Annual Information Form

Fiscal Year Ended April 30, 2017

July 20, 2017

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Note Regarding Forward-Looking Information

Advisories

In this Annual Information Form ("AIF"), 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).

Capitalized terms that are not otherwise defined herein have the meanings given in the Glossary at the end of this AIF.

Unless otherwise noted, all dollar amounts in this AIF are expressed in Canadian dollars.

Scientific and Industry Data

Certain independent third party scientific research and industry data contained in this Annual Information Form is based upon information from government or other independent industry or scientific publications and reports or based on estimates derived from such publications and reports. Government and industry publications and reports generally indicate that they have obtained their information from sources believed to be reliable, but the Corporation has not conducted its own independent verification of such information. While the Corporation believes this information to be reliable, third party information is subject to variations and cannot be verified with complete certainty due to limits on the availability and reliability of raw data, the voluntary nature of the data gathering process, and other limitations and uncertainties inherent in any statistical or scientific survey. In addition, this third party information has been prepared as of a specific date and therefore does not contemplate changes in facts and circumstances following such date. The Corporation has not independently verified any of the research, findings or data from independent third party sources referred to in this Annual Information Form or ascertained the underlying assumptions relied upon by such sources. Unless specifically stated, none of the third party information cited in this Annual Information Form is incorporated by reference herein. All third party information source references are provided for the readers convenience only and do not form a part of this Annual Information Form.

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 prevention of: major adverse cardiovascular events in patients with diabetes mellitus and chronic kidney disease; as well as additional indications including end-stage renal disease treated with hemodialysis, neurodegenerative disease and orphan diseases such as Fabry 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 related to our Phase 3 BETonMACE clinical trial;
- plans relating to our kidney disease program and the planning and design of clinical trials as part of this program;
- plans relating to our orphan disease program and the planning and design of clinical trials as part of this program;

- expectations relating to the timing of significant clinical trial milestones;
- the function and effectiveness of apabetalone, also referred to as 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 clinical trials 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 ability to refinance existing indebtedness on reasonable terms upon maturity;
- the impact of changes in Canadian dollar-US dollar and other foreign exchange rates on our costs and results;
- market competition;
- our ability to attract and retain skilled staff; 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;
- uncertainties related to clinical trials and product development;
- uncertainties related to current economic conditions;
- risks related to rapid technological change;
- uncertainties related 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 related 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 large clinical trials and future commercial production;
- risks related 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, and others to successfully develop and commercialize our technology;

- the willingness of health care insurers and other organizations to pay for our products;
- risks related to our reliance on key personnel;
- risks related 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 us.

The foregoing list of important factors and assumptions is not exhaustive. Events or circumstances could cause our 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.

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 and December 20, 2016, the Company's articles were amended to make certain changes to the dividend entitlement of holders of the Company's 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.

As at April 30, 2017, we employed 28 full-time management, scientific and administration employees.

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.

Description of Business

Since our inception, we have focused on the development of therapeutics for disease states with high unmet medical need. Our lead drug, apabetalone (also referred to as RVX-208), targets Bromodomain and ExtraTerminal domain ("BET") proteins to impact several important biological processes that drive risk in vascular disease patients, namely: (i) reduction of key vascular inflammation markers, (ii) modulation of complement, coagulation and vascular calcification, known drivers in cardiovascular disease ("CVD") and acute cardiac events, (iii) enhancement of reverse cholesterol transport ("RCT"), and (iv) lowering of key markers of metabolic risk. Apabetalone is a first-in-class small molecule currently being investigated in a phase 3 clinical trial for the secondary prevention of major adverse cardiovascular events ("MACE") in high risk patients with diabetes mellitus ("DM") and chronic kidney disease ("CKD"). Additionally, based on the effects of apabetalone on these multiple biological pathways that underlie disease pathology, we are currently exploring the potential for apabetalone in additional indications including end-stage renal disease treated with hemodialysis, neurodegenerative disease and orphan diseases such as Fabry disease.

The Drug Development Process

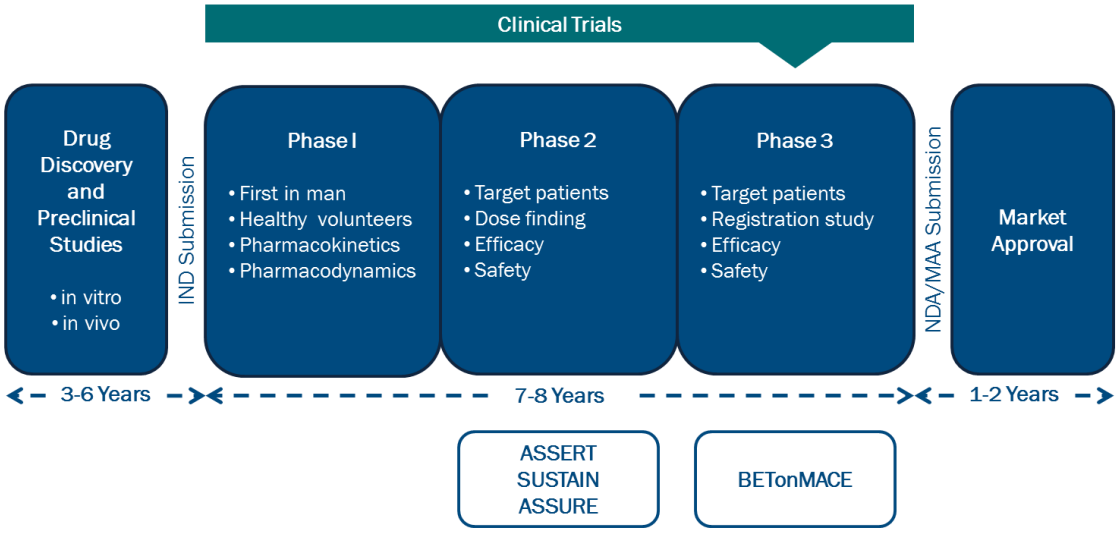
The timeline for a typical experimental drug to go from concept to approval ranges from approximately 12 to 15 years as illustrated in the figure below. The production, manufacture, research and development activities are subject to regulation for safety and efficacy by various governmental authorities around the world. In Europe and the United States, drug products are subject to regulation by the European Medicines Agency ("EMA") and the Federal Drug Administration ("FDA"), respectively.

The FDA has outlined the following guidance for exploratory Investigational New Drug ("IND") studies. During preclinical development, a large number of compounds are created and screened with the objective of identifying the most promising candidates for clinical development. The potential drug candidates are studied with in vitro models (studies performed in an artificial environment outside of the living organism, including primary cells and cell lines) that examine various pharmacologic parameters. Drug candidates yielding favorable data in the early experiments are further tested with in vivo animal (commonly performed in

rodents and dogs) models to evaluate efficacy and safety (toxicity). Results from animal studies include the selection of a safe starting dose for humans, potential toxicity, pharmacokinetic and pharmacodynamic properties. An IND containing all the data collected in the preclinical studies as well as chemistry, manufacturing and controls information must be submitted to applicable regulatory agencies before human clinical testing may begin.

Clinical drug development consists of four sequential phases (Phase I-IV). The steps required for drug approval in the United States, Europe and Canada 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"). Drug development is a step-wise process through which data collected in small early studies are used to rationalize and plan larger, more definitive efficacy studies. To develop new drugs efficiently, it is essential to identify characteristics of the investigational medicine in the early stages of development and to plan an appropriate development based on this profile. Phase 1 clinical trials are "first-in-man" studies with the objective of providing initial safety and tolerability information across multiple doses of the drug, pharmacokinetics data (absorption, distribution, metabolism and excretion of the drug) and pharmacodynamics data (potential efficacy). These studies are generally conducted in healthy volunteer subjects. The main objective of Phase 2 clinical trials is to establish optimal treatment regimen for a Phase 3 trial. Phase 2 trials 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. Phase 2 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, 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 testing a much larger population of patients (several thousand patients) suffering from the targeted condition or disease. Phase 3 trials are usually double-blind using the dose and treatment regimen determined in Phase 2.

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



Drug Development Process

Resverlogix Current Development Stage: Phase 3 Enrolling

Commencing in the fall of 2015, Resverlogix initiated a Phase 3 clinical trial "BETonMACE" with apabetalone in high-risk CVD patients with type 2

BETonMACE
A CARDIOVASCULAR STUDY FOR PEOPLE WITH TYPE 2 DIABETES

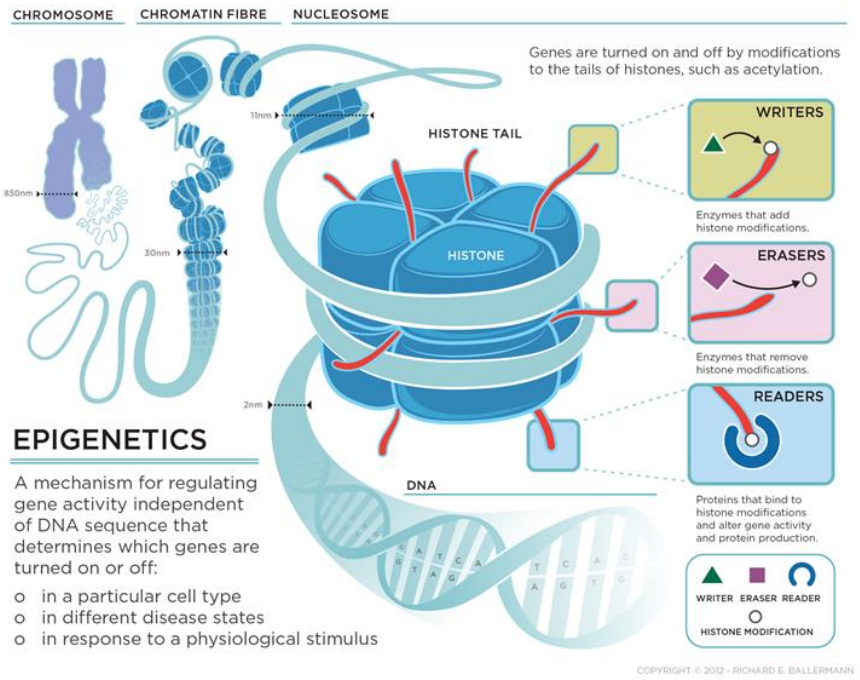
diabetes mellitus and low HDL. Approximately 15% of the patient population will also have CKD. The primary endpoint is the time to first occurrence of MACE. Further details regarding the BETonMACE study are highlighted under "General Development of the Business – Recent Clinical Developments". Further details regarding regulatory submissions relating to apabetalone are highlighted under "General Development of the Business – Regulatory Affairs".

Apabetalone (RVX-208)

Apabetalone is the first BET bromodomain ("BD") inhibitor in clinical trials for high risk vascular disease. As a BET BD inhibitor, apabetalone affects the expression of multiple genes with roles in multiple cellular processes. Apabetalone's 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 biological pathways that are modulated are: vascular inflammation, complement, coagulation and vascular calcification, as well as reverse cholesterol transport. Subgroup analysis of our recent clinical trials has demonstrated that apabetalone significantly reduces MACE in patients with CVD who have a low level of high-density lipoprotein ("HDL") and elevated c-reactive protein ("CRP"), or diabetes mellitus co-morbidity.

Epigenetic Mechanism of Action: Single Therapeutic Target with Multiple Biological Effects

The human body is made up of nearly two hundred different cell types. The differentiation of cell types and cell-specific functions results from the selective production of the proteins encoded by human DNA and, more specifically, human genes. Aberrant levels of proteins can contribute to disease progression and disease states. Epigenetics describes the mechanisms by which gene activity is regulated, thereby affecting levels of messenger RNA ("mRNA") which is then translated into protein. Epigenetics is the study of modifications to chromatin (DNA associated with proteins) that, without affecting the DNA sequence, result in regulation of gene transcription, the first step in producing the proteins that each gene encodes. Such modifications determine whether a gene is "on" or "off" or whether its activity is high or low in a particular cell type, in different disease states or in response to a physiological stimulus. Chromatin modifications are added by enzymes called "writers" and removed by enzymes called "erasers". Other proteins, called "readers", recognize a specific pattern of modifications. In contrast to "writers" and "erasers" that add or remove post translational modifications, "readers" detect the presence or absence of these modifications and serve as a scaffold for the transcriptional machinery directly responsible for gene expression.



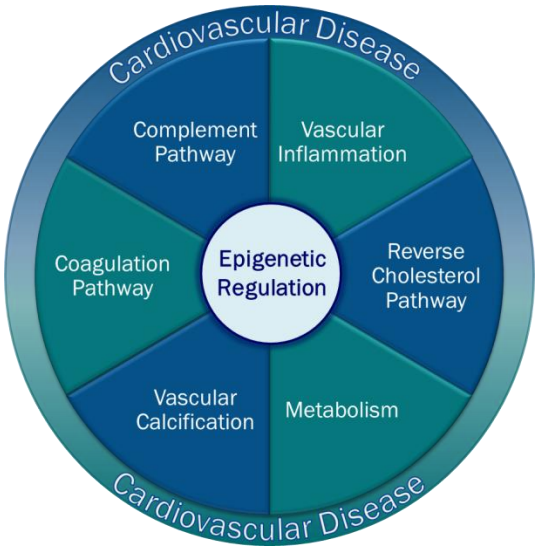
Epigenetic Mechanism of Action

Our compounds target one group of "reader" proteins called BET proteins. Substantial evidence has shown that alterations in the pattern of chromatin modifications underlie multiple disease states. 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 proteins and regulators are promising targets for therapeutic intervention and offer promising treatment advances in diseases with high unmet medical need.

In April 2012, we announced the Mechanism of Action through which apabetalone acts. Our data demonstrates apabetalone to be an inhibitor of the BET proteins. Bromodomains 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, 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. Apabetalone is the first oral agent in the BET inhibitor class that preferentially targets bromodomain 2 ("BD2") of BET protein 4 ("BRD4"). BET proteins play a key role 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.

BET inhibition results in the simultaneous modulation of multiple biological pathways via a single molecular target. BET inhibition holds particular promise for multifactorial diseases including CVD and cancers and represents a novel and promising area of research. Studies highlighting the potential molecular and mechanistic functions of BET inhibitor molecules have begun to shed light into this potential. The ongoing development of BET inhibitors as potential therapeutics in multiple indications would indicate a potential shift from the current drug development paradigm of a single molecular target for a single downstream effect, to a multimodal approach whereby multiple biological processes contributing to a disease state are concurrently modulated via a single molecular target. This is the precise effect of epigenetic modulation and our primary focus.



Epigenetic gene regulation governed by BET proteins is at the core of many CVD pathological processes – dysregulation of multiple pathways contributes to increased risk and worse cardiovascular outcomes

BET Inhibition Targets Pathways and Markers That Are Linked to CVD

Numerous epidemiological and interventional studies have demonstrated that the pathways and biomarkers that apabetalone modulates and improves via select BET inhibition provide biological plausibility and rationale to support the potential of apabetalone to be disruptive in the reduction of residual risk in patients. Below are several key studies that highlight the potential roles of these pathways and biomarkers that have been studied to date that may play a role in the early signal of MACE reduction following apabetalone treatment.

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, apabetalone, will be well-positioned to participate in the critically important global residual risk CVD market.

Vascular Inflammation

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). CRP is recognized as a major cardiovascular risk factor, in addition to providing prognostic information beyond LDL (Ridker, Cannon et al. 2005; Ridker, MacFadyen et al. 2010). CRP is an acute phase reactant produced by hepatocytes (liver cells) in response to proinflammatory mediators (cytokines, in particular interleukin-6, interleukin-17 and interleukin-1a) that reflects different degrees of inflammation (Shrivastava, Singh et al. 2015). Inflammatory processes play a pivotal role in the pathogenesis and pathophysiology of coronary artery disease ("CAD") and atherosclerosis that are largely driven by NF-kB and STAT3 (Brand, Page et al. 1997; Vasamsetti, Karnewar et al. 2015). Traditionally, CRP was thought to be a bystander marker of vascular inflammation, without playing a direct role in CVD (Li and Fang 2004). However, more recent evidence suggests a direct pro-inflammatory mediating role of CRP, associated with all stages of atherosclerosis (most notably unstable plaque development and rupture), and atherogenesis (including activation of the complement pathway, lipid uptake by macrophages, release of proinflammatory cytokines, inducing the expression of tissue factors in monocytes, promoting endothelial dysfunction, and inhibiting nitric oxide production) (Shrivastava, Singh et al. 2015).

Data has shown that an increased level of CRP in patients with unstable coronary syndromes is associated with short-term clinical outcomes (Tataru, Heinrich et al. 1999). Many large scale prospective studies have also demonstrated that CRP strongly and independently predicts adverse CV events, including myocardial infarction, ischemic stroke, and sudden cardiac death in individuals both with and without overt CHD (Shrivastava, Singh et al. 2015). 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). In addition, components of the acute phase response cascade are pro-inflammatory, pro-atherogenic and markers of CVD risk (Lowenstein and Matsushita 2004). These include MBL2, C2, C3 and C5, and SAP (Kulikowski, Gilham et al. 2016).

Vascular Calcification

Medial artery calcification is a characteristic feature of diabetes, CKD, and other high risk cardiovascular patients (Chistiakov, Sobenin et al. 2014). It has been well established that higher levels of alkaline phosphatase ("ALP") are significantly associated with the presence of vascular calcification (Lomashvili, Garg et al. 2008; Ishimura, Okuno et al. 2014), and that vascular calcification in turn is associated with increased cardiovascular morbidity and mortality (Schoppet and Shanahan 2008). ALP breaks down and inactivates inorganic pyrophosphate, a known highly potent inhibitor of vascular calcification and calcific crystal growth (Lomashvili, Garg et al. 2008). Analysis of the CARE and NHANES III study data provide insight into the epidemiological aspect that ALP contributes to all-cause mortality and CVD mortality. Data from the CARE study 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 study reveal that patients 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 calcific state (Tonelli, Curhan et al. 2009). Recently, the authors of a review elucidating the link of ALP to vascular calcification proposed that ALP is an emerging treatment target for CVD and metabolic syndrome and inhibiting ALP represents a promising novel approach to the treatment and prevention of CVD complications in the general population, and acute inflammatory disorders associated with increased mortality in CKD and metabolic syndrome patients (Haarhaus, Brandenburg et al. 2017).

Metabolism

HDL and ApoA-I can directly modulate glucose metabolism through multiple mechanisms. It has been observed in the clinic that HDL elevation through infusion of reconstituted HDL results in the reduction of glucose in individuals with type 2 DM (Drew, Duffy et al. 2009). The first mechanism through which HDL modulates glucose metabolism is increased insulin secretion and the second is increased glucose uptake by skeletal muscle (Drew, Duffy et al. 2009; Fryirs, Barter et al. 2010). HDL may also act through a third mechanism to increase insulin sensitivity via lipid removal and anti-inflammatory actions in metabolic tissues (Carey, Siebel et al. 2013).

Metabolic syndrome refers to a cluster of conditions including high blood pressure (hypertension), high blood sugar (hyperglycemia), high blood cholesterol (hypercholesterolemia), and abnormal lipid levels in the blood (dyslipidemia). These conditions significantly increase the risk of CVD and diabetes. Data taken from 87 studies from the National Cholesterol Education program (NCEP) and the

revised NCEP consisting of over 950,000 patients showed that having metabolic syndrome increases the risk of cardiovascular outcomes by 2-fold and all-cause mortality by 1.5-fold (Mottillo, Filion et al. 2010).

Complement and Coagulation

Analyses of the complement and coagulation 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).

Reverse Cholesterol Transport

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 demonstrated 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 26% reduction of future coronary events. 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 validated 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 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). AMORIS' key findings indicate that improvement of 'cholesterol balance', or the ApoB to ApoA-I ratio, is a robust and specific marker of virtually all ischemic events.

Resverlogix's Drug Development Strategy and Commercial Rationale

Given the high cost, long development times and high attrition rates associated with drug development, many biotechnology companies partner with or license with 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.

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

- BET proteins contain highly-conserved bromodomains that play a key role in epigenetic control of gene expression;
- Apabetalone functions via inhibition of BET bromodomain binding to chromatin thereby modulating transcription of particular targets;
- Apabetalone preferentially binds to the second bromodomain of BET family members, with a 20-fold or higher selectivity for the second bromodomains of BRD2, BRD3 and BRD4 versus the first bromodomain;
- Apabetalone is highly differentiated from other therapies that focus only on single biological targets such as increasing HDL or decreasing low-density lipoprotein ("LDL") in plasma, and has effects on multiple pathways and biomarkers that function in concert to reduce CVD events.
- Apabetalone works via a physiological approach of activating the body's own health-promoting genes 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.
- Apabetalone is the only selective BET inhibitor with no known competitor, providing us with an estimated 7-8 year lead over competitors and significant scarcity value.

Based on the above reasons and clinical data, we believe apabetalone has illustrated potential to become an important and differentiated therapeutic for high risk patients with CVD, DM and CKD.

Cardiovascular Disease

MACE is by far the most important of all clinical endpoints that are analyzed for providing future predictability for CVD risk. Health systems and payer groups study MACE carefully when considering potential reimbursement of a new CVD therapeutic agent. MACE includes a variety of key markers of cardiovascular risk such as death, myocardial infarction, stroke, worsening angina, worsening peripheral artery pain and ischemia, prevention of percutaneous stent procedure, and hospitalization for cardiac-related incidents. According to the 2016 American Heart Association Statistics report, based on 2013 death rate data, over 2,200 Americans die of CVD each day, an average of 1 death every 40 seconds (Mozaffarian, Benjamin et al. 2016). Many of these CVD patients have some form of MACE during or after they have been diagnosed with CVD.

Although treatment of CVD includes many therapeutic agents such as lipid lowering drugs such as statins, heart rate lowering agents such as beta blockers and, 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 2016 American Heart Association Statistics report, more than 85.6 million Americans have one or more vascular disease (Mozaffarian, Benjamin et al. 2016). 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. 2016). CVD related conditions include angina, heart attack, stroke, aortic aneurysms, kidney failure and severe limb ischemia; all of which are contributed to by the increasing prevalence of obesity, hypertension, diabetes and dyslipidemia.

CVD research is expanding its focus from factors driving atherosclerosis (the key underlying cause of CAD and CVD) to additional vascular risk pathways. Other important areas of CVD research that are being carefully examined in high risk patients include vascular inflammation, the innate immune response, coagulation and vascular calcification.

Residual risk in high risk CVD patients represents a large market opportunity. Several pharmaceutical companies have attempted 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 included the cholesteryl ester transfer protein (“CETP”) inhibitors; Torcetrapib (Pfizer) and Dalcetrapib (Roche), and other approaches such as Lp-PLA2 inhibitors; Darapladib (GSK), many of which failed large scale phase 3 clinical trials in recent years. 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. Despite those results, this population remains at substantial risk for cardiovascular events. Despite adequate control of LDL, statin-treated patients with clinically evident CVD maintain a 5-year elevated risk for MACE (Barter, Gotto et al. 2007).

CVD risk factors are also associated with insulin resistance, and 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). 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 CVD events were 70 times more frequent than fatal microvascular complications (Turner, Cull et al. 1996).

Unfortunately, therapeutic interventions designed to raise serum HDL levels (such as niacin and the CETP inhibitors) have not been shown to reduce the risk for MACE or mortality (Barter, Caulfield et al. 2007; Boden, Probstfield et al. 2011). CETP inhibition does not increase ApoA-I production (or new HDL 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. This in part could explain why CETP inhibition treatment is associated with an increase in CRP and lack of positive effects on atherosclerosis and MACE in human studies relative to standard of care inclusive of statins.

High residual risk of MACE, even in patients with controlled LDL, is further evident in the recent EXAMINE 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 (a selective inhibitor of dipeptidyl peptidase 4 that is approved for the treatment of type 2 diabetes) 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. 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 and coronary-artery bypass grafting procedures, the incidence of MACE in DM was 27% and 19% respectively over the subsequent five 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.

We are evaluating the potential use of apabetalone in the Phase 3 BETonMACE clinical study in this therapeutic area.

Chronic Kidney Disease and End-Stage Renal Disease

CKD can result from the long term effects of DM on blood vessels and the filtration apparatus (nephrons) of the kidneys. CKD 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 large, exceeding $34 billion each year in the U.S. The typical cost of dialysis per patient per year is nearly $90,000.

The Company has received regulatory approval from the FDA Cardiovascular and Renal Division and plans to further evaluate CKD and ESRD biomarkers to determine the potential use of apabetalone in this important disease area in a phase 2a study in patients with ESRD treated with hemodialysis. A subgroup analysis of CKD patients is also planned in the Phase 3 apabetalone BETonMACE clinical study in this therapeutic area.

Diabetes Mellitus

Diabetes mellitus 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, and underlie the thinking that ApoA-I/HDL-raising strategies may have benefits beyond vascular disease to not only treat but possibly prevent T2DM. Therefore, we conducted an exploratory study examining the effects of using apabetalone 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.

Fabry Disease

Fabry Disease is an inherited genetic disorder that is classified as a lysosomal storage disease. It results from a mutation leading to a deficiency of the enzyme alpha galactosidase-A, which is responsible for the breakdown of the glycolipid globotriaosylceramide (Gb3). This deficiency causes an accumulation of Gb3 within blood vessels, tissues, and organs. Gb3 buildup starts at childhood, leading to the manifestation of a variety of symptoms in many parts of the body. The major symptoms of Fabry disease include episodes of pain (mainly in the hands and feet), clusters of dark red spots on the skin called angiokeratomas, insufficient or sometimes excessive sweating, cloudiness of the front part of the eye, gastrointestinal issues, and hearing issues. Due to the progressive build-up of Gb3 over time, the disease generally becomes more life-threatening as an individual gets older. Life expectancy for this disease, as reported by the Fabry Registry, was 58.2 years for males and 75.4 years for females. The prevalence is reported to be 1/17,000 people. Fabry disease generally has a poor diagnosis due to the fact that the initial symptoms presented are mild and tend to be misdiagnosed. Two major organs, the heart and kidneys, are affected in the later stages of the disease progression, which ultimately are the major causes of death. Cardiac complications arising from Gb3 buildup in the heart are the leading causes of death followed by stroke and kidney complications. Generally, kidney complications manifest early on and get

treated by renal replacement therapy. It is usually these patients with a history of renal replacement therapy that end up having a serious cardiac complication.

The first-line treatment for Fabry is enzyme replacement therapy (ERT), which involves the intravenous administration of alpha galactosidase into patients to compensate for the deficiency. Adjunctive medications include acetylsalicylic acid, ACE inhibitiors or ARBs, statins, and pain medications. The efficacy and cost-effectiveness of ERT has been questioned due to many long-term studies showing that ERT does not affect the disease progression, but rather only prolongs the time for the incidence of the first complication. Additionally, studies have shown that ERT is beneficial before a patient has major symptoms. However, most healthcare system guidelines require a patient to be symptomatic in order to be eligible to receive ERT. Recently a small molecule chaperone therapy called Galafold™ by Amicus Therapeutics was approved in Europe. This therapy, however, only works for about 35-50% of patients with certain mutations. There is still a considerable unmet need for Fabry patients as ERT does not improve the disease progression and the newly approved small molecule therapy can only target at most half of the Fabry disease patient population. Current medications approved for Fabry disease are not sufficient and there remains a large unmet need.

The Company has received approval from Health Canada and plans to evaluate the potential use of apabetalone in a phase 2a exploratory clinical study to assess the patient safety and effect on key biomarkers of Fabry disease.

Neurodegenerative Disease

Epidemiological and mechanistic evidence indicate a link between CVD and neurodegenerative diseases such as dementia and mild cognitive impairment. 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 these neurodegenerative diseases. There are also links to cognition demonstrating an association between risk of dementia and mild cognitive impairment with a history of stroke, myocardial infarction, peripheral artery disease, and carotid plaques. Other findings demonstrate similar relationships between these neurodegenerative diseases and CAD, myocardial infarction, cardiac arrest, carotid atherosclerosis, and hypercholesterolemia.

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

To evaluate the potential of BET inhibition in neurodegenerative disease, a variety of cell studies are underway to investigate the role of epigenetic modification. Moreover, ongoing preclinical studies are exploring the role of apabetalone mediated modulation of neuroinflammation and innate immunity in this important disease area. The Phase 3 BETonMACE clinical study will include a subgroup analysis evaluating cognitive function in patients over the age of 70.

Competitive Environment

New approaches for residual MACE reduction in CVD patients are constantly being pursued by key stakeholders in the pharmaceutical marketplace. Several new hypotheses such as LDL-lowering (PCSK9 inhibitors), glucose lowering (SGLT2 inhibitors), HDL increasing/LDL-lowering (CETP inhibitors) and epigenetic regulation (BET inhibitors) are being developed in this therapeutic area.

The LDL-lowering hypothesis is being further tested in CVD with aggressive reduction of LDL to unprecedented levels. PCSK9 inhibition is one such approach towards lowering LDL with potential implications for reducing MACE in high risk vascular patients. PCSK9 inhibitors are monoclonal antibodies that inactivate the liver proprotein convertase subtilisin kexin 9 (“PCSK9”) (Seidah 2013). The function of this protein is to bind to LDL receptors on the surface of hepatocytes (liver cells) that transport LDL into the liver for metabolism and clearance. Binding results in reducing LDL clearance from the blood (Seidah, Awan et al. 2014). Therefore, targeted PCSK9 inhibition leads to more LDL receptors remaining active on liver cell surfaces to capture LDL for removal from blood, thereby promoting clearance and reducing blood LDL levels (Gouni-Berthold and Berthold 2014). This mechanism of therapeutically inhibiting PCSK9 to promote the clearance of LDL from the blood by prolonging the life span of the LDL-receptors differs greatly from statins, which inhibit the synthesis of cholesterol. The other key aspect that differentiates PCSK9 inhibitors from many of the other cardiovascular medications is that it is an IV infusion that has to be administered 1-2 times a month.

Praluent ("alirocumab") (Sanofi-Regeneron) was the first PCSK9 inhibitor approved by the FDA (July 24th, 2015) as a second line treatment to lower LDL in adults who have hereditary high cholesterol and patients who require additional LDL lowering when diet and statin treatment have been unsuccessful. Its approval was based on the ODYSSEY LONG TERM phase 3 trial. In a post hoc analysis evaluating MACE rates, a lower rate of events was observed in the alirocumab group compared to the placebo group (Robinson, Farnier et al. 2015). Alirocumab is currently being investigated for secondary prevention of MACE in ACS patients (ODYSSEY OUTCOMES Trial) in a large cohort of over 18,000 patients, expected to be completed in 2017. Repatha ("evolocumab") (Amgen) was the second PCSK9 inhibitor to be approved by the US FDA (August 27th, 2015) for the same second line indication as Praluent. The OSLER phase 3 clinical trials were the basis for this approval. MACE was prospectively adjudicated in an exploratory analysis and patients in the evolocumab group had a lower event rate compared to those in the placebo group (Sabatine, Giugliano et al. 2015). Evolucumab was investigated for secondary prevention of MACE in patients with high cholesterol and clinically evident CVD (FOURIER Outcomes trial) in a large cohort of over 27,000 patients. Relative to placebo, evolocumab treatment significantly reduced the risk of the primary end point (composite of cardiovascular death, myocardial infarction, stroke, hospitalization for unstable angina, or coronary revascularization) with a hazard ratio of 0.85. Although this data outcome was statistically significant from a scientific perspective, reimbursement of such drugs with potentially high cost threshold to prevent a single CVD event are under ever increasing scrutiny from the important stakeholder group, the payers. The reimbursement community offered comments that this approach for residual risk reduction would potentially prove to be too costly. Many key US payers groups have reviewed and reported their perceptions of the data. . Although both ODYSSEY LONG TERM, OSLER and FOURIER 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 inefficient. Independent Payer groups such as ICER have determined this price to be too high and have suggested, "a price of $2,177 per year for this group of new therapeutics [is required] in order to keep overall health cost[s] within bounds" (Tice, Ollendorf et al. 2015). This reported price threshold by a payer group represents a significant price reduction over the planned retail price of these drugs. Due to expensive antibody manufacturing procedures and other cost factors for these types of therapeutic interventions, the commercial model for this class of drugs will face increasing viability pressures in today's ever increasing cost-containment environment. In addition during the regulatory review of this new class of therapeutics, the FDA raised concerns about possible gastrointestinal, metabolic, and neurocognitive adverse effects which are being further evaluated in the phase 3 studies.

Additionally, recent studies on therapeutics designed to improve glycaemic control in diabetes patients (SGLT-2 and GLP-1 inhibitors) have shown that these interventions lower the incidence of MACE (which was defined as the composite of cardiovascular death, nonfatal myocardial infarction, or nonfatal stroke) by 13-14% (Zinman, Wanner et al. 2015; Marso, Daniels et al. 2016). Since the release of these results, however the mechanisms via which the reductions in MACE occur are being debated.

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 the findings of 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, particularly 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 PREVEND study also came to similar conclusions (Borggreve, Hillege et al. 2006). Anacetrapib, a CETP inhibitor being developed by Merck, was evaluated in the REVEAL study. It was recently announced that anacetrapib treatment compared to placebo significantly reduced the primary end point defined as the composite of coronary death, myocardial infarction, and coronary revascularization in 30,000 patients. The results of the REVEAL study will be presented at the European Society of Cardiology meeting in August 2017. This data will be closely scrutinized by payers to ensure compelling value.

Efficient numbers needed to treat ("NNT") are also an important assessment performed by reimbursement and payer groups in the accretive value added of these technologies. There are several advantages to small molecule products and research platform over potential antibody competitors such as the PCSK9 inhibitors. 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. As a result, payers will demand clear efficacy and price value model for risk reduction. If apabetalone 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.

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

Key Products in Development (Resverlogix and Competitors)

Product	Indication	Mechanism of Action	Market Status	Threat Assessment
Apabetalone/RVX-208 (Resverlogix)	Secondary MACE Prevention	BET Inhibition	Phase 3	N/A
Alirocumab (Sanofi/ Regeneron)	CVD	PCSK9 Inhibition	On Market	Moderate / High Cost
Evolocumab (Amgen)	CVD	PCSK9 Inhibition	On Market	Moderate / High Cost
Ezetimibe (Merck)	CVD	Cholesterol Absorption	On Market	Low/ Indication for CVD Outcomes Rejected
Anacetrapib (Merck)	CVD	CETP Inhibition	Phase 3 (Trial Completed)	Low / Long Half Life
Bococizumab (Pfizer)	CVD	PCSK9 Inhibition	Phase 3 (Trial Discontinued)	Moderate / High Cost
Losmapimod (GSK)	CVD	Inflammation Mediators	Phase 3 (Trial Failed)	Low/Poor CVD Outcome Efficacy
Darapladib (GSK)	CVD	Inflammation	Phase 3 (Trial Failed)	Low
A-002 (Anthera)	CVD	Inflammation	Phase 3 (Trial Failed)	Low
Dalcetrapib (Roche)	CVD	CETP Inhibition	Phase 3 (Trial Failed)	Low
Evacetrapib (Lilly)	CVD	CETP Inhibition	Phase 3 (Trial Failed)	Low

General Development of the Business

Recent Clinical Developments

The following principal events have influenced the general development of our business during the most recent three fiscal years. Detailed findings and highlights from the clinical development of apabetalone are highlighted below. As described below, we have reported several important findings over the past three years.

Phase 3 BETonMACE Trial – Enrolling

On January 15, 2014, we announced the pooled MACE results from the SUSTAIN and ASSURE studies. This analysis 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. Results from patients with diabetes mellitus, a high-risk patient population, illustrated similar trends. Based on these findings, our intention with our BETonMACE trial is to reconfirm in a larger prospective setting, with patients that have modifiable vascular disease (i.e. low HDL-C and diabetes), reduction of MACE coupled with favorable effects on markers of vascular risk and renal function.

The BETonMACE study, "Effect of RVX-208 on Time to Major Adverse Cardiovascular Events in High-Risk Type 2 Diabetes Mellitus Subjects with Coronary Artery Disease" is a large international multi-center, double-blind, randomized, parallel group, placebo-controlled clinical trial to determine whether treatment with apabetalone in combination with rosuvastatin or atorvastatin increases the time to MACE compared to treatment with rosuvastatin or atorvastatin alone. The primary endpoint of the BETonMACE trial is designed to show a relative risk reduction ("RRR") of MACE, narrowly defined as a single composite endpoint of CV death, non-fatal myocardial infarction ("MI") and stroke. The study is an event-based trial and will continue until at least 250 MACE events have occurred. MACE will be adjudicated by an independent committee and the study will be monitored by a data and safety monitoring board. Secondary endpoints include, time to first occurrence of the composite broad MACE which includes the addition of hospitalization for CVD events (unstable angina and revascularization procedures), changes in lipoprotein concentrations (HDL and apolipoprotein A-I ("ApoA-I"), changes in DM variables (glucose and glycated hemoglobin), change in ALP, changes in kidney function and additional safety and tolerability of apabetalone. In order to be eligible to participate in the study, patients must have documented history of type 2 DM, experienced a recent (defined in the study as 7-90 days prior to randomization) CAD event

including unstable angina, revascularization procedure or MI and have low levels of HDL (<40 mg/dL for males and <45 mg/dL for females). Standard of care high potency statin therapy consists of daily doses of either atorvastatin 40-80 mg or rosuvastatin 20-40 mg. After an initial screening period of 1 to 2 weeks during which subjects will be treated with standard of care statin therapy, subjects are randomized to either apabetalone 100 mg b.i.d. (twice daily) or matching placebo with continued statin treatment. This combination treatment period continues for up to 104 weeks. We anticipate that a minimum of 2,400 patients will be enrolled. A full detailed protocol for the BETonMACE study can be viewed on clinicaltrials.gov with the following NTC ID, NCT02586155.

BETonMACE Will Enroll Patients From 14 or More Countries Worldwide

On June 22, 2015, we announced that, following meetings with various European regulatory bodies, the first confirmation for our Phase 3 clinical plan was received.

On August 4, 2015, we announced that we had established an international Clinical Steering Committee ("CSC") for the BETonMACE trial, comprised of: Chairman Kausik K. Ray, BSc (hons), MBChB, MD, MPhil (Cantab), Henry N. Ginsberg, MD, Kamyar Kalantar-Zadeh, MD, MPH, PhD, Stephen J. Nicholls, MBBS, PhD, Gregory G. Schwartz, MD, PhD, and Peter P. Toth, MD, PhD. Detailed biographies for the committee members are highlighted under "Clinical and Scientific Advisory Boards". The CSC's role is to advise on the trial design, provide overall supervision of the trial and ensure that it is being conducted in accordance with the principles of Good Clinical Practice. The CSC has oversight of the trial's protocol, any protocol amendments and provides advice to the investigators on all aspects of the trial.

On September 29, 2015, we announced that our clinical, science and business teams, along with two members of the international clinical steering committee for the BETonMACE study, presented a Research and Development Update for apabetalone on September 25, 2015 in New York City. Highlights from the presentation included an in-depth analysis of the large unmet medical need in diabetes patients at high risk for secondary MACE presented by the chair of the BETonMACE clinical steering committee, Kausik K. Ray. In addition, a breakdown of the unmet medical need in CKD and the therapeutic potential of apabetalone in this indication were presented by CSC member, Kamyar Kalantar-Zadeh. The final design of the BETonMACE clinical trial, novel biology and findings from ongoing mechanistic studies focused on cardiovascular risk pathways and apabetalone's potential in reducing MACE and findings from primary market research focusing on primary care physician and payer perceptions of apabetalone were all detailed.

On October 26, 2015, we announced the commencement of the Phase 3 BETonMACE clinical trial. We received initial approval from the regulatory authority and ethics committee in the first three countries: Belgium, Hungary and Israel, which represented approximately 36 investigative sites of an expected 177 sites, including 15 in the first activation wave occuring in 2015. The first site initiation visit was held and enrollment of patients commenced, with additional investigative sites to be activated soon thereafter.

On November 11, 2015, we announced that the first patient in the Phase 3 BETonMACE clinical trial was randomized and dosing had commenced two weeks after the opening of our first sites.

During early calendar 2016, we hosted investigator meetings for BETonMACE in Spain and Latin America to inform investigators about the trial's protocol, safety and efficacy event adjudication, and scientific aspects and mechanism of action of apabetalone.

As of April 30, 2016, all planned countries, namely Argentina, Belgium, Bulgaria, Croatia, Germany, Hungary, Israel, Mexico, Poland, Serbia, and Slovakia had received regulatory approval to open clinical investigator sites and were recruiting patients.

The independent Data and Safety Monitoring Board ("DSMB") for the BETonMACE trial completed four planned safety reviews of the trial (in August 2016, December 2016, March 2017 and June 2017, respectively). As part of each of its planned safety reviews, the DSMB reviewed available study data and noted that no safety or efficacy concerns were identified. Following each review that has been conducted to date the DSMB has recommended that the trial should continue as planned without any modification. The DSMB will conduct additional periodic reviews and will also perform a futility assessment once 125 adjudicated major adverse cardiac events have been observed.

On July 10, 2017, we announced the first randomized patient in Taiwan in BETonMACE.

On July 25, 2017, we announced the receipt of a positive Type C written response from the Division of Metabolism and Endocrinology Products of the FDA. In light of updated information regarding apabetalone, including: human exposure, clinical dosing and established acceptable safety margins, the FDA is allowing us to include United States of America patients in Phase 3 studies, including the global Phase 3 BETonMACE trial. We have agreed to make adjustments to the existing BETonMACE study protocol and to update the Investigator's Brochure and the Informed Consent Documents. With the approval from the FDA, we now have the opportunity to include US patients in BETonMACE. We have enrolled in excess of 70% of the approximately 2,400 patients outlined in the study's protocol.

Phase 1 Pharmacokinetic Trial in Patients with Severe Kidney Function Impairment – Completed

On July 21, 2016, we announced that dosing had commenced in a Phase 1 pharmacokinetics ("PK") study with apabetalone in patients with severe renal impairment. This trial was initiated and designed in accordance with our strategy to expand into new indications such as renal and orphan diseases. The primary objective of the Phase 1 study, based in New Zealand, was to determine if apabetalone treated patients with severe renal impairment had the same PK profile as has been illustrated in previous apabetalone trials. As expected, results showed no significant difference in PK between renal failure patients and age and sex matched controls.

On November 17, 2016, we announced the collection of data from the New Zealand based Phase 1 PK study with apabetalone in patients with severe renal impairment. The primary objective of the Phase 1 study was met by demonstrating that apabetalone treated patients with severe renal impairment have the same favorable PK traits and safety profile as has been observed in previous apabetalone trials. These results allowed us to proceed with the planning of more advanced renal impairment and dialysis trials. The study also explored acute changes in biomarkers relevant to BET inhibition in subjects with severe renal impairment, the data for which is still being analyzed.

On January 23, 2017, we announced preliminary results from the Phase 1 PK study in late stage CKD patients. The data showed remarkable results in reducing CVD and CKD risk factors and disease biomarkers in patients with late stage CKD versus healthy-matched control patients. It is believed that this is the first time in medical history that a direct connection of this type can be made between epigenetic regulation and its potential for positive disease impact. Protein data was collected following a single oral administration of 100mg of apabetalone before and after multiple time points in both cohorts. Initial findings from this study revealed a highly differential protein signature at baseline between CKD patients and controls. Following a single dose administration of apabetalone in the late stage CKD patients, the levels of multiple plasma proteins were changed within 12 hours after dosing, demonstrating a fast onset of drug action. Analysis of the changes in protein levels at the 12-hour time point revealed that, in the late stage CKD patients, 33 percent of proteins had statistically significant changes ($p<0.05$) compared to only 10 percent in the controls. Of these significant proteins, several established renal biomarkers such as interleukin 6 ("IL6") and osteopontin, were regulated positively with respect to disease severity and progression. Ongoing expanded analysis of this exploratory data is also planned which will look at Ingenuity Pathway Analysis (IPA). The quick onset of action and improvement of reported CKD risk factors are encouraging for us in our planned expansion beyond our current cardiovascular and diabetes program. Detailed data will be submitted for future peer reviewed publications.

Phase 2a Pharmacokinetic (Part A) and Efficacy/Safety (Part B) Trial in Patients with End-Stage Renal Disease Treated with Hemodialysis – Planned

On May 24, 2016, we announced the formation of an International Renal Clinical Advisory Board ("RCAB") for the future development of apabetalone into expanded renal indications. The RCAB is comprised of: Chairman Dr. Kamyar Kalantar-Zadeh, MD,

MPH, PhD, Dr. Carmine Zoccali, MD, FASN, FNKF, FERA, Dr. Marcello Tonelli, MD, SM, FRCPC, Dr. Vincent Brandenburg, MD, Dr. Srinivasan Beddhu, MD, and Dr. Mathias Haarhaus, MD, PhD.

On February 23, 2017, we announced the receipt of the final minutes of an in-person Type B meeting with the Cardiovascular and Renal Products Division of the U.S. FDA. The purpose of the meeting was to request written comments, recommendations and feedback on the proposed protocol for a Phase 2a kidney dialysis trial. The primary objective of the study will be to evaluate if treatment with apabetalone in combination with standard of care (SoC) decreases alkaline phosphatase in comparison to placebo and SoC. In light of guidance received from the FDA, the Phase 2a study design will be separated in two parts. Part A will involve a single-dose pharmacokinetic (PK) study in eight patients receiving hemodialysis. Part B will be a double-blind, randomized, placebo-controlled, sequential cross-over study with apabetalone, and is designed to evaluate biomarker changes and safety parameters with apabetalone in up to 30 patients with end-stage renal disease treated with hemodialysis.

On May 15, 2017, we announced the acceptance, by the Cardiovascular and Renal Products Division of the U.S. FDA, of the Company's Investigational New Drug (IND) application to commence a Phase 2a kidney dialysis trial. The details of the study are described above. We intend to proceed with the planned Phase 2a clinical trial in 2017.

Phase 2a Fabry Disease Trial – Planned

On May 30, 2017, we announced that Health Canada, Therapeutic Products Directorate, approved the Company's request to proceed with a clinical trial with its lead compound apabetalone in patients with Fabry disease. This study is an open-label, exploratory clinical study to assess the patient safety and effect on key biomarkers of apabetalone in subjects with Fabry disease for up to 16 weeks. The primary objective of the study is to evaluate the safety and tolerability of apabetalone in patients with Fabry disease. Secondary objectives include evaluating the effect of apabetalone in subjects with Fabry disease as determined by change in key biomarkers including alkaline phosphatase (ALP), high-sensitivity C-reactive protein (hs-CRP), and other well-known markers for chronic kidney disease.The study population will consist of two cohorts: Cohort 1: Patients with Fabry disease receiving enzyme replacement therapy (ERT) and Cohort 2: Patients with Fabry disease not receiving ERT. We intend to proceed with the planned Phase 2a clinical trial in 2017. Patients with Fabry disease experience various heart, kidney, and dermatological complications with stroke, heart disease and kidney complications being the top causes of mortality. Current medications approved for Fabry disease are not sufficient and there remains a large unmet need.

ASSURE and SUSTAIN Analysis

Our Phase 2 clinical program in CVD patients with varying degrees of disease severity is comprised of three trials: ASSERT, SUSTAIN and ASSURE. These studies provided us with a repository of samples that enable the interrogation of multiple biomarkers that are affected by treatment with apabetalone. The data generated from the analysis of these samples is the first and largest integrated dataset of the response of multiple vascular risk markers to an epigenetic drug treatment. To date, our BET database of hundreds of thousands of data points provides insight into how epigenetics and select BET inhibition affect target risk markers for vascular disease. We continue to add to these important data with ongoing sample analyses to further elucidate the role of epigenetics in this biology and its role in vascular disease.

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 apabetalone to impact MACE over a short time period of six months.

Combined data from both SUSTAIN and ASSURE trials (n=499) demonstrated that treatment with apabetalone led to a significant reduction in MACE. Patients treated with apabetalone (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 apabetalone 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 apabetalone 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 treated with apabetalone had a 55% (p=0.02) relative risk reduction ("RRR") in MACE. More importantly, this beneficial effect of apabetalone 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 apabetalone 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 apabetalone BET inhibition effects on RCT 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 presentation at the American College of Cardiology Scientific Session and Expo ("ACC") entitled, "Apabetalone 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 apabetalone vs. placebo (change, p value) in: serum ALP (-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 apabetalone were those with DM or CKD. In those with DM (n=192) treated with apabetalone, 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, apabetalone 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 eGFR <60 mL/min/1.73m^2) treated with apabetalone 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 apabetalone. This treatment demonstrated significant changes in cellular pathways or networks characterized by: attenuation in inflammation, coagulation, complement and cholesterol synthesis. These clinical findings illustrate the effects of apabetalone 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 apabetalone 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 ASSERT, 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 patients treated with apabetalone (n=331) compared to a reduction of -3.23% in patients treated with placebo (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 group treated with apabetalone (n=127) compared to -4.49% in the group treated with placebo (n=65) (p<0.0001). Further analysis was performed on patients with CKD defined by an eGFR of below 60 mL/min/1.73 m^2. In this group, patients treated with apabetalone (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 treatment with apabetalone, an increase in eGFR of +3.4% (p=0.04 vs. baseline) in the group treated with apabetalone was observed compared to a decrease of -5.9% in the group treated with placebo. 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 CVD and risk.

Analysis of the Phase 2b program pooled data continues to not only broaden our understanding but also provides a more targeted pathway for our future clinical trials with apabetalone. We plan to perform further detailed analysis on potential new biomarkers and biological pathways that apabetalone may affect through its select BET inhibition mechanism. New findings in these analyses will seek 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 2 Pre-Diabetes Mellitus Trial – Completed

In October 2012, we initiated an exploratory Phase 2 clinical trial in patients with pre-diabetes mellitus to examine the effects of apabetalone and ApoA-I production on glucose metabolism. The foundation of this trial built upon the actions of apabetalone and the knowledge that apabetalone 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 DM. The trial was conducted in collaboration with Baker IDI Heart & Diabetes Institute in Melbourne, Australia.

On June 8, 2015, we announced findings from this study 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 apabetalone (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 treatment with apabetalone 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 International Society of Atherosclerosis ("ISA") meeting in May 2015 in Amsterdam, NL. In 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 apabetalone 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 apabetalone has the ability to affect glucose and lipid metabolism in ways that will be of benefit to patients with CVD risks.

On June 2, 2016, we announced that findings from the above-mentioned study had been published in the journal 'Metabolism', titled: "Effects of the BET-inhibitor, RVX-208 on the HDL lipidome and glucose metabolism in individuals with prediabetes: A randomized controlled trial." Data summarized in the publication was gathered from patients (n=23) with pre-diabetes mellitus characterized by abnormal blood glucose level treatedt with apabetalone (200 mg/day) for 29-33 days.

Summary of Phase 2 Clinical Trials

Three Phase 2 studies in patients with CVD have been completed:

- 12-week ASSERT study enrolled 299 patients,
- 24-week SUSTAIN study enrolled 176 patients, and
- 26-week ASSURE study enrolled 323 patients.

Based on our clinical trials, we have developed a broader and more integrated view of the effects of treatment with apabetalone with safety and efficacy results for up to 6 months of treatment.

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 apabetalone induced ApoA-I production on atherosclerotic plaque regression in the setting of patients with CAD who have a low level of high density cholesterol ("HDL-C") using intravascular ultrasound ("IVUS"). Secondary objectives for ASSURE were safety and tolerability of apabetalone as reflected by adverse events, and effects of apabetalone on HDL, ApoA-I, Large HDL particles and non-HDL lipid parameters such as CRP and other markers of interest in CVD risk reduction.

Findings drawn from ASSURE included:

- low baseline HDL were the best responders;
- elevated baseline hsCRP were strong responders; and
- there were fewer MACE events in subjects treated with apabetalone (7.4%) vs. subjects treated with placebo (13.8%).

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 apabetalone 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 apabetalone for 24 weeks vs. placebo. Secondary endpoints included change in ApoA-I, LDL-C, non-HDL-C, apoB, triglycerides and HDL subclasses.

Findings drawn from SUSTAIN included:

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

Phase 2 ASSERT Clinical Trial – Completed

ASSERT was a 12-week randomized, double-blind, placebo-controlled, multi-center US study with 299 patients enrolled with stable CAD. The primary endpoint of the study was increased plasma ApoA-I levels compared to placebo group after three months of

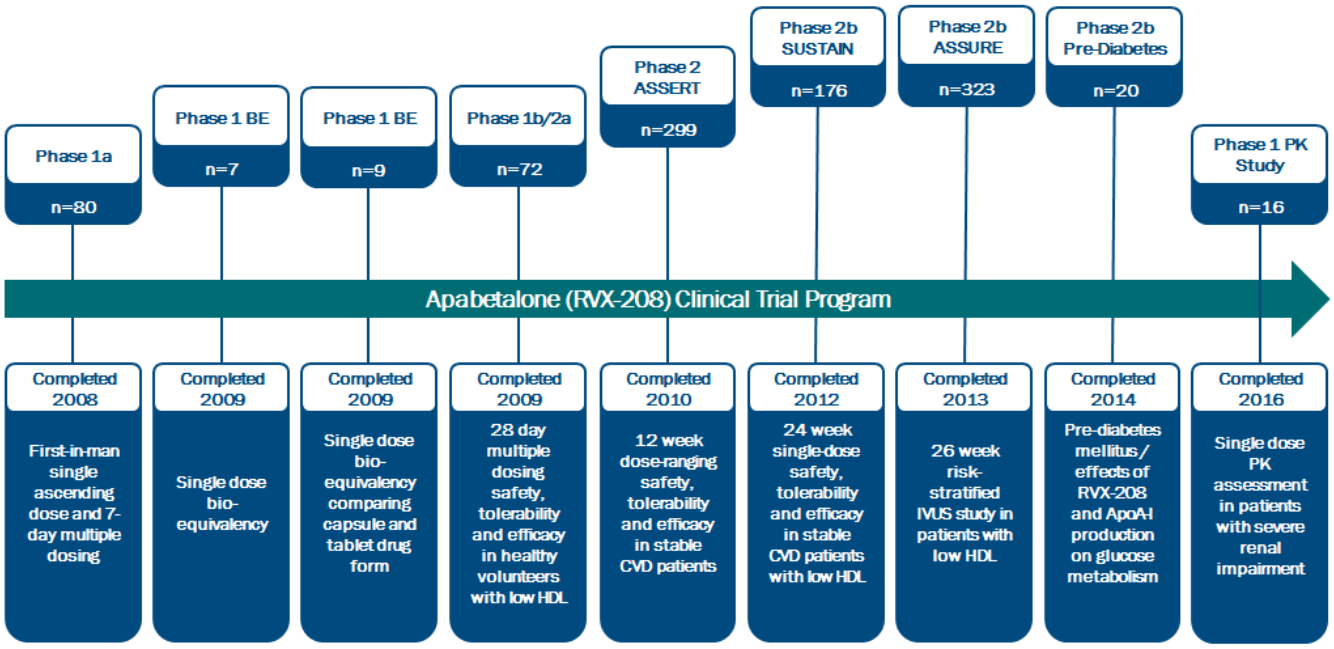
treatment with apabetalone. Other objectives were to examine the safety and tolerability of apabetalone and to compare the dose and time response relationship for ApoA-I as well as to examine key RCT markers involved with HDL functionality.

Findings drawn from ASSERT included:

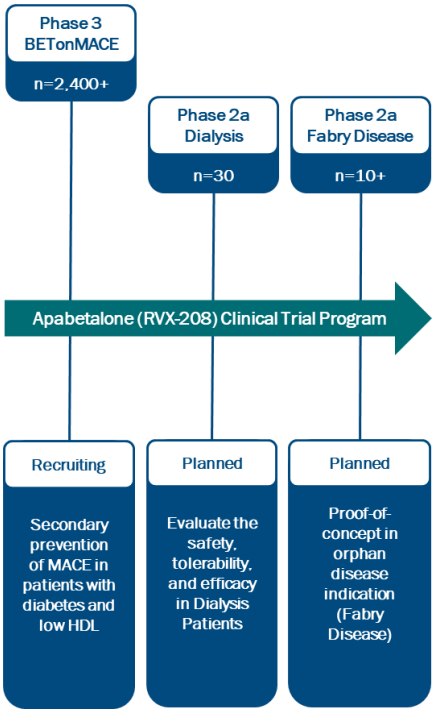
- Data illustrated that 200 mg/day of apabetalone was the optimal dose, based on safety and efficacy;
- Data illustrated that those patients with a low level of HDL-C at baseline had a better response for HDL-C and ApoA-I increases when treated with apabetalone; and
- Data illustrated that the best response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

These key findings contributed to determining a therapeutics window and targeted patient group for apabetalone.

Completed Apabetalone (RVX-208) Clinical Trials



Current or Planned Apabetalone (RVX-208) Clinical Trials



Scientific Developments

Based on our observed MACE reduction data from the pooled analysis of the SUSTAIN and ASSURE clinical trials, a number of hypotheses were generated to help investigate the driving factors responsible for the MACE reductions observed. Based on our research and in-depth analysis of the activity of apabetalone in multiple cell types, a combination of BET responsive activities was identified as likely underlying the MACE reductions observed in the clinic, including, reverse cholesterol transport, directional changes towards normalization of perturbed vascular inflammation, vascular calcification, complement and coagulation.

We performed microarray-based gene expression analysis using primary human hepatocytes treated with apabetalone. Cells from multiple donors were assessed in independent experiments. Results demonstrated that apabetalone downregulates pathways that contribute to cardiovascular risk or MACE such as atherosclerosis, thrombosis and inflammation. Specifically, apabetalone downregulated the complement, fibrin clotting, acute phase response, cholesterol and fatty acid synthesis pathways, illustrating repression of most of the pathway components. Overactivation of the complement pathway and acute phase response participate in plaque development and destabilization. Fibrin clotting is fundamental in the formation of thrombi and emboli. Downregulation of these pathways by apabetalone may avoid catastrophic vascular events leading to occlusion and death. Results for several components of the complement and coagulation pathways were verified by real-time polymerase chain reaction (“PCR”), a more sensitive and robust method of measuring mRNA expression, as well as using enzyme-linked immunosorbent assay (“ELISAs”) to measure protein levels. Results were also recapitulated in human hepatocarcinoma cell lines and with BET inhibitors with different chemical scaffolds. In addition, in patients’ serum from the SUSTAIN and ASSURE clinical trials, apabetalone reduced levels of specific complement, coagulation and acute phase response proteins. Further analysis of protein levels in patients’ serum from our previous clinical trials is in progress.

Multiple cell types present in blood, including monocytes, lymphocytes and neutrophils, contribute to CVD. To assess the effect of apabetalone, we treated human whole blood from healthy volunteers ex vivo and analyzed gene expression using microarrays. The analysis identified multiple pro-inflammatory and pro-atherosclerotic genes that are downregulated by apabetalone. Many of the apabetalone targets predict reduction in atherosclerosis, CVD and risk of MACE in patients. Other groups have shown that these proteins are upregulated in blood from sites of occlusion during acute myocardial infarction. These genes either play a direct role in the acute event or they constitute a tissue response to the occlusion. In any event, data shows that apabetalone may impact pathways underlying CVD and/or an acute event.

Apabetalone-mediated BET inhibition affects multiple processes important for CVD. In addition to effects on lipoproteins, apabetalone 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 apabetalone treatment, or select BET inhibition, attenuates the inflammatory process that contributes to disease initiation and progression. Furthermore, we hypothesize that treatment with apabetalone induces directional changes towards normalization of perturbed inflammatory states, restores basal activity of the innate immune response and clotting cascade with immediate benefits to atherosclerosis and cardiovascular disease. We are currently performing further detailed analysis on potential new biomarkers and biological pathways that apabetalone 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. These new findings have been presented at various prestigious conferences including the American College of Cardiology Scientific Session, American Heart Association Scientific Sessions and the World Diabetes Congress as highlighted under "Clinical Trial Developments."

During the year, we presented several findings including the following:

- data demonstrating that apabetalone treatment reduced basal and cytokine-induced expression of complement factors in hepatocytes. Furthermore, in samples from CVD patients, the complement pathway was identified to be the most downregulated by apabetalone treatment. This was supported by an observed reduction in the levels of complement proteins, which have been linked to CVD and MACE. A reduction in the overall function of the complement cascade in plasma from cardiovascular disease patients treated with apabetalone was also presented. As complement components are linked to CVD and metabolic syndrome, including major acute cardiac events, modulating their levels and activity by apabetalone may alleviate risks associated with these diseases;
- two potential new indications recently identified by third party academic research involving our lead drug, apabetalone, including degenerative diseases of the eye (retinal), and Facioscapulohumeral Muscular Dystrophy (FSHD). Both indications are areas of interest and licensing potential for us;
- new data from the recently completed phase 1 PK study in patients with severe renal impairment highlighted an upregulation of pathways known to be activated in CKD such as the inflammatory response, immune response, thrombosis, calcification and oxidative stress, in the renal impaired patients (stage 4 CKD) compared to controls. These pathways were robustly and highly significantly downregulated in the stage 4 CKD patients 12 hours after a single oral administration of apabetalone. Apabetalone treatment also downregulated the abundance of circulating CKD biomarkers involved in vascular inflammation, endothelial dysfunction, acute phase response, coagulation and vascular calcification. The data presented provides the rationale for our CKD program, with apabetalone, which is aimed at assessing the cardiovascular risk reducing potential, in addition to any improved kidney function, observed in prior Phase 2 trials.

Potential Orphan Disease Indications

Based on expanded publications and knowledge of epigenetics, BET and BRD4 inhibition, our additional proprietary analysis of BET-treated human patients and our recent proteomics assessment, new observed pathways, genes and biomarkers that are known to play a role in orphan diseases have been elucidated. New data generated in our research laboratory has demonstrated that BET inhibition by treatment with apabetalone has effects on multiple biological pathways that underlie disease pathology. Based on these recent advancements and scientific knowledge gained, we intend to expand our research and development efforts to explore orphan diseases. We will perform detailed commercial and scientific analysis in all of these opportunities to build the best possible rationale for advancing any of these opportunities forward. In addition to apabetalone, preclinical testing with other BET inhibitors from within our compound library has demonstrated similar effects on important markers known to play a role in orphan diseases. These compounds are under consideration as follow-on compounds.

Orphan Disease Fact Sheet

- Defined as rare diseases and disorders.
- Affect fewer than 200,000 people in the US.
- An estimated 7,000 rare diseases have been identified affecting over 30 million patients in the US.
- 400 drugs and biologics have been FDA approved.
- Due to the difficulty in recovering the therapeutic development costs associated with small patient segments, the Orphan Drug Act (ODA) was introduced in 1983 to foster research into rare diseases.
- The ODA provides for granting special status to a drug or biological product to treat a rare disease. This status is referred to as orphan designation.
- Orphan designation allows the drug sponsor to benefit from incentives for the development of these products.
- Incentives include tax credits on clinical research, technical assistance during new drug application (NDA) filing and exclusivity of 7 years after the marketing approval is granted.

Source: NIH Rare Diseases Clinical Research Network Fact Sheet

Regulatory Affairs

Apabetalone is being investigated for the secondary prevention of MACE including emergency/urgent revascularization in patients with type 2 DM, and low HDL. An IND was filed and accepted by the FDA in 2007 to commence clinical testing in humans. Subsequently, clinical trial applications were submitted and accepted by national, central and local health authorities in Poland, Belgium, Spain, Netherlands, Russia, Brazil, Argentina, South Africa and Australia in connection with Phase 2 clinical trials. 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 submitted safety data and other pertinent information in the form of annual reports to the FDA and applicable ex-US health authorities. We continue to provide all applicable health authorities with the required nonclinical, manufacturing and clinical information as outlined in country specific regulations.

Meetings with health authorities in 2015 in Germany, Sweden and UK provided scientific insight useful in the design of a Phase 3 study to support filing a Marketing Authorisation Application ("MAA") within Europe; the learnings were incorporated into the Phase 3 BETonMACE clinical study which commenced recruitment in the fall of 2015. The BETonMACE study has received clinical trial approval from regulatory authorities in 13 countries.

Future product development activities related to apabetalone and/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.

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 cardiovascular, inflammatory, autoimmune disease, cancers and fibrotic indications.

As of July 20, 2017, we own and/or have rights to numerous patent families, comprising fifteen issued US patents and a number of pending applications. The portfolio also includes a number of pending US applications and world-wide equivalents. In fiscal 2012, we were issued five patents within four patent families in two jurisdictions, including a United States Patent covering composition of matter claims to apabetalone, and a United States Patent covering the manufacturing of apabetalone. In fiscal 2013, we were issued eight patents within four patent families in six jurisdictions, including a United States Patent related to inflammatory diseases. In fiscal 2014, we were issued fourteen patents within five patent families in nine jurisdictions, including a patent in Europe containing composition of matter claims to apabetalone. In fiscal 2015, we were issued eighteen patents within seven patent families in eleven jurisdictions, including United States patents related to use claims for apabetalone and manufacturing of apabetalone. In fiscal 2016, we were issued seventeen patents within seven patent families in twelve jurisdictions, including United States patents related to use claims for apabetalone. In fiscal 2017, we were issued eighteen patents within six patent families in thirteen jurisdictions, including a United States patent related to formulation of apabetalone.

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 disclosure of proprietary knowledge.

We also believe that our proprietary know-how will provide a significant competitive advantage; we 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 collaborators to execute confidentiality agreements. Employees, consultants and contract research organizations specify that all inventions resulting from work performed utilizing our property, business strategies, and work completed pursuant to their employment/services performed remains our exclusive property to the fullest extent permitted by law.

Clinical and Scientific Advisory Boards

BETonMACE Clinical Steering Committee

Our Clinical Steering Committee ("CSC") for the BETonMACE trial, established in August 2015, provides overall supervision of the clinical trial and ensures that the trial is conducted in accordance with the principles of Good Clinical Practice and applicable regulations. The CSC established the trial protocols, any protocol amendments and provides advice to the investigators on various aspects of the trial. The members of the CSC are as follows:

Professor Kausik Ray is Professor of Public Heath, 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 CVD using observational methods and intervention studies including large clnical trials. Professor Ray established the first global registry of Familial Hypercholesterolaemia in conjunction with the European Atherosclerosis Society (EAS) named the FH studies collaboration (FHSC) and is the Principal Investigator for the TOGETHER study investigating cardiometabolic risk factors and clinical outcomes in approximately 250,000 people using electronic health records in London, England.

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 220 publications in medical and scientific journals and textbooks. He is Editor-in-Chief of the *Year in Lipid Disorders* 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* with Michael Davidson of *Therapeutic Lipidology*, with Dominic Sica of *Current Controversies in Dyslipidemia Management*, with Kevin Maki of *Practical Lipid Management*, with Christopher Cannon of *Comprehensive Cardiovascular Care in the Primary Care Setting*, and Domenic Sica of *Clinical Challenges in Hypertension* vols I and II. 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 IVUS 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 National Institute of Health ("NIH") funded Clinical Translational Science Awards. Dr. Ginsberg is also Principal Investigator on two R01 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.

Renal Clinical Advisory Board

Our Renal Clinical Advisory Board ("RCAB") was established in May 2016 for the future development of apabetalone into expanded renal indications. The members of the RCAB are as follows:

Dr. Kamyar Kalantar-Zadeh (Chair): Dr. Kalantar-Zadeh is Professor and Chief, Division of Nephrology and Hypertension at University of California, Irvine. Dr. Kalantar-Zadeh is the founder and director of the Harold Simmons Center for Kidney Disease Research and Epidemiology. Among his numerous appointments in the renal field, Dr. Kalantar-Zadeh is Associate Editor of several peer-reviewed journals including Nephrology Dialysis Transplantation, American Journal of Kidney Diseases, Cardiorenal Medicine, Seminars in Dialysis, Sarcopenia and Muscle, and a member of the editorial board of Journal of Kidney International, Journal of American Society Nephrology, Nature Reviews Nephrology, American Journal of Nephrology. Dr. Kalantar-Zadeh has authored 3 textbooks and over 500 peer-reviewed publications.

Dr. Carmine Zoccali: Dr. Zoccali is a specialist in Renal Diseases (Pisa University) and Hypertension. Dr. Zoccali's appointments include: Director, Division of Nephrology, Hypertension and Renal Transplantation, Ospedali Riuniti, Reggio Cal, Italy; Chief, CNR-IBIM

Clinical Epidemiology and Pathophysiology of Renal Diseases and Hypertension; Professor, Postgraduate Schools of Nephrology, Palermo, Catania and Messina Universities. Dr. Zoccali's current editorial positions include: Editor in Chief, Nephrology Dialysis and Transplantation, Academic Editor, (Nephrology) PlosOne, and Editorial Board member, Journal of the American Society of Nephrology. Editorial Board member, Clinical Journal of the American Society of Nephrology and Editorial Board member, Kidney International. Dr. Zoccali has over 402 international peer-reviewed publications.

Dr. Marcello Tonelli: Dr. Tonelli is Associate Vice-President (Research) at the University of Calgary. Dr. Tonelli was the recipient of the 2013 United States National Kidney Foundation Medal for Distinguished Service and the Kidney Foundation of Canada's 2013 Medal for Research Excellence for changing nephrology practice in Canada and beyond. Along with the two other team co-leads, he received a Top Canadian Achievements in Health Research Award from the CIHR-CMAJ in 2013 for his work with the Interdisciplinary Chronic Disease Collaboration. He was elected a fellow of the Canadian Academy of Health Sciences in 2012 and a member of the American Society for Clinical Investigation in 2014. He was named a "Highly Cited" researcher in 2015 by Thomson-Reuters, representing a ranking in the top 1% by citations of all researchers worldwide for field and publication year.

Dr. Vincent Brandenburg: Dr. Brandenburg is Nephrologist, Associate Professor and Senior Consultant at the Department of Cardiology, Intensive Care Medicine and Vascular Medicine, University Hospital of the RWTH Aachen, Germany. Dr. Brandenburg has been leader of the German Calciphylaxis registry since 2007. He is a board member of the ERA-EDTA scientific working group Chronic Kidney Disease – Mineral and Bone Disorder ("CKD-MBD"). Dr. Brandenburg has authored or co-authored over 140 articles in peer-reviewed journals. The primary focus of these articles has been chronic kidney disease – mineral and bone disorder, cardiorenal syndrome, and calciphylaxis. Dr. Brandenburg is also a member of the German and European Societies of Nephrology and the Societies of Cardiology.

Dr. Srinivasan Beddhu: Dr. Beddhu, MD is a tenured Professor of Medicine at the University of Utah School of Medicine. He is Board Certified in Internal Medicine and Nephrology. Dr. Beddhu received his medical degree from Stanley Medical College, Chennai, India. His clinical and research interests include hypertension, CKD progression and complications and end-stage renal disease. Dr. Beddhu's research is funded primarily by NIH grants. He has served in several national committees including NIH panels, American Society of Nephrology Research Committee and National Kidney Foundation clinical practice guidelines committee. Dr. Beddhu has published approximately 100 articles including peer-reviewed publications, editorials and book chapters.

Dr. Mathias Haarhaus: Dr. Haarhaus is a Consultant Nephrologist at the Department of Nephrology, Karolinska University Hospital, Stockholm, Sweden, where he is Head of the Bone and Mineral Program. His research at the Division of Renal Medicine, Karolinska Institutet focuses primarily on the link between skeletal disorders and cardiovascular complications in CKD, with a special focus on ALP. He is an active member of the CKD-MBD working group of the ERA-EDTA and a member of the Guidelines Committee of the Swedish Society of Nephrology.

Neurodegenerative Clinical and Scientific Advisory Board

In July 2012, we established 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.

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

Dr. Henrik Zetterberg, MD, PhD is professor of neurochemistry at the University of Gothenburg and 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 laboratory. Her primary 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.

Financing

Private Placements

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 at the time, subscribed for 1,230,769 common shares. Directors and officers of the Company subscribed for a total of 1,080,522 common shares.

In July 2015, we and Shenzhen Hepalink Pharmaceutical Co., Ltd. ("Hepalink") entered into a definitive stock purchase agreement and closed an exclusive license of apabetalone within China, Hong Kong, Taiwan and Macau (the "Territories"), for all indications. On July 20, 2015, we closed the private placement. Under the terms of the transaction, Hepalink subscribed for 13,270,000 Resverlogix common shares and 1,000,000 common share purchase warrants for gross proceeds of CAD$35.4 million (US$27.3 million), or CAD$2.67 per unit. In addition, Eastern Capital Limited (“Eastern”) purchased 5,600,000 common shares and 422,005 common share purchase warrants for gross proceeds of CAD$15.0 million (US$11.5 million), or CAD$2.67 per unit. The 1,422,005 warrants issued on July 20, 2015 have an exercise price of CAD$2.67 and expire on July 20, 2020.

As a condition of the investment by Hepalink, we entered into a Nomination Rights Agreement dated July 20, 2015 with Hepalink whereby Hepalink is entitled to nominate one representative to the board of directors of the Company for successive terms during the period ending as of the commencement of the Company’s 2018 annual meeting, provided that the license agreement between the Company and Hepalink has not been terminated for any reason. In addition, as a further condition of the investment by Hepalink, we entered into a Share Restriction Agreement dated July 20, 2015 whereby Hepalink agreed not to sell the securities it purchased pursuant to the private placement for a period of three years and it agreed to certain additional contractual restrictions on resale thereafter.

In April 2017, we closed a private placement of 150,000 common shares at a price of CAD$2.00 per common share for gross proceeds of CAD$0.3 million (US$0.2 million).

In June 2017, we issued a total of CAD$10 million in equity units. Eastern and Hepalink purchased 1,617,980 and 1,333,333 equity units, respectively at a price of CAD$1.80 per unit for aggregate proceeds of CAD$5.3 million pursuant to a private placement. In addition, pursuant to a prospectus supplement dated June 13, 2017 to the Company’s base shelf prospectus dated October 1, 2015, subscribers purchased an additional 2,552,489 equity units at a price of CAD$1.80 per unit for aggregate proceeds of an additional CAD$4.6 million. Each equity unit consists of one common share and one common share purchase warrant. Each warrant is exercisable at a price of CAD$2.05 per underlying common share for a period of four years from the closing of the offering.

Citibank Loan

In August 2012, we entered into a CAD$25 million Loan Agreement with Citibank, N.A. ("Citibank"). 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 is repayable upon maturity on August 28, 2017 and may be repaid prior to maturity in whole or in part without penalty. Our cash as at April 30, 2017 will be insufficient to repay our loan. We continue to engage in discussions regarding extending or replacing the loan; however, there is no assurance that the loan will be extended or replaced. Interest on the loan is 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, 2016, the annual interest rate was reset from 3.7643% to 4.0560%. 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 research and development activities related to the epigenetics platform technology to

Zenith, as described under "Licensing" herein, the exercise prices of the August 2012 and March 2013 warrants were adjusted to CAD$1.44 and CAD$2.16, respectively.

Base Shelf Prospectus

On October 5, 2015, we obtained a receipt for a final short-form base shelf prospectus filed on October 1, 2015 with the securities commissions in each of the provinces of Canada except Quebec. Subject to securities regulations, the short form base shelf prospectus allows us to make offerings of common shares, preferred shares, debt securities, warrants, units, or any combination of such securities up to an aggregate offering price of CAD$125 million during the 25 month period that the base shelf prospectus remains effective.

Licensing

Licensing Agreement

As stated above, in July 2015, we closed an exclusive license of apabetalone for all indications in the Territories with Hepalink. The license between us and Hepalink provides for certain milestone payments based on net sales of apabetalone in the Territories. The annual sales milestones range from 500 million renminbi ("RMB") to 10 billion RMB (US$73 million to US$1.5 billion), 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 apabetalone in the Territories. Hepalink is responsible for all clinical and development costs in the Territories, including a patient population that is expected to be included in the our Phase 3 BETonMACE clinical trial. We are contractually obligated to pay a fee to the financial advisor involved with the transaction equal to 3.5% on the first $10.0 million of payments, if any, received from Hepalink pursuant to the license, and 2.5% on amounts above $10.0 million, up to a maximum of $1.0 million of fees. As at April 30, 2017, these potential payments do not satisfy the criteria for recognition as a liability.

Services and Licensing Agreements with Zenith Capital Corp.

In 2013, we reorganized into two companies and spun off research and development activities related to the epigenetics platform technology with the potential to impact multiple diseases, 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 our CVD and DM clinical programs and our neurodegenerative diseases program. RVX Therapeutics Inc. ("RVX Therapeutics"), which was a wholly-owned subsidiary of Resverlogix prior to the reorganization, held all of the assets spun off and was acquired by Zenith as part of the reorganization.

Services Agreements

Pursuant to an Assignment and Services Agreement dated June 3, 2013 and effective May 1, 2012 between us and RVX Therapeutics, which was subsequently assumed by Zenith, we perform research and related administrative and support services requested by Zenith from time to time. The agreement was for an initial term of three (3) years and automatically renews 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.

Pursuant to a Management Services Agreement dated June 3, 2013 between us and Zenith, we provide management and administrative services pertaining to Zenith as required. We receive a management fee from Zenith 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.

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

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.

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. 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 a development stage company, 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 and negative cash flow. We expect to continue to incur substantial net losses for the foreseeable future, and we may never achieve or maintain positive cash flow.

To date, we have not recorded any revenues from the sale of biopharmaceutical products, and have incurred significant negative cash flows in many periods since our inception. As at April 30, 2017, we had a deficit of US$314.0 million. We expect to incur substantial net losses and negative cash flow for the foreseeable future. Such losses and negative cash flow 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 positive cash flow. 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 negative cash flow 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 report net losses and negative cash flow 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, net losses and cash flow are also expected. Even if we do achieve profitability, we may not be able to sustain positive cash flow on an ongoing basis.

We will need to raise additional capital in the future to fund our operations and repay our debt. 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 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.

Our cash as at April 30, 2017 will be insufficient to fund our contractual commitments for the next year and our planned business operations over the next year based on anticipated patient enrollment for BETonMACE. We will have to raise additional capital. If we are not able to raise capital or extend or replace our debt, we would also have to reduce our cash requirements by eliminating or deferring spending on research, development and corporate activities. These conditions result in a material uncertainty which casts significant doubt on our ability to continue as a going concern.

We will also require additional capital to fund research, development and corporate activities beyond the next year. We will continue to explore alternatives to generate additional cash including raising additional equity and product licensing; however, there is no assurance that these initiatives will be successful. We intend to raise capital from equity and/or debt offering and/or partnering in the future.

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.

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 in 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, and/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. We agreed to indemnify Eastern 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.

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 apabetalone, 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 apabetalone. Those patients taking rosuvastatin and apabetalone had a highly statistically significant PAV plaque regression of -1.43% with probability value of $p<0.002$ vs. baseline. This PAV regression exceeded the trial's pre-specified PAV endpoint (-0.6%) by more than two-fold. However, those patients taking atorvastatin (Lipitor®) together with apabetalone had a PAV plaque progression of +0.19% with a non-significant probability value vs. 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 apabetalone, 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 product candidates 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 product candidates in clinical studies, including apabetalone, 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 product candidates 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, apabetalone, 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 apabetalone 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 apabetalone. These clinical trials would be expensive and could delay any commercialization of apabetalone. Adverse results in these trials could delay or prevent commercialization of apabetalone 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 apabetalone. 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 apabetalone or other product candidates. 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 apabetalone 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 may 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 candidate, 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 product candidates. 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 to 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 product candidates. These agreements generally require that the third party pays to us 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, we 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 us 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 our 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 product candidates, if approved for marketing, will achieve market acceptance. If our product candidates, 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 product candidates;
- our product candidates' 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 European countries, the United States, Canada, China, or any other jurisdictions. 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 countries' numerous statutes and regulations. We are required to obtain various regulatory approvals prior to being able to study, commercialize and distribute our product candidates. The regulatory review and approval process required to perform a clinical study in any country 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 apabetalone, or to manufacture or market our products in reasonable time frames, if at all.

Governmental authorities in any country 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 apabetalone 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 apabetalone or any other product candidate 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 complications

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 apabetalone is approved for reduction of CVD risk and MACE and it improves other biomarkers such as eGFR, Albumin and ALP, apabetalone would potentially compete with, or be added to, novel and existing CKD products in clinical development.

We anticipate that, if approved for neurodegenerative disorders, apabetalone 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, apabetalone 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 polices 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 MACE, 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.

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, 2017, the closing market price of our common shares ranged from CAD$1.10 to CAD$2.47 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, 2017, we had 105,642,444 Common Shares, 75,202,620 Royalty Preferred Shares, and no preferred shares are issued and 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 our remaining assets, subject to the rights of any class of preferred shares.

Preferred Shares

We may issue preferred shares 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 our Board. Preferred shares shall rank senior to 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 the Company in the event of a dissolution, liquidation or winding up of the Company.

Royalty Preferred Shares

The Royalty Preferred Shares were issued to Zenith on June 3, 2013 as part of the spin-off transaction that resulted in the Company's epigenetic platform technology (excluding apabetalone) being transferred to Zenith and shareholders of the Company at the time of that transaction receiving common shares of Zenith.

The terms of the Royalty Preferred Shares were amended on July 2, 2015 to limit the dividends payable to holders of Royalty Preferred Shares in a particular period to amounts received by the Company during that period and to include certain additional deductions in the calculation of net revenue subject to the royalty. These amendments were necessary to align the terms of the Royalty Preferred Shares with the terms of the royalties to which the Company would be entitled pursuant to a license agreement entered into with Shenzhen Hepalink Pharmaceutical Co., Ltd. on July 8, 2015.

The terms of the Royalty Preferred Shares were further amended on December 20, 2016 to provide that the holder of the Royalty Preferred Shares is entitled to a dividend calculated based on a percentage of net revenue earned from the sale or licensing of any pharmaceutical product in which the Company holds an intellectual property right and remove the requirement that the pharmaceutical product elevate plasma levels of a certain lipoprotein associated with a decreased risk of atherosclerosis and coronary heart disease. The Company determined that this amendment was necessary and appropriate based on detailed analysis of the results of the Company's phase 2 clinical program.

The Royalty Preferred Shares, after giving effect to the foregoing amendments, entitle Zenith to cumulative preferential dividends in an amount ranging from 6% to 12% of Net Revenue during any year, subject to an adjustment for tax payable on the dividend. The dividend amount is calculated based on 6% of the aggregate Net Revenue of up to US$1 billion, 8% of the aggregate Net Revenue of between US$1 billion and US$2 billion, 10% of the aggregate Net Revenue between US$2 billion and US$5 billion and 12% of the aggregate Net 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 us or our affiliates in respect of and including Net Revenue in such period.

Net Revenue is defined as the aggregate of the following amounts: (i) amounts received by us or our affiliates from any person who is not us or our 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 products and/or 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 us, any licensee or their respective affiliates from the sale of any product (other than consideration received by us, any licensee or their respective affiliates from a licensee of such 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 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 us, any licensee or their respective affiliates); and (iii) amounts received from a third party by us or an affiliate in consideration for the sale of any 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 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 our assets among 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 our assets 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, 2017

Date	High ($)	Low ($)	Volume
4/28/2017	2.38	1.81	547,700
3/31/2017	2.41	1.95	909,300
2/28/2017	2.08	1.81	672,500
1/31/2017	2.39	1.65	1,154,700
12/31/2016	1.89	1.60	393,000
11/30/2016	2.07	1.61	571,000
10/31/2016	2.47	1.87	769,900
9/30/2016	2.16	1.19	1,485,000
8/31/2016	1.33	1.12	512,700
7/29/2016	1.28	1.13	337,600
6/30/2016	1.35	1.10	354,000
5/31/2016	1.47	1.25	296,000

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, 2017:

Date	Type of Security	Issue Price or Exercise Price of Securities	Number of Securities	Type of Issuance
December 2016	Stock Options	$1.73	155,000	Pursuant to stock option plan
July 2016	Stock Options	$1.19	10,000	Pursuant to stock option plan
May 2016	Stock Options	$1.32	528,800	Pursuant to stock option plan
May 2016	Stock Options	$1.39	150,000	Pursuant to stock option plan
May 2016	RSUs	N/A	405,000	Pursuant to long term incentive plan

Escrowed Securities and Securities Subject to Contractual Restrictions on Transfer

The following table indicates our securities 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)	11.93%
Common Share Purchase Warrants	1,000,000(1)	Not applicable

Notes:

(1) On July 20, 2015, we 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 committed 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 six 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
Donald J. McCaffrey Calgary, Alberta, Canada	Chairman of the Board, President, CEO and Secretary	President, Chief Executive Officer and Secretary of Resverlogix since 2003. Chairman of Resverlogix since April 2016. President, Chief Executive Officer and Secretary of Zenith Capital Corp. since 2013.	2003
Dr. Eldon Smith (2), (3) Calgary, Alberta, Canada	Lead Director and Chair of the Corporate Governance and Nominating Committee	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.	2010
Kenneth Zuerblis, CPA(1) Sarasota, Florida, U.S.A.	Director and Chair of the Audit and Finance Committee	Currently a director of the Corporation, Zenith Capital Corp. and Stemline Therapeutics.	2010
Kelly McNeill, CPA, CA (1), (2),(3) Winnipeg, Manitoba, Canada	Director and Chair of the Compensation and HR Committee	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.	2009
Norma Biln (2), (3) Vancouver, British Columbia, Canada	Director	Chief Executive Officer of and Co-Founder of Augurex Life Sciences Corp. since 2006.	2016
Shawn Lu (1) Toronto, Ontario Canada	Director	Chief Financial Officer of Hepalink USA Inc. (a subsidiary of Hepalink) since 2014. From 2013 to 2014, Mr. Lu served as Area Manager for BMO, Bank of Montreal, and from 2005 to 2013 Mr. Lu served as Residential Mortgage Manager for TD Bank.	2016
A. Brad Cann, CPA, CA, CBV Calgary, Alberta, Canada	Chief Financial Officer	Chief Financial Officer of Resverlogix since 2009. Chief Financial Officer of Zenith Capital Corp. since 2013.	N/A
Dr. Michael Sweeney, MD Menlo Park, California, U.S.A.	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

Name and Municipality of Residence	Position	Principal Occupation During Past 5 Years	Director Since
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
Dr. Ewelina Kulikowski, PhD Calgary, Alberta, Canada	Senior Vice President Scientific Development	Senior Vice President Scientific Development of Resverlogix since 2016. From 2005 to 2016, Dr. Kulikowski held various positions at Resverlogix of increasing responsibility.	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 8,301,025, or 7.5%, of issued and outstanding Common Shares as of July 20, 2017.

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 comprised of three independent, unrelated directors – Mr. Zuerblis as Chair, Mr. McNeill and Mr. Lu. 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 of Stemline Therapeutics, Inc. (since 2012) and Zenith Capital Corp. (since 2013).

Kelly McNeill

Mr. McNeill holds a Masters of Accountancy and a Bachelor of Commerce (Honours), and is a Chartered Professional 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 Capital Corp. (since 2013).

Shawn Lu

Mr. Lu has extensive experience in the areas of corporate finance, capital markets and investment financing spanning over 24 years. Prior positions include: Area Manager for BMO Bank of Montreal; TD Bank Residential Mortgage Manager; TD Bank Senior Financial Advisor; Chief Financial Officer and Vice President of Corporate Finance, Hepalink; Vice President of Investment and Corporate Finance, Shenzhen FuTianXin Investment Co.; General Manager of Corporate Finance Department and Manager of Investment & Finance Department, China Merchant Shekou Port Co. Ltd. Mr. Lu holds the following designations: Canadian Investment Manager (CIM) and a Certified Accountant and Certified Corporate Economist in China. He has Master of Finance Management and a Master of Corporate Economics and Business Administration. Mr. Lu currently also serves on the boards of directors (since 2015) of Quest PharmaTech Inc.

Pre-approval of Audit Fees

We 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 us:

Year	Audit Fees (1)	Audit-Related Fees (1)	Tax Fees (2)	Other Fees
2017	$138,000	$30,000	$5,100	$Nil
2016	$133,000	$10,000	$4,800	$Nil

Notes:

(1) Audit fees were for professional services for the audit of our annual financial statements and reviews of our unaudited interim financial statements. Audit-related fees were for our short form base shelf prospectus, 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 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 that 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".

On June 10, 2014, NGN, a limited partnership that was under the control or direction of Dr. Peter Johann, our chairman at that time, 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. Dr. Johann resigned from the Company's Board of Directors on April 2, 2016.

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 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 apabetalone for the Territories for all indications. The license provides for certain milestone payments based on net sales of apabetalone in the Territories and for Hepalink to pay a royalty of 6% of annual net sales of apabetalone in the Territories. See "General Development of the Business – Licensing".

In June 2017, we completed a private placement of 1,333,333 equity units to Hepalink and 1,617,980 equity units to Eastern for aggregate proceeds of CAD$5.3 million, or CAD$1.80 per unit. Each equity unit consists of one common share and one common share purchase warrant. Each warrant is exercisable at a price of CAD$2.05 for a period of four years.

Transfer Agent and Registrar

The transfer agent and registrar for our Common Shares is Computershare 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. Nomination Rights Agreement dated July 20, 2015, between us and Hepalink, as more particularly described under "General Development of the Business – Financing – Private Placements".
7. 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, 2017.

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. 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.

21. 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.

22. 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

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.
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
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 *(also known as atheroma).*
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 *(also see drug).*
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 ("DM")	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.
European Medicines Agency ("EMA")	is the European governmental agency responsible for the approval for manufacture, usage and sale of food, human diagnostics and therapeutic products within the European Union.
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.
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 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 *(also known as "bad cholesterol").*
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 Revenue	the aggregate of the following amounts: (i) amounts received by us or our affiliates from any person who is not us or our 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 products and/or 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 us, any licensee or their respective affiliates from the sale of any product (other than consideration received by us, any licensee or their respective affiliates from a licensee of such 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 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, us, any licensee or their respective affiliates); and (iii) amounts received from a third party by us or its affiliates in consideration for the sale of any 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")	*(see "New Drug Application")*
Pharmaceutical Agent	a compound or composition covered by a Valid Claim.
Pharmacological Agent	*(see "Drug").*

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 event.
Polymerase Chain Reaction ("PCR")	the technique uses thermocycling to amplify a region of DNA. Resverlogix uses real-time PCR as a method to assess gene expression.
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.
Triglycerides	a type of fat found in the blood and other parts of the body.
Type II Diabetes	*(see "Diabetes Mellitus")*

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 Capital Corp. (formerly Zenith Epigenetics Corp.), a corporation incorporated under the ABCA, and its subsidiaries.

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