”) in excess of 3 times the upper limit of normal (“ULN”) were experienced by 18 of 225 treated patients in ASSERT. It is important to note that elevated ALTs alone are not considered indicative of liver injury. Increases in bilirubin, which would indicate liver injury, were not experienced in any patient. In high risk patients, HDL below 45 mg/dl who were on newer leading statin agents at select doses, 1 patient of 45 experienced ALTs in excess of 3 times ULN. The single subject who had the ALT in excess of 3 times ULN had a surgery during the trial and was administered an agent as well as high dose Tylenol post-surgery, two agents known to cause excessive liver ALT signals. When measuring biochemical safety measures, in all doses, ALTs were highest at week 8 and declined at weeks 10 and 12, possibly indicating adaptation by the liver or that elevated ALTs are transient. Incidents of elevated ALTs were significantly less frequent with patients who were on new leading statin agents at low and medium doses. Target levels for key RCT markers in these patients maintained or improved on these dosing combinations over co-administration with high doses of statins. We considered these findings important, novel and understandable given RVX-208 site of action to induce the production of de-novo ApoA-I particles is in the liver; we incorporated these important findings from the ASSERT trial into the design of the ASSURE trial and made safety and targeted efficacy a primary focus for the future development of the drug.

Findings drawn from ASSERT included:

- Data illustrated that 200 mg/day of RVX-208 was the optimal dose, based on safety and efficacy;
- Data illustrated that those patients with a low level of HDL-C at baseline had a better response for HDL-C and ApoA-I increases when treated with RVX-208; and
- Data illustrated that the best response were those patients given RVX-208 in combination with non-max doses of second generation statins such as Rosvastatin (Crestor®) or Atorvastatin (Lipitor®).

These key findings contributed to determining a therapeutics window and targeted patient group for RVX-208.

Summary of Clinical Trials

Trial	Summary	Patients	Status	Initiated (Calendar)	Data Release (Calendar)
Phase 3	Secondary prevention of MACE in patients with diabetes and low HDL	2,400 +	Enrolling Fall 2015	TBD	TBD
Phase 2a	Alzheimer's disease	45-60	Pending	TBD	TBD
Phase 2b	Pre-diabetes mellitus / effects of RVX-208 and ApoA-I production on glucose metabolism	20	Completed	Q4 2012	Q3 2014
Phase 2b ASSURE	26 week risk-stratified IVUS study in patients with low HDL	323	Completed	Q2 2011	Q2 2013
Phase 2b SUSTAIN	24 week single-dose safety, tolerability and efficacy in stable CVD patients with low HDL	176	Completed	Q3 2011	Q3 2012
Phase 2 ASSERT	12 week dose-ranging safety, tolerability and efficacy in stable CVD patients	299	Completed	Q4 2009	Q4 2010
Phase 1b/2a	28 day multiple dosing safety, tolerability and efficacy in healthy volunteers with low HDL	72	Completed	Q3 2008	Q3 2009
Phase 1 BE	Single dose bio-equivalency comparing capsule and tablet drug form	9	Completed	Q3 2009	N/A
Phase 1 BE	Single dose bio-equivalency	7	Completed	Q3 2009	Q4 2009
Phase 1a	First-in-man single ascending dose and 7-day multiple dosing	80	Completed	Q4 2007	Q1 2008

Regulatory Matters

RVX-208 is being investigated for the secondary prevention of major adverse cardiac events including emergency/urgent revascularization in patients with type 2 diabetes mellitus, and low HDL-cholesterol. An IND was filed and accepted by the FDA in 2007 to commence clinical testing. Subsequently, clinical trial applications were submitted for phase 2 trial conduct and accepted by national, central and local health authorities in Poland, Belgium, Spain, Netherlands, Russia, Brazil, Argentina, South Africa and Australia. To support the IND and ex-US applications, we submitted manufacturing, nonclinical study and clinical study information (data, protocols, processes, reports, etc.) to the FDA and the ex-US health authorities. We have also provided safety data and other pertinent information in the form of annual reports to the FDA and required ex-US health authorities. We continue to provide all health authorities with the appropriate nonclinical, manufacturing and clinical information as outlined in country specific regulations.

Recent meetings with health authorities in Germany, Sweden and UK have provided scientific advice to help design a Phase 3 study to support filing an MAA within Europe. The scientific advice has been incorporated into the Phase 3 RVX-208 BETonMACE clinical study which is scheduled to commence recruitment in the fall of 2015. Discussions with FDA on the design of additional future studies are currently ongoing.

Product development activities related to RVX-208 or related products will be subject to extensive regulation by various government authorities, including the FDA and comparable regulatory authorities in other countries, which regulate the design, research, clinical and non-clinical development, testing, manufacturing, storage, distribution, import, export, labeling, advertising and marketing of pharmaceutical products and devices.

Mechanism of Action

In April 2012, we announced the Mechanism of Action through which RVX-208 acts. Our data shows RVX-208 to be an inhibitor of the Bromodomain and Extraterminal Domain ("BET") proteins. RVX-208 acts on BET proteins, including BRD4, a member of the BET-protein family. BET proteins are key players in 'epigenetics', a critical mechanism for regulating the expression of genes. Epigenetic processes are mediated by proteins, including the BET-proteins that act in concert with the DNA to make it transcriptionally active or dormant. This epigenetic control of gene expression plays a key role in the development and progression of many human diseases.

RVX-208 mediated inhibition affects multiple processes important for CVD. In addition to effects on lipoproteins, RVX-208 represses pathways underlying the pathogenesis of atherosclerosis and acute coronary events, including inflammation, complement, coagulation (thrombosis) and atherogenesis. Based on mechanistic data, we believe that RVX-208 treatment, or select BET inhibition, attenuates the inflammatory process that contributes to disease initiation and progression. Furthermore, we hypothesize that RVX-208 treatment restores basal activity of the inflammatory and innate immune response and clotting cascade with immediate benefits to atherosclerosis and cardiovascular disease. This is consistent with observed reductions in MACE, especially in patients with an acute inflammatory component in CVD.

Intellectual Property

We devote significant resources to ensure protection of the ideas and inventions related to core areas of our business. Our intellectual property portfolio covers compositions, methods and treatments for the treatment of unmet medical needs related to major adverse cardiovascular events in patients with higher risk such as acute coronary syndrome, peripheral arterial disease, diabetes mellitus and chronic kidney disease.

As of July 27, 2015, we own and/or have rights to numerous patent families, comprising ten issued US patents and a number of pending applications. This includes non-provisional US and provisional applications. The pending patent applications are largely interrelated and assert rights to substantially similar inventions in different jurisdictions. In fiscal 2012, Resverlogix was granted four patents, including a United States Patent covering composition of matter claims to RVX-208, and a United States Patent covering the manufacturing of RVX-208. In fiscal 2013, Resverlogix was granted seven patents including a United States Patent related to inflammatory diseases. In fiscal 2014, Resverlogix was granted eleven patents, including a patent in Europe containing composition of matter claims to RVX-208. In fiscal 2015, Resverlogix was granted eighteen patents including United States patents related to use claims for RVX-208 and manufacturing of RVX-208.

Our intellectual property strategy is to build a strong patent portfolio around core technology that is important to development of leading edge medicines. Our strategies include being the first to identify, isolate, and patent therapeutic agents with commercial importance, to seek out and license intellectual property believed to be useful in connection with potential products, and to control public disclosures.

We also believe that our know-how will provide a significant competitive advantage, and intend to continue to develop and protect our proprietary tools, methods and trade secrets. Our policy is to require employees, consultants, members of our boards and other

third parties in collaborative agreements to execute confidentiality agreements. Employee, consultant and contract research organization agreements specify that all inventions resulting from work performed utilizing our property, business strategies, and work completed during employment/services performed are our exclusive property to the extent permitted by law.

Spin Out of Epigenetics Drug Discovery Platform

On June 3, 2013, we reorganized into two companies and spun off all research and development activities related to the epigenetics platform technology, including cancer and autoimmune diseases, to Zenith, a newly-formed company. We retained research and development activities related to the development of compounds for applications with indications involving a therapeutic increase in ApoA-I, including the cardiovascular disease clinical program related to RVX-208. We also retained the neurodegenerative diseases and diabetes mellitus clinical programs related to RVX-208. The spin-out was implemented by way of a court-approved Plan of Arrangement ("the Arrangement") pursuant to the Business Corporations Act (Alberta).

We developed, and spun out to Zenith, an epigenetic drug development platform with the potential to impact multiple diseases including cancers, cardiovascular disease, neurodegenerative diseases and diabetes mellitus. Zenith's epigenetic research and development program is focused on cancer and autoimmune diseases.

RVX Therapeutics Inc. ("RVX Therapeutics"), which was a wholly-owned subsidiary of Resverlogix prior to the effective time of the Arrangement, held all of the assets spun off. Pursuant to the Plan of Arrangement, all of Resverlogix's entire legal and beneficial right, title and interest in and to all of the shares of RVX Therapeutics was transferred to Zenith, and Zenith issued to shareholders of Resverlogix, fully paid and non-assessable shares of Zenith on the basis of one (1) Zenith share for each Resverlogix common share issued and outstanding immediately prior to the effective time of the Arrangement. Upon completion of the Arrangement, shareholders of Resverlogix owned one share of Resverlogix and one share of Zenith for each common share of Resverlogix held immediately prior to the effective time of the Arrangement. Pursuant to the Arrangement, RVX Therapeutics became a wholly-owned subsidiary of Zenith.

In addition to the transfer of the shares of RVX Therapeutics, as part of the Arrangement Resverlogix transferred US\$19.1 million owed to Resverlogix by RVX Therapeutics and CAD\$10 million of cash to Zenith such that at the effective time of the Arrangement RVX Therapeutics was no longer indebted to Resverlogix and Zenith was provided with initial capitalization.

Pursuant to the Arrangement, Zenith was also issued Royalty Preferred Shares, which provide Zenith with dividends based on a percentage of Net Apo Revenue, if any, as follows:

- 6% of the aggregate Net Apo Revenue for the applicable period that is less than or equal to US\$1 billion;
- 8% of the aggregate Net Apo Revenue for the applicable period that is greater than US\$1 billion but less than or equal to US\$2 billion;
- 10% of the aggregate Net Apo Revenue for the applicable period that is greater than US\$2 billion but less than or equal to US\$5 billion; and
- 12% of the aggregate Net Apo Revenue for the applicable period that is greater than US\$5 billion; and

For additional information regarding the terms of the Royalty Preferred Shares see "Description of Share Capital". On July 2, 2015, the Royalty Preferred Shares were amended to limit the dividends payable to holders of the Resverlogix Royalty Preferred Shares in a particular period to amounts received by us during that period.

Assignment and Services Agreement

Pursuant to an Assignment and Services Agreement dated June 3, 2013 and effective May 1, 2012 between us and RVX Therapeutics, we sold, assigned and transferred to RVX Therapeutics all of our right, title and interest in and to the Transferred IP Rights and the Transferred Technology for US\$8.5 million, payable by RVX Therapeutics to us through the issuance of a promissory note.

We also agreed, on a non-exclusive basis, to perform research services for RVX Therapeutics, as well as related administrative and support services requested by RVX Therapeutics from time to time or as set forth in any statement of work executed by the parties, for an initial term of three (3) years and which will automatically renew for successive one (1) year periods unless a party provides the other party with written notice of non-renewal at least sixty (60) days prior to the expiration of the then-current term. The Assignment and Services Agreement may be found on SEDAR at www.sedar.com.

License Agreement

Pursuant to an Amended and Restated License Agreement dated June 3, 2013 between us and RVX Therapeutics (the “License Agreement”), we granted RVX Therapeutics the worldwide license to develop, make, administer, sell, distribute or otherwise commercialize Licensed Products in any field other than the ApoA-I Therapeutic Field.

On March 17, 2014, we and RVX Therapeutics entered into a Waiver Agreement whereby we agreed to waive our right under the License Agreement to license any method or pharmaceutical agent within the scope of certain patents owned or controlled by RVX Therapeutics that may be determined to come within the ApoA-I Therapeutic Field (as defined in the License Agreement), and RVX Therapeutics agreed not to develop any patents and/or compounds for any indication within the ApoA-I Therapeutic Field for a period of five years. RVX Therapeutics agreed to pay us \$2.5 million in cash and granted to us a right of first refusal for a period of three years thereafter in respect of the license or sale of such patents and/or compounds that are determined to come within the ApoA-I Therapeutic Field. Entering into the Waiver Agreement generated cash for us without impacting negatively on our core assets. The \$2.5 million paid to us by RVX Therapeutics was a negotiated amount agreed upon by the two parties as the fair value and, having received independent financial advice, we concluded that the Waiver Agreement was fair from a financial point of view.

On January 31, 2015, we terminated the License Agreement in order to enhance our freedom to operate and pursue expanded indications. As consideration for the termination, we agreed to pay Zenith \$1.05 million, the estimated fair value of the License Agreement.

Management Services Agreement

Pursuant to a Management Services Agreement dated June 3, 2013 between us and Zenith, we provide management and administrative services pertaining to Zenith as are required, but not including any research services requested by Zenith pursuant to the Assignment and Services Agreement. We are paid a management fee based on the cost of our personnel and the proportionate time worked on behalf of Zenith. We are also reimbursed for general and administrative costs.

Services Agreement

On January 1, 2015, we entered into a Services Agreement with Zenith whereby Zenith will perform research services on an ongoing basis on our behalf. As consideration for these services, we paid a \$0.25 million deposit to Zenith against which expenditures, at cost, will be applied as they are incurred.

Employees

As at April 30, 2015, we employed 22 full-time management, scientific and administration employees.

Board of Directors, Scientific Advisory Board, Clinical Advisory Board and Management

In February 2014, Arthur Higgins resigned from our Board of Directors.

In November 2014, we appointed Dr. Michael Sweeney as Senior Vice President of Clinical Development.

In 2015, we formed a Phase 3 BETonMACE clinical study steering committee comprised of: Dr. Kausik Ray, Imperial College London; Dr. Greg Schwartz, University of Colorado Denver; Dr. Henry Ginsberg, Columbia University New York; Dr. Peter Toth University of Illinois, Peoria; and Dr. Stephen Nicholls, SAHMRI Adelaide Australia.

Financing

Private Placements

In August 2013, we closed a private placement of 1,765,307 units comprised of one common share of Resverlogix and 0.3 of a common share purchase warrant at a price of CAD\$0.90 per unit for gross proceeds of CAD\$1.6 million (US\$1.6 million) to Eastern Capital Limited (“Eastern”). Each whole warrant was exercisable at a price of CAD\$0.90 for a term of five years.

In June 2014, we closed a private placement of 3,485,137 common shares at a price of CAD\$0.65 per common share for gross proceeds of CAD\$2.3 million (US\$2.1 million). NGN BioMed Opportunity II, L.P. (“NGN”), an entity under the control or direction of the Chairman of Resverlogix Corp, subscribed for 1,230,769 common shares. Directors and officers of the Company subscribed for a total of 1,080,522 common shares.

Equity Distribution Agreement

In January 2012, we entered into an equity distribution agreement (the "EDA") with JonesTrading Canada Inc. as agent to sell up to 15 million common shares of Resverlogix ("ATM Shares"), solely at our discretion, from time to time at the market prices prevailing at the time of the sales, without discount, during the period that the EDA remains effective. Pursuant to the EDA, we also appointed JonesTrading Institutional Services LLC and MLV & Co LLC as US agents to sell up to an additional 10 million common shares of Resverlogix, solely at our discretion, from time to time at a fixed price, determined at that time, to subscribers in certain jurisdictions outside Canada during the period that the EDA remained effective. The EDA remained effective until November 2013. The ATM Shares were sold by way of transactions that are "at-the-market distributions", including sales on the Toronto Stock Exchange and other existing trading markets in Canada. The timing of any sale of ATM Shares and the number of ATM Shares sold was at our discretion. The agents did not purchase shares of Resverlogix as principal under the EDA.

We were permitted to sell up to a maximum of approximately CAD\$11.0 million of ATM Shares, and the number of ATM Shares sold on any trading day could not exceed 25% of the total trading volume of the common shares on that trading day. During the year ended April 30, 2014, we sold 5,053,300 ATM Shares at an average price of CAD\$1.02 for gross proceeds of US\$4.9 million (CAD\$5.2 million). No shares were sold under the EDA during the year ended April 30, 2015.

Citibank Loan

In August 2012, we entered into a CAD\$25 million Loan Agreement with Citibank, N.A. ("Citibank"). We received the CAD\$25 million upon closing of the loan. In March 2013, we entered into an Amended and Restated Loan Agreement with Citibank, increasing the previous loan granted to us by Citibank from CAD\$25 million to CAD\$38.8 million. In August 2014, we entered into a Second Loan Amendment with Citibank which provided for the existing loan granted to us by Citibank to be increased by CAD\$30 million to CAD\$68.8 million. The entire CAD\$68.8 million loan will be repayable upon maturity on August 28, 2017 and may be repaid in whole or in part without penalty. Interest on the loan will be payable annually in arrears at a rate equal to the per annum Canadian one-year LIBOR swap rate plus 3.14%, to be reset annually. Effective August 27, 2014, the annual interest rate was reset from 4.4473% to 4.4410%. The loan is secured by an irrevocable CAD\$68.8 million Standby Letter of Credit arranged by Eastern which will be maintained until maturity of the loan.

In connection with the Standby Letter of Credit, we agreed to indemnify Eastern for all liabilities, costs and expenses arising from any payments made to Citibank under the CAD\$68.8 million Standby Letter of Credit and we pledged our issued patents and certain tax losses and pools to Eastern as security for our obligations under the indemnity. We issued 1,320,000 share purchase warrants to Eastern in August 2012, an additional 728,640 share purchase warrants in March 2013, and 5,000,000 share purchase warrants in August 2014. We also agreed to pay a guarantee fee to Eastern in the amount of 0.03% per annum on the average daily aggregate principal amount of the issued and undrawn letter of credit. The warrants issued in August 2012 are exercisable at a price of CAD\$1.58 and expire on August 27, 2017. The warrants issued in March 2013 are exercisable at a price of CAD\$2.38 and expire on March 8, 2018. The warrants issued in August 2014 are exercisable at a price of CAD\$0.75 and expire on August 15, 2019. In June 2013, in connection with the spin out of our epigenetics drug discovery platform to Zenith, as described under "Spin Out of Epigenetics Drug Discovery Platform" herein, the exercise prices of the August 2012 and March 2013 warrants were adjusted to CAD\$1.44 and CAD\$2.16, respectively.

Private Placements

On April 27, 2015, we and Shenzhen Hepalink Pharmaceutical Co., Ltd. ("Hepalink") entered into a Framework Agreement which sets forth the principal business terms for an equity investment and a license of RVX-208 for all indications to Hepalink. On July 20, 2015, we closed an equity investment with Hepalink. Under the terms of the transaction, Hepalink subscribed for 13,270,000 Resverlogix common shares and 1,000,000 common share purchase warrants, for aggregate proceeds of CAD\$35 million, or CAD\$2.67 per unit. Each warrant is exercisable at a price of CAD\$2.67 for a period of five years. The common shares and warrants issued to Hepalink are subject to a three year lock-up period. Pursuant to a Share Restriction Agreement, Hepalink is also entitled to nominate one mutually agreed representative for election to our board of directors, pursuant to a Nomination Rights Agreement.

In addition, Eastern purchased 5,600,000 common shares and 422,005 common share purchase warrants for aggregate consideration of approximately CAD\$15 million, or CAD\$2.67 per unit. Each warrant is exercisable at a price of CAD\$2.67 for a term of five years.

Licensing

Licensing Agreement

As stated above, on April 27, 2015, we and Hepalink entered into a Framework Agreement which sets forth the principal business terms for an equity investment and a license of RVX-208 for all indications to Hepalink. On July 8, 2015, we and Hepalink entered into a License Agreement covering a license of RVX-208 for China, Hong Kong, Taiwan and Macau (the "Territories"), for all indications. The license between us and Hepalink provides for certain milestone payments based on net sales of RVX-208 in the licensed territories. The annual sales milestones range from 500 million renminbi ("RMB") to RMB 10 billion (US\$81 million to US\$1,611 million), with Resverlogix being eligible to receive sales-based milestone payments from Hepalink ranging from US\$5 million to US\$90 million. In addition, Hepalink shall pay a royalty of 6% of annual net sales of RVX-208 in the Territories, subject to certain adjustments. Hepalink will be responsible for all clinical and development costs in the Territories, including a patient population that is expected to be included in our planned Phase 3 BETonMACE clinical study.

Recent Developments

As described under "General Development of the Business – Clinical Development – Pooled ASSURE and SUSTAIN Analysis - Ongoing", on June 1, 2015, we announced new information from the analysis of data pooled from the ASSURE and SUSTAIN clinical trials relating to specific biomarkers relevant to CKD. As described under "Description of Business – Chronic Kidney Disease", on June 1, 2015, we announced results of an analysis examining the effects of RVX-208 on key renal parameters.

As described under "General Development of the Business – Clinical Development – Pre-Diabetes Mellitus - Completed", on June 8, 2015, we announced findings from a study performed by the Baker IDI Heart and Diabetes Institute, Melbourne, Australia and our scientists which were reported at the American Diabetes Association Scientific Sessions.

As described under "General Development of the Business – Financing", on July 20, 2015 we closed an equity investment with Hepalink.

As described under "General Development of the Business – Licensing", on April 27, 2015 we and Hepalink entered into a Framework Agreement which sets forth the principal business terms for an equity investment and a license of RVX-208 for all indications to Hepalink, and on July 8, 2015, we and Hepalink entered into a License Agreement.

Risk Factors

An investment in the Company should be considered highly speculative due to the nature of its activities and the stage of its development. The biotechnology industry generally may be regarded as uncertain given the nature of the industry. Accordingly, investments in biotechnology companies should be regarded as speculative. Biotechnology research and development involves a significant degree of risk. The risks and uncertainties set forth below are not the only ones we will face. Additional risks and uncertainties not presently known to us or that we currently consider immaterial may also impair our business and operations and cause the price of the Common Shares to decline. If any of the following risks actually occur, our business may be harmed and our financial condition and results of operations may suffer significantly. In that event, the value of the Common Shares could decline and purchasers of the Common Shares may lose all or part of their investment. Readers should carefully consider the following risk factors in addition to the other information contained herein before investing in the Common Shares.

Risks Relating to Our Business

We are a development stage company. If we do not develop commercially successful products, we may be forced to cease operations.

We are in an early stage of development, which may require significant additional investment for research and development, manufacturing, clinical testing, and regulatory submissions prior to commercialization. Investors must evaluate our business in light of the uncertainties and complexities affecting a development stage biotechnology company and there can be no assurance that any such product will eventually be developed. Any product would be unsuccessful if it:

- does not demonstrate acceptable safety and efficacy in preclinical studies and clinical trials or otherwise does not meet applicable regulatory standards for approval;
- does not offer therapeutic or other improvements over existing drugs used to treat the same or similar conditions;
- is not capable of being produced in commercial quantities at an acceptable cost, or at all; or
- is not accepted by patients, the medical community or third party payors.

A commitment of substantial time and resources is required to conduct research and clinical trials if we are to complete the development of any product. We have not proven our ability to develop and commercialize products. It is not known whether any of these products will meet applicable health regulatory standards and obtain required regulatory approvals, or (i) whether such products can be produced in commercial quantities at reasonable costs and be successfully marketed, (ii) whether our products will achieve market acceptance, or (iii) if our investment in any such products will be recovered through sales or royalties. Problems frequently encountered in connection with the development and utilization of new and unproven technologies and the competitive environment in which we operate might limit our ability to develop commercially successful products.

Results of early research and development may not be indicative of the results that will be obtained in later stages of research and development. If regulatory authorities do not approve the products or if regulatory compliance is not maintained, we would have limited ability to commercialize our products, and our business and results of operations would be harmed. We may fail to develop any products, to obtain regulatory approvals, to enter clinical trials, or to commercialize any products. If we are unable to make our product candidates commercially available, we will not generate product revenues, and we may be forced to cease operations.

We have a history of net losses. We expect to continue to incur substantial net losses for the foreseeable future, and we may never achieve or maintain profitability.

To date, we have not recorded any revenues from the sale of biopharmaceutical products, and have incurred significant net losses in each year since our inception. As at April 30, 2015, we had a deficit of US\$248.2 million. We expect to incur substantial net losses and negative cash flow for the foreseeable future. Such losses have had, and will continue to have, an adverse effect on our shareholders' equity and working capital.

The process of developing and commercializing our products requires significant preclinical testing and clinical trials as well as regulatory approvals for commercialization and marketing before we could begin product sales. In addition, commercialization of our products would require us to establish a sales and marketing organization or contractual relationships to enable product manufacturing and other related activities. Because of the numerous risks and uncertainties associated with pharmaceutical product development and commercialization, we are unable to accurately predict the timing or amount of future expenses or when, or if, we will be able to achieve or maintain profitability. Currently, we have no products approved for commercial sale, and to date we have not generated any product revenue. We have financed our operations primarily through the sale of equity securities and a credit facility. The size of our future net losses will depend, in part, on the rate of growth or contraction of our expenses and the level and rate of growth, if any, of our revenues. We expect to incur losses unless and until such time as payments, if any, from corporate collaborations, product sales and/or royalty payments generate sufficient revenues to fund our continuing operations. Quarter to quarter fluctuations in revenues, expenses and losses are also expected. Even if we do achieve profitability, we may not be able to sustain or increase profitability on an ongoing basis.

We will need to raise additional capital in the future to fund our operations. If we cannot raise additional capital, we will have to delay, reduce or cease operations.

We will need to raise additional capital to fund our operations and to develop our products. We expect to attempt to raise additional funds through public or private equity or debt financing and/or from other sources. Our future capital requirements will be substantial and will depend on many factors, such as the following:

- the scope, rate of progress, results and costs of any clinical and preclinical programs;
- timing, costs and outcomes of regulatory proceedings;
- the cost and timing of developing sales and marketing operations or partnerships;
- payments received under any future partnerships;
- prosecution or defense of patent claims;
- the cost and timing of developing manufacturing capacity;
- costs associated with commercialization of our products; and
- competing technological and market developments, including the introduction by others of new therapies in our market.

On July 20, 2015, we closed a CAD\$50 million (US\$39 million) private placement with Hepalink and Eastern.

We believe our cash as at April 30, 2015, in combination with cash received subsequent to April 30, 2015 from the private placement described above, is sufficient to fund our contractual commitments for at least the next year, and is sufficient to fund substantially all of our planned business operations for at least the next year. We may raise additional capital through private placements and/or prospectus offerings.

Further, changing circumstances may cause us to consume capital significantly faster or slower than we currently anticipate. We have based these estimates on assumptions that may prove to be wrong, and we could utilize our available financial resources sooner than we currently expect.

There can be no guarantee that we will be able to access capital markets in the future to fund our ongoing operations. If we cannot access capital markets in the future we may be forced to cease operations. Any financing transaction may contain unfavorable terms. If we raise additional funds by issuing equity securities, our stockholders' equity will be diluted. If we raise additional funds through strategic partnerships, we may be required to relinquish rights to our products, or to grant licenses on terms that are not favorable to us.

Unstable market conditions may have serious adverse consequences on our business.

Our business strategy may be adversely affected by unpredictable and unstable market conditions. If the current equity and credit markets deteriorate it may make any necessary equity or debt financing more difficult, more costly, and more dilutive. Failure to secure any necessary financing in a timely manner and on favorable terms could have a material adverse effect on our growth strategy, financial performance and stock price and could require us to delay or abandon clinical development plans. There is a risk that one or more of our current or future strategic partners may encounter difficulties during challenging economic times, which would directly affect our ability to attain our operating goals on schedule and on budget.

If our clinical trials fail to establish the safety and efficacy of our products, including RVX-208, we will not be able to commercialize our products.

Drug discovery and development has inherent risk and the historical failure rate is high. Failures in the HDL cholesterol market by some pharmaceutical companies have highlighted the risk of these types of therapies.

On June 27, 2013, we announced topline ASSURE data and that ASSURE did not meet its primary endpoint of a -0.6% change in PAV. However, on September 3, 2013 we announced the results of subgroup analysis of 281 treated patients in ASSURE. Current findings show that the subgroup with below median HDL (<39 mg/dL) baseline population consisted of 92 patients who were taking either rosuvastatin (Crestor®) or atorvastatin (Lipitor®) together with RVX-208. Those patients taking rosuvastatin and RVX-208 had a highly statistically significant PAV plaque regression of -1.43% with probability value of $p < 0.002$ v.s. baseline. This PAV regression exceeded the trial's pre-specified PAV endpoint (-0.6%) by more than two-fold. But those patients taking atorvastatin (Lipitor®) together with RVX-208 had a PAV plaque progression of +0.19% with a non-significant probability value v.s. baseline.

To obtain regulatory approval to market and sell any of our products, we must satisfy the United States Federal Drug Administration ("FDA"), Health Canada's Therapeutic Products Directorate (the "TPD"), and other regulatory authorities, through extensive clinical trials and preclinical studies, that our products are safe and efficacious. If we cannot demonstrate that our drugs, including RVX-208, are safe and effective for human use, we may need to abandon one or more of our drug development programs.

We may not have conducted or may not conduct in the future the types of testing ultimately required by regulatory authorities, or future tests may indicate that our products are not safe for use in humans. Preclinical testing and clinical trials are expensive, can take many years and have an uncertain outcome. In addition, success in initial preclinical testing does not ensure that later preclinical testing or clinical trials will be successful. There are a number of factors that could cause a clinical trial to fail or be delayed including:

- the clinical trials may produce negative or inconclusive results, which may require us to conduct additional preclinical testing or to abandon product candidates that we believed to be promising;
- the regulators may require that we hold, suspend or terminate clinical research for noncompliance with regulatory requirements;
- we, our potential partners, the FDA, the TPD or foreign regulatory authorities could suspend or terminate a clinical trial due to adverse side effect of a drug on subjects or patients in the trial;
- we may decide, or regulators may require us, to conduct additional preclinical testing or clinical trials;
- enrollment in our clinical trials may be slower than anticipated;
- the cost of our clinical trials may be greater than anticipated;
- our products may have unfavorable pharmacology, toxicology or carcinogenicity;
- our product candidates may cause undesirable side effects; and
- the supply or quality of our drugs or other materials necessary to conduct clinical trials may be insufficient, inadequate or delayed.

If any of our drugs in clinical studies, including RVX-208, do not show sufficient efficacy in patients with the targeted indication, it could negatively impact our development and commercialization or goals for this and other drugs and, as a result, materially adversely affect our business, financial condition and results of operations.

We may be required to conduct additional clinical trials to address concerns that the use of our leading product, RVX-208, might increase the risk of liver injury. This may materially adversely affect our business, financial condition and results of operations.

In our Phase 2 ASSERT clinical trial, some patients had elevations in serum enzymes which are sensitive markers of liver injury; however other clinical laboratory tests indicate there was no impairment in liver function and patients were asymptomatic for liver injury. Most of these liver signals occurred between weeks five and ten with less occurrence between weeks ten and thirteen. In our subsequent Phase 2b clinical trials, SUSTAIN and ASSURE I, increases in ALTs were observed in a small group of patients. Those who had ALT elevations of 3X ULN all dosed through the trial which potentially illustrated adaptability to the drug. Those who had elevation greater than 5XULN, a high number of those patients had pre-existing liver condition such as hepatitis and took known agents that cause ALT elevations such as acetaminophen, clavulanic acid, diclofenac, and Augmentin. These increases were all observed within weeks 12 and 24 of the trial. Upon stopping RVX-208 ALT elevations returned to ULN quickly which further illustrates a lack of hepatotoxicity. We also performed the FDA's liver analysis tool ("eDISH") which further illustrated that there were no Hy's Law (elevated ALT and total bilirubin) cases. With these learnings, we believe that the current therapeutic regimen can be safe with regard to effects on the liver. However, if further tests were to determine such risk did exist, the FDA may require us to conduct additional clinical trials to address these concerns prior to receiving FDA or foreign regulatory approval for RVX-208. These clinical trials would be expensive and could delay any commercialization of RVX-208. Adverse results in these trials could delay or prevent commercialization of RVX-208 or could jeopardize existing development in other indications.

If our testing assumptions are incorrect our products may not be approved for marketing.

The design of our clinical trials is based on many assumptions about the expected effect of our product candidates. If those assumptions prove incorrect, the clinical trials may not produce statistically significant results. We cannot assure you that the design of, or data collected from, the clinical trials of our product candidates will be sufficient to support the FDA and foreign regulatory approvals.

We are dependent on third parties to conduct our clinical trials and to provide services for certain important aspects of our business. If these third parties do not perform as contractually required or expected, we may not be able to obtain regulatory approval for our products, or we may be delayed in doing so.

We rely on third parties, such as contract research organizations, medical institutions, academic institutions, independent clinical investigators and contract laboratories, to conduct our clinical trials and preclinical studies, and we expect to continue to do so in the future. We rely heavily on these parties for successful execution of our clinical trials, but do not control many aspects of their activities. As a result, many important aspects of our product development are outside our direct control. We are responsible for confirming that our preclinical studies are conducted in accordance with applicable regulations and that each of our clinical trials is conducted in accordance with its general investigational plan and protocol. The FDA requires us to comply with regulations and standards, commonly referred to as good laboratory practices, or GLP, for conducting and recording the results of our preclinical studies and good clinical practices for conducting, monitoring, recording and reporting the results of clinical trials, to assure that data and reported results are accurate and that the clinical trial participants are adequately protected. Our reliance on third parties does not relieve us of these responsibilities. If the third parties conducting our clinical trials do not perform their contractual duties or obligations, do not meet expected recruitment or other deadlines, fail to comply with the FDA's good clinical practice regulations, do not adhere to our clinical trial protocols or otherwise fail to generate reliable clinical data, development, approval and commercialization of our products, including RVX-208, may be extended, delayed or terminated or may need to be repeated, and we may not be able to obtain regulatory approval.

We do not currently own or operate manufacturing facilities for clinical or commercial production of the active pharmaceutical ingredient, or API, used in RVX-208. As a result, we rely on third parties to supply the API. We expect to continue to depend on third parties to supply the API for our lead product candidate and any additional product candidates we develop in the foreseeable future. An API manufacturer must meet high precision and quality standards for that API to meet regulatory specifications and comply with regulatory requirements. A contract manufacturer's failure to comply with applicable regulations and requirements could result in refusal to approve or a delay in approval of RVX-208 or other product candidate. We are ultimately responsible for confirming that the APIs used in our product candidates are manufactured in accordance with applicable regulations. Furthermore, if our third party drug suppliers fail to achieve and maintain high manufacturing standards in compliance with applicable regulations, we could be subject to certain product liability claims in the event such failure to comply resulted in defective products that caused injury or harm.

We rely on partnerships and strategic relationships for our success. The failure to successfully collaborate with third parties may delay, prevent or otherwise impair the development or commercialization of our products or revenue expectations.

As a result of the costs associated with commercializing a product candidate we seek strategic partnerships with corporate and academic collaborators, licensors, licensees and others for the research, development, clinical testing, manufacturing, marketing and commercialization of our products, and we intend to attract corporate partners and enter into additional research collaborations. Our goal is to partner RVX-208 so that it may be developed for clinical conditions. There can be no assurance, however, that such collaborations will be established, that such collaborations will be established on favourable terms, if at all, or that future collaborations will be successful. In particular, failures in HDL cholesterol therapies may negatively impact our potential partners' willingness to enter into partnering agreements due to the potential risks in the cholesterol market and the high clinical costs to bring such drugs to market. Failure to attract commercial partners for our products may result in our incurring substantial clinical testing, manufacturing and commercialization costs prior to realizing any revenue from product sales or result in delays or program discontinuance if funds are not available in sufficient quantities, and this may materially adversely affect our business, financial condition and results of operations.

Should a collaborative partner fail to develop, manufacture, or commercialize successfully any product to which it has rights, or any partner's product to which we have rights, the business may be adversely affected. Failure of a collaborative partner to continue to participate in any particular program could delay or halt the development or commercialization of products generated from such program. In addition, there can be no assurance that the collaborative partners will not pursue other technologies or develop alternative products either alone or in collaboration with others, including our competitors, as a means for developing treatments for the diseases targeted by our programs.

Furthermore, we will hold licenses for certain technologies and there can be no assurance that these licenses will not be terminated, or that they will be renewed on conditions acceptable to us. We may negotiate additional licenses in respect of technologies developed by other companies and academic institutions. Terms of license agreements to be negotiated may include, inter alia, a requirement to make milestone payments, which may be substantial. We may also be obligated to make royalty payments on the sales, if any, of products resulting from licensed technology and, in some instances, are responsible for the costs of filing and prosecuting patent applications.

We face significant competition in seeking appropriate strategic partners, and these strategic partnerships can be intricate and time consuming to negotiate and document. We may not be able to negotiate additional strategic partnerships on acceptable terms, or at all. We are unable to predict when or if we will enter into any additional strategic partnerships because of the numerous risks and uncertainties associated with establishing strategic partnerships. If we are unable to negotiate additional strategic partnerships for our products, we may be forced to delay or terminate development or commercialization of one or more of our products. If we elect to increase our expenditures to fund development or commercialization activities on our own, we may need to obtain additional capital, which may not be available to us.

If we enter into partnerships or other strategic relationships, we may lose important rights to and control over the development of our products.

As a result of the costs and risks associated with commercializing a product we will seek strategic partnerships in order to continue to develop and, if approved, market our products. Such strategic partnerships may require us to relinquish control over the timing and manner of clinical trials and commercialization of our products. Strategic partners may experience financial difficulties or choose to terminate the arrangement or independently work on a competing product resulting in the delay or discontinuation of development or commercialization of our product candidates. Furthermore, disputes may arise between us and our strategic partners that result in the delay or termination of the research, development or commercialization of our product candidates or that result in costly litigation or arbitration that diverts management's attention and consumes resources. Strategic partners may not properly maintain or defend our intellectual property rights or may use our proprietary information in a manner that could jeopardize or invalidate our proprietary information or expose us to potential litigation.

We may not receive the full payment of all milestone or royalty payments pursuant partnerships or strategic relationships.

We may enter into license agreements and other forms of agreements with third parties regarding the development and commercialization of our products. These agreements generally require that the third party pays to the Company certain amounts upon the attainment of various milestones and possibly include royalties on the sale of the developed product. There can be no guarantee that we will receive the payments described in those agreements since the development of the products may be cancelled if clinical trials do not yield positive results. Under such circumstances, the Company would not receive royalties as well. Even if the development of a product yields positive results, all of the risks described herein with respect to the obtaining of regulatory approval and market acceptance of the product are applicable. Finally, if there occurs a disagreement between the

Company and the third party, the payment relating to the attainment of milestones or of royalties may be delayed. The occurrence of any of these circumstances could have a material adverse effect on the Company's financial condition and operating results.

If our products fail to achieve market acceptance for any reason, such failure may materially adversely affect our business, financial condition and results of operations.

There can be no assurance that any of our products, if approved for marketing, will achieve market acceptance. If our products, once approved, do not receive market acceptance for any reason, it will adversely affect our business, financial condition and results of operations. The degree of market acceptance of any products we develop will depend on a number of factors, including:

- the clinical efficacy and safety of our products;
- our products' potential advantages over existing and future treatment methods;
- the price of our products; and
- reimbursement policies of government and third-party payers, including hospitals and insurance companies.

If after we obtain regulatory approval to sell our products, physicians, and healthcare payors fail to adopt our products or conclude that our products are not safe and effective, physicians could choose not to use them to treat patients. Our competitors may also develop new technologies or products which are more effective or less costly, or that seem more cost-effective than our products.

In addition, regulations affecting the pricing of pharmaceutical products may change in ways adverse to us. While we cannot predict the likelihood of any regulatory proposals, if a government agency were to adopt proposals limiting market or third-party payor pricing for pharmaceutical products, it could materially adversely affect our business, financial condition and results of operations.

We cannot be certain that we will ever obtain regulatory approvals in Canada, the United States, or other countries. The failure to obtain such approvals may materially adversely affect our business, financial condition and results of operations.

Biotechnology, medical device and pharmaceutical companies operate in a high-risk regulatory environment. The study, manufacture and sale of products are governed by numerous statutes and regulations in the United States, Canada and other countries. We are required to obtain various regulatory approvals prior to being able to study, commercialize and distribute our products. The regulatory review and approval process required to perform a clinical study in Canada, the United States and other countries includes evaluation of preclinical studies and clinical studies, as well as the evaluation of our manufacturing process. This process is complex, lengthy, expensive, resource intensive and uncertain. Securing regulatory approval to market our products also requires the submission of extensive preclinical and clinical data, manufacturing information regarding the process and facility, scientific data characterizing our product and other supporting data to the regulatory authorities in order to establish its safety and effectiveness. We, or our collaborators, may fail to obtain the necessary approvals to commence or continue preclinical or clinical testing of our product candidates, including RVX-208, or to manufacture or market our products in reasonable time frames, if at all.

Governmental authorities in Canada, the United States, or other countries may enact regulatory reforms or restrictions on the development of new therapies that could adversely affect our ability to develop our products. Many of the products and processes that are being currently developed by us require significant development, testing and the investment of significant funds prior to their commercialization. There can be no assurance that RVX-208 or any other drugs we attempt to develop will actually be developed to a commercial level. Completing clinical testing through late stage trials and obtaining required approvals is expected to take several years and to require the expenditure of substantial resources. There can be no assurance that clinical trials will be completed successfully within any specified period of time, if at all. Furthermore, clinical trials may be delayed or suspended at any time by us or by the FDA, the TPD or foreign regulatory authorities if it is determined that the subjects or patients are being exposed to unacceptable risks. We may encounter delays or rejections based on varying regulatory interpretations or changes in regulatory agency policies, during the period in which we develop a product.

No assurance can be given that RVX-208 or any of the other product candidates will prove to be safe and effective in clinical trials or that we will receive the requisite regulatory approval. Moreover, any regulatory approval of a drug which is eventually obtained may be granted with specific limitations on the indicated uses for which that drug may be marketed or may be withdrawn if problems occur following initial marketing or if compliance with regulatory standards is not maintained. We have limited experience in filing and pursuing applications necessary to gain these regulatory approvals.

The approval procedures vary among countries and can involve additional product testing and administrative review periods. The time required to obtain approval in various countries vary from one another. Approval in one country does not ensure approval by the regulatory authorities of other countries. The failure to obtain such approvals may materially adversely affect our business, financial condition and results of operations.

Regulatory authorities may not approve our products even if they meet safety and efficacy endpoints in clinical trials.

The FDA, the TPD and other foreign regulatory agencies can delay, limit or deny marketing approval for many reasons, including finding a product may not be considered safe and effective; the manufacturing processes or facilities may not meet applicable requirements; or changes in approval policies or regulations. Our product candidates may not be approved even if they achieve their endpoints in clinical trials. Regulatory agencies, including the FDA, or their advisors may disagree with our trial design and our interpretations of data from preclinical studies and clinical trials. Regulatory agencies may change requirements for approval even after a clinical trial design has been approved. Regulatory agencies also may approve a product candidate for fewer or more limited indications than requested or may grant approval subject to the performance of post-marketing studies. In addition, regulatory agencies may not approve the labeling claims that are necessary or desirable for the successful commercialization of our product candidates.

Our products will remain subject to ongoing regulatory review even if they receive marketing approval. If we fail to comply with continuing regulations, we could lose these approvals, and the sale of any future products could be suspended.

In the event we receive regulatory approval to market a particular product candidate, United States, Canadian or other foreign regulatory authority could condition approval on conducting additional costly post-approval studies or could limit the scope of approved uses. In addition, the product may later cause adverse effects that limit or prevent its widespread use, force us to withdraw it from the market or prevent or delay our ability to obtain regulatory approvals in additional countries. In addition, we will continue to be subject to regulatory review and periodic inspections to ensure adherence to applicable regulations. After receiving marketing approval, the FDA imposes extensive regulatory requirements on the manufacturing, labeling, packaging, adverse event reporting, storage, advertising, promotion and record keeping related to the product. Failure to comply with the regulatory requirements could result in:

- civil or criminal penalties or fines;
- injunctions;
- product seizures, detentions or import bans;
- voluntary or mandatory product recalls and publicity requirements;
- suspension or withdrawal of regulatory approvals; and
- refusal to approve pending applications for marketing approval of new drugs or supplements to approved applications.

We may be subject to product liability claims if our products harm people, and we do not have product liability insurance.

The manufacture and sale of pharmaceutical products involve an inherent risk of product liability claims and associated adverse publicity. We have entered into human clinical trials that involve inherent risks in the testing of unproven products. We currently have only clinical trial liability insurance for our products; we do not have product liability insurance. We do not know if we will be able to maintain existing or obtain additional clinical trial liability insurance or obtain product liability insurance on acceptable terms or with adequate coverage against potential liabilities. This type of insurance is expensive and may not be available on acceptable terms. If we are unable to obtain or maintain sufficient insurance coverage on reasonable terms or to otherwise protect against potential clinical trial and product liability claims, we may be unable to commercialize our products. A successful clinical trial liability or product liability claim brought against us in excess of our insurance coverage, if any, may require us to pay substantial amounts. This could have a material adverse effect on our business, financial condition and results of operations.

The pharmaceutical industry is extremely competitive. If our competitors develop and market products that are more effective, safer or less costly than any future products that we may develop, our commercial opportunity will be reduced or eliminated.

The technological competition we face from new and established pharmaceutical companies, biopharmaceutical companies and universities is intense and is expected to increase, in particular in the market for therapeutic products to treat, mitigate or prevent cardiovascular disease. Competitors may develop products more quickly and obtain regulatory approval for such products more rapidly, or develop products which are more effective than those which we intend to develop. Our commercial opportunity will be reduced or eliminated if our competitors develop and commercialize products that are safer, more effective, have fewer side effects or are less expensive than any future products that we may develop and commercialize. In addition, significant delays in the development of our product candidates could allow our competitors to bring products to market before us and impair our ability to commercialize our product candidates. Research and development by others may render our technology or products obsolete or noncompetitive or produce treatments or cures superior to any therapy developed or to be developed by us.

We anticipate that, if approved for the reduction of MACE in cardiovascular / atherosclerotic disease, RVX-208 would be positioned to be used in conjunction with leading standard of care statin treatments such as Lipitor and Crestor to further reduce major adverse cardiac events such as myocardial infarction, stroke and death and potentially compete with other therapeutic programs in development, such as, the LDL reduction programs (PCSK9), peptide programs, ApoA-I infusion treatments, delipitated HDL programs and cholesteryl transfer protein (“CETP”) inhibitors.

We anticipate that, if RVX-208 is approved for reduction of CVD risk and MACE and it improves other biomarkers such as eGFR and ALP, it would then potentially compete with, or be added to, novel and existing CKD products in clinical development.

We anticipate that, if approved for neurodegenerative disorders, including mild cognitive impairment / AD / dementia, RVX-208 would potentially be used in conjunction with standard of care therapies such as Aricept to improve therapeutic outcomes and/or compete with other agents and novel approaches to this disease such as small molecules, Namenda and PBT2, and monoclonal antibody technologies (“MOABs”) such as Bapineuzumab.

We anticipate that, if approved for reduction of CVD risk and MACE in diabetes mellitus patients, RVX-208 would potentially be a complimentary agent added to standard of care diabetes mellitus agents in clinical development.

Many of our competitors have significantly greater financial resources and expertise in research and development, manufacturing, preclinical testing, conducting clinical trials, obtaining regulatory approvals and marketing approved products than we do. Established pharmaceutical companies may invest heavily to discover quickly and develop novel compounds or drug delivery technology that could make our product candidates obsolete. Smaller or early stage companies may also prove to be significant competitors, particularly through strategic partnerships with large and established companies. In addition, these third parties compete with us in recruiting and retaining qualified scientific and management personnel, establishing clinical trial sites and patient registration for clinical trials, as well as in acquiring technologies and technology licenses complementary to our programs or advantageous to our business. Accordingly, our competitors may succeed in obtaining patent protection, receiving FDA approval or discovering, developing and commercializing products before we do. If we are not able to compete effectively against our current and future competitors, our business will not grow and our financial condition will suffer.

We are dependent upon retaining and attracting key personnel and others, the loss of which could materially adversely affect our business, financial condition and results of operations.

We depend on certain members of our management and scientific staff, the loss of whose services might significantly delay or prevent the achievement of research, development or business objectives and would materially adversely affect our business, financial condition and results of operations. We do not have employment agreements with any of our senior management that would prevent them from leaving us. In addition, our success depends, in large part, on our ability to improve our management systems and attract and retain qualified management, scientific and medical personnel, and on our ability to develop and maintain important relationships with commercial partners, leading research institutions and key distributors. We face intense competition for such personnel and relationships. We cannot assure that we will attract and retain such persons or maintain such relationships. In addition, failure to succeed in clinical trials may make it more challenging for us to recruit and retain qualified scientific personnel.

We may not be able to attract, train and retain a sufficient number of qualified employees to maintain and grow our business.

We expect that our potential expansion into areas and activities requiring additional expertise, such as further clinical trials, governmental approvals, manufacturing, sales, marketing and distribution will place additional requirements on our management, operational and financial resources. We expect these demands will require an increase in management and scientific personnel and the development of additional expertise by existing management personnel. There is currently aggressive competition for employees who have experience in technology and engineering. The failure to attract and retain such personnel or to develop such expertise could materially adversely affect our business, financial condition and results of operations.

Confidentiality agreements with employees and others may not adequately prevent disclosure of trade secrets and other proprietary information. Disclosure of this information may materially adversely affect our business, financial condition and results of operations.

We rely on trade secrets, which we seek to protect, in part, through confidentiality and non-disclosure agreements with our employees, collaborators, suppliers, and other parties. There can be no assurance that these agreements will not be breached, that we would have adequate remedies for any such breach or that our trade secrets will not otherwise become known to or independently developed by our competitors. We might be involved from time to time in litigation to determine the enforceability, scope and validity of our proprietary rights. Any such litigation could result in substantial cost and divert management’s attention from our operations.

We may need to implement additional finance and accounting systems, procedures and controls in the future as we grow and to satisfy new reporting requirements.

As we grow we may access capital markets more broadly which could require us to implement additional finance and accounting systems along with enhanced internal control systems. This will result in increased costs to us as we continue to undertake efforts to comply with best practices and applicable rules and requirements applicable to public companies. These rules may make it more difficult and costly for us to obtain certain types of insurance, including director and officer liability insurance, and we may be forced to accept reduced policy limits and coverage or incur substantially higher costs to obtain the same or similar coverage as compared to the policies previously available to public companies. The impact of these events could also make it more difficult for us to attract and retain qualified persons to serve on our board of directors or our board committees or as executive officers. In addition, we may need to hire additional legal and accounting staff with appropriate experience and technical knowledge, and we cannot assure that if additional staffing is necessary that we will be able to do so in a timely fashion.

Our products may not be eligible for reimbursement from government or private third-party payors, or may be eligible for reimbursement at lower prices than we currently anticipate, which could materially adversely affect our business, financial condition and results of operations.

Our ability to successfully market therapeutic products depends in part on the extent to which reimbursement for the cost of such products and related treatments will be available from government health administration authorities, private health insurers and other healthcare organizations. Significant uncertainty exists as to whether newly-approved pharmaceutical products will qualify for reimbursement from these organizations. Furthermore, challenges to the price of medical products continue to grow in frequency due to increased focus on cost containment and pharmacoeconomic issues. These recent changes will become more pronounced as leading therapeutics in the atherosclerosis market such as statins continue to come off patent. Health authorities will continue to increase their scrutiny and pharmacoeconomic diligence on new products in all disease areas including those for the cardiovascular market. These rapid changes in the healthcare reimbursement marketplace will potentially have a significant impact on the future marketability of new drugs in development and could materially adversely affect our business, financial condition and results of operations. It is expected that new drug entrants will not only have to be effective and safe but also have to provide a clear value proposal to health systems, such as risk reduction in major adverse cardiovascular events, over the current standard of care therapy, statin therapy.

In light of these market changes in drug development, pricing of drug therapies has come under significant pressure with government authorities and private health insurers around the world. The top current leading reimbursed markets; USA, Japan, Germany, UK, France, Spain, Italy, and Canada, have implemented healthcare reforms that focus specifically on value and reimbursement. Reforms such as reference based pricing, pharmacoeconomics, and numbers needed to treat are a few of the many instruments that healthcare organizations utilize to ensure maximum value for reimbursed therapeutics. Healthcare reform is underway in these top global markets and there is additional uncertainty about the viability of current pricing methodologies for reimbursement. There can be no assurance that adequate third-party coverage will be available to establish price levels which would allow us to realize an acceptable return on our investment in product development. If we cannot realize an acceptable return on our investment in product development we may need to delay or cease our product development.

We have been advanced funds under our loan agreement with Citibank and we must repay such funds when they become due and payable.

Under our loan agreement with Citibank, Citibank advanced to us a total of CAD\$68.8 million in August 2012, March 2013, and August 2014. We are required to pay annual interest payments on the funds advanced to us under the loan agreement and to repay the loan in full by August 2017. Our ability to repay our indebtedness to Citibank under the loan agreement when principal and interest payments are due and payable will depend upon our available capital resources at such time. If we do not have sufficient capital resources to make such payments, we may need to seek additional funding through public or private equity or debt financing, or we may be required to divert capital that would otherwise have been used for research or development projects, which could adversely affect our business, financial condition, prospects and results of operations.

Failure to repay our indebtedness could result in a loss of our intellectual property.

If we are unable to repay amounts owing under the loan agreement with Citibank, Citibank could proceed to foreclose or otherwise realize upon the collateral granted to them to secure the indebtedness. The collateral consists of a CAD\$68.8 million Standby Letter of Credit arranged by Eastern Capital Limited. We agreed to indemnify Eastern Capital Limited for all liabilities, costs and expenses arising from any payments made to Citibank under the Standby Letter of Credit and we have pledged our patents and certain tax losses and pools to Eastern as security for our obligations under the indemnity. In the event that we are unable to satisfy any indemnity obligation to Eastern, Eastern would be entitled to foreclose or otherwise realize upon our patents and certain tax losses and pools to satisfy the indemnity obligation.

Variations in interest rates could adversely affect our financial condition.

Our indebtedness under the loan agreement with Citibank is at variable rates of interest and exposes us to interest rate risk. If interest rates increase, our debt service obligations on the indebtedness and our net loss both would increase and our cash flows would decrease.

It may be difficult or impossible for U.S. investors to enforce judgments against us, our directors or our officers in Canada.

We were formed under the laws of the Province of Alberta. Some of the members of our board of directors and our officers are residents of countries other than the United States. As a result, it may be impossible for U.S. investors to affect service of process within the United States upon us or these persons or to enforce against us or these persons any judgments in civil and commercial matters, including judgments under U.S. federal or state securities laws. In addition, a Canadian court may not permit U.S. investors to bring an original action in Canada or to enforce in Canada a judgment of a state or federal court in the United States.

Risks Relating to our Intellectual Property**We may not accurately predict the protection afforded by our patents and proprietary technology and if our predictions are wrong, this may materially adversely affect our business, financial condition and results of operations.**

Our success will depend in part on our ability to obtain, maintain, and enforce patent rights, maintain trade secret protection and operate without infringing the proprietary rights of third parties, both domestically and internationally.

Our patent positions are highly uncertain, and any future patents we receive for our potential products will be subject to this uncertainty, which may adversely affect our business, financial condition and results of operations. There can be no assurance that pending patent applications will be allowed and that we will develop additional proprietary products that are patentable, that issued patents will provide any competitive advantage or will not be challenged by any third parties, or that patents of others will not have an adverse effect on the ability to do business. Furthermore, there can be no assurance that others will not independently develop similar products, duplicate any of the products, or design around the products patented by us. In addition, we may be required to obtain licenses under patents or other proprietary rights of third parties. No assurance can be given that any licenses required under such patents or proprietary rights will be available on terms acceptable to us. If such licenses are not obtained we could encounter delays in introducing one or more of our products to the market, while we attempt to design around such patents, or could find that the development, manufacturing or sale of products requiring such licenses could be foreclosed. In addition, we could incur substantial costs in defending suits brought against us on such patents or in suits in which we attempt to enforce our own patents against other parties. Such disputes could involve arbitration, litigation or proceedings declared by the United States Patent and Trademark Office or International Trade Commission or other foreign patent authorities. Intellectual property litigation can be extremely expensive, and this expense, as well as other consequences should we not prevail, could seriously harm our business. With respect to any infringement claim asserted by a third party, we can give no assurances that we will be successful in the litigation or that such litigation would not have a material adverse effect on our business, financial condition and results of operation.

Until such time, if ever, that patent applications are filed and/or approved, our ability to maintain the confidentiality of the described technology may be crucial to our ultimate possible commercial success. While procedures have been adopted to protect the confidentiality of our technology through signed invention and service agreements, no assurance can be given that such arrangements will be effective, that third parties will not gain access to trade secrets or disclose the technology, or that we can meaningfully protect our rights to our trade secrets.

Even if valid and enforceable patents cover our products and technologies, such patents will provide protection only for a limited amount of time.

Even if patents are issued regarding our product candidates or methods of using them, those patents can be challenged by our competitors who can argue that our patents are invalid and/or unenforceable. Third parties may challenge our rights to, or the scope or validity of, our patents. Patents also may not protect our products if competitors devise ways of making these or similar product candidates without legally infringing our patents. The Federal Food, Drug and Cosmetic Act and the FDA regulations and policies provide incentives to manufacturers to challenge patent validity or create modified, non-infringing versions of a drug or device in order to facilitate the approval of generic substitutes. These same types of incentives encourage manufacturers to submit new drug applications that rely on literature and clinical data not prepared for or by the drug sponsor.

We also rely on trade secrets to protect our technology, especially where we do not believe that patent protection is appropriate or obtainable. However, trade secrets are difficult to protect. The employees, consultants, contractors, outside scientific collaborators and other advisors of our company and our strategic partners may unintentionally or willfully disclose our confidential information to competitors. Enforcing a claim that a third party illegally obtained and is using our trade secrets is expensive and time consuming

and the outcome is unpredictable. Failure to protect or maintain trade secret protection could adversely affect our competitive business position.

Our research and development collaborators may have rights to publish data and other information in which we have rights. In addition, we sometimes engage individuals or entities to conduct research that may be relevant to our business. The ability of these individuals or entities to publish or otherwise publicly disclose data and other information generated during the course of their research is subject to certain contractual limitations. These contractual provisions may be insufficient or inadequate to protect our trade secrets and may impair our patent rights. If we do not apply for patent protection prior to such publication or if we cannot otherwise maintain the confidentiality of our technology and other confidential information, then our ability to receive patent protection or protect our proprietary information may be jeopardized.

Litigation or other proceedings or third party claims of intellectual property infringement could require us to spend time and money and could shut down some of our operations.

Our commercial success depends in part on not infringing patents and proprietary rights of third parties. Others have filed, and in the future are likely to file, patent applications covering products that are similar to our product candidates, as well as methods of making or using similar or identical products. If these patent applications result in issued patents and we wish to use the claimed technology, we would need to obtain a license from the third party. We may not be able to obtain these licenses at a reasonable cost, if at all.

In addition, administrative proceedings, such as interferences and reexaminations before the U.S. Patent and Trademark Office, could limit the scope of our patent rights. We may incur substantial costs and diversion of management and technical personnel as a result of our involvement in such proceedings. In particular, our patents and patent applications may be subject to interferences in which the priority of invention may be awarded to a third party. We do not know whether our patents and patent applications would be entitled to priority over patents or patent applications held by such a third party. Our issued patents may also be subject to reexamination proceedings. We do not know whether our patents would survive reexamination in light of new questions of patentability that may be raised following their issuance.

We may be subject to claims for intellectual property infringement from former employers of our key employees, which could result in loss of intellectual property, our key employees or both.

Many of our employees were previously employed at universities or other biotechnology or pharmaceutical companies, including competitors or potential competitors. We could be subject to claims that these employees or we have inadvertently or otherwise used or disclosed trade secrets or other proprietary information of their former employers. In many cases, litigation may be necessary to defend against these claims.

Even if we are successful in defending against these claims, litigation could result in substantial costs and be a distraction to management. If we fail in defending such claims, in addition to paying money claims, we may lose valuable intellectual property rights or personnel. A loss of key research personnel or their work product could hamper or prevent the ability to commercialize certain product candidates, which could severely harm our business, financial condition and results of operations.

Risks Relating to Owning our Common Shares

Our share price has been and may continue to be extremely volatile. It may be difficult to resell our common shares.

The market price of our common shares has fluctuated substantially in the past, including subsequent to our June 27, 2013 announcement concerning our Phase 2b ASSURE clinical trial, and could fluctuate substantially in the future. During the twelve months preceding April 30, 2015, the closing market price of our common shares ranged from CAD\$0.42 to CAD\$3.00 per share. In addition, the trading prices of life science and biotechnology company stocks in general have experienced extreme price fluctuations in recent years. The valuations of many life science companies without consistent product revenues and earnings are high based on conventional valuation standards, such as price to revenue ratios. These trading prices and valuations may not be sustained. Any negative change in the public's perception of the prospects of life science or biotechnology companies could depress our stock price regardless of our results of operations. In addition our stock may fluctuate based on a variety of factors, including actual or anticipated regulatory approvals or disapprovals of our products or competing products, actual or anticipated results and timing of our clinical trials, changes in the expected or actual timing of our development programs, changes in our operating results, conditions or trends in the life science and biotechnology industries, announcements by us or our competitors of significant acquisitions, strategic partnerships, joint ventures or capital commitments, additions or departures of key personnel, sales and distributions of our common shares by us or our shareholders, changes in general conditions in the economy or other developments affecting us, our clients, or our competitors, some of which may be unrelated to our performance.

Among other things, volatility in our share price could mean that investors will not be able to sell their shares at or above prices at which they were acquired. The volatility also could impair our ability in the future to offer common stock as a source of additional capital. In addition, in the past, stockholders have often instituted securities class action litigation after periods of volatility in the market price of a company's securities. If a stockholder files a securities class action suit against us, we would incur substantial legal fees, and our management's attention and resources would be diverted from operating our business in order to respond to the litigation.

If we sell common shares in the future, existing common shareholders will experience immediate dilution and our stock price may decrease.

We will need to raise additional capital to fund our operations and to develop our products. We will likely raise such additional capital through the sale of our common shares and/or warrants from time to time. Any such financing transaction will result in our existing common shareholders experiencing immediate dilution.

If our estimates regarding timing of milestones are incorrect our share price may decrease.

For planning purposes, we estimate and may disclose timing of a variety of clinical, regulatory and other milestones. We base our estimates on present facts and a variety of assumptions. Many underlying assumptions are outside our control such as the ability to recruit patients, obtain access to clinical sites as expected or obtain approval from regulatory bodies such as the FDA to enter into trials. If we do not achieve milestones consistent with investors' expectations, the price of our shares would likely decline.

We do not currently intend to pay dividends on our common shares and, consequently, investors' ability to achieve a return on investment will depend on appreciation in the price of our common shares.

We have not to date paid any dividends on our Common Shares. We currently intend to invest our future earnings, if any, to fund the development and growth of our business. The payment of dividends will be at the discretion of our board of directors and will depend on our results of operations, capital requirements, financial condition, future prospects, contractual arrangements, restrictions imposed by applicable law, any limitations on payments of dividends present in any debt agreements we may enter into and other factors our board of directors may deem relevant. If we do not pay dividends, your ability to achieve a return on your investment in the Company will depend on any future appreciation in the market price of our common shares. There is no guarantee that our Common Shares will appreciate in value or even maintain the price at which our holders have purchased their Common Shares.

Dividends

We have not declared or paid any dividends on our Common Shares in our past fiscal years or current financial year.

The ABCA does not permit a corporation to pay dividends if the corporation is, or would after the payment, be unable to pay its liabilities as they become due or the realizable value of the corporation's assets would thereby be less than the aggregate of its liabilities and stated capital of all classes. Our directors may issue preferred shares that have preference over the Common Shares with respect to the payment of dividends, in which case such preference may prevent us from paying dividends on the Common Shares. There are 75,202,620 royalty preferred shares outstanding as at the date hereof. There are no other restrictions on our ability to pay dividends.

We intend to retain any earnings to finance growth and do not expect to pay dividends on our Common Shares in the near future. The Board will review this policy from time to time having regard for our financial condition, financing requirements and other factors considered relevant.

Description of Capital Structure

We are authorized to issue an unlimited number of Common Shares, an unlimited number of Preferred Shares issuable in series and 75,202,620 royalty preferred shares. As at April 30, 2015, we had 86,106,938 Common Shares issued and outstanding, 75,202,620 royalty preferred shares issued and outstanding and no preferred shares are outstanding.

The following is a summary of the rights, privileges, restrictions and conditions attaching to our Common Shares, Preferred Shares, and Royalty Preferred Shares.

Common Shares

The holders of Common Shares are entitled, subject to the rights of holders of any class of preferred shares, to dividends declared by the Board, to one vote per share at meetings of the shareholders and, upon liquidation, dissolution or winding up, to receive pro rata the remaining assets of Resverlogix, subject to the rights of any class of preferred shares.

Preferred Shares

The preferred shares may be issued from time to time in one or more series. The terms of each series of preferred shares, including the number of shares, the designation, rights, privileges, restrictions and conditions, will be determined at the time of creation of each such series by the Board. The preferred shares shall rank senior to the Common Shares and the shares of any other class ranking junior to the preferred shares with respect to the payment of dividends or distribution of capital of Resverlogix in the event of a dissolution, liquidation or winding up of Resverlogix.

Royalty Preferred Shares

On June 3, 2014, 75,202,620 royalty preferred shares were issued to Zenith in connection with the Arrangement. The Royalty Preferred Shares entitle Zenith to cumulative preferential dividends in an amount ranging from 6% to 12% of Net Apo Revenue during any year, subject to an adjustment for tax payable on the dividend. The dividend amount is calculated based on 6% of Net Apo Revenue of up to US\$1 billion, 8% of Net Apo Revenue of between US\$1 billion and US\$2 billion, 10% of Net Apo Revenue between US\$2 billion and US\$5 billion and 12% of Net Apo Revenue in excess of US\$5 billion. The dividend amount in a prescribed dividend payment period may not exceed the aggregate of all amounts received by the Company or its affiliates in respect of and including Net Apo Revenue in such period.

Net Apo Revenue is defined as the aggregate of the following amounts: (i) amounts received by the Company or its affiliates from any person who is not the Company or its affiliate (a “third party”) in consideration for granting a license or other rights to the third party which entitle the third party to research, develop, make, manufacture, modify, administer, offer to sell, sell or distribute one or more of the Apo Products and/or Apo Intellectual Property Rights or amounts received under the terms of such license or other right that are granted to the third party; (ii) the gross consideration received from a third party by the Company, any licensee or their respective affiliates from the sale of any Apo Product (other than consideration received by the Company, any licensee or their respective affiliates from a licensee of such Apo Product or its affiliate); less (A) credits or allowances, if any, actually granted; (B) discounts actually allowed; (C) freight, postage, and insurance charges and additional special packaging charges; (D) customs duties, and excise sales taxes, duties or other taxes imposed upon and paid with respect to such sales (excluding what is commonly known as income taxes); (E) rebates and chargebacks or retroactive price reductions made to federal, state or local governments (or their agencies), or any third party payor, administrator or contractor, including managed health organizations; and (F) commissions related to import, distribution or promotion of any Apo Product paid to third parties (specifically excluding any commissions paid to sales personnel, sales representatives and sales agents who are employees or consultants of, or members of a contract sales force engaged by or on behalf of, the Company, any licensee or their respective affiliates); and (iii) amounts received from a third party by the Company or its affiliates in consideration for the sale of any Apo Intellectual Property Right.

In the event we do not declare and pay the dividend on the applicable payment date, holders of Royalty Preferred Shares are entitled to receive additional cumulative preferential dividends in an amount equal to twenty percent (20%) per annum of the dividend payable on such payment date, subject to a tax adjustment, calculated daily and compounded monthly.

Subject to the ABCA, holders of Resverlogix Royalty Preferred Shares are not entitled to receive notice of or attend meetings of the shareholders of the Company and are not entitled to vote at any such meetings other than in respect of separate meetings of the holders of the Royalty Preferred Shares.

In the event of the liquidation, dissolution or winding-up of the Company, whether voluntary or involuntary, or any other return of capital or distribution of assets of the Company among its shareholders for the purpose of winding up its affairs, the holders of the Royalty Preferred Shares are entitled to receive in respect of each such share, before any distribution of any part of the assets of the Company among the holders of Common Shares or other shares of the Company ranking junior to the Royalty Preferred Shares, an amount equal to the greater of \$1.00 divided by the number of outstanding Royalty Preferred Shares and the amount of any accrued, but unpaid dividends.

Market for Securities

Our Common Shares are listed and posted for trading on the TSX under the symbol “RVX”. Our securities are not listed on any stock exchange in the United States and there is no established trading market for our securities in the United States.

Trading Prices and Volume by Month for Fiscal Year Ended April 30, 2015

Date	High (\$)	Low (\$)	Volume
4/30/2015	3.13	1.55	6,805,900
3/31/2015	1.93	0.70	4,655,400
2/27/2015	1.00	0.63	1,307,400
1/30/2015	0.80	0.45	1,273,600
12/31/2014	0.56	0.41	921,700
11/28/2014	0.64	0.54	309,600
10/31/2014	0.75	0.49	611,700
9/30/2014	0.80	0.58	668,600
8/29/2014	0.84	0.73	800,000
7/31/2014	0.89	0.75	1,124,400
6/30/2014	0.78	0.68	820,800
5/30/2014	0.76	0.61	510,500

Prior Sales

We issued the following securities that are not listed or quoted in the marketplace at the prices set out below during the fiscal year ended April 30, 2015:

Date	Type of Security	Issue Price or Exercise Price of Securities	Number of Securities	Type of Issuance
April 2015	Stock Options	\$1.66	20,000	Pursuant to stock option plan
March 2015	Stock Options	\$1.04	105,000	Pursuant to stock option plan
January 2015	Stock Options	\$0.55	10,000	Pursuant to stock option plan
December 2014	RSUs	N/A	200,000	Pursuant to long term incentive plan
October 2014	Stock Options	\$0.72	215,000	Pursuant to stock option plan
August 2014	Warrants	\$0.75	5,000,000	Pursuant to private placement
June 2014	Stock Options	\$0.75	75,000	Pursuant to stock option plan
May 2014	Stock Options	\$0.65	229,200	Pursuant to stock option plan
May 2014	RSUs	N/A	354,400	Pursuant to long term incentive plan

Escrowed Securities and Securities Subject to Contractual Restrictions on Transfer

The following table indicates securities of the Company that are subject to escrow or contractual restrictions on transfer.

Designation of Class	Number of Securities Held in Escrow or that are subject to contractual restrictions on transfer	Percentage of Class
Common Shares	13,270,000 ⁽¹⁾	12.63%
Common Share Purchase Warrants	1,000,000 ⁽¹⁾	Not applicable

Notes:

- (1) On July 20, 2015, the Company completed a private placement of 13,270,000 common shares and 1,000,000 common share purchase warrants to Hepalink. As a condition of that transaction, Hepalink agreed with the Company not to sell the common shares and warrants for a period of three years.

Directors and Executive Officers

Name, Occupation and Security Holdings

The following table sets forth the name, municipality of residence, year of appointment as a director or executive officer of the Company, and position held with us and principal occupation of each of the directors or executive officers of the Company.

The Board is comprised of five directors. The directors are elected annually by the shareholders and serve until the next annual meeting of shareholders unless their successors are duly elected or appointed prior thereto.

Name and Municipality of Residence	Position	Principal Occupation During Past 5 Years	Director Since
Dr. Peter Johann ^{(2),(3)} Heidelberg, Germany	Director and Chairman	Managing General Partner of NGN Capital, a venture capital firm since 2004.	2009
Donald J. McCaffrey Calgary, Alberta, Canada	Director, President, CEO and Secretary	President, Chief Executive Officer and Secretary of Resverlogix since 2003. President, Chief Executive Officer and Secretary of Zenith Epigenetics since 2013.	2003
Kenneth Zuerblis ⁽¹⁾ Sarasota, Florida, U.S.A.	Director and Chair of the Audit and Finance Committee	Currently a director of the Corporation, Zenith Epigenetics and Stemline Therapeutics. From 2011 to 2012, Executive Vice President and Chief Financial Officer of Savient Pharmaceuticals, Inc., a specialty biopharmaceutical company. From 2008 to 2009, Mr. Zuerblis served as Chief Financial Officer and Senior Vice President of ImClone Systems, a biopharmaceutical company developing biologic medicines in the area of oncology.	2010
Dr. Eldon Smith ^{(1), (2), (3)} Calgary, Alberta, Canada	Director	President and CEO of Eldon R. Smith & Associates Ltd. (a private health care consulting company) since 2001, and is Emeritus Professor of Medicine and Former Dean, Faculty of Medicine, University of Calgary. From 1997 until 2010, Dr. Smith served as Editor-in-Chief of the <i>Canadian Journal of Cardiology</i> .	2010
Kelly McNeill, CA ^{(1),(2),(3)} Winnipeg, Manitoba, Canada	Director	Chief Financial Officer of RTDS Technologies Inc. since 2014. From 2009 to 2014, Mr. McNeill served as Executive Vice President, Finance and Administration, Chief Financial Officer and Secretary of IMRIS Inc., a provider of fully integrated image guided therapy solutions. From 2006 to 2009, Mr. McNeill was Resverlogix's Chief Financial Officer.	2009

Name and Municipality of Residence	Position	Principal Occupation During Past 5 Years	Director Since
A. Brad Cann, CA, CBV Calgary, Alberta, Canada	Chief Financial Officer	Chief Financial Officer of Resverlogix since 2009. Chief Financial Officer of Zenith Epigenetics since 2013.	N/A
Dr. Michael Sweeney, MD	Senior Vice President of Clinical Development	Senior Vice President of Clinical Development of Resverlogix since 2014. From 2007 to 2014, Dr. Sweeney served as Chief Medical Officer and Vice President of Research and Development at Depomed, Inc	N/A
Kenneth Lebioda, BA Calgary, Alberta, Canada	Senior VP Business & Corporate Development	Senior Vice President Business and Corporate Development of Resverlogix since 2005.	N/A
Dr. Norman Wong, BSc, MSc, MD, FRCP(C) Calgary, Alberta, Canada	Co-Founder, Chief Scientific Officer and Chairman of the Scientific Advisory Board	Acted in capacity of Chief Scientific Officer of Resverlogix since 2003; Professor, Departments of Medicine and Biochemistry and Molecular Biology within the Faculty of Medicine the University of Calgary since 1987.	N/A
Dr. Jan Johansson, MD, PhD San Ramon, California, U.S.A.	Senior Vice President Medical Affairs	Senior Vice President Medical Affairs of Resverlogix since 2004.	N/A

Notes:

- (1) Member of the Audit and Finance Committee
- (2) Member of the Corporate Governance and Nominating Committee
- (3) Member of the Compensation and HR Committee

The directors and executive officers, in the aggregate, beneficially own, directly or indirectly, or exercise control or direction over 15,976,045, or 18.5%, of issued and outstanding Common Shares as of July 27, 2015.

Clinical Steering Committee

The Company's Steering Committee for the BETonMACE trial will provide overall supervision of the clinical trial and ensure that it is being conducted in accordance with the principles of Good Clinical Practice and FDA regulations. The Steering Committee will establish the trial protocols, any protocol amendments and provided advice to the investigators on various aspects of the trial. The members of the Steering Committee are as follows:

Professor Kausik Ray is Professor of Public Health, Department of Primary Care and Public Health, School of Public Health at Imperial College London. A clinical cardiologist by training Professor Ray received his medical education (MB ChB, 1991) at the University of Birmingham, his MD (2004) from the University of Sheffield, a postdoctoral fellowship at Harvard Medical School (2004-2005) an MPhil in epidemiology (2007) from the University of Cambridge and was Chair in Preventive Cardiology at St Georges University of London from 2010. Professor Ray's research interests focus on the prevention of cardiovascular disease using observational methods and intervention studies including large trials. Professor Ray established the first global registry of Familial Hypercholesterolaemia in conjunction with the European Atherosclerosis Society (EAS) called the FH studies collaboration (FHSC) and is PI for the TOGETHER study looking at cardiometabolic risk factors and clinical outcomes in approximately 250 000 people using electronic health records in London.

Gregory G. Schwartz is Professor of Medicine in the Division of Cardiology of the University of Colorado Denver. He earned MD and PhD degrees at Duke University School of Medicine, and served as resident and Chief Resident in Internal Medicine at the University of Colorado and fellow in Cardiology at the University of California San Francisco. His research interests include clinical trials investigating new lipid and metabolic treatments to improve outcomes after heart attacks. Dr. Schwartz has been the lead

investigator in large global CVD trials such as MIRACL and dal-OUTCOMES, and is co-Chair of the ongoing Odyssey Outcomes trial. Dr. Schwartz serves as Section Editor for Clinical Trials on the Editorial Board of the Journal of the American College of Cardiology. He is a fellow of the American Heart Association and the American College of Cardiology.

Peter P. Toth, MD, PhD is director of preventive cardiology, CGH Medical Center, Sterling, Ill., and professor of clinical family and community medicine at University of Illinois, Peoria. Dr. Toth has authored and coauthored over two hundred twenty publications in medical and scientific journals and textbooks. He is Editor-in-Chief of the *Year in Lipid Disorders* (Atlas Publishing, Oxford, UK) and an Associate Editor for the *Year Book of Endocrinology*. He is coeditor with Antonio Gotto of the textbook, *Comprehensive Management of High Risk Cardiovascular Patients* (Taylor and Francis, New York) with Michael Davidson of *Therapeutic Lipidology* (Humana, Philadelphia), with Dominic Sica of *Current Controversies in Dyslipidemia Management* (Atlas Publishing, Oxford), with Kevin Maki of *Practical Lipid Management* (Wiley-Blackstone, London), with Christopher Cannon of *Comprehensive Cardiovascular Care in the Primary Care Setting* (Springer Humana, Philadelphia), and Domenic Sica of *Clinical Challenges in Hypertension* vols I and II (Clinical Publishing, Oxford, UK). He has lectured on many topics in cardiovascular medicine throughout the United States.

Dr. Stephen J. Nicholls, MBBS, PhD is the inaugural SAHMRI Heart Foundation Heart Disease Theme Leader at the South Australian Health & Medical Research Institute. Dr. Nicholls is also Professor of Cardiology at the University of Adelaide. He has authored more than 350 original manuscripts, meeting abstracts and book chapters. His current research interests include the functional properties of HDL, the role of inflammation and oxidative stress in atherogenesis and the development of new imaging modalities to assess factors that influence the natural history of atherosclerosis. He plays a lead role in clinical trials that employ intravascular ultrasound to investigate the impact of novel anti-atherosclerotic therapies.

Henry N. Ginsberg, MD is the Irving Professor of Medicine at Columbia University College of Physicians and Surgeons, Associate Dean for Clinical and Translational Research, and Director of the Irving Institute for Clinical and Translational Research at Columbia University Medical Center in New York, New York. He is the Principal Investigator of one of the first 12 NIH-funded Clinical Translational Science Awards. Dr. Ginsberg is also principal investigator on two RO1 research grants from the NIH, National Heart, Lung, and Blood Institute. He is also the co-principal investigator at Columbia on the ACCORD Trial. His research interests have focused on the regulation of plasma cholesterol and triglyceride blood levels, particularly the metabolism of apolipoprotein B-containing lipoproteins in cells, mice, and humans. He has authored or coauthored more than 200 articles, reviews, and chapters related to lipids, diabetes, and heart disease.

Neurodegenerative Clinical and Scientific Advisory Board

In July 2012, we formed a Neurodegenerative Clinical and Scientific Advisory Board. The board, chaired by Dr. Bengt Winblad, provides insight and guidance on all aspects of the development program. Appointed members of the clinical and scientific advisory board are:

Dr. Bengt Winblad, MD, PhD, Chairman, is professor of geriatric medicine and chief physician at the Karolinska University Hospital, Huddinge and the Karolinska Institute in Stockholm, Sweden. Professor Winblad is co-chair of the European Alzheimer Disease Consortium and chairs the Medical Scientific Advisory Panel of the Alzheimer Disease International. In 2009, Dr. Winblad was ranked the world's most prolific researcher in the Alzheimer's disease field (J Alzheimer's Disease 2009).

Dr. Jeffrey Cummings, MD, ScD is director of the Cleveland Clinic Lou Ruvo Center for Brain Health in Las Vegas and Cleveland. Dr. Cummings received the Ronald and Nancy Reagan Research Award from the National Alzheimer's Association in 2008.

Dr. Henrik Zetterberg, MD, PhD is professor of neurochemistry at the University of Gothenburg. He was a Fulbright Scholar and research fellow in neurology at the Harvard Institutes of Medicine, Boston between 2004 and 2005. Dr. Zetterberg is an established leader in the field of neurochemistry, biomarkers and diagnostics.

Dr. Rada Koldamova, MD, PhD is an associate professor and lead researcher at the School of Public Health, University of Pittsburgh's Koldamova and Lefterov lab. Her interest is in cellular and molecular mechanisms of neurodegeneration, the role of ABCA1 transporter and apolipoproteins A-I and E in the pathogenesis of Alzheimer's disease.

Audit Committee Matters

Audit and Finance Committee Charter

The Audit and Finance Committee Charter is attached hereto as Schedule "A".

Composition of the Audit and Finance Committee

The Audit and Finance Committee is composed of three independent, unrelated directors – Mr. Zuerblis as Chair, Mr. McNeill and Dr. Smith. All three members of the Committee are considered financially literate. Each of the members have held board and executive positions on behalf of several companies, and have a wealth of experience in leading and managing companies.

Relevant Education & Experience

Kenneth Zuerblis

Mr. Zuerblis received a BS in Accounting and is a Certified Public Accountant with nearly 30 years of experience, has held senior financial positions with three publicly-traded companies and has held directorships with numerous organizations. Mr. Zuerblis served as Executive Vice President and Chief Financial Officer of Savient Pharmaceuticals, Inc. from 2011 to 2012. Prior to joining Savient, Mr. Zuerblis served as Chief Financial Officer and Senior Vice President at ImClone Systems from 2008 through 2009. From 1994 through 2005, Mr. Zuerblis served as Chief Financial Officer of Enzon Pharmaceuticals Inc., and held the position of Corporate Controller from 1991 through 1994. Mr. Zuerblis began his career at KPMG, LLP in 1982 where he held management positions of increasing responsibility over a 10 year period. Mr. Zuerblis currently serves on the boards of directors (since 2012) of Stemline Therapeutics, Inc. and Zenith Epigenetics Corp. (since 2013).

Kelly McNeill

Mr. McNeill holds a Masters of Accountancy and a Bachelor of Commerce (Honours), and is a Chartered Accountant with over 20 years of experience. Mr. McNeill has served as Chief Financial Officer of RTDS Technologies Inc. since 2014. Mr. McNeill served as Executive Vice President, Finance and Administration, Chief Financial Officer and Secretary of IMRIS Inc. between 2009 and 2014. From 2006 to 2009, Mr. McNeill was Resverlogix's Chief Financial Officer. Prior thereto, Mr. McNeill held senior financial positions with two multinational companies. Mr. McNeill also serves on the board of directors of Zenith Epigenetics Corp. (since 2013).

Dr. Eldon Smith

Dr. Smith is a physician and President and CEO of Eldon R. Smith and Associates Ltd. (a private healthcare consulting company), and was for many years involved in senior administration at the University of Calgary. From 1992 to 1997, Dr. Smith served as the Dean (Chief Executive Officer) of the Faculty of Medicine, where he was responsible for approximately 1,600 employees and an annual budget of more than \$100 million. Dr. Smith holds a Doctor of Medicine degree from Dalhousie University.

Dr. Smith has also served as the Chairman of a publicly listed company (TSX and NASDAQ) and over the past 15 years has served on the audit committees of seven (7) publicly traded companies in Canada and the USA, in addition to Resverlogix. Dr. Smith currently is chairman and member of the audit committee of Aston Hill Financial Inc. (since 2005) (TSX), serves on the board of directors (since 2009) and is a member of the audit committee of Intellipharma International Inc. (TSX/NASDAQ), and serves on the board of directors and is a member of the audit committee of Zenith Epigenetics Corp. (since 2013).

Pre-approval of Audit Fees

The Company will not engage external auditors to carry out any Prohibited Service as defined in the CICA revised Rules of Professional Conduct.

The Board, upon recommendation from the Audit and Finance Committee, will consider the pre-approval of permitted services to be performed by the external auditors in each of the following broad categories:

- Audit Services
- Audit Related Services
- Tax Services

Engagements of external auditors will only commence subsequent to Board pre-approval of audit services, and only a member of the Audit and Finance Committee, or the President and CEO or Chief Financial Officer shall be authorized to request services of external auditors.

External Auditor Service Fees

The following table sets out the aggregate fees billed by our external auditor in each of the last two financial years for services provided to the Company:

Year	Audit Fees ⁽¹⁾	Audit-Related Fees	Tax Fees ⁽²⁾	Other Fees
2015	\$146,800	\$Nil	\$4,500	\$Nil
2014	\$201,800	\$Nil	\$4,800	\$Nil

Notes:

- (1) Audit fees were for professional services for the audit of our annual financial statements, reviews of our unaudited interim financial statements, audits of RVX Therapeutics Inc., French translations, our short form base shelf prospectus and prospectus supplements, as well as services provided in connection with securities, statutory and regulatory filings or engagements paid to KPMG LLP.
- (2) Tax Fees were for professional services for corporate reorganization advise, tax planning and compliance services paid to KPMG LLP.

Cease Trade Orders, Bankruptcies, Penalties or Sanctions

Other than as set out below, no director or executive officer is as at the date hereof, or has been within ten years before the date hereof, a director or chief executive officer or chief financial officer of any company (including the Company) that, while he was acting in such capacity: (i) was the subject of a cease trade or similar order, or an order that denied the relevant company access to any exemption under securities legislation for a period of more than 30 consecutive days; (ii) was subject to an event that resulted, after the director or executive officer ceased to be a director or chief executive officer or chief financial officer, in the company being the subject of a cease trade or similar order or an order that denied the relevant company access to any exemption under securities legislation, for a period of more than 30 consecutive days.

In addition, other than set out below, no director or executive officer or shareholder holding a sufficient number of securities of the Company to affect materially the control of the Company is as at the date hereof, or has been within ten years before the date hereof, a director or executive officer of any company (including the Company) that, while that person was acting in that capacity or within a year of that person ceasing to act in that capacity, became bankrupt, made a proposal under any legislation relating to bankruptcy or insolvency or was subject to or instituted any proceedings, arrangement or compromise with creditors or had a receiver, receiver manager, or trustee appointed to hold its assets.

Mr. Cann was a director of Banff Rocky Mountain Resort Ltd., General Partner for the Banff Rocky Mountain Resort Limited Partnership, which was subject to cease trade orders between May and November 2008 from the Alberta and Ontario Securities Commissions for a delay in filing audited annual financial statements of the Partnership.

Mr. McNeill was the Chief Financial Officer of IMRIS Inc. ("IMRIS") from 2009 until his resignation on September 5, 2014. IMRIS is a biomedical company that is a reporting issuer in all provinces of Canada and at the time of Mr. McNeill's resignation was listed on TSX and NASDAQ. On May 26, 2015, IMRIS and certain of its subsidiaries filed voluntary petitions under Chapter 11 of the United States Bankruptcy Code in the United States Bankruptcy Court for the District of Delaware which granted a stay of proceedings against IMRIS. On June 3, 2015, the Manitoba Court of Queen's Bench granted an initial recognition order under the Companies' Creditors Arrangement Act (Canada) recognizing the Chapter 11 proceedings and granting a stay of proceedings against IMRIS.

No director, executive officer or shareholder holding a sufficient number of our securities to affect materially the control of the Company has, within the past ten years, become bankrupt, made a proposal under any legislation relating to bankruptcy or insolvency, or became subject to or instituted any proceedings, arrangement or compromise with creditors, or had a receiver, receiver manager or trustee appointed to hold the assets of such person.

No director, executive officer or shareholder holding a sufficient number of our securities to affect materially the control of the Company has been subject to any penalties or sanctions imposed by a court relating to Canadian securities legislation or by a Canadian securities regulatory authority or has entered into a settlement agreement with a Canadian securities regulatory authority or been subject to any other penalties or sanctions imposed by a court or regulatory body that would likely be considered important to a reasonable investor in making an investment decision.

Conflicts of Interest

Certain directors and officers of the Company and our subsidiary are associated with other reporting issuers or other corporations which may give rise to conflicts of interest. In accordance with the ABCA directors who have a material interest or any person who is a party to a material contract or a proposed material contract with us are required, subject to certain exceptions, to disclose that interest and abstain from voting on any resolution to approve the contract.

Interests of Management and Others in Material Transactions

Other than as described below, there are no material interests, direct or indirect of directors, executive officers, any shareholders that beneficially own, directly or indirectly, more than 10% of our outstanding Common Shares, or any known associates or affiliates of such persons, in any transaction within the last three years or in any proposed transaction which has materially affected or is reasonably expected to materially affect us.

Eastern arranged for an irrevocable standby letter of credit to secure our loan from Citibank and we agreed to indemnify Eastern in respect of its obligations under the standby letter of credit. We pledged our patents and certain tax losses and pools as security for its indemnity obligations and also agreed to issue common share purchase warrants and pay certain fees to Eastern, as more particularly described under “General Development of Business – Financing – Citibank Loan”.

In August 2013, Eastern subscribed for 1,765,307 units of the Company at a price of \$0.90 per unit, each unit being comprised of one Common Share and 0.3 common share purchase warrants. Each whole warrant was exercisable at a price of \$0.90 per share for a period of five years.

On June 10, 2014, NGN, a limited partnership that is under the control or direction of Dr. Peter Johann, our chairman, subscribed for 1,230,769 Common Shares of the Company at a price of \$0.65 per share and certain other directors and officers of the Company subscribed for an aggregate of 1,080,522 Common Shares at a price of \$0.65 per share.

In July 2015, we completed a private placement of 13,270,000 common shares and 1,000,000 common share purchase warrants to Hepalink and 5,600,000 common shares and 422,005 common share purchase warrants to Eastern Capital Limited for aggregate proceeds of CAD\$50 million, or CAD\$2.67 per unit. Each warrant is exercisable at a price of CAD\$2.67 for a period of five years.

In conjunction with the private placement, we entered into a license agreement with Hepalink whereby we granted Hepalink a license of RVX-208 for the Territories for all indications. The license provides for certain milestone payments based on net sales of RVX-208 in the Territories and for Hepalink to pay a royalty of 6% of annual net sales of RVX-208 in the Territories. See “General Development of the Business – Recent Developments – Licensing”.

Transfer Agent and Registrar

The transfer agent and registrar for our Common Shares is Valiant Trust Company at its transfer offices in Calgary and Toronto.

Material Contracts

Material contracts which we entered into within the most recently completed financial year or before the most recently completed financial year which remain in effect, other than contracts entered into in the ordinary course of business are as follows:

1. Investor Rights Agreement (“IRA”) dated April 15, 2009 and amended April 30, 2013 among us, NGN and certain other investors and each of Donald McCaffrey, Wayne Chiu and Norman Wong. The IRA provides that we shall not issue any new securities without offering a proportionate share to NGN and the other investors. The IRA also provides NGN with certain approval rights including, any offering of securities that rank senior to the Common Shares, any increase or decreases from the intended composition of seven Board members, any amendment to our constating documents and any related party transactions.
2. Second Amended and Restated Loan Agreement dated July 3, 2014 among the Company and Citibank, N.A., as more particularly described under “General Development of the Business – Financing – Citibank Loan”.
3. Amended and Restated Agreement and Indemnity dated July 3, 2014 and Covenant Agreement dated April 29, 2013 among us and Eastern, as more particularly described under “General Development of the Business – Financing – Citibank Loan”.
4. Amended and Restated Intellectual Property Security Agreement dated July 3, 2014 granted by us in favour of Eastern, as more particularly described under “General Development of the Business – Financing – Citibank Loan”.

5. Amended and Restated Patent Security Agreement dated July 3, 2014 granted by the Company in favour of Eastern, as more particularly described under “General Development of the Business – Financing – Citibank Loan”.
6. Assignment and Services Agreement dated June 3, 2013 and effective May 1, 2012 between us and RVX Therapeutics, as more particularly described under “General Development of the Business – Spin Out of Epigenetics Drug Discovery Platform – Assignment and Services Agreement”.
7. Management Services Agreement dated June 3, 2013 between us and Zenith, as more particularly described under “General Development of the Business – Spin Out of Epigenetics Drug Development Platform – Management Services Agreement”.
8. Nomination Rights Agreement dated July 20, 2015, between us and Hepalink, as more particularly described under “General Development of the Business – Financing – Private Placements”.
9. Share Restriction Agreement dated July 20, 2015, between us and Hepalink, as more particularly described under “General Development of the Business – Financing – Private Placements”.

Interests of Experts

Our auditors are KPMG LLP, Chartered Accountants of Calgary, Alberta, Canada. KPMG LLP has confirmed that it is independent with respect to us in accordance with the rules of professional conduct of the Institute of Chartered Accountants of Alberta.

Additional Information

Additional information, including directors’ and executive officers’ remuneration and indebtedness, principal holders of our securities and securities authorized for issuance under equity compensation plans is contained in the Management Information Circular with respect to the most recent annual meeting of shareholders. Additional financial information is provided in our audited financial statements and MD&A for the year ended April 30, 2015.

Additional information relating to us may be found on SEDAR at www.sedar.com. In addition, we maintain updated information on our website at www.resverlogix.com.

Schedule "A" – Audit and Finance Committee Charter

RESVERLOGIX CORP.

AUDIT & FINANCE COMMITTEE CHARTER

PART I ESTABLISHMENT OF COMMITTEE

1. Committee Purpose

The Audit and Finance Committee (the "**Committee**") is established by the board of directors (the "**Board of Directors**") of Resverlogix Corp. ("Resverlogix") primarily for the purpose of overseeing the accounting and financial reporting processes of Resverlogix and the reviews and audits of the financial statements of Resverlogix.

The Committee shall assist the Board of Directors in fulfilling its oversight responsibilities by monitoring, among other things:

- (a) the quality and integrity of the financial statements and related disclosure of Resverlogix;
- (b) compliance by Resverlogix with legal and regulatory requirements that could have a material effect upon the financial position of Resverlogix which are not subject to the oversight of another committee of the Board of Directors or the Board of Directors as a whole;
- (c) the independent auditor's qualifications and independence; and
- (d) performance of Resverlogix's independent auditor.

2. Composition of Committee

The Committee shall consist of as many members as the Board shall determine, but in any event not fewer than three directors, provided that all members of the Committee shall be determined by the Board to be independent within the meaning of National Instrument 52-110 (Audit Committees), Rule 10A-3(b)(1) under the United States Securities Exchange Act of 1934 and the rules of any stock exchange or market on which Resverlogix's shares are listed or posted for trading (collectively, "**Applicable Governance Rules**"). In this Charter, the term "independent" includes the meanings given to similar terms by Applicable Governance Rules, including the terms "non-executive", "outside" and "unrelated" to the extent such terms are applicable under Applicable Governance Rules. No member of the Audit Committee shall have participated in the preparation of the financial statements of the Corporation or any current subsidiary of the Corporation at any time during the past three (3) years.

All members of the Audit Committee must be able to read and understand fundamental financial statements (including a balance sheet, income statement and cash flow statement) and read and understand a set of financial statements that present a breadth and level of complexity of accounting issues that are generally comparable to the breadth and level of complexity of the issues that can reasonably be expected to be raised by the Corporation's financial statements. In addition: (i) at least one member of the Audit Committee must have past employment experience in finance or accounting, requisite professional certification in accounting or any other comparable experience or background that results in the individual's financial sophistication, including service as a chief executive officer, chief financial officer, or other senior officer with financial oversight responsibilities or otherwise satisfy standards for financial expertise required for audit committees of companies listed on the Toronto Stock Exchange and/or NASDAQ Stock Market, and (ii) at least one member of the Audit Committee must be an "audit committee financial expert" as defined by the Applicable Governance Rules.

3. Appointment of Committee Members

The members of the Committee shall be appointed by the Board of Directors on the recommendation of the Corporate Governance and Nominating Committee. The members of the Committee shall be appointed at the time of each annual meeting of shareholders and shall hold office until the next annual meeting, until they are removed by the Board of Directors or until their successors are earlier appointed, or until they cease to be directors of Resverlogix.

PART II COMMITTEE PROCEDURE

4. Vacancies

Where a vacancy occurs at any time in the membership of the Committee, it may be filled by the Board of Directors on the recommendation of the Corporate Governance and Nominating Committee and shall be filled by the Board of Directors if the membership of the Committee is fewer than three directors. The Board of Directors may remove and replace any member of the Committee.

5. Committee Chair

The Board of Directors shall appoint a chair (the "**Chair**") for the Committee. The Chair may be removed and replaced by the Board of Directors.

6. Absence of Chair

If the Chair is not present at any meeting of the Committee, one of the other members of the Committee present at the meeting shall be chosen by the Committee to preside at the meeting.

7. Secretary of Committee

The Committee shall appoint a Secretary who need not be a director of Resverlogix.

8. Regular Meetings

The Chair, in consultation with the Committee members, shall determine the schedule and frequency of the Committee meetings, provided that the Committee shall meet at least quarterly. The Committee at any time may, and at each regularly scheduled Committee meeting shall, meet without management present and shall meet periodically with management and the independent auditor. The Committee shall also meet separately with the independent auditor at every regularly scheduled meeting of the Committee at which the independent auditor is present. The Committee shall record and maintain minutes of meetings.

9. Special Meetings

The Chair, any two members of the Committee, the independent auditor or the Chief Executive Officer of Resverlogix may call a special meeting of the Committee.

10. Quorum

A majority of the members of the Committee, present in person or by telephone or other telecommunication device that permits all persons participating in the meeting to speak to each other, shall constitute a quorum.

11. Notice of Meetings

Notice of the time and place of every meeting shall be given in writing or by e-mail or facsimile communication to each member of the Committee at least 48 hours prior to the time fixed for such meeting; provided, however, that a member may, in any manner, waive notice of a meeting and attendance of a member at a meeting is a waiver of notice of the meeting, except where a member attends a meeting for the express purpose of objecting to the transaction of any business on the grounds that the meeting is not lawfully called.

12. Agenda

The Chair shall develop and set the Committee's agenda, in consultation with other members of the Committee, the Board of Directors and management of Resverlogix. The agenda and information concerning the business to be conducted at each Committee meeting shall, to the extent practicable, be communicated to the members of the Committee sufficiently in advance of each meeting to permit meaningful review.

13. Delegation

Subject to subsection PART III19(e), the Committee shall have the power to delegate its authority and duties to subcommittees or individual members of the Committee as it deems appropriate.

14. Access

In discharging its oversight role, the Committee shall have full access to all books, records, facilities and personnel of Resverlogix.

15. Attendance of Others at a Meeting

At the invitation of the Chair, one or more officers, directors or employees of Resverlogix may, and if required by the Committee shall, attend a meeting of the Committee.

16. Procedure, Records and Reporting

The Committee shall fix its own procedure at meetings, keep records of its proceedings and report to the Board of Directors when the Committee may deem appropriate (but not later than the next meeting of the Board of Directors).

17. Outside Consultants or Advisors

The Committee, when it considers it necessary or advisable, may retain, at Resverlogix's expense, outside consultants or advisors (including independent counsel) to assist or advise the Committee independently on any matter within its mandate. The Committee shall have the sole authority to retain or terminate such consultants or advisors, including the sole authority to approve the fees and other retention terms for such persons.

PART III MANDATE OF COMMITTEE

18. Appointment of Resverlogix's Independent Auditor

Subject to confirmation by the independent auditor of its compliance with Canadian regulatory registration requirements, the Committee shall recommend to the Board of Directors the appointment of the independent auditor for the purpose of preparing or issuing any audit report or performing other audit, review or attest services for Resverlogix, such appointment to be confirmed by Resverlogix's shareholders at each annual meeting. The Committee shall also recommend to the Board of Directors the engagement letter with the independent auditor, the approval of fees to be paid to the independent auditor for audit services and shall pre-approve the retention of the independent auditor for any permitted non-audit service. The Committee shall also be directly responsible for overseeing the work of the independent auditor (including resolution of disagreements between management of Resverlogix and the independent auditor regarding financial reporting) for the purpose of preparing or issuing an audit report or performing other audit, review or attest services for Resverlogix. The Committee shall communicate directly with the independent auditor. The independent auditor shall report directly to the Committee.

The Committee shall review the independence of the independent auditor including a written report from the independent auditor delineating all relationships between the auditor and Resverlogix, considering whether the advisory services performed by the independent auditor during the course of the year have affected its independence, and ensuring that no relationship or service between the independent auditor and Resverlogix is in existence that may affect the objectivity and independence of the auditor, or recommending appropriate action to ensure the independence of the independent auditor.

19. Specific Mandates

The Committee, to the extent required by applicable laws or rules, or otherwise considered by the Committee to be necessary or appropriate, shall:

(a) Oversight in Respect of Financial Disclosure

(i) review, discuss with management of Resverlogix and the independent auditor, and recommend to the Board of Directors for approval:

A. the annual and interim financial statements;

- B. the annual information form;
 - C. the annual and interim management's discussion and analysis;
 - D. the portions of the management proxy circular, for any annual or special meeting of shareholders, containing significant financial information respecting Resverlogix;
 - E. all financial statements included in prospectuses or other offering documents;
 - F. any significant financial information contained in all prospectuses and all documents which may be incorporated by reference in a prospectus;
 - G. any significant financial information respecting Resverlogix contained in a material change report or a business acquisition report;
- (ii) review and discuss with management of Resverlogix:
- A. each press release which contains significant financial information respecting Resverlogix (including, without limitation, annual and interim earnings press releases) or contains earnings guidance, prior to public dissemination thereof;
 - B. the use of "pro forma" or "adjusted" non-IFRS information;
 - C. financial information and earnings guidance provided to analysts and rating agencies; provided, however, that such discussion may be done generally (consisting of discussing the types of information to be disclosed and the types of presentations to be made), and the Committee need not discuss in advance each instance in which Resverlogix may provide earnings guidance or presentations to rating agencies;
- (iii) review with management and the independent auditor the scope of the audit, in particular the independent auditor's view of Resverlogix's accounting principles as applied in the financial statements in terms of disclosure quality and evaluation methods, inclusive of the clarity of Resverlogix's financial disclosure and reporting, degree of conservatism or aggressiveness of Resverlogix's accounting principles and underlying estimates, and other significant decisions made by management in preparing the financial disclosure and reviewed by the independent auditor;
- (iv) review with management of Resverlogix and the independent auditor major issues regarding accounting and auditing principles and practices as well as the adequacy of internal controls and procedures for financial reporting and management information systems and inquire of management and the independent auditor about significant risks and exposures to the Corporation that could significantly affect Resverlogix's financial statements;
- (v) review with management of Resverlogix and the independent auditor, and satisfy itself as to the adequacy of the procedures that are in place for the review of Resverlogix's disclosure of financial information extracted or derived from Resverlogix's financial statements, and periodically assess the adequacy of those procedures;
- (vi) review with management of Resverlogix and the independent auditor (including those of the following that are contained in any report of the independent auditor): (a) all critical accounting policies and practices to be used by Resverlogix in preparing its financial statements; (b) all alternative treatments of financial information within IFRS that have been discussed with management, ramifications of the use of these alternative treatments, and the independent auditor's assessment of the alternatives; and (c) other material communications between the independent auditor and management of Resverlogix, such as any management letter or schedule of unadjusted differences;
- (vii) review with management of Resverlogix and the independent auditor the effect of regulatory and accounting initiatives as well as off-balance sheet transactions on Resverlogix's financial statements;
- (viii) review the plans of management of Resverlogix and the independent auditor regarding any significant changes in accounting practices or policies and the financial and accounting impact thereof;
- (ix) review with management of Resverlogix, the independent auditor and, if necessary, legal counsel, any litigation, claim or contingency, including tax assessments, that could have a material effect upon the financial position of Resverlogix, and the manner in which these matters have been disclosed in the financial statements;

- (x) review disclosures by Resverlogix's Chief Executive Officer and Chief Financial Officer with respect to any required certification for Resverlogix's financial statements by such individuals; and
 - (xi) discuss with management Resverlogix's material financial risk exposures and the steps management of Resverlogix has taken to monitor and control such exposures, including Resverlogix's financial risk assessment and financial risk management policies.
- (b) **Oversight in Respect of Legal and Regulatory Matters**
- (i) review, if necessary, with legal counsel, Resverlogix's compliance policies, legal matters and any material reports or inquiries received from regulators or governmental agencies that could have a material effect upon the financial position of Resverlogix and which are not subject to the oversight of another committee of the Board of Directors or the Board of Directors as a whole.
- (c) **Oversight in Respect of the Chief Financial Officer**
- (i) consult with management on management's appointment, replacement, reassignment or dismissal of the Chief Financial Officer of Resverlogix; and
 - (ii) ensure the Chief Financial Officer of Resverlogix has access to the Chair, the Chairman of the Board of Directors and the Chief Executive Officer of Resverlogix, and shall meet separately with the Chief Financial Officer of Resverlogix to review any problems or difficulties he or she may have encountered in the performance of his or her responsibilities and report to the Board of Directors on such meetings.
- (d) **Oversight in Respect of the Independent Auditor**
- (i) meet with the independent auditor prior to the annual audit to review the planning and staffing of the audit;
 - (ii) review annually the independent auditor's formal written statement of independence delineating all relationships between itself and Resverlogix and review all such relationships;
 - (iii) receive confirmation from the independent auditor as to its standing as a "participating audit firm" and its compliance with any restrictions or sanctions imposed by the Canadian Public Accountability Board as those concepts are set forth in National Instrument 52-108 of the Canadian Securities Administrators;
 - (iv) review and evaluate the independent auditor, including the lead partner of the independent auditor team and shall confirm compliance by the independent auditors with laws and regulations relating to audit partner rotation;
 - (v) meet separately with the independent auditor to review with them any problems or difficulties they may have encountered and specifically:
 - A. any difficulties which were encountered in the course of the audit work, including any restrictions on the scope of activities or access to required information, and any disagreements with management of Resverlogix; and
 - B. any changes required in the planned scope of the audit;and report to the Board of Directors on such meetings;
 - (vi) review the engagement reports of the independent auditor on unaudited financial statements of Resverlogix; and
 - (vii) review and approve Resverlogix's hiring policies regarding partners, employees, former partners and former employees of Resverlogix's present and former independent auditor.

(e) **Oversight in Respect of Audit and Non-Audit Services**

- (i) have the sole authority to pre-approve all audit services (which may entail providing comfort letters in connection with securities underwritings) and all permitted non-audit services, other than non-audit services where:
 - A. the aggregate amount of all such non-audit services provided to Resverlogix or its subsidiaries constitutes not more than 5% of the total amount of fees paid by Resverlogix (and its subsidiaries) to the independent auditor during the fiscal year in which the non-audit services are provided;
 - B. such services were not recognized by Resverlogix (or any subsidiary) at the time of the engagement to be non-audit services; and
 - C. such services are promptly brought to the attention of the Committee and approved, prior to the completion of the audit, by the Committee or by one or more members of the Committee to whom authority to grant such approvals has been delegated by the Committee; and
- (ii) delegate to one or more designated members of the Committee the authority to grant pre-approvals required by this section; provided that the decision of any member to whom authority is delegated to pre-approve an activity shall be presented to the Committee at the first scheduled meeting following such decision, and provided further that, if the Committee approves an audit service within the scope of the engagement of the independent auditor, such audit service shall be deemed to have been pre-approved for purposes of this section

(f) **Oversight in Respect of Certain Policies**

- (i) establish procedures for: (a) the receipt, retention and treatment of complaints received by Resverlogix regarding accounting, internal accounting controls or auditing matters; and (b) the confidential, anonymous submission by employees of Resverlogix of concerns regarding questionable accounting or auditing matters; and
- (ii) periodically review Resverlogix's public disclosure policy.

20. Evaluation of Code of Business Conduct and Ethics

The Committee shall conduct an annual assessment of management's adherence to Resverlogix's Code of Business Conduct and Ethics.

21. Non-Exhaustive List

The foregoing list of duties is not exhaustive, and the Committee may, in addition, perform such other functions as may be necessary or appropriate for the performance of its oversight responsibilities.

22. Review of Committee's Charter

The Committee shall assess the adequacy of this Charter on an annual basis and recommend any changes to the Board of Directors.

23. Oversight Function

While the Committee has the responsibilities and powers set forth in this Charter, it is not the duty of the Committee to plan or conduct audits or to determine that Resverlogix's financial statements are complete and accurate or are in accordance with IFRS. These are the responsibilities of management of Resverlogix and the independent auditor. The Committee and its Chair are members of the Board of Directors, appointed to the Committee to provide broad oversight of the financial risk and control related activities of Resverlogix, and are specifically not accountable nor responsible for the day to day operation or performance of such activities. The role of all Committee members is to oversee the process, not to certify or guarantee the accuracy or completeness of the external audit of Resverlogix's financial information or public disclosure.

Glossary

In this Annual Information Form, unless the context otherwise requires, references to “Resverlogix”, “we”, “us”, “our” or similar terms, or to the “Company” refer to Resverlogix Corp. (either alone or together with its subsidiaries) and the following terms shall have the following meanings, unless otherwise defined elsewhere in this Annual Information Form:

ABCA	means the Business Corporations Act (Alberta).
Acetylated Lysine	an acetyl-derivative of the amino acid lysine (also known as Acetyllysine). In proteins, the acetylation of lysine residues is an important mechanism of epigenetics. It plays a role in regulating the transcription of genes through recruitment of additional proteins to histones associated with DNA.
Acetylation	the process by which an acetyl functional group is transferred onto a molecule
Acute Coronary Syndrome (“ACS”)	a term used for any condition brought on by the sudden reduced blood flow to the heart. Acute coronary syndromes may include a heart attack, unstable angina. The first sign of acute coronary syndrome can be sudden stopping of your heart (cardiac arrest). Acute coronary syndrome is often diagnosed in an emergency room or hospital.
Acute Phase Response Cascade	a series of systemic events that occur within hours of an inflammatory stimulus. The most important component of this response comprises the acute phase proteins. Acute phase response takes place in response to a variety of stimuli including bacterial infection, trauma and myocardial infarction.
Alkaline Phosphatase (“ALP”)	a hydrolase enzyme responsible for removing phosphate groups from many types of molecules, including proteins. Data suggests that elevated serum alkaline phosphatase levels are associated with increased mortality and morbidity in diseases such as diabetes, chronic kidney disease, heart failure and Alzheimer’s disease.
ALTs	Alanine transaminase, also called serum glutamic pyruvic transaminase (“SGPT”) or alanine aminotransferase (“ALAT”), is found in serum and most commonly associated with the liver, measurements are used as a part of a diagnostic evaluation of hepatocellular injury.
Alpha1 HDL	mature lipid-rich particles that are involved in reverse cholesterol transport whereby cholesterol is removed from cell membranes to the liver for excretion.
Alzheimer’s disease (“AD”)	a disease marked by the loss of cognitive ability, generally over a period of 10 to 15 years, and associated with the development of abnormal tissues and protein deposits in the cerebral cortex.
Amyloid-beta40 (“A β -40”)	Amyloid beta 40 is one isoform formed by the cleavage of the amyloid precursor protein (“APP”). Along with A β 42, A β 40 has been identified in cerebral spinal fluid and plasma and may play a role in the pathology of Alzheimer’s disease. Both isoforms are therapeutic targets for AD, and are being studied as experimental biomarkers for the disease.
Angiography	a medical imaging technique used to visualize the inside (lumen) of blood vessels and organs of the body, with particular interest in the arteries, veins and the heart chambers.
Apabetalone	generic name of RVX-208

Apo Intellectual Property Right	any right, whether under a patent, patent application or invention disclosure or otherwise, to use, or to prevent others from using, any product, device, process, substance, composition or service that falls within an ApoA-I Therapeutic Field;
Apo Products	any product, device, process, substance, composition or service that falls within the ApoA-I Therapeutic Field and in respect of which the Company has an Apo Intellectual Property Right;
ApoA-I Therapeutic Field	the prevention, treatment or mitigation of any disease via the administration of a Pharmaceutical Agent that results in therapeutic relevant elevation in the plasma levels of ApoA-I that in a predictable model of ApoA-I expression, using either a human or nonhuman primate model, the Pharmaceutical Agent is demonstrated to have at least a seven percent (7%) increase in humans and fifty percent (50%) increase in nonhuman primates in the ApoA-1 plasma level in two consecutive weeks of treatment using less than 30 milligrams – b.i.d. (60 milligrams per day) of the Pharmaceutical Agent per kilogram of the weight of the subject;
Apolipoprotein	the protein combined with a lipid to form a lipoprotein, a component of HDL and LDL.
ApoA-I	is one of the apolipoprotein components of the HDL particle.
ApoB	is one of the apolipoprotein components of the LDL particle.
Atherosclerosis	a disease in which the deposition of lipids and inflammatory cells in the arterial wall creates a plaque resulting in the hardening and decrease of arterial lumen size.
Atherosclerotic Plaque	the deposit or accumulation of lipid and lipid-containing cells (plaque) in the arterial wall (<i>also known as atheroma</i>).
BET proteins	BET proteins (Bromodomain and ExtraTerminal domain) are proteins that contain bromodomains, which regulate gene transcription through binding to acetylated lysines within the histones bound to DNA.
b.i.d.	“bis in die” (Latin) refers to twice a day dosing.
Bilirubin	the yellow breakdown product of normal heme catabolism, that is excreted in bile and urine; elevated levels may indicate a disease state.
Bioavailability	the degree and rate at which a drug is absorbed into a living system or is made available at the site of activity after administration.
Biopharmaceuticals	a medical drug developed by biotechnology to improve human or animal health.
Bromodomain (“BRD”)	(see BET proteins)
Cancer	a disease characterized by abnormal and uncontrolled cell growth.
Coagulation Cascade	a series of events that culminate in the formation of a blood clot and its subsequent breakdown. This process is controlled by a signaling cascade consisting of coagulation factors which interact and activate each other.
Complement Cascade	the complement system contains a network of tightly regulated proteins that together are a key part of the innate immune system response. The principal roles of complement include defending against invading pathogens, bridging innate and adaptive immunity, eliminating immune complexes and the products of inflammatory injury.

Coronary artery disease (“CAD”)	Coronary artery disease is the most common type of heart disease. It is the leading cause of death in the United States in both men and women. CAD occurs when arteries that supply blood to heart muscle become hardened and narrowed. This is due to the buildup of cholesterol and other material, called plaque, on their inner walls.
C-Reactive Protein (“CRP”)	a biomarker of cardiovascular inflammation
Cardiovascular disease (“CVD”)	a group of diseases of the heart and blood vessels.
Cholesterol	a fatty molecule essential for normal body functions, including the production of hormones and bile acids; it is also an important component of a cell membrane.
Common Shares	common shares in the capital of Resverlogix Corp.
Compound	a chemical substance formed from two or more elements (<i>also see drug</i>).
Contract Research Organization (“CRO”)	an organization (commercial, academic or other), contracted by the sponsor to conduct research or development activities.
Chromatin	the combination of DNA and proteins that make up the contents of the nucleus of a cell. The primary functions of chromatin are: to package DNA into a smaller volume to fit in the cell, to strengthen the DNA to allow mitosis and meiosis and prevent DNA damage, and to control gene expression and DNA replication. The primary protein components of chromatin are histones that compact the DNA.
Clinical Trial/Study	a research study in human subjects to evaluate a new drug, medical device, biologic or other intervention under a strictly controlled scientific setting.
Chronic Kidney Disease (“CKD”)	a progressive loss in renal function over a period of months or years, also known as chronic renal disease (CRD). Chronic kidney disease is also associated with other chronic diseases such as diabetes and or cardiovascular disease. Profession guidelines classify the severity of chronic kidney disease in five stages, with stage 1 being the mildest and usually causing few symptoms and stage 5 being a severe illness with poor life expectancy if untreated. Stage 5 CKD is often called end stage renal disease.
Deoxyribonucleic Acid (“DNA”)	the material inside the nucleus of cells that carries genetic information.
Diabetes Mellitus	the most common metabolic disease and currently is a worldwide epidemic fueled by the wave of modernization swiping across much of the developing countries. There are two types of diabetes, Type-1 and Type-2. The difference between these two types of diabetes is that there is an absence of insulin (Type-1) or a deficiency in the amount of insulin (Type-2). While Type-1 affects less people and mostly younger individuals, Type-2 most commonly accounts for roughly 90% of the cases. The cause of Type-1 Diabetes is believed to lie in defects within the immune system. In the pathogenesis of Type-2, there is direct connection between dietary habits, sedentary life styles and obesity. One of the most feared consequences of either form DM is that it is one of many major risk factors leading to the development of CVD, the number one cause of premature death in modern societies.
Drug	is any substance that can be used to modify a chemical process or processes in the body to mitigate, treat or prevent a medical condition.
Dyslipidemia	a disorder associated with abnormal levels of blood lipids and lipoproteins.

End Stage Renal Disease (“ESRD”)	the last stage of chronic kidney disease. The stage at which the kidneys have incurred permanent damage and lost nearly all function and the treatments include dialysis or a transplant.
Endogenous	is a process whereby a molecule is produced within the body.
Enzyme	a protein that acts as a catalyst in mediating and accelerating a specific chemical reaction.
Epigenetics	the study of heritable traits not caused by a change in the genetic code. These are typically mediated through secondary modifications to the DNA and its bound proteins, which regulate expression of genes contained within the DNA.
Estimated Glomerular Filtration Rate (“eGFR”)	a rate calculated using the results of a blood creatinine test, age and gender. The result indicates the severity and stage of chronic kidney disease. An eGFR below 60 for three months or more indicates CKD.
Food and Drug Administration (“FDA”)	is the United States governmental agency responsible for the approval for manufacture, usage and sale of food, human diagnostics and therapeutic products within the US.
Gene	a sequence of DNA encoding a protein.
Good Clinical Practice (“GCP”)	is the international ethical and scientific quality standard for designing, conducting, recording and reporting trials that involve human subjects.
Good Laboratory Practice (“GLP”)	is the international regulation which embodies a set of principles which provide a framework for laboratory studies, ensuring high quality experimental standards and reliable data.
Good Manufacturing Practice (“GMP”)	is the international set of regulations, codes and guidelines for the manufacture of drugs, medical devices, diagnostics and food products.
High-density Lipoprotein (“HDL”)	a complex of lipids and proteins (ApoA-I) that function in the transport of cholesterol away from the tissues to the liver and is associated with a decreased risk of atherosclerosis and coronary heart disease (also known as “good cholesterol”).
Histones	highly alkaline proteins found in eukaryotic cell nuclei that package and order the DNA into structural units called nucleosomes. Histones are the chief protein components of chromatin, acting as spools around which DNA winds, and play a role in gene regulation.
Health Canada	is the governmental agency which regulates the manufacture, use and sale of human diagnostics and therapeutic products in Canada, and oversees safety of foods.
Hepatic Transaminases	are variables analyzed in plasma that describe liver function and liver cell integrity. They include, for example, Alanine Transaminase (“ALT”) and Aspartate Transaminase (“AST”).
IND-Enabling Studies	a toxicology package, including general acute and repeated-dose toxicity and genotoxicity studies, and safety pharmacology studies, conducted under GLP and in accordance with the International Conference of Harmonization guideline (M3(R1)) to support the filing of an IND application (21.CFR.312). Initiation of the toxicology package will occur when protocols have been written and a contract laboratory has been contracted to conduct the studies.
Interleukin 6 (“IL-6”)	an inflammatory mediator that acts in many diseases as a pro-inflammatory cytokine. IL-6 is known to stimulate the inflammatory and auto-immune processes in many diseases such as diabetes, atherosclerosis, Alzheimer’s disease and rheumatoid arthritis.

Investigational New Drug (“IND”)	the application submitted to the FDA to permit a drug to be tested in humans in clinical trials in the US.
Intravascular Ultrasound (“IVUS”)	an invasive procedure, performed along with cardiac catheterization; a miniature sound probe (transducer) on the tip of a coronary catheter is threaded through the coronary arteries and, using high-frequency sound waves, produces detailed images of the interior walls of the arteries. Where angiography shows a two-dimensional silhouette of the interior of the coronary arteries, IVUS shows a cross-section of both the interior, and the layers of the artery wall itself.
Licensed Know-How	any and all proprietary technology and copyrights owned or controlled by the Company relating to the Licensed Patents, including without limitation, manufacturing processes or protocols, know-how, trade secrets, writings, documentation, data, technical information, techniques, results of experimentation and testing, diagnostic and prognostic assays, specifications, databases, any and all laboratory, research, pharmacological, toxicological, analytical, quality control, pre-clinical and clinical data, and other information and materials, whether or not patentable.
Licensed Product	any product, device, process, substance, composition or service in a Residual Field and which is encompassed within the scope of a Valid Claim.
Low-density Lipoprotein (“LDL”)	a complex of lipids and proteins (ApoB) that function by transporting cholesterol to the tissues, in particular the arteries, and is associated with an increased risk of atherosclerosis and coronary heart disease (<i>also known as “bad cholesterol”</i>).
Lipids	are fatty substances, including cholesterol and triglycerides, that are present in cell membranes and body tissues.
Lipoproteins	a complex of proteins and lipids that are the principal means by which fat and cholesterol is transported in the blood; major lipoproteins are LDL and HDL.
Major Adverse Cardiovascular Events (“MACE”)	a commonly used end point for cardiovascular research. MACE is a composite of clinical events that usually are measured in clinical trials of cardiovascular patients. It may include a variety of end points such as death, myocardial infarction (heart attack), stroke, worsening angina, hospitalization for heart disease and operative treatments for heart disease.
Medical Device	a diagnostic or therapeutic article that does not work by chemical action.
Metabolism	is the biochemical modification or degradation of a drug, often readily removing the drug from the body.
Method	any method covered by a Valid Claim.
Methylation	the process by which an methyl functional group is transferred onto a molecule
Net Apo Revenue	the aggregate of the following amounts: (i) amounts received by the Company or its affiliates from any person who is not the Company or its affiliate (a “third party”) in consideration for granting a license or other rights to the third party which entitle the third party to research, develop, make, manufacture, modify, administer, offer to sell, sell or distribute one or more of the Apo Products and/or Apo Intellectual Property Rights or amounts received under the terms of such license or other right that are granted to the third party; (ii) the gross consideration received from a third party by the Company, any licensee or their respective affiliates from the sale of any Apo Product (other than consideration received by the Company, any licensee or their respective affiliates from a licensee of such Apo Product or its affiliate); less (A) credits or allowances, if any, actually granted; (B) discounts actually allowed; (C) freight, postage, and

insurance charges and additional special packaging charges; (D) customs duties, and excise sales taxes, duties or other taxes imposed upon and paid with respect to such sales (excluding what is commonly known as income taxes); (E) rebates and chargebacks or retroactive price reductions made to federal, state or local governments (or their agencies), or any third party payor, administrator or contractor, including managed health organizations; and (F) commissions related to import, distribution or promotion of any Apo Product paid to third parties (specifically excluding any commissions paid to sales personnel, sales representatives and sales agents who are employees or consultants of, or members of a contract sales force engaged by or on behalf of, the Company, any licensee or their respective affiliates); and (iii) amounts received from a third party by the Company or its affiliates in consideration for the sale of any Apo Intellectual Property Right.

New Drug Application (“NDA”)	the documentation submitted to the FDA, Health Canada or other local regulatory authorities to obtain approval to market a new drug.
New Drug Submission (“NDS”)	<i>(see “New Drug Application”)</i>
Pharmaceutical Agent	a compound or composition covered by a Valid Claim.
Pharmacological Agent	<i>(see “Drug”).</i>
Pharmacodynamics	the study of the biological actions of a drug in the body, specifically the relationship between how much drug is present and its effects.
Pharmacoeconomics	the scientific discipline that compares the monetary value of one pharmaceutical drug or drug therapy to another. It is a sub-discipline of Health economics. A pharmacoeconomic study evaluates the cost (expressed in monetary terms) and effects (expressed in terms of monetary value, efficacy or enhanced quality of life) of a pharmaceutical product.
Pharmacokinetics	the study of how a drug is absorbed, distributed, metabolized and eliminated (“ADME”) by the body over time.
Pharmacology	the study of pharmacological agents and their origin, nature, properties and effects on living organisms.
Phase 2 Clinical Trial	a study in patients (not healthy volunteers) with the main objective to establish a safe and efficacious dose for phase 3 clinical trials.
Phase 3 Clinical Trial	a study or studies in a defined patient population designed to demonstrate effect to support use for a special indication, for example treatment of patients with previous coronary artery disease to prevent the occurrence of a major adverse coronary.
Phosphorylation	the process by which an phosphate functional group is transferred onto a molecule
Preclinical Studies	the studies conducted in animals to evaluate the pharmacology, toxic effects, pharmacokinetics and metabolism of a drug to provide evidence for safety, efficacy and bioavailability of the drug prior to its administration to humans in clinical studies.
Proprotein convertase subtilisin/ kexin type 9 (“PCSK9”)	an enzyme that has medical significance because it functions in cholesterol homeostasis. PCSK9 binds to a domain of the LDL receptor, inducing degradation. Reduced levels of the LDL receptor result in decreased metabolism of LDL, and thus increased LDL levels, a known risk factor for CVD

Reader, writer, eraser	proteins that bind to histone modifications and alter gene activity and protein production (reader); enzymes that add histone modifications (writer); enzymes that remove histone modifications (eraser).
Residual Field	any field other than the ApoA-I Therapeutic Field.
Reverse Cholesterol Transport (“RCT”)	the term that signifies the process whereby cholesterol, an insoluble molecule, is packaged and transported by special particles in the plasma called lipoproteins for movement from peripheral tissues through the blood and back to the liver for excretion from the body. Cholesterol that moves from peripheral tissues to the liver is considered to be moving in the reverse direction.
RVX-208	our drug candidate for the treatment of atherosclerosis in patients at high risk for cardiovascular disease.
Statin	a class of drugs that block cholesterol production in the body by inhibiting an enzyme called HMG-CoA reductase.
Therapeutic	a biopharmaceutical useful for treating a disease.
Toxicology	the study of the harmful effects of substances in the body, including the level of toxicity, the mechanism by which toxicity occurs and how it can be controlled.
Therapeutic Products Directorate (“TPD”)	the Canadian governmental agency that is responsible for the regulation and approval of the sale of drugs and diagnostics in Canada.
Transferred IP Rights	any intellectual property rights that protect or otherwise cover the Transferred Technology, and without limitation includes the rights to the inventions covered by the patent applications listed in Schedule A of the Assignment and Services Agreement dated June 3, 2013 and effective May 1, 2012 and all divisionals, continuations, continuations-in-part or foreign counterparts of such patent applications and all patents issuing from such applications, divisionals, continuations, continuations-in-part or foreign counterparts, and all reissues, renewals, re-examination certificates and extensions of such patents or patent applications.
Transferred Technology	any technology conceived, invented or developed to the date of the Assignment and Services Agreement dated June 3, 2013 and effective May 1, 2012 that relates to pharmaceutical agents and methods for treating diseases and health conditions with a therapeutic agent that acts as a BET inhibitor (excluding compound RVX000222 (RVX-208) which increase the production ApoA-I protein used for the treatment of atherosclerosis), including without limitation, manufacturing processes or protocols, know-how, trade secrets, writings, documentation, data, technical information, techniques, results of experimentation and testing, diagnostic and prognostic assays, specifications, databases, any and all laboratory research, pharmacological, toxicological, analytical, quality control, pre-clinical and clinical data, and other information and materials, whether or not patentable.
Triglycerides	a type of fat found in the blood and other parts of the body.
Type II Diabetes	(see “ <i>Diabetes Mellitus</i> ”)
Valid Claim	a patent claim in a Licensed Patent (as extended by a Supplementary Protection Certificate, where applicable) that has not expired, where the claim has not been disclaimed or cancelled from the Licensed Patent.
Zenith	Zenith Epigenetics Corp., a corporation incorporated under the ABCA, which acquired RVX Therapeutics Inc. from the Company pursuant to a Plan of Arrangement completed on June 3, 2013.

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