



Annual Information Form

Fiscal Year Ended April 30, 2019

July 26, 2019

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Advisories

Conventions

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).

The information contained in this AIF is presented as at April 30, 2019, except where otherwise noted.

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 Company has not conducted its own independent verification of such information. While the Company 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 Company 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 reader’s convenience only and do not form a part of this Annual Information Form.

Forward-Looking Information

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

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

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

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

- aim to commercialize or license to a pharmaceutical partner our products for the treatment of unmet medical needs related to 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;
- projected competitive conditions with respect to our products;
- anticipated sources of revenue and the estimated market for our products;
- 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 agreements with Shenzhen Hepalink Pharmaceutical Co., Ltd. and Medison Pharma 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;
- risks relating to compliance with covenants to secured lenders and our ability to repay or refinance secured indebtedness when it comes due;
- 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, 2019, we employed 27 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 (“RVX-208”), targets BET proteins to impact several important biological processes that are contributors to the pathophysiology of chronic vascular diseases such as cardiovascular disease (“CVD”), diabetes mellitus (“DM”), and chronic kidney disease (“CKD”), namely: (i) vascular inflammation, (ii) acute phase response, (iii) vascular calcification, (iv) complement and coagulation, and (v) reverse cholesterol transport (“RCT”). Apabetalone is a first-in-class small molecule in development for the secondary prevention of major adverse cardiovascular events (“MACE”) in high risk CVD patients with a DM co-morbidity. Based on the above-mentioned effects of apabetalone, we are currently exploring the potential for apabetalone to modulate disease-related pathology in other indications including CKD, end-stage renal disease (late stage CKD), neurodegenerative disease and orphan diseases such as Fabry disease.

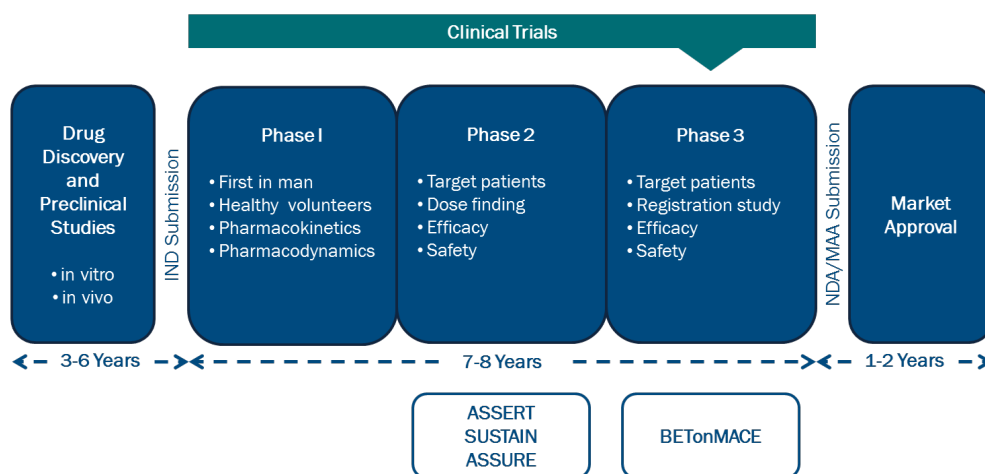
The Drug Development Process

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, manufacturing, 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 Food and Drug Administration (“FDA”), respectively.

The FDA has outlined the following guidance for exploratory Investigational New Drug (“IND”) studies. During preclinical development, compounds are created and screened with the objective of identifying their potential for further 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 models (commonly performed in rodents) to evaluate efficacy and safety (toxicity). Results from animal studies provide information on the selection of a safe starting dose for humans, potential toxicity, as well as pharmacokinetic and pharmacodynamic properties of the drug. An IND containing all the data collected in the preclinical studies as well as chemical, manufacturing and control 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 properties 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 thousands required in Phase 3) suffering from the targeted condition or disease. These trials commonly include randomization of patients as well as a placebo or comparator arm. The primary aim is to determine the drug’s efficacy, optimal doses, treatment regimens, and pharmacokinetics, pharmacodynamics and dose response relationships. Phase 2 trials also provide additional safety data and serve to identify possible common short-term side effects and risks in a larger group of patients and controls. 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. Phase 4 clinical trials are conducted after approval to identify and evaluate the long-term effects of new drugs and treatments for a greater number of patients and a more diverse patient population. Phase 4 research only takes place after the Regulatory Authorities approve the marketing of a new drug and addresses the generation of new data in the approved indication. Through Phase 4 clinical studies, new drugs can be tested continuously to uncover more information about efficacy, safety and side effects after being approved for marketing.



Drug Development Process

Resverlogix Current Development Stage: Phase 3 Nearing Completion

In the fall of 2015, Resverlogix initiated a Phase 3 clinical trial "BETonMACE" with apabetalone in high-risk CVD patients with type 2 diabetes mellitus ("DM") and low levels of high-density lipoprotein ("HDL"). Approximately 11% of the patient population will also have CKD. The primary endpoint is the time to first occurrence of MACE. 2,425 patients were enrolled (surpassing the approximately 2,400 outlined in the study's protocol). The BETonMACE trial is nearing completion. On July 8, 2019, we announced that all patients had completed their final follow-up safety visits; remaining third party adjudications of MACE events continue. Topline results of the study are currently expected in the fall of 2019. 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".



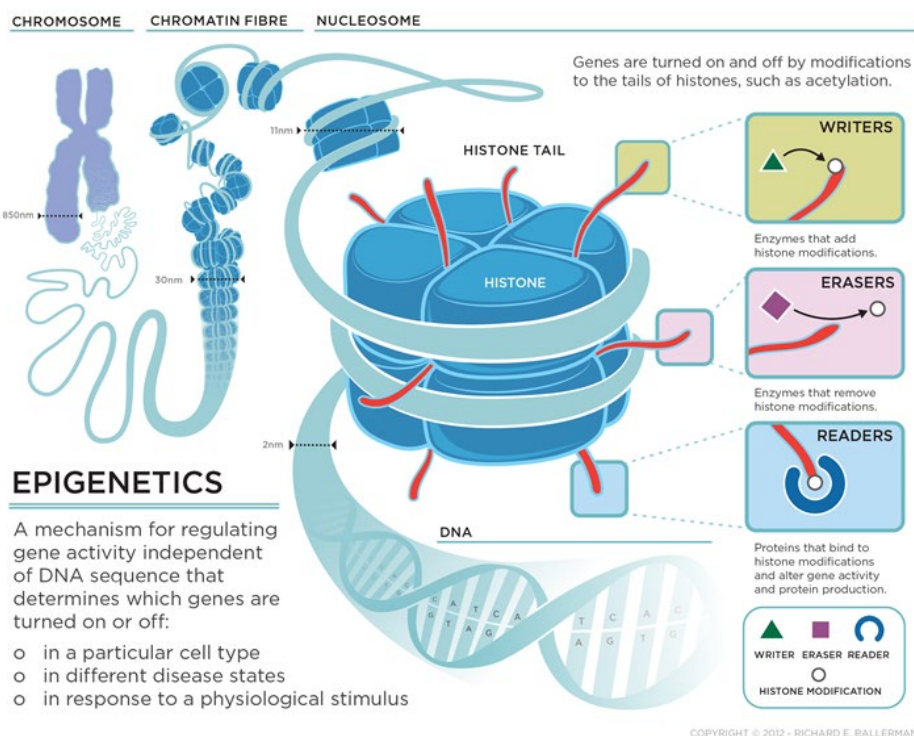
Apabetalone (RVX-208)

Apabetalone is the first BET inhibitor in clinical trials for high risk vascular disease. A hallmark of many diseases such as cancer, inflammation and more recently cardiovascular disease, is aberrant gene transcription. Bromodomains ("BRDs") are a family of evolutionary conserved protein-interaction modules that play key functions in chromatin organisation and regulation of gene transcription. One recognised family of bromodomain-containing proteins is the BET family. Apabetalone's "Epigenetic Mechanism of Action" (below) illustrates that it functions as an inhibitor of the BET proteins. Apabetalone is the first oral agent in the BET inhibitor class that preferentially targets bromodomain 2 ("BD2") of BET proteins. In binding to this bromodomain, apabetalone affects the expression of multiple genes with roles in a variety of cellular processes.

Analysis of our phase 2 (ASSERT, SUSTAIN and ASSURE) clinical trials has demonstrated that, based on the modulation of risk factors, apabetalone significantly reduces MACE in patients with CVD, particularly those who have a low level of HDL and elevated c-reactive protein ("CRP"), a well-known marker of inflammation, and in those with diabetes mellitus co-morbidity (Nicholls, Ray et al. 2018). Insight into the clinical benefits of apabetalone has been provided by experimental investigations detailing the multiple physiological activities affected by apabetalone.

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

The human body is made up of nearly two hundred different cell types that have cell-specific functions resulting 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 transcription into 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



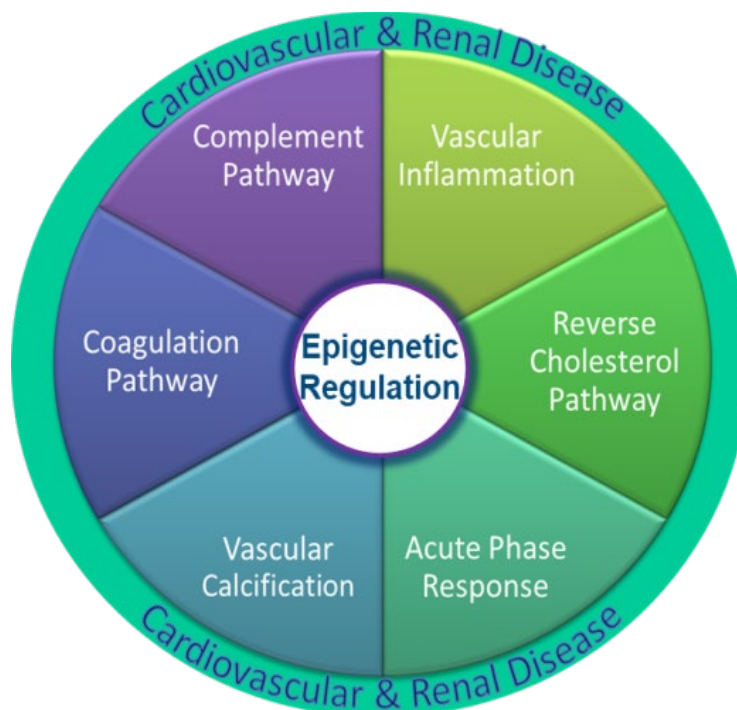
Epigenetic Mechanism of Action

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. BET proteins are “readers”, proteins that recognize a specific pattern of modifications and bind to the chromatin at these sites. The BET proteins then serve as a scaffold, recruiting the necessary transcriptional machinery to the chromatin to drive gene expression and ultimately protein production.

Our compounds target one group of “reader” proteins called the Bromodomain and Extra Terminal (“BET”) proteins. BET inhibition represents an important new area of drug development, since epigenetic modification is a known hallmark of several complex pathologies, including cardiovascular disease (“CVD”), metabolic disorders, and neurological diseases. Substantial evidence has shown that alterations in the pattern of chromatin modifications underlie these multiple disease states. Epigenetic regulators are promising targets for therapeutic intervention, and hold significant potential for treatment advances in important diseases of high unmet medical need.

BET inhibition results in the simultaneous modulation of multiple biological pathways via a single molecular target. Studies highlighting the molecular and mechanistic functions of BET inhibitor molecules and the ongoing development of new BET inhibitors as potential therapeutics in multiple indications are initiating a shift from the current drug development paradigm. From 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, epigenetic modulation is a novel approach to targeting disease pathology.

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 which are improved by select BET inhibition, provide biological plausibility and rationale to support the potential of apabetalone to reduce residual risk in CVD patients. Below are several key studies that highlight the potential roles of these pathways and biomarkers that have been studied to date, and 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 and acquire therapies that modulate the multiple risk pathways associated with the pathogenesis of CVD and MACE. These pathways include: (i) vascular inflammation, (ii) acute phase response, (iii) vascular calcification, (iv) complement and coagulation, and (v) reverse cholesterol transport (“RCT”). 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

Vascular inflammation (“VI”) is a powerful driver of atherosclerosis, coronary artery disease, hypertension, hypercholesterolemia, and diabetes mellitus (Mestas and Ley 2008). A hallmark of the VI process involves greater adhesion of white blood cells (leukocytes, including monocytes, lymphocytes and granulocytes) to the vessel wall, or vascular endothelium (Mestas and Ley 2008). The vascular endothelial cells themselves contribute to VI by promoting adhesion, where endothelial cell surface receptors (such as E-selectin, ICAM1 and VCAM1) bind to monocyte adhesion receptors (L-selectin and carbohydrate moieties) on circulating monocytes to form atherosclerotic plaques. VI occurs as a result of sustained high levels of harmful circulating factors (acute phase reactants

such as CRP, cytokines, glucose, and Trimethylamine-N-oxide (“TMAO”)) which lead to activation of endothelial cells, upregulation of circulating cell adhesion molecules, and recruitment of circulating leukocytes. In CVD and related diseases including DM, the combination of these factors continually promote VI and contribute to atherosclerotic plaque development, growth and rupture.

Acute Phase Response

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). Multiple epidemiological, prospective and intervention studies (MRFIT, PHS, CHS/RHPP, WHS), have shown that minor CRP elevations are associated with future major CVD risk (Ridker 2001), and that CRP strongly and independently predicts adverse CVD events, including myocardial infarction, ischemic stroke, and sudden cardiac death in individuals both with and without overt CHD (Shrivastava, Singh et al. 2015). CRP is thus recognized as a major cardiovascular risk factor, and provides prognostic information beyond LDL (Ridker, Cannon et al. 2005, Ridker, MacFadyen et al. 2010). 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). Furthermore, elevated CRP is associated with increased risk of DM development (Ridker 2001). Additional acute phase response components, such as MBL2, C2, C3 and C5, and SAP (Kulikowski, Gilham et al. 2016) are pro-inflammatory, pro-atherogenic, and markers of CVD risk (Lowenstein and Matsushita 2004).

Vascular Calcification

Medial artery calcification is a characteristic feature of diabetes, CKD, and identifies 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 finding 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 a 21% increased risk of coronary heart disease death, nonfatal myocardial infarction, symptomatic heart failure or stroke and a 43% increased risk of all-cause mortality. 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 and that higher ALP levels represent 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 that 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).

Complement and Coagulation

The complement and coagulation cascades have been shown to play a significant 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 coronary artery disease (“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. These studies suggest 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 acute myocardial infarction patients and age and sex matched controls demonstrated that the ratio of apolipoprotein B ("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 individuals (referred for clinical laboratory testing as part of a health checkup) 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 continue 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 all contain highly-conserved bromodomains that play a key role in epigenetic control of gene expression (in many cell types);
- 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 (BRD2, BRD3 and BRD4), with a 20-fold or higher selectivity for the second bromodomain 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; and
- Apabetalone is the only selective BET inhibitor in the field of CVD 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 pain from peripheral arterial disease (ischemia), percutaneous stent procedures, and hospitalization for cardiac-related incidents. According to the 2018 American Heart Association Statistics report, based on 2015 death rate data, CVD claims more lives each year than cancer and chronic lower respiratory disease combined. (Benjamin, Virani et al. 2018). 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, for example 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 2018 American Heart Association Statistics report, more than 85.6 million Americans have one or more vascular disease (Benjamin, Virani et al. 2018). By the year 2035, the total projected economic burden and direct costs of CVD in the United States is estimated at \$748.7 billion annually. 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). The lead products in the CVD pipeline at the time of publication of the report 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 the present time, the proprotein convertase subtilisin/kexin type 9 (“PCSK9”) inhibitor programs; Alirocumab (Sanofi/Regeneron) and Evolocumab (Amgen) and the sodium/glucose cotransporter 2 (“SGLT2”) inhibitor programs; Canagliflozin (Janssen), Dapagliflozin (AstraZeneca), and Empagliflozin (Boehringer Ingelheim and Eli Lilly) 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).

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 could partially 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 including statins.

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 excreted 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 where 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 to further evaluate CKD and ESRD biomarkers to determine the potential use of apabetalone in this important disease area. The Company plans to assess these biomarkers and associated pathways 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 BETonMACE clinical study in this therapeutic area.

Diabetes Mellitus

Diabetes mellitus is the most common metabolic disease in the world. A primary defect in DM is the inability of the body to provide or efficiently utilize insulin, thus leading to increased blood glucose and numerous related health problems. CVD risk factors are also associated with insulin resistance. In fact, patients with DM have two to four times greater risk of death or serious cardiovascular morbidity compared to individuals without DM and approximately 75% of deaths in patients with DM are due to CVD (Wu and Parhofer 2014). High residual risk of MACE in DM was evident in the recent EXAMINE study of patients with DM receiving statin therapy. In a cohort of 5,380 DM 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. The incidence of MACE 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 trial add to many other preceding studies (UKPDS, ACCORD, ADVANCE AND VADT) to further underscore that, despite intensive glucose control and standard of care, the incidence of MACE remains significant in the setting of DM. Revascularization in patients with DM leads to additional 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). Similar residual risk post ACS for patients with diabetes was demonstrated in the AleCardio Study (Lincoff, Tardif et al. 2014) (approximately 10% of individuals' experienced cardiovascular death, nonfatal myocardial infarction, or nonfatal stroke during the two year study period) and the ELIXA study (Pfeffer, Claggett et al. 2015) (approximately 13% of individuals' experienced cardiovascular death, nonfatal myocardial infarction, or nonfatal stroke during the 25 month study period).

The role of inflammation in DM has also been highlighted in recent years. It is now well-known that inflammation is one of the main reasons why people with diabetes experience heart attacks, strokes, kidney problems and other related complications. A variety of inflammatory markers have been linked to DM. Elevated circulating inflammatory markers such as CRP and interleukin-6 (IL-6) consistently predict the development of type 2 diabetes (Pickup 2004), and there is increasing evidence that an ongoing cytokine-induced acute-phase response, sometimes called low-grade inflammation, is closely involved in the pathogenesis of DM and associated complications such as dyslipidemia and atherosclerosis. There is clearly an urgent need for new approaches to reduce MACE in patients with DM. Known effects of apabetalone on inflammation, and CRP, IL-6, and other acute phase reactants specifically, hold significant promise for reducing the risk of MACE in DM patients. We are evaluating the potential use of apabetalone in a subgroup of DM patients in the Phase 3 BETonMACE clinical study.

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 in 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 disease progression, and ultimately become the major causes of death. Cardiac complications arising from Gb3 buildup in the heart are the leading cause of death in Fabry patients, followed by stroke and kidney complications. Although, kidney complications generally manifest early 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 to compensate for the deficiency. Adjunctive medications include acetylsalicylic acid, angiotensin converting enzyme ("ACE") inhibitors or angiotensin II receptor blockers ("ARB"), 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 to 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, the United States, and Canada. 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 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 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 in neurodegeneration. Moreover, ongoing preclinical studies are exploring the role of apabetalone mediated modulation of neuroinflammation and innate immunity in this important disease area. As the incidence of dementia and neurodegenerative diseases increases significantly with age, the Phase 3 BETonMACE clinical study includes 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 they require an IV infusion 1-2 times a month.

Praluent® ("alirocumab") (Sanofi-Regeneron) was the first PCSK9 inhibitor approved by the FDA (July 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. Alirocumab was investigated for secondary prevention of MACE in ACS patients (ODYSSEY OUTCOMES Trial) in a large cohort of over 18,000 patients. Relative to placebo, alirocumab treatment significantly reduced the risk of the primary end point (composite of cardiovascular death, myocardial infarction, stroke, hospitalization for unstable angina) with a hazard ratio of 0.85. Repatha® ("evolocumab") (Amgen) was the second PCSK9 inhibitor to be approved by the US FDA (August 2015) for the same second line indication as Praluent. Evolocumab 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 both therapeutics illustrated statistically significant outcome reductions from a scientific perspective, reimbursement of drug classes with potentially high cost thresholds are under ever-increasing scrutiny from a key stakeholder group, the payers. The reimbursement community has offered comments that this approach for preventing CVD events would potentially prove to be too costly. Many key US payer groups have reviewed and reported their perceptions of the data. These drugs were launched into the market with a reported annual treatment cost per patient of US\$7,000-12,000 (Staton 2015). Independent Payer groups such as

the Institute for Clinical and Economic Review have determined this price to be too high and have suggested, “based on the results of the ODYSSEY Outcomes trial, [a] price benchmark, for alirocumab in patients with a recent acute coronary event [of] \$2,300-\$3,400 per year,” and “given the evidence available from FOURIER, to reasonably align with clinical benefit the annual price of evolocumab would need to drop by 85-88%, to a range between \$1,725 and \$2,242” (Tice JA 2017, Tice JA 2018). This reported price threshold by a payer group represents a significant price reduction over the initial 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.

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.

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 epidemiological study to examine 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, and noted high HDL levels but also 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 also came to similar conclusions (Borggreve, Hillege et al. 2006). Anacetrapib, a CETP inhibitor being developed by Merck, was evaluated in the REVEAL study. The results of the REVEAL study illustrated a significant relative risk reduction of 11% between the anacetrapib treatment group and placebo. The primary end point was defined as the composite of coronary death, myocardial infarction, and coronary revascularization. The mechanism by which anacetrapib reduced the risk of major coronary events in this trial is currently being debated. This data is also being 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 the associated research platform over potential antibody competitors such as the PCSK9 inhibitors. The key issues that need to be addressed are 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 relative risk reduction of 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.

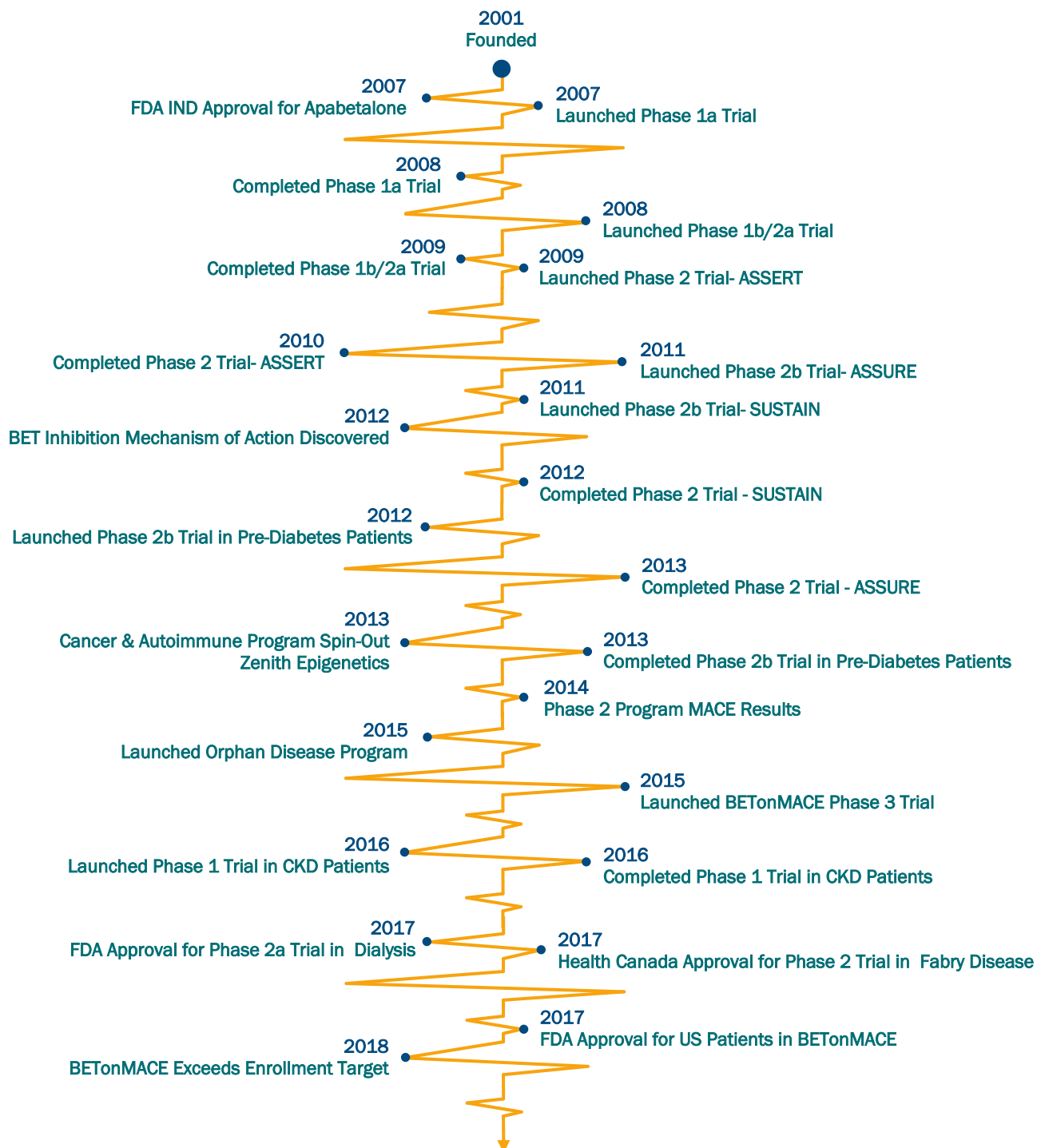
The current landscape of drugs in development for CVD risk management is presented in the table below.

Key Products in Development (Resverlogix and Competitors)

Product	Mechanism of Action	Market Status	Threat Assessment
Apabetalone/ RVX-208 (Resverlogix)	BET Inhibition	Phase 3	N/A
Praluent® (Alirocumab) (Sanofi/ Regeneron)	PCSK9 Inhibition	On Market (2015)	Moderate: IV Infusion High Cost
Repatha® (Evolocumab) (Amgen)	PCSK9 Inhibition	On Market (2015)	Moderate: IV Infusion High Cost
Invokana® / Sulisent® (Canagliflozin) (Johnson and Johnson)	SGLT2 Inhibition	On Market (2013)	Moderate: High Cost
Jardiance® (Empagliflozin) (Boehringer Ingelheim and Eli Lilly and Company)	SGLT2 Inhibition	On Market (2014)	Moderate: High Cost
Anacetrapib (Merck)	CETP Inhibition	Phase 3 (Trial Completed)	Low: Long Half Life
Bempedoic Acid (Esperion)	ATP Citrate Lyase inhibitor	Phase 3 (Trial Completed)	Moderate: Lack of CVD Outcomes Data
Canakinumab (Novartis)	IL-1 β inhibitor	Phase 3	Moderate: Lack of CV Outcomes Data (CANTOS MACE study currently underway)
Vascepa (Amarin)	To be determined – perhaps Triglyceride Reduction	Priority review (FDA) September 2019	Moderate: High triglyceride population; Stable CAD

General Development of the Business

History of Apabetalone Clinical Development



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 – Nearing Completion

The pooled MACE analysis from our phase 2 studies focused on the potential benefit of apabetalone and BET inhibition to reduce MACE over a short treatment time period of up to six months. The data from 798 patients demonstrated that treatment with apabetalone led to a significant reduction in MACE. Results from patients within these studies whom also had diabetes mellitus, a high-risk patient population, illustrated similar significance. Based on these findings, our intention with the BETonMACE trial is to reconfirm in a larger prospective setting, in patients with modifiable vascular disease (i.e. low HDL and diabetes), the 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 must have accrued at least 250 MACE events. MACE is adjudicated by an independent committee and the study is 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 ApoA-I), changes in DM variables (glucose and glycated hemoglobin), change in ALP, changes in kidney function as well as 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 were treated with standard of care statin therapy, subjects were randomized to either apabetalone 100 mg b.i.d. (twice daily) or matching placebo with continued statin treatment. This combination treatment period continues for the duration of the study. We have enrolled 2,425 patients (surpassing the 2,400 outlined in the study’s protocol) and have reached the endpoint of 250 MACE events; as of the date of this AIF, study closure procedures are underway. A full detailed protocol for the BETonMACE study can be viewed on clinicaltrials.gov with the following NCT ID, NCT02586155.



BETonMACE Approved in 14 Countries

On November 11, 2015, we announced that the first patient in the Phase 3 BETonMACE clinical trial was randomized and dosing had commenced.

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 submitted to the FDA regarding apabetalone, including: human exposure, clinical dosing and established acceptable safety margins, the FDA is allowing us to include U.S. patients in Phase 3 studies, including the BETonMACE trial. We agreed to make adjustments to the existing BETonMACE study protocol and to update the Investigator's Brochure and the Informed Consent Documents.

On March 19, 2018, we announced that recruitment in the BETonMACE study has successfully surpassed the planned enrollment target of 2,400 patients. A sample size re-estimation analysis ("SSRA") was planned when 75% of the planned 250 MACE events, being 188 events in total, had occurred. Subsequently, it was determined that a SSRA would no longer be performed given the expectation for BETonMACE trial completion and that conducting such an analysis would offer no practical value and would also cause a statistical penalty in regards to final powering of the trial.

On June 14, 2018, we announced that we received confirmation from the FDA that BETonMACE, if successful, is likely to support the filing and approval of a New Drug Application ("NDA"). The statistical analysis plan and proposed endpoints were reviewed and accepted by the Division of Cardiovascular and Renal Products ("DcaRP") of the FDA. The exact indication for approval will be reviewed by the FDA, and will be driven by the study's results. Notwithstanding the foregoing, no U.S. patients were required; therefore, no U.S. patients were enrolled.

The independent Data and Safety Monitoring Board ("DSMB") for the BETonMACE trial completed nine planned safety reviews of the trial between August 2016 and March 2019, and recommended that the study continue as planned without any modifications and noted that no safety concerns were identified. We, the clinical steering committee, and all investigators remain blinded to the study data.

On April 18, 2019, we announced that BETonMACE had successfully reached the targeted 250 MACE, strictly defined as cardiovascular death, non-fatal myocardial infarction and stroke, and a summary of the remaining steps in the trial.

On June 12, 2019, we announced that all study participants in BETonMACE had completed their last treatment visits, and follow-up safety visits were progressing.

On July 8, 2019, we announced that all patients active in BETonMACE had completed their final follow-up safety visits, and we outlined the remaining steps required to finalize BETonMACE. We announced anticipating being in a position to provide top-line results in September 2019.

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

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. The PK results from Part A will influence the dose selection for Part B. 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 2019.

Phase 2a Fabry Disease Trial – Planned

On May 30, 2017, we announced that Health Canada, Therapeutic Products Directorate ("TPD"), approved our 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 2019. 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.

Phase 1 Pharmacokinetic Trial in Patients with Severe Renal Impairment – Completed

On June 1, 2015, we presented the analysis of data pooled from the ASSERT, SUSTAIN and ASSURE clinical trials relating to specific biomarkers relevant to CKD. 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”, was presented at the European Renal Association-European Dialysis and Transplant Association (“ERA-EDTA”) Congress. It highlighted the statistically significant reductions in ALP observed in the apabetalone (RVX-208) treatment group compared to placebo and the early signal of improved estimated glomerular filtration rate (“eGFR”) in patients with eGFR <60 mL/min/1.7m² at baseline and following 6 months of treatment. Dr. Kamyar Kalantar-Zadeh, MD, MPH, PhD, Professor and Chief, Division of Nephrology and Hypertension at University of California in Irvine, examined these findings. These findings generated the hypothesis that apabetalone may be beneficial in the treatment of CKD and that additional clinical trials for target responder CKD and/or dialysis populations who have a high burden of cardiovascular disease and risk may be warranted.

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 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 have the same favourable PK traits 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 favourable PK traits and safety profile as has been observed in previous apabetalone trials. These results allowed us to proceed with 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.

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 inflammatory protein biomarkers in patients with late stage CKD versus healthy control patients. Protein data was collected following a single oral administration of 100mg of apabetalone before and after multiple time points in both cohorts. Protein levels of 288 proteins were significantly different at baseline between the two groups ($p < 0.05$), revealing a highly differential protein signature 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 significantly 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 positively regulated with respect to disease severity and progression. These and additional data were published in the peer reviewed journal *Kidney International Reports* in May of 2018. The article was titled: “Benefit of Apabetalone on Plasma Proteins in Renal Disease”, authored by Wasiak et al. 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.

Phase 2 ASSERT, SUSTAIN and ASSURE 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.

Combined data from ASSERT, SUSTAIN and ASSURE trials (n=798) demonstrated that treatment with apabetalone led to a significant reduction in MACE. Patients treated with apabetalone (n=556) had a lower cumulative MACE rate of 5.9% vs. 10.4% (p=0.02) in the placebo treated group (n=242). In exploratory subgroups the benefit of apabetalone treatment appeared more striking with MACE occurring less frequently in association with apabetalone than with placebo among patients with diabetes (5.4% vs. 12.7%; p=0.02). Similarly, MACE occurred less frequently in association with apabetalone than with placebo in patients with baseline HDL-C < 39 mg/dL (5.5 vs. 12.8%; p=0.01) or with baseline hsCRP levels > 2 mg/L (5.4 vs. 14.2%; p = 0.02). This data was published in the American Journal of Cardiovascular Drugs by Nicholls et al (Nicholls, Ray et al. 2018).

Additional combined data was published in Kidney and Blood Pressure, by Kulikowski et al in March of 2018. These data showed that apabetalone treatment of SUSTAIN and ASSURE patients with CKD was associated with favourable kidney function and ALP profile. Patients who received apabetalone over 6 months showed significantly (p=0.02) lowered serum ALP -14.0% (p<0.0001 versus baseline) versus -6.3% (p=0.9 versus baseline) compared to placebo treated patients. The eGFR in the apabetalone treated group increased by 3.4% (1.7 mL/min/1.73 m²) (p=0.04 versus baseline), and decreased by 5.8% (2.9 mL/min/1.73 m²) (p=0.6 versus baseline) in the placebo group.

Follow-up analyses of the impact of apabetalone on attenuated coronary atherosclerotic plaque ("AP") in ASSURE patients were published by Shishikura et al in the American Journal of Cardiovascular Drugs (August 28, 2018)(Shishikura, Kataoka et al. 2019). The study employed serial intravascular ultrasound ("IVUS") measures of coronary atheroma in patients treated with apabetalone or placebo. Apabetalone treated patients demonstrated significant reductions in AP length and AP index compared to baseline (pre-treatment) measures. The change in AP index correlated with on-treatment concentration of high-density lipoprotein ("HDL") particles as well as with very low-density lipoprotein ("LDL") particles. These correlations were independent of each other. This study indicates that apabetalone favourably modulated ultrasonic measures of plaque vulnerability.

Analysis of the phase 2 program 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 guide selection of potential additional indications that can broaden the scope of diseases that BET inhibition can benefit. Appropriate intellectual property will be developed in concert with any novel findings.

Apabetalone Phase 2 Clinical Trial History

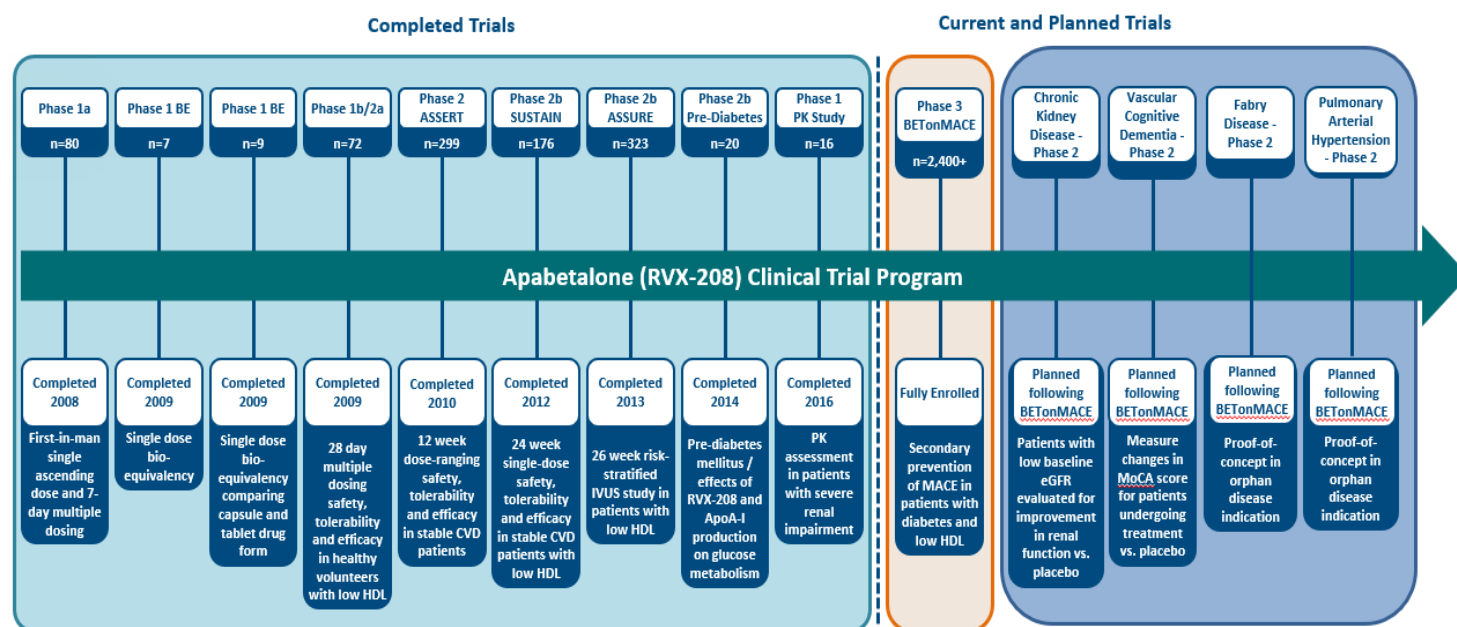
A total of 1,001 subjects have participated in our Phase 1 and Phase 2 clinical trials, of which 722 received treatment with apabetalone and 279 received placebo. We have completed three Phase 2 studies in patients with cardiovascular disease and one Phase 2 study in patients with pre-diabetes.

- 1) The Phase 2 ASSERT study enrolled 299 patients. Findings demonstrated by ASSERT included:
 - 200 mg/day of apabetalone was the optimal dose, based on safety and efficacy;
 - 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
 - Best responses were observed in those patients given apabetalone in combination with second generation statins such as Rosuvastatin (Crestor®) or Atorvastatin (Lipitor®).
- 2) The Phase 2b SUSTAIN study enrolled 176 patients. Findings demonstrated by SUSTAIN included:
 - Low baseline HDL and low baseline ApoA-I were the best responders; and
 - There was one MACE event in subjects treated with apabetalone compared to six in subjects treated with placebo.
- 3) The Phase 2b ASSURE study enrolled 323 patients. Findings demonstrated by 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%).
- 4) The Phase 2b Pre-diabetes study enrolled 20 patients. Findings demonstrated by this study included:
 - Short duration of apabetalone treatment had effects on glucose metabolism; and
 - Both the reduction in glucose absorption and production are expected to be of benefit in patients with prediabetes mellitus.

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

Based on our clinical trials, we have developed a broader and more integrated view of the effects of treatment with apabetalone across the vascular and coronary artery disease spectrums with safety and efficacy results for up to six months of treatment.

Completed, Current and Planned Apabetalone (RVX-208) Clinical Trials



Recent Scientific Developments

Based on our observed MACE reduction data from the pooled post-hoc analysis of the ASSERT, 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 were identified as probable underlying contributors to 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, NF- κ B signaling¹, 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. NF- κ B plays a pivotal role in inflammatory responses, regulating multiple aspects of innate and adaptive immunity, and inducing the expression of various pro-inflammatory genes. 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, acute phase response pathways were verified by real-time 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' plasma from the ASSERT, SUSTAIN and ASSURE clinical trials, apabetalone reduced levels of specific complement, coagulation and acute phase response proteins. Further proteomic analysis of protein levels in patients' plasma from our previous clinical trials is in progress.

¹ NF- κ B is 'nuclear factor kappa-light-chain-enhancer of activated B cells'

Multiple cell types present in blood, including monocytes, lymphocytes and neutrophils, contribute to CVD. To assess the effect of apabetalone in these cells, 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. Effects on many 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. These data provide further evidence that apabetalone is affecting pathways underlying CVD and/or acute events. We are also pursuing investigation of apabetalone's specific effects on individual cell types in blood from patients with other diseases.

Apabetalone-mediated BET inhibition affects multiple processes important for CVD, CKD and DM. Based on mechanistic data, we believe that apabetalone treatment, or select BET inhibition, attenuates the inflammatory process that contributes to disease initiation and progression. We have recently published data in cell models of vascular inflammation which support the anti-inflammatory effect of apabetalone treatment in endothelial cell lines, and prevention of cellular adhesion that occurs between endothelial cells and monocytes at the site of plaque formation. In addition, the reduction in vascular calcification seen in osteogenic vascular smooth muscle cells treated with apabetalone supports apabetalone's potential in treating CKD and related complications. We have also begun to compile results on the effects of *ex vivo* treated DM patient blood, with particular focus on the effects of apabetalone on the immune response in relevant cell types. Data collected to date has also supported investigation of apabetalone effects in models of neurodegenerative diseases, due to apabetalone's peripheral anti-inflammatory effects and the known links between CVD, DM and cognitive deficits. These analyses show consistent regulation of markers and pathways known to contribute to CVD, CKD and DM, and are highlighted in the publications below.

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, European Society of Cardiology Congress, American Society of Nephrology Kidney Week, European Renal Association Congress, American Diabetes Association Scientific Sessions, Vascular Discovery: From Genes to Medicine (ATVB), Clinical Trials on Alzheimer's Disease Conference, Alzheimer's Association International Conference, International Conference on Alzheimer's & Parkinson's Diseases (AD/PD), Keystone Symposia on Molecular and Cellular Biology (Drivers of Type 2 Diabetes: From Genes to Environment), and Gordon Research Conference: Epigenetic Regulation of Cardiovascular Disease.

During the year, we published several findings in peer reviewed journals, including the following:

- We collaborated with experts in the field of pulmonary arterial hypertension ("PAH") research to develop a high impact manuscript titled "Multicenter preclinical validation of BET inhibition for the treatment of pulmonary arterial hypertension", published in the American Journal of Respiratory and Critical Care Medicine. This data demonstrates the benefits of apabetalone treatment in a number of rodent and cell models of PAH. Research was conducted at both Canadian and Netherlands institutions with similar results. These findings have supported the initiation of a pilot trial in PAH patients.
- We recently published a manuscript entitled "The Effect of Bromodomain and Extra-Terminal Inhibitor Apabetalone on Attenuated Coronary Atherosclerotic Plaque: Insights from the ASSURE Trial" in the American Journal of Cardiovascular Drugs. This publication reports, for the first time, the observed effect of 26 weeks apabetalone treatment directly on plaque parameters and measurements as visualized by IVUS. Results from this study suggest that apabetalone may have the potential to stabilize high-risk atherosclerotic plaque. We also published an article titled: "Apabetalone downregulates factors and pathways associated with vascular calcification" in Atherosclerosis, the official peer-reviewed scientific journal of the European Atherosclerosis Society. Data in this manuscript show that apabetalone inhibits calcium deposition in human coronary artery vascular smooth muscle cells (VSMCs) under osteogenic conditions, as well as reducing levels of other factors involved in vascular calcification in relevant cell types, via an epigenetic mechanism.
- A manuscript titled "Apabetalone (RVX-208) reduces vascular inflammation *in vitro* and in CVD patients by a BET-dependent epigenetic mechanism" was recently accepted by Clinical Epigenetics (In Press). This publication highlights the effects of apabetalone treatment on markers of vascular inflammation and cellular adhesion *in vitro* and in clinical plasma samples, as well as showing a reduction of monocyte adhesion to endothelial cells in cell models, with additional data supporting the BET dependent nature of these processes.
- Currently, an additional manuscript has been accepted with revisions to the Journal of Translational Medicine, titled "Epigenetic modulation by apabetalone counters cytokine driven acute phase response *in vitro*, in mice and in cardiovascular disease patients". This work compiles all findings to date regarding apabetalone's effects on the acute phase response, related targets and pathways.

- A manuscript has been submitted to the American Heart Journal titled “Effect of selective BET protein inhibitor apabetalone on cardiovascular outcomes in patients with acute coronary syndrome and diabetes: Rationale, design, and baseline characteristics of the BETonMACE trial” outlining details of BETonMACE study design and patient characteristics collected at enrollment.
- We have submitted a manuscript titled “Apabetalone Lowers Serum Alkaline Phosphatase and Improves Cardiovascular Risk in Patients with Cardiovascular Disease”, led and written in collaboration with clinical experts in the area ALP and CKD. This publication has been re-submitted with requested edits to Atherosclerosis. Data contained in this manuscript highlights the role of ALP in cardiovascular risk, and the benefits of apabetalone induced reduction of ALP in reducing cardiovascular risk in treated CVD patients.

On June 10, 2019, we announced participation at leading, international scientific conferences including:

- The American Diabetes Association’s (ADA) 79th Scientific Sessions 2019: “Apabetalone (RVX-208) attenuates an inflammatory milieu that enhances adhesion of monocytes to endothelial cells in type 2 diabetes mellitus with cardiovascular disease patients”
- The 56th European Renal Association - European Dialysis and Transplant Association (ERA-EDTA) Congress 2019. We supported an educational symposium titled: “Vascular calcification in kidney disease: Epigenetics as a novel approach” where key opinion leaders reviewed the role of epigenetics and BET-inhibition as a novel strategy to improve outcomes in cardiovascular and chronic kidney disease patients. Additionally, Dr. Kamyar Kalantar-Zadeh, MD, UC Irvine School of Medicine, Irvine, CA, presented a poster titled: “Apabetalone, a Selective Bromodomain and Extra-Terminal (BET) Inhibitor, Reduces Serum FGF23 in Cardiovascular Disease and Chronic Kidney Disease Patients” and Dr. Ewelina Kulikowski, Senior Vice President, Research & Development also delivered an oral presentation titled: “Apabetalone, an Inhibitor of BET Proteins, Improves Cardiovascular Risk and Reduces Alkaline Phosphatase in both CVD Patients and Primary Human Cell Culture Systems”.
- The 2019 FSHD International Research Congress: Dr. Christopher Sarsons, Applied Research Scientist, delivered the oral presentation: “Apabetalone, a CVD Phase 3 Clinical-stage BET Inhibitor, Opposes DUX4 Expression in Primary Human FSHD Muscle Cells”.
- The Alzheimer’s Association International Conference (AAIC) 2019: Dr. Cummings, Founding Director of the Cleveland Clinic Lou Ruvo Brain Health Center, Las Vegas, NV, presented a poster titled: Effects of the Epigenetic BET-Inhibitor Small Molecule Apabetalone on Cognition in Patients with Diabetes and Cardiovascular Disease.

Through participation at major conferences and events, we continue to highlight apabetalone’s ability to regulate multiple biological pathways that underlie and contribute to CVD, diabetes, and neurodegenerative diseases.

Potential Orphan Disease Indications

Based on the literature and knowledge of epigenetics and BET inhibition, our analysis of apabetalone treated human plasma and our recent proteomics assessment, new pathways, genes and biomarkers known to play a role in orphan diseases have been investigated. New data generated in our research laboratory has demonstrated that BET inhibition by apabetalone has effects on multiple biological pathways that underlie disease pathology. Based on these recent advancements and scientific knowledge gained, we intend to continue to expand our research and development to explore orphan diseases. We will continue to 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 coronary artery disease 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 14 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 April 30, 2019, we own and/or have rights to thirteen patent families, comprising 121 allowed/issued patents (including eighteen issued US patents). Our intellectual property portfolio includes a number of pending US applications and world-wide equivalents. Our patent portfolio covers compositions, methods of use, manufacturing and formulation claims to apabetalone.

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. In fiscal 2018, we were issued seventeen patents within five patent families in twelve jurisdictions, including a United States patent related to apabetalone for treating anti-inflammatory diseases. In fiscal 2019, we were issued ten patents within five patent families in six jurisdictions, including a United States patent related to apabetalone for treating complement-associated diseases.

Our patents and patent applications have been filed in numerous jurisdictions, including the USA, Europe, China, Japan, South Korea, Canada and India; the details for each patent vary depending on the nature of the claims and importance to the Company. The patent containing compound claims to apabetalone has been granted in all jurisdictions in which we applied (the USA, Australia, Canada, China, Europe, Hong Kong, India, Japan, South Korea, Macau, Mexico, and New Zealand). One of the core patents around apabetalone, titled “Novel Compounds Useful in the Synthesis of Benzamide Compounds” (Patent No. ZL201380053651.0), issued in China on September 7, 2016 with an expiry date in 2033.

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 advisory

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 Health, Department of Primary Care and Public Health, School of Public Health at Imperial College London. A clinical cardiologist by training, Professor Ray received his medical education (MB ChB, 1991) at the University of Birmingham, his MD (2004) from the University of Sheffield, a postdoctoral fellowship at Harvard Medical School (2004-2005) an MPhil in epidemiology (2007) from the University of Cambridge and was Chair in Preventive Cardiology at St Georges University of London from 2010. Professor Ray’s research interests focus on the prevention of CVD using observational methods and intervention studies including large clinical 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) 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 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 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 has been named a "Highly Cited" researcher in 2015, 2016 and 2017 by Clarivate Analytics, representing a ranking in the top 1% by citations of all researchers worldwide for field and publication year.

Dr. Vincent 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 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 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 2017, we issued a total of CAD\$10 million (US\$7.5 million) in equity units. Eastern Capital Limited ("Eastern") and Shenzhen Hepalink Pharmaceutical Co., Ltd. ("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 (US\$4.0 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 (US\$3.5 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.

On September 8, 2017 we issued a total of 3,418,744 equity units, pursuant to a private placement for gross proceeds of CAD\$5.1 million (US\$4.2 million), or CAD\$1.50 per unit, whereby each unit is comprised of one common share and one-half common share purchase warrant. Each warrant is exercisable at a price of \$1.50 per share for a period of 4 years from the closing of the offering.

On December 1, 2017 we closed a private placement of 60,416,667 equity units to Hepalink at a price of CAD\$1.44 per unit for gross proceeds of CAD\$87.0 million (US\$68.5 million). Each unit was comprised of one common share and 0.082759 of a common share purchase warrant. Each full warrant is exercisable at a price of CAD\$1.64 per share for a period of four years from the closing of the offering.

During the three months ended April 30, 2018, we issued 1,776,322 common shares at a weighted average price of CAD\$1.78 pursuant to additional private placements for gross proceeds of CAD\$3.2million (US\$2.5 million). In addition, on April 12, 2018 we issued 364,914 equity units at CAD\$1.75 per unit pursuant to a private placement for gross proceeds of CAD\$0.6 million (US\$0.5 million). Each unit consists of one common share and 0.5 of a common share purchase warrant. Each warrant is exercisable at a price of CAD\$2.00 per underlying common share for a period of two years from the closing of the private placement.

In August 2018, we issued 10,403,216 equity units at CAD\$2.50 per unit pursuant to a private placement for gross proceeds of CAD\$26.0 million (US\$20.0 million). Each unit consisted of one common share and one-half of a common share purchase warrant. Each warrant is exercisable at a price of CAD\$3.00 per underlying common share for a period of three years from the closing of the private placement.

In November 2018, we issued 4,500,000 equity units at CAD\$3.00 per unit pursuant to a private placement for gross proceeds of CAD\$13.5 million (US\$10.3 million). Each unit consisted of one common share and one-half of a common share purchase warrant. Each warrant is exercisable at a price of CAD\$3.10 per underlying common share for a period of three years from the closing of the private placement.

In January 2019, we issued 2,213,398 equity units to Hepalink at CAD\$3.00 per unit pursuant to a private placement for gross proceeds of CAD\$6.6 million (US\$5.1 million). Each unit consisted of one common share and one-half of a common share purchase warrant. Each warrant is exercisable at a price of CAD\$3.21 per underlying common share for a period of three years from the closing of the private placement.

On March 29, 2019, we issued 4,479,793 equity units to Hepalink at CAD\$3.00 per unit pursuant to a private placement for gross proceeds of CAD\$13.4 million (US\$10.1 million). In addition, during the three months ended April 30, 2019, we issued 559,444 units at CAD\$3.00 per unit pursuant to additional private placements to other subscribers for gross proceeds of CAD\$1.7 million

(US\$1.3 million). Each unit consisted of one common share and one-half of a common share purchase warrant. Each warrant is exercisable at a price of CAD\$3.21 per underlying common share for a period of three years from the closing of the private placements.

Repayment of Loan

On December 6, 2017, we repaid our CAD\$68.8 million (US\$54.2 million) loan, originally obtained from Citibank, N.A. and subsequently assigned to an affiliate of Eastern, and all related interest and fees (CAD\$6.1 million, or US\$4.8 million).

Third Eye Loan

On May 7, 2018, we announced we had closed a US\$30 million senior secured loan (the "Third Eye Loan") with Third Eye Capital ("Third Eye"). The loan bears interest at 10% per annum. On April 30, 2019, we entered into a loan amendment to extend the maturity date of the loan from May 4, 2019 to August 4, 2019. We are currently in discussions with Third Eye regarding the repayment and/or extension of the Third Eye Loan beyond August 4, 2019. The Third Eye Loan also includes covenants that we must comply with including a minimum cash balance of US\$3.0 million, a current ratio of 1:1 (with the current ratio calculation excluding warrant liability, unearned licensing rights fee and debt from the current liabilities denominator), and a minimum market capitalization of CAD\$150 million. The Third Eye Loan is subject to mandatory prepayment provisions requiring at least 50% of the net cash proceeds of asset dispositions, licensing, distribution or partnership agreements, royalties, debt or equity issuances, grants and tax refunds to be applied to repayment of the Third Eye Loan (unless waived by Third Eye). During the year ended April 30, 2019, we repaid approximately US\$15.5 million of the Third Eye Loan, corresponding to 50% of the net cash proceeds totaling approximately US\$30.9 million from private placements and warrant exercises in the period. During the year ended April 30, 2019, Third Eye waived repayments associated with net cash proceeds totaling approximately US\$16.4 million from private placements.

Licensing

Licensing Agreements

On July 8, 2015, we entered in to a licensing agreement with Shenzhen Hepalink Pharmaceutical Co., Ltd. ("Hepalink"), Under the terms of the agreement, Hepalink has the exclusive rights to distribute and market apabetalone in China, Hong Kong, Taiwan and Macau (the "Territories"), for all indications.

The license between us and Hepalink provides for certain milestone payments based on net sales of RVX-208 in the Territories. The annual sales milestones range from 500 million renminbi ("RMB") to 10 billion RMB (US\$74 million to US\$1.5 billion, incorporating the period end spot exchange rate), with Resverlogix being eligible to receive sales-based milestone payments from Hepalink ranging from US\$5 million to US\$90 million. In addition, Hepalink shall pay a royalty of 6% of annual net sales of RVX-208 in the Territories. The royalty is subject to an adjustment mechanism that may reduce the royalty rate to a minimum of 4% in the event that certain annual sales milestones are achieved and applicable regulatory authorities in the Territories reduce the approved selling price of RVX-208. Hepalink will be responsible for all clinical and development costs in the Territories, including a patient population that is expected to be included in the Company's Phase 3 BETonMACE 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, 2019, these potential payments do not satisfy the criteria for recognition as a liability.

On October 23, 2017, we entered into a Right of First Refusal Agreement with Hepalink USA Inc. (Hepalink USA), a subsidiary of Hepalink. Under the Agreement, Hepalink USA was granted a right of first refusal in connection with the licensing of the right to develop, manufacture, and commercialize pharmaceutical products containing RVX-208 (apabetalone) in the United States until April 15, 2019. Hepalink USA paid CAD\$8.0 million to us in consideration for the right of first refusal granted (the "Fee"). Pursuant to the Agreement, if Resverlogix and Hepalink USA entered into a license agreement with respect to the US Licensing Rights, the Fee would have been credited against any payment obligations of Hepalink USA thereunder. Otherwise, the Fee was refundable, in whole or in part, until termination of the Agreement. The Unearned Licensing Rights Fee was recognized as a current liability from the commencement of the Agreement until the April 15, 2019 termination date (at which time it was no longer refundable), at which time the Fee was considered earned, and recorded as other income.

On January 8, 2018, we entered into a licensing agreement with Medison Pharma Ltd. Under the terms of the agreement, Medison has the exclusive rights to distribute and market apabetalone in Israel. Resverlogix is eligible to receive from Medison, ascending double digit royalties based on future net sales of the product in the licensed territory. Medison will be responsible for all regulatory, sales and marketing costs for apabetalone in the Israel licensed territory.

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. The purpose of the agreement is to enable each party to achieve greater utilization of their respective resources.

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. The purpose of the agreement is to enable us to obtain access to specialized research services on a more cost-effective basis than other alternatives.

Recent Developments

Public Offering of Units

In June 2019, we completed a public offering of approximately 3.8 million equity units at a price of CAD\$4.00 per unit for gross proceeds of approximately CAD\$15.2 million (US\$11.4 million). Each unit consisted of one common share and one common share purchase warrant. Each warrant is exercisable at a price of CAD\$4.60 per underlying common share for a period of four years from the closing of the offering.

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 or securities convertible into 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 Company.

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, 2019, we had a deficit of US\$537.0 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 advances pursuant to credit facilities. 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 and comply with 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, 2019, in combination with the \$11.4 million raised subsequently, will not be sufficient to fund our contractual commitments or our planned business operations over the next year and repay the Third Eye Loan upon maturity (on August 4, 2019 unless extended) or comply with our loan covenants for the entire remaining term of the Third Eye loan.

We will have to raise additional capital. If we are not able to raise sufficient capital to fund our operations, we would also have to reduce our cash requirements by eliminating or deferring spending on research, development and corporate activities, or may be forced to cease operations. 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 unfavourable 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 favourable to us.

We have been advanced funds under a secured loan agreement and failure to pay all amounts when they become due could result in a loss of all of our assets.

In May 2018, we obtained a US\$30 million senior secured term loan from Third Eye Capital Corporation, as agent for a syndicate of lenders. The loan bears interest at a rate of 10% per annum, payable monthly. On April 30, 2019, we entered into a loan amendment to extend the maturity date of the loan from May 4, 2019 to August 4, 2019. We are currently in discussions with Third Eye regarding the repayment and/or extension of the Third Eye Loan beyond August 4, 2019. The loan is secured by all of our assets and contains certain restrictive financial covenants, covenants relating to the completion of the BETonMACE clinical trial (top line data on or before January 31, 2019 or a later date at Third Eye's discretion) and other customary covenants. Pursuant to the loan amendment with Third Eye, the Company must maintain a cash balance greater than \$3 million, a current ratio greater than 1:1 (with the current ratio calculation excluding warrant liability, unearned licensing rights fee and debt from the current liabilities denominator), and a market capitalization greater than CAD\$150 million, as well as other customary covenants. The Third Eye Loan is subject to mandatory prepayment provisions requiring at least 50% of the net cash proceeds of asset dispositions, licensing, distribution or partnership agreements, royalties, debt or equity issuances, grants and tax refunds to be applied to repayment of the Third Eye Loan (unless waived by Third Eye).

We do not currently have sufficient cash available to repay the principal amount of the loan when it becomes due. We will need to raise additional capital to remain in compliance with covenants applicable to the Third Eye Loan, to repay the balance of the Third Eye Loan at the maturity date of August 4, 2019 (unless extended). We may be unable to remain in compliance with covenants applicable to the Third Eye Loan. In addition, events beyond our control, including the results of clinical trials, regulatory restrictions, competitive conditions and changes in general economic, business and market conditions, may affect our ability to observe or satisfy financial, operational and other covenants, which could result in a default under the loan.

If an event of default under the loan occurs, the lenders could elect to declare all principal amounts outstanding under the loan at such time, together with accrued interest and applicable fees, to be immediately due and payable. If we are unable to repay amounts owing under the loan, the lenders could proceed to foreclose or otherwise realize upon all of our assets, including our intellectual property, that is security for the indebtedness.

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 favourable 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.

To obtain regulatory approval to market and sell any of our products, we must satisfy the FDA, 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 unfavourable 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 fewer occurrences between weeks ten and thirteen. In our subsequent Phase 2b clinical trials, SUSTAIN and ASSURE, 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 policies previously available to public companies. The impact of these events could also make it more difficult for us to attract and retain qualified persons to serve on our board of directors or our board committees or as executive officers. In addition, we may need to hire additional legal and accounting staff with appropriate experience and technical knowledge, and we cannot assure that if additional staffing is necessary that we will be able to do so in a timely fashion.

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

Our ability to successfully market therapeutic products depends in part on the extent to which reimbursement for the cost of such products and related treatments will be available from government health administration authorities, private health insurers and other healthcare organizations. Significant uncertainty exists as to whether newly-approved pharmaceutical products will qualify for reimbursement from these organizations. Furthermore, challenges to the price of medical products continue to grow in frequency due to increased focus on cost containment and pharmacoeconomic issues. These recent changes will become more pronounced as leading therapeutics in the atherosclerosis market such as statins continue to come off patent. Health authorities will continue to

increase their scrutiny and pharmacoeconomic diligence on new products in all disease areas including those for the cardiovascular market. These rapid changes in the healthcare reimbursement marketplace will potentially have a significant impact on the future marketability of new drugs in development and could materially adversely affect our business, financial condition and results of operations. It is expected that new drug entrants will not only have to be effective and safe but also have to provide a clear value proposal to health systems, such as risk reduction in 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.

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 U.S. 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, and could fluctuate substantially in the future. During the twelve months preceding April 30, 2019, the closing market price of our common shares ranged from CAD\$1.12 to CAD\$4.15 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.

We may consolidate our share capital without seeking further approval from shareholders

We obtained approval from shareholders at the annual and special meeting of shareholders held on September 12, 2018 to consolidate our common shares on the basis of one post-consolidation common share for up to every five pre-consolidation common shares at any time within 12 months following the shareholder approval. We may determine to proceed with the consolidation at any time during such 12 month period at an exchange ratio to be determined by the board of directors, provided such exchange ratio is equal to or greater than one post-consolidation common share for every five pre-consolidation common shares, and without seeking any further approval from shareholders. Any consolidation of the common shares would reduce the total number of common shares outstanding and the number of common shares owned by each shareholder and may adversely impact the market value and the liquidity of the common shares.

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, the dividends that we may be required to be pay to holders of the royalty preferred shares in accordance with the terms of such securities 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, 2019, we had 200,327,919 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, 2019

Date	High (\$)	Low (\$)	Volume
4/30/2019	4.50	3.13	1,938,100
3/29/2019	3.70	2.98	1,899,200
2/28/2019	3.28	2.90	778,300
1/31/2019	3.49	3.11	774,500
12/31/2018	3.43	2.94	820,800
11/30/2018	3.38	3.02	671,600
10/31/2018	3.80	3.00	1,441,800
9/28/2018	4.31	2.90	2,570,100
8/31/2018	3.77	2.62	2,768,500
7/31/2018	2.73	2.25	1,504,100
6/29/2018	2.75	1.21	5,057,100
5/31/2018	1.68	1.11	3,279,800

In addition, on June 20, 2017 we issued 5,503,802 warrants expiring June 20, 2021, each exercisable for one Common Share at an exercise price of \$2.05 (the “June 2017 Warrants”). The June 2017 warrants are listed for trading on the TSX under the symbol “RVX.WT”. The following table shows the high and low trading prices and the volume of June 2017 Warrants traded on the TSX during the year ended April 30, 2019 (as reported by the TSX).

Date	High (\$)	Low (\$)	Volume
4/30/2019	3.05	2.00	15,890
3/29/2019	2.50	2.10	27,500
2/28/2019	2.60	2.00	8,200
1/31/2019	2.60	2.25	8,850
12/31/2018	2.50	2.50	1,300
11/30/2018	2.55	2.30	3,490

Date	High (\$)	Low (\$)	Volume
10/31/2018	3.00	2.32	8,601
9/28/2018	2.95	1.83	62,237
8/31/2018	2.10	1.19	109,368
7/31/2018	1.25	1.05	90,550
6/29/2018	1.10	0.50	305,379
5/31/2018	0.68	0.60	27,740

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, 2019:

Date	Type of Security	Issue Price or Exercise Price of Securities	Number of Securities	Type of Issuance
March 2019	Warrants	\$3.21	2,239,897	Pursuant to private placement
March 2019	Warrants	\$3.21	77,000	Pursuant to private placement
March 2019	RSUs	N/A	62,500	Pursuant to long term incentive plan
March 2019	Stock Options	\$3.35	25,000	Pursuant to stock option plan
February 2019	Warrants	\$3.21	83,334	Pursuant to private placement
February 2019	Warrants	\$3.21	16,666	Pursuant to private placement
February 2019	Warrants	\$3.21	102,723	Pursuant to private placement
February 2019	Stock Options	\$3.01	25,000	Pursuant to stock option plan
January 2019	Warrants	\$3.21	1,106,699	Pursuant to private placement
January 2019	DSUs	N/A	9,353	Pursuant to deferred share unit plan
November 2018	Warrants	\$3.10	2,250,000	Pursuant to private placement
October 2018	DSUs	N/A	10,098	Pursuant to deferred share unit plan
September 2018	DSUs	N/A	135,550	Pursuant to deferred share unit plan
September 2018	RSUs	N/A	1,883,600	Pursuant to long term incentive plan
August 2018	Warrants	\$3.00	5,201,608	Pursuant to private placement
May 2018	Warrants	\$1.40	3,500,000	Pursuant to loan transaction

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. Chairman and Chief Medical Officer of Cardiol Therapeutics Inc. and Director of Zenith Capital Corp.	2010
Kenneth Zuerblis, CPA ⁽⁴⁾ 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, Inc.	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 (as well as the Chief Executive Officer since May 2018). 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
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

Name and Municipality of Residence	Position	Principal Occupation During Past 5 Years	Director Since
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 8,584,325, or 4.2%, of issued and outstanding Common Shares as of July 26, 2019.

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 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.

The Board has determined that Messrs. Zuerblis and McNeill are both independent within the meaning of National Instrument 52-110 - Audit Committees and that Mr. Lu, although not independent due to his role as an executive officer of a subsidiary of a major shareholder, is exempt from the requirement to be independent under NI 52-110 pursuant to Section 3.3(2) of that instrument. The Corporation has relied on this exemption in order that Mr. Lu may be a member of the Audit and Finance Committee in accordance with NI 52-110. The Board has determined in its reasonable judgment that Mr. Lu is able to exercise the impartial judgment necessary to fulfill his responsibilities as an Audit and Finance Committee member and that his appointment is required in the best interests of the Corporation and its shareholders.

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 (as well as the Chief Executive Officer since May 2018). 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 25 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 USA Inc.; 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 board 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
2019	\$163,000	\$36,000	\$4,800	\$Nil
2018	\$138,000	\$37,500	\$26,733	\$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. McNeill was the Chief Financial Officer of IMRIS Inc. ("IMRIS") from 2009 until his resignation on September 5, 2014. IMRIS is a biomedical company that is a reporting issuer in all provinces of Canada and at the time of Mr. McNeill's resignation was listed on TSX and NASDAQ. On May 26, 2015, IMRIS and certain of its subsidiaries filed voluntary petitions under Chapter 11 of the United States Bankruptcy Code in the United States Bankruptcy Court for the District of Delaware which granted a stay of proceedings against IMRIS. On June 3, 2015, the Manitoba Court of Queen's Bench granted an initial recognition order under the Companies' Creditors Arrangement Act (Canada) recognizing the Chapter 11 proceedings and granting a stay of proceedings against IMRIS.

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

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

Conflicts of Interest

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

Interests of Management and Others in Material Transactions

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

Eastern arranged for an irrevocable standby letter of credit to secure our loan from Citibank and we agreed to indemnify Eastern in respect of its obligations under the standby letter of credit. We pledged our patents and certain tax losses and pools as security for its indemnity obligations and also agreed to issue common share purchase warrants and pay certain fees to Eastern. The loan was repaid in 2017 as more particularly described under “General Development of Business – Financing – Repayment of Loan”.

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 consisted 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.

In December 2017, we completed a private placement of 60,416,667 equity units to Hepalink for aggregate proceeds of CAD\$87.0 million (US\$68.5 million), or CAD\$1.44 per unit. Each unit was comprised of one common share and 0.082759 of a common share purchase warrant. Each full warrant is exercisable at a price of CAD\$1.64 for a period of four years.

In January 2019, we completed a private placement of 2,213,398 equity units to Hepalink for aggregate proceeds of CAD\$6.6 million (US\$5.1 million), or CAD\$3.00 per unit. Each unit consisted of one common share and one-half of a common share purchase warrant. Each warrant is exercisable at a price of CAD\$3.21 per underlying common share for a period of three years from the closing of the private placement.

On March 29, 2019, we completed a private placement of 4,479,793 equity units to Hepalink for aggregate proceeds of CAD\$13.4 million (US\$10.1 million), or CAD\$3.00 per unit. Each unit consisted of one common share and one-half of a common share purchase warrant. Each warrant is exercisable at a price of CAD\$3.21 per underlying common share for a period of three years from the closing of the private placement.

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, subsequent to the most recently completed financial year and prior to the date of this AIF, 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. Loan Agreement dated May 4, 2018, as amended, among the Company, as borrower, Resverlogix Inc., as guarantor, Third Eye Capital Corporation, as agent for certain lenders and each of such lenders, as more particularly described under “General Development of the Business – Third Eye Loan”.

Interests of Experts

KPMG LLP are the auditors of the Company and have confirmed with respect to the Company, that they are independent within the meaning of the relevant rules and related interpretations prescribed by the relevant professional bodies in Canada and any applicable legislation or regulations.

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, 2019.

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
Apolipoprotein	the protein combined with a lipid to form a lipoprotein, a component of HDL and LDL.
ApoA-I	is one of the apolipoprotein components of the HDL particle.
ApoB	is one of the apolipoprotein components of the LDL particle.
Atherosclerosis	a disease in which the deposition of lipids and inflammatory cells in the arterial wall creates a plaque resulting in the hardening and decrease of arterial lumen size.
Atherosclerotic Plaque	the deposit or accumulation of lipid and lipid-containing cells (plaque) in the arterial wall (<i>also known as atheroma</i>).
BET proteins	BET proteins (Bromodomain and ExtraTerminal domain) are proteins that contain bromodomains, which regulate gene transcription through binding to acetylated lysines within the histones bound to DNA.
b.i.d.	“bis in die” (Latin) refers to twice a day dosing.
Bilirubin	the yellow breakdown product of normal heme catabolism, that is excreted in bile and urine; elevated levels may indicate a disease state.
Bioavailability	the degree and rate at which a drug is absorbed into a living system or is made available at the site of activity after administration.
Biopharmaceuticals	a medical drug developed by biotechnology to improve human or animal health.
Bromodomain (“BRD”)	(see BET proteins)

Cancer	a disease characterized by abnormal and uncontrolled cell growth.
Coagulation Cascade	a series of events that culminate in the formation of a blood clot and its subsequent breakdown. This process is controlled by a signaling cascade consisting of coagulation factors which interact and activate each other.
Complement Cascade	the complement system contains a network of tightly regulated proteins that together are a key part of the innate immune system response. The principal roles of complement include defending against invading pathogens, bridging innate and adaptive immunity, eliminating immune complexes and the products of inflammatory injury.
Coronary artery disease ("CAD")	Coronary artery disease is the most common type of heart disease. It is the leading cause of death in the United States in both men and women. CAD occurs when arteries that supply blood to heart muscle become hardened and narrowed. This is due to the buildup of cholesterol and other material, called plaque, on their inner walls.
C-Reactive Protein ("CRP")	a biomarker of cardiovascular inflammation
Cardiovascular disease ("CVD")	a group of diseases of the heart and blood vessels.
Cholesterol	a fatty molecule essential for normal body functions, including the production of hormones and bile acids; it is also an important component of a cell membrane.
Common Shares	common shares in the capital of Resverlogix Corp.
Compound	a chemical substance formed from two or more elements (<i>also see drug</i>).
Contract Research Organization ("CRO")	an organization (commercial, academic or other), contracted by the sponsor to conduct research or development activities.
Chromatin	the combination of DNA and proteins that make up the contents of the nucleus of a cell. The primary functions of chromatin are: to package DNA into a smaller volume to fit in the cell, to strengthen the DNA to allow mitosis and meiosis and prevent DNA damage, and to control gene expression and DNA replication. The primary protein components of chromatin are histones that compact the DNA.
Clinical Trial/Study	a research study in human subjects to evaluate a new drug, medical device, biologic or other intervention under a strictly controlled scientific setting.
Chronic Kidney Disease ("CKD")	a progressive loss in renal function over a period of months or years, also known as chronic renal disease (CRD). Chronic kidney disease is also associated with other chronic diseases such as diabetes and or cardiovascular disease. Professional 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.
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.
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”)
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).
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.
SGLT2	Sodium-glucose Cotransporter-2 (SGLT2). SGLT2 is a glucose transporter located in the kidneys and it is responsible for 90% of glucose reabsorption. Inhibition of SGLT2 leads to the decrease in blood glucose due to the increase in renal glucose excretion. SGLT2 inhibitors are a class of prescription medicines that are FDA-approved for use with diet and exercise to lower blood sugar in adults with type 2 diabetes. Medicines in the SGLT2 inhibitor class include canagliflozin, dapagliflozin, and empagliflozin.
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 " <i>Diabetes Mellitus</i> ")
Zenith	Zenith Capital Corp. (formerly Zenith Epigenetics Corp.), a corporation incorporated under the ABCA, and its subsidiaries.

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