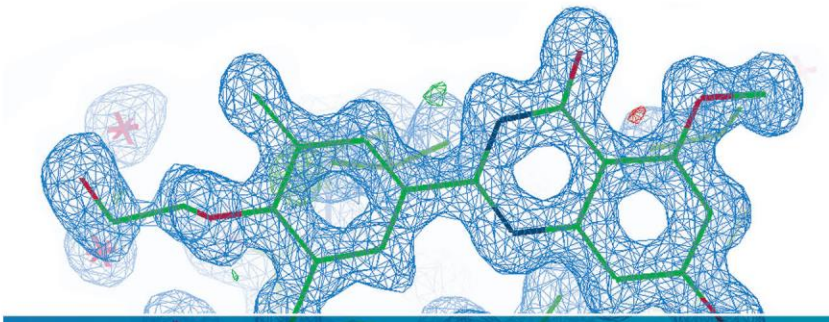


MANAGEMENT'S DISCUSSION & ANALYSIS – Q1 2018 (July 31, 2017)

This Management's Discussion and Analysis ("MD&A") of Resverlogix Corp.'s operations and financial position should be read in conjunction with the unaudited condensed interim consolidated financial statements and the notes thereto for the three months ended July 31, 2017 and 2016 and the audited consolidated financial statements and the notes thereto and the Management's Discussion and Analysis for the years ended April 30, 2017 and 2016. Our financial statements have been prepared by management in accordance with International Financial Reporting Standards ("IFRS") and comprise Resverlogix Corp. (the "Company") and its wholly-owned subsidiary Resverlogix Inc. (together referred to as the "Group"). An advisory with respect to the use of non-IFRS measures is set out in this MD&A under "Non-IFRS Measures". All amounts in the following MD&A are stated in US dollars unless otherwise stated. References to "we", "us" or "our" mean Resverlogix Corp. and its subsidiary unless the context otherwise requires.

Cautionary Statement Regarding Forward-Looking Information

This MD&A contains forward-looking information within the meaning of applicable Canadian securities legislation. Forward-looking information is often, but not always, identified by the use of words such as "believes", "anticipates", "plans", "intends", "will", "should", "expects", "continue", "estimate", "forecasts" and other similar expressions. In particular, this MD&A includes forward-looking information related to: our belief that apabetalone, formerly/also referred to as RVX-208, is a first-in-class, small molecule selective Bromodomain and ExtraTerminal Domain ("BET") inhibitor with potentially important benefits for patients with high-risk cardiovascular disease, diabetes mellitus, chronic kidney disease, end-stage renal disease treated with hemodialysis, neurodegenerative disease, Fabry disease, peripheral artery disease and other orphan diseases; our plans to establish apabetalone for treatment of clinical conditions; our belief that our human clinical trials will provide an understanding of the drug properties in humans; our belief that our patent and patent applications will protect our ideas and inventions related to composition of matter, methods and treatments in our core areas of science and business; and our expectation that we will be able to raise additional capital through external financing or partnering to provide additional funds for our programs.

Readers are cautioned that our expectations, beliefs, projections and assumptions used in preparation of such information, although considered reasonable at the time of preparation, may prove to be wrong, and as such, undue reliance should not be placed on forward-looking statements. With respect to forward-looking statements contained in this MD&A, we have made key assumptions including:

- BET proteins play a critical role in the epigenetic regulation of transcription of particular genes.
- BET proteins all contain highly conserved bromodomains that play a key role in their epigenetic control of gene expression.
- Our small molecules, including apabetalone, function via inhibition of BET bromodomains and, therefore, specifically modulate transcription of particular targets.
- Our patents and patent applications will protect our ideas and inventions related to composition of matter, methods and treatments in our core areas of science and business.
- We anticipate that we will be able to raise additional capital through external financing or partnering to provide additional funds for our programs; and
- The anticipated expenditures required to complete clinical trials.

Our actual results, events or developments could be materially different from those expressed or implied by these forward-looking statements. We can give no assurance that any of the events or expectations will occur or be realized. By their nature, forward-looking statements are subject to numerous known and unknown risks and uncertainties including but not limited to those associated with the success of research and development programs, clinical trial programs, the regulatory approval process, competition, securing and maintaining corporate alliances, market acceptance of our products, the availability of government and insurance reimbursements for our products, the strength of our intellectual property, our financing capability, the potential dilutive effects of any financing, reliance on subcontractors and key personnel and additional risk factors discussed in our Annual

Information Form and other documents we file from time to time with securities authorities, which are available through SEDAR at www.sedar.com. Additionally, risks and uncertainties are discussed on page 23 of this MD&A.

The forward-looking statements contained in this MD&A are expressly qualified by this cautionary statement. We disclaim any intention and have no obligation or responsibility, except as required by law, to update or revise any forward-looking statements, whether as a result of new information, future events or otherwise.

Non-IFRS Measures

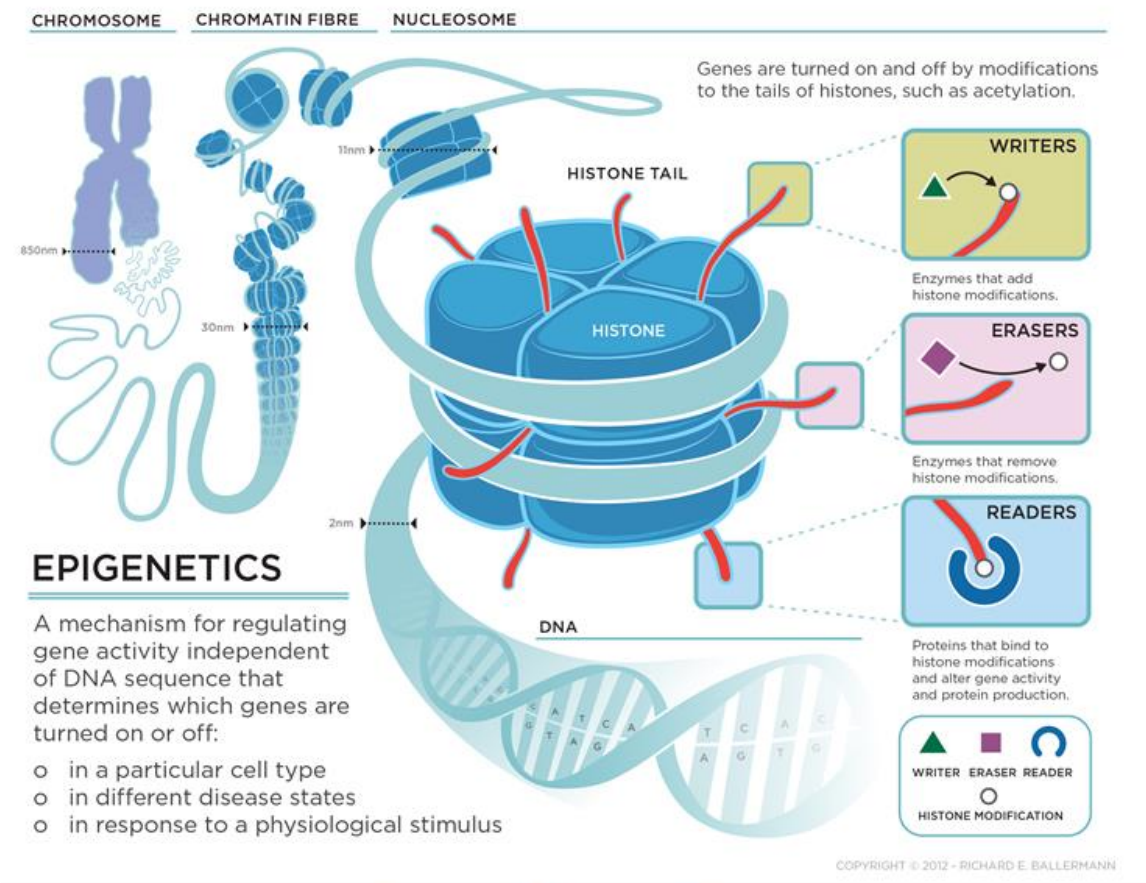
To supplement our consolidated financial statements presented in accordance with IFRS, we use the non-IFRS measure average monthly Cash Burn Rate. This measure is provided to enhance readers' overall understanding of our current use of cash resources and is included to provide investors and management with an alternative measure for assessing our operating results in a manner that is focused on the use of cash for operations and to provide a more consistent basis for comparison between quarters. This measure is based on the cash flow used in operations prior to changes in non-cash working capital from the Consolidated Statements of Cash Flows, as presented on page 16 herein. The average monthly amount is determined using the applicable period total divided by the number of months in the period. This measure is not in accordance with and does not have a standardized meaning under IFRS and is unlikely to be comparable to a similar measure used by other entities.

Overview

What is Epigenetics?

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

Our compounds target one group of "reader" proteins called the Bromodomain and Extra Terminal ("BET") proteins. Substantial evidence has shown that alterations in the pattern of chromatin modifications underlie multiple disease states. Epigenetics represents an important new area of drug development and is now a hallmark of several complex pathologies, including metabolic disorders, cardiovascular and neurological diseases. Epigenetic proteins and regulators are promising targets for therapeutic intervention, and offer the promise of treatment advances in important diseases of high unmet medical need.



Epigenetic Mechanism of Action

Resverlogix Corp.

Since our inception, we have focused on developing 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 drive risk in vascular disease patients, namely: (i) reduction of key vascular inflammation markers, (ii) modulation of complement, coagulation and acute phase response cascades, known drivers in cardiovascular disease ("CVD") and acute cardiac events, (iii) enhancement of reverse cholesterol transport ("RCT"), and (iv) lowering of key markers of metabolic risk. Apabetalone is a first-in-class small molecule in development for the secondary prevention of major adverse cardiovascular events ("MACE") in high risk patients and other chronic diseases such as diabetes mellitus ("DM"), chronic kidney disease ("CKD") and neurodegenerative diseases. Additionally, based on the effects of apabetalone on these multiple biological pathways that underlie disease pathology, an orphan disease program specific for complement mediated diseases represents another potential opportunity for the Company and has been initiated.

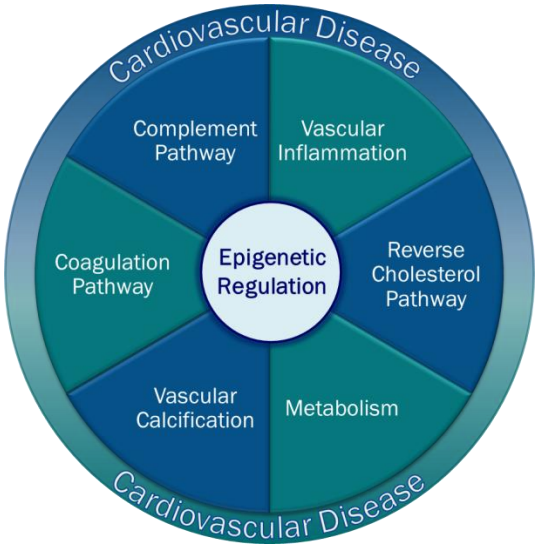
Commencing in the fall of 2015, Resverlogix initiated a Phase 3 clinical trial "BETonMACE" with apabetalone in high-risk CVD patients with type 2 diabetes mellitus and low HDL. The primary endpoint is the time to first occurrence of MACE. We received regulatory approval to open clinical investigator sites in all proposed countries, namely Argentina, Belgium, Bulgaria, Croatia, Germany, Hungary, Israel, Mexico, Poland, Serbia, and Slovakia. Expansion of the trial into clinical sites in Taiwan and Russia is currently underway. With recent communication from the U.S. Food and Drug Administration ("FDA"), we now have the opportunity to include U.S. patients in BETonMACE. We have enrolled in excess of 75% of the approximately 2,400 patients outlined in the study's protocol. A futility analysis is planned after 50–75% of the primary MACE events have been adjudicated.

BETonMACE
A CARDIOVASCULAR STUDY FOR PEOPLE WITH TYPE 2 DIABETES

Apabetalone (RVX-208)

As part of ongoing commercial development for RVX-208, we undertook an official process through the World Health Organization ("WHO") International Nonproprietary Names ("INN") and the United States Adopted Names ("USAN") Council to obtain a nonproprietary name for RVX-208. The name, apabetalone, has received formal approval as an INN and a USAN. As such, the nonproprietary name, apabetalone, can be used globally for internal and external communications. As part of ongoing commercial development for RVX-208, we launched an official process to obtain the official global brand name for apabetalone.

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 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. BET inhibition represents a novel, epigenetic approach to treat multiple diseases. Apabetalone is the first oral agent in the BET inhibitor class that preferentially targets bromodomain 2 ("BD2") of BET protein 4 ("BRD4"). In binding to this bromodomain, apabetalone affects the expression of multiple genes with roles in multiple cellular processes. Its primary mode of action appears to result in the modulation of vascular risk pathways and markers that drive MACE in high risk CVD patients. Some of the biological pathways that are modulated in experiments are: vascular inflammation, complement, coagulation and acute phase response cascades as well as reverse cholesterol transport. Subgroup analysis of our recent clinical trials has demonstrated that apabetalone significantly reduces MACE in patients with CVD who have a low level of high-density lipoprotein ("HDL") and elevated c-reactive protein ("CRP"), especially in those patients with diabetes mellitus co-morbidity. The experimental investigations that have uncovered the multiple activities affected by apabetalone treatment have provided insight into these clinical findings.



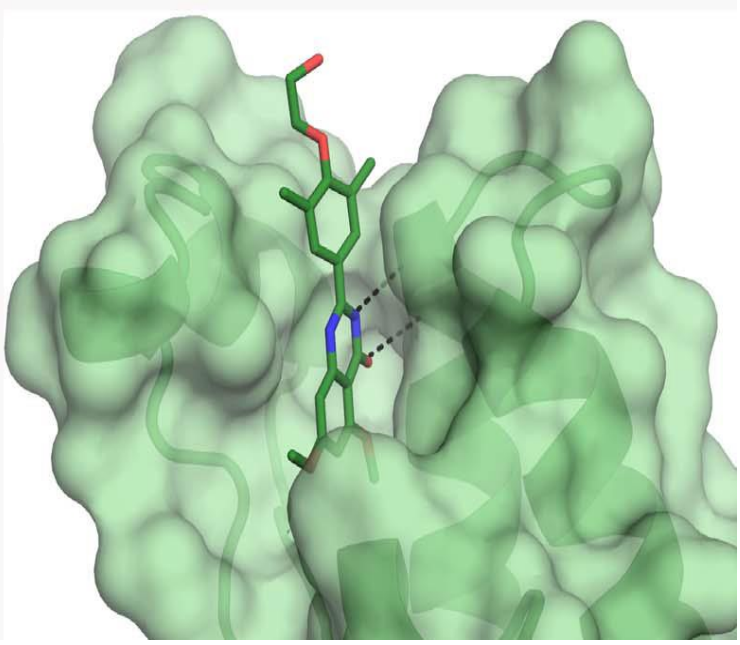
Epigenetic gene regulation governed by BET proteins is at the core of many CVD pathological processes – dysregulation of multiple pathways contributes to increased risk and worse cardiovascular outcomes

Single Therapeutic Target with Multiple Biological Effects

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

We believe that this approach is 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;
- Apabetalone functions via inhibition of BET bromodomain binding to chromatin thereby modulating transcription of particular targets;
- Apabetalone preferentially binds to the second bromodomain of BET family members, with a 20-fold or higher selectivity for the second bromodomains of BRD2, BRD3 and BRD4 versus the first bromodomain;
- Apabetalone is highly differentiated from other therapies that focus only on single biological targets such as increasing HDL or decreasing low-density lipoprotein ("LDL") in plasma, and has effects on multiple pathways and biomarkers that function in concert to reduce CVD events; and
- Apabetalone is the only selective BET inhibitor with no known competitor, providing Resverlogix with an estimated 7-8 year lead over competitors and significant scarcity value.



Apabetalone (RVX-208) Bound in the BET Binding Pocket

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

Clinical Sample Repository of BET Inhibitor Treated Patients

Our Phase 2 clinical program in CVD patients with varying degrees of disease severity consisted 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 apabetalone treatment. 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. Our BET database, which is comprised 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.

Highlights

Clinical Trial Developments

Phase 3 BETonMACE Trial - Enrolling

On January 15, 2014, we announced the pooled MACE results from the SUSTAIN and ASSURE studies (described in further detail herein). This analysis focused on the potential benefit of apabetalone and select BET inhibition to reduce MACE over a short treatment time period of six months. When MACE data (n=499) from both SUSTAIN and ASSURE trials were combined, it demonstrated that treatment with apabetalone led to a significant reduction in MACE. Results from patients with diabetes mellitus, a high-risk patient population, illustrated similar trends.

Based on these findings, our intention with our BETonMACE trial is to reconfirm in a larger prospective setting, with patients that have modifiable vascular disease (i.e. low HDL-C and diabetes), reduction of MACE coupled with favorable effects on markers of vascular risk and renal function. The BETonMACE study, "Effect of RVX-208 on Time to Major Adverse Cardiovascular Events in High-Risk Type 2 Diabetes Mellitus Subjects with Coronary Artery Disease", commenced in October 2015. The study is a large international multi-center, double-blind, randomized, parallel group, placebo-controlled clinical trial to determine whether treatment with apabetalone in combination with rosuvastatin or atorvastatin increases the time to MACE compared to treatment with rosuvastatin or atorvastatin alone. The primary endpoint of the BETonMACE trial is designed to show a relative risk reduction ("RRR") of MACE, narrowly defined as a single composite endpoint of CV death, non-fatal myocardial infarction ("MI") and stroke. The study is

an event-based trial and will continue until at least 250 MACE events have occurred. MACE will be adjudicated by an independent committee and the study will be monitored by a data safety monitoring board. Secondary endpoints include: time to first occurrence of the composite broad MACE which includes the addition of hospitalization for CVD events (unstable angina and revascularization procedures), changes in lipoprotein concentrations (HDL and apolipoprotein A-I ("ApoA-I")), changes in diabetes mellitus variables (glucose and glycated hemoglobin), change in alkaline phosphatase ("ALP"), changes in kidney function and additional safety and tolerability of apabetalone (RVX-208). In order to be eligible to participate in the study, patients must have documented history of type 2 Diabetes Mellitus, experienced a recent (defined as 7-90 days prior to randomization) Coronary Artery Disease ("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 shall consist of daily dose of either atorvastatin 40-80 mg or rosuvastatin 20-40 mg. After an initial screening period of 1 to 2 weeks during which subjects will be treated with standard of care statin therapy, subjects will be randomized to either apabetalone (RVX-208) 100 mg b.i.d. (twice daily) or matching placebo with continued statin treatment. This combination treatment period will continue for up to 104 weeks. We anticipate that a minimum of 2,400 patients will be enrolled. A full detailed protocol for the BETonMACE study can be viewed on www.clinicaltrials.gov with the following NTC ID, NCT02586155.

BETonMACE Will Enroll Patients From 14 or More Countries Worldwide

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

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

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

On October 26, 2015, we announced the commencement of the Phase 3 BETonMACE clinical trial. We received initial approval from the regulatory authority and ethics committee in the first three countries: Belgium, Hungary and Israel, which would represent approximately 36 investigative sites of an expected 177 site trial, including 15 in the first activation wave occurring in 2015. The

first site initiation visit was held and enrollment of patients would commence, with additional investigative sites being activated soon thereafter.

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

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

As of July 31, 2016, all proposed countries, namely Argentina, Belgium, Bulgaria, Croatia, Germany, Hungary, Israel, Mexico, Poland, Serbia, and Slovakia had received regulatory approval to open clinical investigator sites and were recruiting patients.

On August 11, 2016, we announced that that the independent Data and Safety Monitoring Board ("DSMB") for the BETonMACE study in high-risk cardiovascular ("CVD") patients completed a planned safety review and verbally recommended that the study should continue as planned without any modifications.

On December 5, 2016, we announced that the independent DSMB for the BETonMACE trial in high-risk CVD patients completed a second planned safety review and recommended that the study should continue as planned without any modifications. The DSMB reviewed available study data and noted that no safety or efficacy concerns were identified.

On March 17, 2017 and June 28, 2017, respectively, we announced that the independent DSMB confirmed that consistent with the previous DSMB reviews, the BETonMACE study should continue as planned without any modifications and permits the trial to remain on schedule. The DSMB will conduct additional periodic reviews and a futility analysis is planned after 50-75% of the primary MACE events have been adjudicated. We, the clinical steering committee, and all investigators remain blinded to the actual safety and efficacy results.

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 FDA regarding apabetalone, including: human exposure, clinical dosing and established acceptable safety margins, the possibility to include U.S. patients in Phase 3 studies, including the global Phase 3 BETonMACE trial is now open. We have agreed to make adjustments to the existing BETonMACE study protocol and to update the Investigator's Brochure and the Informed Consent Documents. With the approval from the FDA, we now have the opportunity to include U.S. patients in BETonMACE.

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 (the final stage of chronic kidney disease) treated with hemodialysis.

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

Phase 2a Fabry Disease Trial- Planned

On May 30, 2017, we announced that Health Canada, Therapeutic Products Directorate ("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 2017. Patients with Fabry disease experience various heart, kidney, and dermatological complications with

stroke, heart disease and kidney complications being the top causes of mortality. Current medications approved for Fabry disease are not sufficient and there remains a large unmet need.

Phase 1 PK Trial in Patients with Severe Renal Impairment - Completed

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 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 favorable 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 favorable 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, the data for which is still being analyzed.

On January 23, 2017, we announced preliminary results from the Phase 1 PK study in late stage CKD patients. The data showed remarkable results in reducing inflamed protein biomarkers in patients with late stage CKD versus healthy control patients. It is believed that this is the first time in medical history that a direct connection of this type can be made between epigenetic regulation and its potential for positive disease impact. Protein data was collected following a single oral administration of 100mg of apabetalone before and after multiple time points in both cohorts. Protein levels of 289 proteins were significantly different at baseline between the two groups ($p<0.05$). Initial findings from this study revealed a highly differential protein signature at baseline between CKD patients and controls. Following a single dose administration of apabetalone in the late stage CKD patients, the levels of multiple plasma proteins were changed within 12 hours after dosing, demonstrating a fast onset of drug action. Analysis of the changes in protein levels at the 12-hour time point revealed that, in the late stage CKD patients, 33 percent of proteins had statistically significant changes ($p<0.05$) compared to only 10 percent in the controls. Of these significant proteins, several established renal biomarkers such as interleukin 6 (IL6) and osteopontin, were regulated positively with respect to disease severity and progression. Ongoing expanded analysis of this exploratory data is also planned which will look at Ingenuity Pathway Analysis (IPA). The quick onset of action and improvement of reported CKD risk factors are encouraging for us in our planned expansion beyond our current cardiovascular and diabetes program. Detailed data will be submitted for future peer reviewed publications.

Phase 2 Pre-Diabetes Mellitus Trial – Completed

On June 8, 2015, we presented findings from a pre-diabetes mellitus study performed by the Baker IDI Heart and Diabetes Institute, Melbourne, Australia and our scientists which were reported at the American Diabetes Association Scientific Sessions. The presentation was entitled "Effects of the ApoA-I Inducer, RVX-208 on Glucose Metabolism in Individuals with Pre-diabetes Mellitus." The data presented was based on patients with pre-diabetes mellitus who already had abnormal blood glucose levels. Treatment with apabetalone (200 mg/day) for 29-33 days led to a statistically significant reduction in glucose absorption and a statistically significant suppression of endogenous glucose production. The possible significance of these findings is as follows: (1) short duration of apabetalone treatment had effects on glucose metabolism, and (2) both the reduction in glucose absorption and production are expected to be of benefit in patients with pre-diabetes mellitus. The above findings provoke intellectual interest when viewed in the light of additional new data arising from the same study reported at the recent International Society of Atherosclerosis ("ISA") meeting, in May 2015. At the ISA meeting, in a presentation entitled, "The effects of a novel apoA-I transcriptional regulator (RVX-208) on whole plasma and HDL lipidomes," the same team of investigators detailed the ability of apabetalone to change the lipid profile within the HDL favoring normalization of the composition towards that observed in healthy individuals. Together, the data contained in the two presentations contribute to the concept that apabetalone has the ability to affect glucose and lipid metabolism in ways that will be of benefit to patients with high risk vascular disease such as DM.

On June 2, 2016, we announced that findings of a pre-diabetes mellitus study performed by the Baker IDI Heart and Diabetes Institute and the Company had been published in the journal 'Metabolism', titled: "Effects of the BET-inhibitor, RVX-208 on the HDL lipidome and glucose metabolism in individuals with prediabetes: A randomized controlled trial." Data summarized in the publication was gathered from patients with prediabetes mellitus characterized by abnormal blood glucose levels. Treatment with apabetalone (200 mg/day) for 29-33 days led to a delay and a reduction in glucose absorption and endogenous production. The significance of

these findings are as follows: (1) short duration of apabetalone treatment had effects on glucose metabolism, and (2) both the reduction in glucose absorption and production are expected to be of benefit in patients with prediabetes mellitus.

ASSURE and SUSTAIN Analysis

On June 1, 2015, we presented the analysis of data pooled from the ASSURE and SUSTAIN 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/1.7m^2 at baseline 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 and has also contributed to additional abstracts that have been submitted for peer review presentation. These findings may warrant additional clinical trials for target responder CKD and/or dialysis populations who have a high burden of cardiovascular disease and risk. In the BETonMACE trial, a specified subgroup of patients with baseline eGFR <60 mL/min/1.7m^2 will be examined under the guidance of Dr. Kalantar-Zadeh.

On November 9, 2015, Dr. Kam Kalantar Zadeh, at an American Society of Nephrology event highlighted apabetalone's lowering effect on alkaline phosphatase ("ALP"). Dr. Zadeh reported large epidemiology studies on the correlation of ALP and CVD risk in diabetes, CKD and Dialysis patients. In addition Dr. Zadeh reported on apabetalone effects on ALP including its statistically significant correlation to MACE reduction when ALP was lowered by the molecule versus placebo. This post-hoc analysis was performed from our Phase 2 SUSTAIN and ASSURE clinical trials.

Potential Orphan Disease Indications

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

Orphan Disease Fact Sheet

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

Source: NIH Rare Diseases Clinical Research Network Fact Sheet

Scientific Developments

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

We performed microarray-based gene expression analysis using primary human hepatocytes treated with apabetalone. Cells from multiple donors were assessed in independent experiments. Results demonstrated that apabetalone downregulates pathways that contribute to cardiovascular risk or MACE such as atherosclerosis, thrombosis and inflammation. Specifically, apabetalone downregulated the complement, fibrin clotting, acute phase response, cholesterol and fatty acid synthesis pathways, illustrating repression of most of the pathway components. Over activation of the complement pathway and acute phase response participate in plaque development and destabilization. Fibrin clotting is fundamental in the formation of thrombi and emboli. Downregulation of these pathways by apabetalone may avoid catastrophic vascular events leading to occlusion and death. Results for several

components of the complement and coagulation pathways were verified by real-time PCR, a more sensitive and robust method of measuring mRNA expression, as well as using enzyme-linked immunosorbent assay ("ELISAs") to measure protein levels. Results were also recapitulated in human hepatocarcinoma cell lines and with BET inhibitors with different chemical scaffolds. In addition, in patients' serum from the SUSTAIN and ASSURE clinical trials, apabetalone reduced levels of specific complement, coagulation and acute phase response proteins. Further proteomic analysis of protein levels in patients' serum from our previous clinical trials is in progress.

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

Apabetalone mediated BET inhibition affects multiple processes important for CVD and renal risk. In addition to effects on lipoproteins, apabetalone represses pathways underlying the pathogenesis of atherosclerosis and acute coronary events, including inflammation, complement, coagulation (thrombosis), vascular calcification and atherogenesis. Based on mechanistic data, we believe that apabetalone treatment, or select BET inhibition, attenuates the inflammatory process that contributes to disease initiation and progression. Furthermore, we hypothesize that apabetalone treatment induces directional changes towards normalization of perturbed inflammatory states, restores basal activity of the innate immune response and clotting cascade with immediate benefits to atherosclerosis and cardiovascular disease. We are currently performing further detailed analysis on potential new biomarkers and biological pathways that apabetalone may affect through its select BET inhibition mechanism. New findings in these analyses will seek out potential additional indications that can be applied to broadening the scope of diseases that BET inhibition can benefit.

On August 31, 2015, we presented new data at the European Society of Cardiology ("ESC") Congress 2015 in a poster presentation titled: "RVX-208, an orally active BET inhibitor, lowers CVD risk by activities beyond raising ApoA-I/HDL". The presentation summarized studies designed to further understand the reductions in MACE observed in apabetalone treated patients.

On January 25, 2016, we announced that a manuscript focused on apabetalone had been accepted in the Atherosclerosis Journal. The manuscript titled, "RVX-208, a BET-inhibitor for treating atherosclerotic cardiovascular disease, raises ApoA-I/HDL and represses pathways that contribute to cardiovascular disease" details some of the beneficial actions of apabetalone. The data presented emphasized not only that apabetalone raises the levels of ApoA-I and HDL, but also BET inhibition modulates pathways known to be major contributors to CVD risk including complement, fibrin clotting, acute phase response, cholesterol and fatty acid synthesis. The accepted manuscript is currently in electronic press and will appear in a future print issue of Atherosclerosis. The manuscript is available at: http://www.atherosclerosis-journal.com/article/S0021-9150(16)30035-1/fulltext.

On April 28, 2016, we participated in the symposium, Epigenetics: Cancer and Beyond, at the New York Academy of Sciences. An international assembly of biomedical investigators convened to explore the therapeutic potential of pharmacologic modulation of the epigenome. Abnormalities in the epigenome have been identified in many diseases, providing a promising new path for drug discovery. Internationally renowned expert in the field of cancer research, Dr. Craig B Thompson, Memorial Sloan Kettering Cancer Center, delivered the keynote address at the symposium which was chaired by Dr. Norman Wong, the Company's Chief Scientific Officer and Dr. Dominique Verhelle of Third Rock Ventures. Dr. Ewelina Kulikowski, Senior Vice President of Research & Development presented on behalf of the Company. Additional speakers include: Dr. Michael Elowitz, California Institute of Technology, Dr. Keiko Ozato, National Institute of Child Health and Human Development, Dr. Roberto Pili, Indiana University School of Medicine, Dr. Patrick Trojer, Constellation Pharmaceuticals, Dr. Christopher Vakoc, Cold Spring Harbor Laboratory, Daniel Vitt, 4SC AG, and Dr. Eric Campeau, Zenith Capital Corp. ("Zenith").

On May 24, 2016, we presented new data at the ERA-EDTA Congress in Vienna, Austria in a poster titled: "Apabetalone (RVX-208), a Selective Bromodomain and Extra-Terminal (BET) Protein Inhibitor, Decreases Abundance and Activity of Complement Proteins in Vitro, in Mice and in Clinical Studies". The presentation summarized the effects of apabetalone treatment on complement component expression and cascade activity.

In May and June 2016, new findings were presented at the ATVB Scientific Sessions, EAS Congress and the ADA Scientific Sessions. These presentations summarized studies designed to further understand the reductions in MACE observed in apabetalone treated patients.

On August 28, 2016, we hosted a symposium entitled: "A novel approach for high CV risk patients with diabetes: The potential of epigenetics," at the European Society of Cardiology (ESC) Congress in Rome, Italy. Speakers included: Stefano Del Prato, MD - Università di Pisa, Pisa, Italy; Kausik Ray, MD - Imperial College London, United Kingdom; Jorge Plutzky, MD - Brigham and Women's

Hospital, Harvard Medical School, Boston, USA; and John Kastelein, MD - Academic Medical Centre, Amsterdam, The Netherlands. These renowned experts in the field of CVD presented on topics including, “The High Risk Diabetes patient with CVD: What else can we target to reduce cardiovascular risk?”, “Understanding BET inhibition as a novel pathway for cardiovascular risk modulation”, and “BET inhibition in CVD: A new dawn?” The presentations highlighted the opportunity and need for a novel approach to the treatment of high risk cardiovascular disease and diabetes. Moreover, data presented demonstrated the potentially important role for epigenetics in the underlying pathology of these diseases.

On August 29, 2016, we presented new data at the 2016 ESC Congress in a poster presentation titled: "Modulation of the complement cascade in cardiovascular disease patients by a BET protein inhibitor". The poster contained data demonstrating that apabetalone treatment reduced basal and cytokine-induced expression of complement factors in hepatocytes. Furthermore, in samples from CVD patients, the complement pathway was identified to be the most downregulated by apabetalone treatment. This was supported by an observed reduction in the levels of complement proteins, which have been linked to CVD and MACE. A reduction in the overall function of the complement cascade in plasma from cardiovascular disease patients treated with apabetalone was also presented.

Throughout September, October and November 2016, new findings were presented at the European Association for the Study of Diabetes Annual Meeting, AHA Scientific Sessions and the ASN Kidney Week Conference. These presentations summarized studies designed to further explore the mechanisms underlying the reductions in MACE observed in apabetalone treated patients.

In October 2016, we hosted R&D update events in New York, NY and London, UK, featuring presentations from two internationally recognized key opinion leaders. The featured key opinion leaders presenting at both events included Professor Kausik Ray, MBCHB, MD, MPHIL, FACC, FAHA, FESC, FRCP. Professor of Public Health, Department of Primary Care and Public Health, School of Public Health, Imperial College London, UK. Chairman, Resverlogix BETonMACE Clinical Steering Committee and Dr. Kamyar Kalantar-Zadeh, MD, MPH, PhD. Professor of Medicine, Epidemiology, Pediatrics and Public Health, University of California, Irvine School of Medicine, Chairman, Resverlogix Renal Clinical Advisory Board. We detailed an update of the BETonMACE clinical trial, novel biology and findings from ongoing mechanistic studies focused on cardiovascular risk pathways and apabetalone's potential in reducing MACE.

On February 13, 2017, we highlighted two potential new indications recently identified by third party academic research involving our lead drug, apabetalone. The first indication was published in the Journal of Neuroinflammation which described the inhibition of BET epigenetic readers, including apabetalone, as having therapeutic potential in degenerative diseases of the eye (retinal). The second indication was highlighted by research conducted at Saint Louis University, demonstrating apabetalone mediated modulation of important targets in Facioscapulohumeral Muscular Dystrophy (“FSHD”). Both indications are areas of interest and licensing potential for us. In January 2017, the Journal of Neuroinflammation published an article titled "Photoreceptor protection via blockade of BET epigenetic readers in a murine model of inherited retinal degeneration" (Zhao et al. Journal of Neuroinflammation (2017) 14:14) where BET inhibitors, including apabetalone, were tested in an animal model of retinal degeneration. The authors concluded that inhibition of the epigenetic readers rescued photoreceptor degeneration in the eye, likely via the suppression of microglial activation. Moreover, the findings demonstrated differential effects of BET inhibitors based on their bromodomain selectivity, which were suggested to have distinct functions in this pathogenic process. BET protein inhibition was identified as a potential novel therapeutic strategy to treat neurodegenerative disease states where microglial activation or pathogenic cell state transformation plays a role, with potential impact for diseases such as retinitis pigmentosa. In research conducted at Saint Louis University and subsequently used in a patent application, BET inhibitors were shown to inhibit expression of the DUX4 gene, which is expected to reduce the severity of symptoms in this disease. Apabetalone treatment, when compared to other BET inhibitors, demonstrated a similar repression of DUX4, however no transient suppression of MYH2 (a differentiation marker for myotubes) was observed. This differential effect on the marker of muscle cell differentiation was unique to apabetalone and demonstrated the distinct properties of this BET inhibitor. Treatment of FSHD via BET protein inhibition offers a novel therapeutic indication for apabetalone and this class of compounds.

On May 23, 2017, we highlighted two additional works involving our lead drug, apabetalone, one recently published by third party academics and one in the form of a patent application by Pfizer Inc. (Pfizer). The publication, titled "Alkaline phosphatase: a novel treatment target for cardiovascular disease in CKD," is published in the prominent journal Nature Reviews Nephrology. This article describes the mechanisms that link ALP to vascular calcification, inflammation, and cardiovascular disease. The authors discuss, "new drugs that target ALP, which have the potential to improve cardiovascular outcomes without inhibiting skeletal mineralization." The article dedicates a paragraph to apabetalone and cites four different publications involving apabetalone. The Pfizer patent application was filed for the purpose of protecting their invention of using BET-family bromodomain inhibitors as a method of increasing frataxin in the treatment of patients with Friedreich's ataxia. Apabetalone was listed as a potentially effective agent against this disease which is present in about 1 in 50,000 people. The ataxia of Friedreich's ataxia occurs from the degeneration of nerve tissue in the spinal cord. Symptoms usually begin between 5 to 15 years of age, leading to wheelchair requirements and can eventually lead to early death often related to cardiovascular disease.

On May 31, 2017, we announced that a recently submitted scientific paper titled, "Downregulation of the Complement Cascade In Vitro, in Mice and in Patients with Cardiovascular Disease by the BET Protein Inhibitor Apabetalone (RVX-208)" was published in the Journal of Cardiovascular Translational Research. This publication discusses how through transcriptional regulation, apabetalone modulates pathways that underlie CVD including reverse cholesterol transport, vascular inflammation, coagulation, and complement a pathway which includes many CVD risk factors that are dysregulated. Using advanced technologies such as transcriptomics and proteomics the data clearly illustrated that complement is one of the pathways most downregulated by apabetalone in cells and in plasma from CVD patients previously treated with apabetalone. Plasma proteomics of CVD patients shows that apabetalone significantly decreases complement proteins and regulators indicating decreased activity of complement in patients. As complement components are linked to CVD and metabolic syndrome, including major acute cardiac events, modulating their levels and activity by apabetalone may alleviate risks associated with these diseases.

On June 5, 2017, we hosted a symposium entitled: "Managing CKD, Diabetes & CVD: Is epigenetics a new way forward?", at the European Renal Association – European Dialysis and Transplant Association (ERA-EDTA) Congress in Madrid, Spain. Speakers included: Vincent M Brandenburg, MD – Aachen, Germany; Luis M Ruilope, MD – Hospital 12 de Octubre, Madrid, Spain; Marta Ruiz-Ortega – Foundación Jiménez Diaz, Madrid, Spain; Kamyar Kalantar-Zadeh, MD – UC Irvine School of Medicine, Irvine, USA and; Carmine Zoccali, MD – Reggio Cal, Italy. These renowned experts in the field of CKD presented on topics including, “The high risk diabetes patient: What is the need for novel approaches to reduce cardiovascular and renal risk?”, “Epigenetics as a novel strategy in cardiovascular and renal risk reduction: A closer look at BET as a pathway for inhibition”, and “BET inhibition in renal and cardiovascular disease: What is the clinical roadmap?” The presentations highlight the opportunity and need for a novel approach to reduce risk in kidney and cardiovascular disease, and demonstrate the important role for epigenetics in the underlying pathology of these diseases.

In addition to the symposium at the ERA-EDTA Congress, we presented new data from the recently completed phase 1 PK study in patients with severe renal impairment in a oral presentation titled, “Apabetalone (RVX-208) Impacts Key Biomarkers and Pathways Associated with Chronic Kidney Disease in Patients with Severe Renal Impairment.” This presentation highlighted an upregulation of pathways known to be activated in CKD such as the inflammatory response, immune response, thrombosis, calcification and oxidative stress, in the renal impaired patients (stage 4 CKD) compared to controls. These pathways were robustly and highly significantly downregulated in the stage 4 CKD patients 12 hours after a single oral administration of apabetalone. Apabetalone treatment also downregulated the abundance of circulating CKD biomarkers involved in vascular inflammation, endothelial dysfunction, acute phase response, coagulation and vascular calcification. Concurrently, we presented new data in two poster presentations at the ERA-EDTA Congress titled: Effects of Apabetalone (RVX-208) on Serum Albumin in Subjects with CVD, Diabetes and Chronic Kidney Disease; A Post-hoc Analysis of the ASSURE and SUSTAIN Clinical Trials and “Apabetalone, a Bromodomain and Extraterminal Protein Inhibitor, Decreases Key Factors in Vascular Calcification in vitro and in Clinical Trials”. The data presented provides the rationale for our CKD program, with apabetalone, which is aimed at assessing the cardiovascular risk reducing potential, in addition to any improved kidney function, observed in prior Phase 2 trials.

On August 26, 2017, we hosted a symposium entitled: "Managing Diabetes & CVD: Is epigenetics a new way forward?" at the ESC Congress ib Barcelona, Spain. Speakers included: Lina Badimon, MD – Barcelona, Spain; Kausik Ray, MD - Imperial College London, United Kingdom; Erik Stroes, MD - Academic Medical Centre, Amsterdam, Netherlands; and Stephen Nicholls, MD - Adelaide, Australia. These renowned experts in the field of CVD presented on topics including, “Managing high risk diabetes patients with cardiovascular disease: What works, and what else can we do?”, “Promise of epigenetic modulation as a target in atherosclerotic patients”, and “Insights from the first trials in epigenetics in human: What is the way forward?” The presentations highlighted the opportunity and need for a novel approach to the treatment of high risk cardiovascular disease and diabetes. Moreover, data presented demonstrated the potentially important role for epigenetics in the underlying pathology of these diseases.

On August 27 and 29, 2017, we presented two poster presentations titled: “Lowering the neutrophil to lymphocyte ratio by the BET inhibitor, apabetalone: potential implications for cardiovasculer events in high risk patients” and “Apabetalone (RVX-208) impacts key biomarkeers and pathways associated with cardiovascular disease in patients with severe renal impairment”. The first poster contained data demonstrating that neutrophil/lymphocyte (“NLR”) ratio levels, a well-known marker of inflammation in cardiovascular patients, were higher in patients with established CVD that experienced a MACE compared to those who did not experience a MACE in the ASSURE and SUSTAIN studies. In addition, it was shown that apabetalone treatment reduced the NLR ratio after 6-months of treatment, highlighting the impact of apabetalone on inflammatory pathways implicated in CVD. The second poster highlighted data from the Phase 1 PK trial in patients with severe renal impairment. Data presented illustrated that a single oral dose of apabetalone rapidly reduces circulating markers and predicted pathway activation linked to the progression of renal disease and accompanying CVD complications. Together these posters highlight the potential of apabetalone in modulating the multiple pathways underlying CVD, including inflammation.

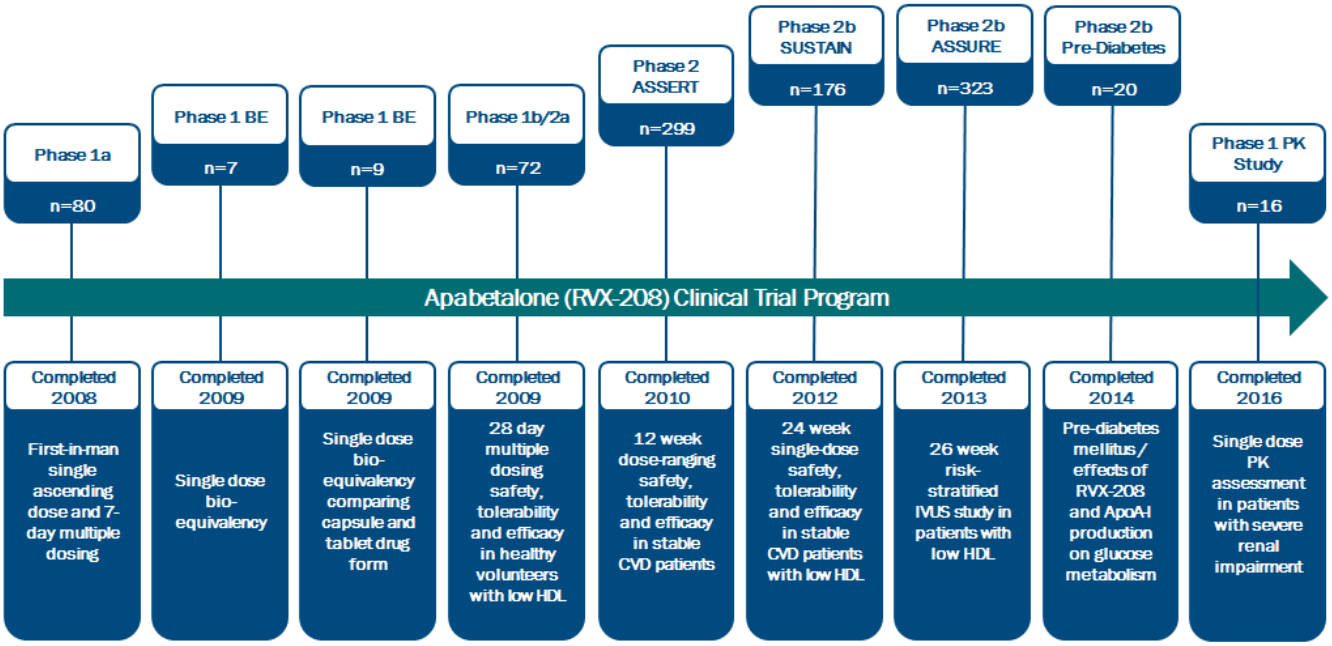
Apabetalone (RVX-208) Clinical Trial History

A total of 1,001 subjects have participated in our completed clinical trials, of which 722 received treatment with apabetalone and 279 received placebo. Three Phase 2 studies in patients with cardiovascular disease have been completed:

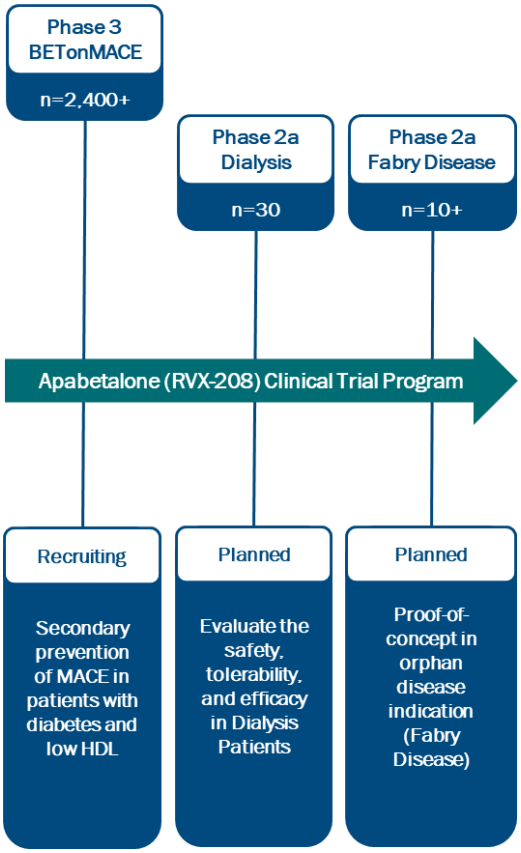
- 12-week ASSERT study enrolled 299 patients,
- 24-week SUSTAIN study enrolled 176 patients, and
- 26-week ASSURE study enrolled 323 patients.

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

Completed Apabetalone (RVX-208) Clinical Trials



Current or Planned Apabetalone (RVX-208) Clinical Trials



Corporate Developments

Board of Directors

On April 4, 2016, we announced that Ms. Norma K. Biln, Chief Executive Officer of Life Sciences Corp. ("Augurex"), and Mr. Shawn Lu, Chief Financial Officer of Hepalink USA Inc. (a subsidiary of Shenzhen Hepalink Pharmaceutical Co., Ltd.), were appointed to Resverlogix's Board of Directors.

Ms. Biln is the Chief Executive Officer and Co-Founder of Augurex. She has 22 years of experience in the pharmaceutical and biotech industries, commencing her career in clinical research with Pfizer Pharmaceuticals and held several positions in sales management, marketing and market access with Pfizer, Amgen and Abbott Laboratories where she won several regional and national level awards for top performance. Ms. Biln later joined Stressgen Biotechnologies as Director of Corporate Market Development and Director of Sales and Marketing for Stressgen Bioreagents. Before her role of CEO at Augurex, she worked in a senior consulting capacity with Aspreva Pharmaceuticals on Business Development, Commercial and Communications initiatives. Ms. Biln holds a Bachelor of Science and Master of Business Administration and is Chair of the Board of Directors of BioTalent Canada.

Mr. Lu has extensive experience in the areas of corporate finance, capital markets and investment financing spanning over 24 years. Prior positions include: Area Manager for BMO Bank of Montreal; TD Bank Residential Mortgage Manager; TD Bank Senior Financial Advisor; Chief Financial Officer and Vice President of Corporate Finance, Shenzhen Hepalink Biopharmaceutical Co.; 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 also has Master of Finance Management and a Master of Corporate Economics and Business Administration.

On April 2, 2016, Dr. Peter Johann, Resverlogix's former chairman, stepped down from our Board of Directors.

Private Placements, Prospectus Offering and Licensing Agreement

On July 8, 2015, we formally entered into a definitive stock purchase agreement with Shenzhen Hepalink Pharmaceutical Co., Ltd. ("Hepalink") and closed a license of RVX-208 for China, Hong Kong, Taiwan and Macau (the "Territories"), for all indications with Hepalink. On July 20, 2015, we closed the private placement. Under the terms of the transaction, Hepalink subscribed for 13,270,000 Resverlogix common shares and 1,000,000 common share purchase warrants for gross proceeds of $27.3 million (CAD$35.4 million), or CAD$2.67 per unit. After giving effect to the transaction, Hepalink held approximately 12.63% of Resverlogix's common shares. The common shares and warrants issued to Hepalink are subject to a three year lock-up period.

In addition, Eastern Capital Limited ("Eastern") purchased 5,600,000 common shares and 422,005 common share purchase warrants for gross proceeds of $11.5 million (CAD$15.0 million), or CAD$2.67 per unit. After giving effect to the transaction, Eastern held approximately 19.57% of Resverlogix's common shares.

The license between us and Hepalink provides for certain milestone payments based on net sales of RVX-208 in the licensed territories. The annual sales milestones range from 500 million renminbi ("RMB") to 10 billion RMB (US$73 million to US$1.5 billion), with Resverlogix being eligible to receive sales-based milestone payments from Hepalink ranging from US$5 million to US$90 million. In addition, Hepalink shall pay a royalty of 6% of annual net sales of RVX-208 in the licensed territories. 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 July 31, 2017, these potential payments do not satisfy the criteria for recognition as a liability.

On June 20, 2017, we issued a total of $7.5 million (CAD$10 million) of equity units pursuant to a private placement and prospectus offering. Eastern and Hepalink purchased 1,617,980 and 1,333,333 equity units, respectively at a price of CAD$1.80 per unit pursuant to a private placement for gross proceeds of $4.0 million (CAD$5.3 million). Other subscribers purchased an additional 2,552,489 equity units at a price of CAD$1.80 per unit pursuant to a prospectus offering for gross proceeds of an additional $3.5 million (CAD$4.6 million). Each equity unit consists of one common share and one common share purchase warrant. Each warrant is exercisable at a price of CAD$2.05 per underlying common share for a period of four years from the closing of the private placement and prospectus offering.

As described under "Debt" herein, subsequent to July 31, 2017, the maturity date of our CAD$68.8 million loan was extended from August 28, 2017 to December 26, 2017.

Also, as described under "Liquidity" herein, subsequent to July 31, 2017 we issued $4.2 million (CAD$5.1 million) of equity units pursuant to a private placement.

Royalty Preferred Shares

On July 2, 2015, the Company's articles were amended to make certain changes to the dividend entitlement of holders of royalty preferred shares. The amendments to the Royalty Preferred Shares limit the dividends payable to holders of royalty preferred shares in a particular period to amounts received by us during that period. We determined that this amendment was necessary in the course of negotiating the terms of the license agreement with Hepalink.

On December 20, 2016, the Company's articles were further amended to make certain additional changes to the dividend entitlement of holders of royalty preferred shares. The amendments provided that the holder of royalty preferred shares is entitled to a dividend, calculated based on a percentage of net revenue earned from the sale of licensing of any pharmaceutical product in which Resverlogix holds an intellectual property right, and removed the requirement that the pharmaceutical product elevate plasma levels of certain lipoprotein associated with a decreased risk of atherosclerosis and coronary heart disease. We determined that the amendments were necessary and appropriate based on detailed analysis of the results of our phase 2 clinical program.

Zenith Royalty

During the year ended April 30, 2016, we entered into a letter of understanding with Zenith in connection with a proposal that Zenith grant royalty rights to us related to some or all of Zenith's intellectual property, we paid Zenith $2.3 million. The letter of understanding prescribed that in the event that a transaction does not close, any consideration paid by us to Zenith in connection with the transaction would remain payable by Zenith to us. On May 18, 2016, the letter of understanding was terminated, as Zenith decided to pursue other alternatives, and therefore, the $2.3 million was repaid by Zenith.

Results of Operations for the Three Months Ended July 31, 2017 and 2016

(in thousands of US dollars unless otherwise noted)	2017	2016
Expenses	$ 8,909	$ 7,114
Financing costs (income)	6,438	(666)
Loss before income taxes	15,347	6,448
Income taxes	9	31
Net and total comprehensive loss	15,356	6,479
Net loss per share		
Basic and diluted	$ 0.14	$ 0.06

Cash Burn Rate

The average monthly Cash Burn Rate, a non-IFRS measure as described on page 2 herein, for the three months ended July 31, 2017 was $2.9 million (2016 - $2.6 million). The increase was primarily attributable to the advancement of the BETonMACE Phase 3 clinical trial.

	Three months ended July 31,	
(in thousands of US dollars unless otherwise noted)	2017	2016
Cash flow used in operations	$ 8,141	$ 2,904
Changes in non-cash working capital	665	4,827
	8,806	7,731
Number of months	3	3
Average Monthly Cash Burn Rate	2,935	2,577

Our historical Cash Burn Rate is not necessarily indicative of our future Cash Burn Rate. Our burn rate has increased, reflecting the Phase 3 BETonMACE clinical trial progressing. Based on our planned business operations for the next year, we expect our Cash Burn Rate to continue to fluctuate each quarter for the duration of the trial based on the specific activities, most notably enrollment rates, occurring in each quarter.

Research and Development

In addition to the costs associated directly and indirectly with clinical programs, research and development includes other product development costs such as drug development and manufacturing, pharmacology, toxicology and other studies, and costs associated with discovery research. R&D expenses also include salaries and benefits for R&D staff, consulting fees, supplies and general laboratory operating expenses.

During the three months ended July 31, 2017, gross R&D expenditures totaled $8.1 million (2016 - $6.2 million). Clinical costs totaled approximately $4.3 million (2016 - $4.1 million), including $3.8 million on the BETonMACE clinical trial net of cost recoveries, reflecting the continued progression and expansion of the trial and $0.2 million on the Renal Dialysis clinical trial (2016 - $3.7 million on the BETonMACE clinical trial and $0.2 million on the Renal PK trial), $0.1 million on regulatory costs (primarily related to the BETonMACE clinical trial) (2016 - $0.1 million) and $0.2 million (2016 - $0.1 million) of other clinical costs including sample analysis, consultants and insurance. BETonMACE costs included those related to country selection, investigative site evaluation; central lab start-up, set-up of electronic systems, training, site initiation visits, and patient recruitment.

During the three months ended July 31, 2017, chemistry costs (comprised of CMC, or chemistry, manufacturing and controls) totaled approximately $2.3 million (2016 - $0.8 million). The increase was due to the shipment of clinical supplies to sites for the BETonMACE clinical trial.

During the three months ended July 31, 2017, preclinical costs were approximately $0.6 million (2016 - $0.5 million). Preclinical costs include research, pharmacology, toxicology and DMPK (drug metabolism, and pharmacokinetics). The increase was attributable to various small studies related in part to the potential broadening of additional indications.

Research and development compensation and related costs (related primarily to our research, preclinical and clinical teams), for the three months ended July 31, 2017 were $0.6 million (2016 - $0.5 million). The increase was due to the expansion of clinical staff for the oversight of the BETonMACE clinical trial and lower compensation-related recoveries from Zenith in the current period.

General and Administrative

General and administrative expenses includes compensation and related costs, operating costs not directly involved in research and development, as well as professional fees for legal, audit, communications, medical affairs and business development services.

During the three months ended July 31, 2017, general and administrative expenditures decreased to $0.9 million (2016 - $1.0 million). The decrease was due to a reduction in share-based payments corresponding to a forefeiture of certain stock options in the current period.

Share-based Payments

Share-based payments and depreciation and amortization are included in research and development and general and administrative rather than being presented separately in the statements of comprehensive loss (income).

During the three months ended July 31, 2017, we recognized share-based payments of $0.1 million (2016 - $0.3 million). The expense recognized in a given period reflects the fair value of past and newly-granted stock options outstanding during the period, and is impacted by factors such as vesting and fluctuations in share price. Share-based payments are a non-cash expense which does not impact operating cash flows.

During the three months ended July 31, 2017, we granted nil stock options (2016 – 688,800 stock options with a weighted average exercise price of CAD$1.33 and a weighted average fair value of $0.91), and we granted nil restricted stock units (2016 – 405,000).

Change in Fair Value of Warrant Liability

We have issued warrants in connection with various securities offerings. Warrants issued as part of an equity unit, or in connection with a debt financing, with an exercise price denominated in a foreign currency are reported as a liability until they are exercised or expire. These warrants are adjusted to fair value at each reporting period and any change in fair value between reporting periods is recorded in the statement of comprehensive loss.

During the three months ended July 31, 2017, we recognized a $2.1 million gain on the change in the fair value of our warrant liability (2016 - $3.3 million gain). The changes in fair value were based on several factors including changes in the market price of our shares to CAD$1.36 on July 31, 2017 from CAD$1.99 on April 30, 2017, and to CAD $1.19 on July 31, 2016 from CAD$1.45 on April 30, 2016, the issuance of 5.5 million new liability classified warrants in the current period, as well as decreases in the remaining terms of the various series of warrants, and changes in estimated future volatility of our common shares. Gains and losses resulting from the revaluation of warrant liability are non-cash and do not impact our cash flows.

Change in Fair Value of Royalty Preferred Shares

During the three months ended July 31, 2017, we recognized a $1.5 million loss on the change in the fair value of our royalty preferred shares (2016 - $1.8 million loss). For fair value measurement purposes, the royalty preferred shares liability has been categorized within level 3 of the fair value measurement hierarchy. The fair value of the royalty preferred shares is based on management's judgments, estimates and assumptions which include significant unobservable inputs including the timing and amounts of the Company's discounted risk adjusted future net cash flows, which incorporate: a cumulative probability rate of generating forecasted future cash flows of 35% as at July 31, 2017 and April 30, 2017 (July 31, 2016 and April 30, 2016 – 35%) reflecting in each case, among other factors, our clinical results and communication with regulatory bodies; a discount rate of 23.6% as at July 31, 2017 and 23.4% as at April 30, 2017 (23.1% as at July 31, 2016 and 23.5% as at April 30, 2016); commencement of revenue in between 2021 and 2023, based on clinical development paths across various jurisdictions, as at July 31, 2017 and April 30, 2017 (July 31, 2016 and April 30, 2016 - between 2021 and 2023); projected apabetalone market shares percentages; and projected product pricing. The passage of time during the three months ended July 31, 2017 to future cash flows based on the estimated timing and commencement of revenue, as well as the change in the risk-free rate of return, affected the fair value of our royalty preferred shares.

The fair value of the royalty preferred shares is subject to significant volatility. Small changes in the aforementioned assumptions may have a significant impact on the fair value of the royalty preferred shares. For instance, holding all other assumptions constant, a 1% increase in the discount rate would result in a $3.9 million decrease in the fair value of the royalty preferred shares. Assuming commencement of revenue one year later would result in a $3.6 million decrease in the fair value of the royalty preferred shares.

Interest and Accretion

During the three months ended July 31, 2017, interest on our loan totaled $0.6 million (2016 - $0.5 million); changes in interest rates during the periods were offset by changes in USD/CAD exchange rates during the periods. Accretion (of the discount on the debt and debt issuance costs) totaled $1.4 million (2016 - $1.2 million). Interest on the loan is payable annually in arrears and the interest rate is reset annually to a rate equal to Canadian one-year LIBOR swap rate plus 3.14%. Effective August 27, 2016, the annual interest rate was reset from 3.7643% to 4.0560%; effective August 27, 2015, the annual interest rate was reset from 4.4410% to 3.7643%. The discount on the debt and debt issuance costs are accreted over the term of the loan using the effective interest method.

Liquidity and Capital Resources

Cash

As at July 31, 2017, we had $0.4 million of cash, $12.7 million of trade and other payables, and $2.2 million of accrued interest. Our cash and liquidity is described further under “Liquidity”.

Debt

Subsequent to July 31, 2017, the maturity date of our CAD$68.8 million loan was extended from August 28, 2017 to December 26, 2017.

The entire loan may be repaid prior to maturity in whole or in part without penalty. We do not currently have the funds to repay the loan. In connection with the loan extension, the loan was assigned from Citibank to a lender affiliated, directly or indirectly with Eastern Capital Limited (“the Lender”). The loan is secured by an irrevocable CAD$68.8 million Standby Letter of Credit (the “Letter of Credit”) in favour of the Lender arranged by Eastern Capital Limited (“Eastern”), which will be maintained until maturity of the loan. In connection with the extension of the loan, we granted to the Lender a security interest in all of our assets as security for the loan.

Interest on the loan shall continue to accrue, but shall not be payable until the new maturity date. Effective August 27, 2017, the annual interest rate was reset from 4.0560% to 4.6046%, and the fee payable to Eastern on the undrawn amount of the Letter of Credit increased from 0.03 percent to 12 percent per annum.

Liquidity

We are a development stage company; our primary capital requirements relate to funding research and development activities, including preclinical and clinical trials, and for general working capital purposes. Our operations have been financed in recent years primarily through the sale of common shares or units (consisting of common shares and warrants) and the Citibank loan.

Our primary objective when managing capital is to ensure we have sufficient funds available to carry out our research, development and commercialization programs based, in part, on continuous monitoring.

As at July 31, 2017, we had $0.4 million of cash, $12.7 million of trade and other payables, $2.2 million of accrued interest and were committed to pay $8.2 million for research and development and $0.7 million of lease obligations over the following twelve months. In addition, aggregate expenditures over the next twelve months under cancellable agreements with contract research organizations and central laboratories conducting the BETonMACE and other trials are estimated to total approximately $20 - 25 million. Our CAD$68.8 million (US$55.2 million) loan is repayable upon maturity on December 26, 2017, at which time we are also committed to pay interest and fees related to the loan which would total CAD$6.7 million if paid upon maturity of the loan on December 26, 2017. Our average monthly Cash Burn Rate, a non-IFRS measure, as described on page 16 herein, for the three months ended July 31, 2017 was $2.9 million. Our historical Cash Burn Rate is not indicative of our future Cash Burn Rate. Our cash burn rate has increased in recent quarters, reflecting the BETonMACE trial progressing. Based on our planned business operations for the next year which reflects the BETonMACE trial, we expect our Cash Burn Rate to fluctuate each quarter for the duration of the trial based on the specific activities, most notably enrollment rates, occurring in each quarter.

Our cash as at July 31, 2017 will be insufficient to fund our contractual commitments for the next year and our planned business operations over the next year based on anticipated patient enrollment for BETonMACE, or to repay our loan. We will have to raise additional capital. If we are not able to raise capital, we will have to reduce our cash requirements by eliminating or deferring spending on research, development and corporate activities.

Subsequent to July 31, 2017 we issued $4.2 million (CAD$5.1 million) of equity units pursuant to a private placement. Each unit is comprised of one common share and one-half common share purchase warrant. Each warrant is exercisable at a price of CAD$1.50 per share for a period of 4 years from the closing of the private placement.

We recently received a letter of intent regarding a significant potential regional licensing arrangement and/or a significant equity investment in Resverlogix. We are also pursuing or examining both non-dilutive and dilutive arrangements, with a preference for non-dilutive alternatives, in the following priority: co-development, licensing, rights (on indications or potential follow-on compounds, for instance) or other partnering arrangements, private placements and/or public offerings (equity and/or debt).

These conditions result in a material uncertainty which may cast 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.

We intend to perform additional human clinical trials, including a severe renal impairment trial, and such trials and regulatory approvals likely require several years to complete. We may not generate operating cash inflows in the foreseeable future, and we will require additional financial resources to ensure that we have sufficient capital to fund our long-term research, development and corporate activities. Our long-term capital requirements will depend on, among other considerations, whether we commence additional clinical trials, the size of any trials, and whether the trials are funded entirely by us or, partially or entirely, by a strategic partner.

We continuously investigate and assess financing alternatives and expect to be able to raise additional capital to fund our capital requirements. However, there is no assurance that initiatives to raise additional capital will be successful. If we are unable to raise additional capital, we may need to defer or discontinue some or all of our research and development activities.

During the three months ended July 31, 2017, all of our treasury funds were invested in high interest deposit accounts.

Cash Flows Used In Operating Activities

Cash flows used in operating activities for the three months ended July 31, 2017 totaled $8.1 million (2016 - $2.9 million), reflecting increased research and development costs in the current period and changes in various components of our working capital.

Cash Flows from Financing Activities

As described under “Corporate Developments” during the three months ended July 31, 2017 we issued a total of $7.5 million (CAD$ 10 million) of equity units pursuant to a private placement and prospectus offering. Each unit consisted of one common share and one share purchse 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 private placement and prospectus offering.

During the three months ended July 31, 2017, 59,099 stock options were exercised for proceeds of CAD$0.1 million (2016 – no stock options were exercised).

Cash Flows Used In Investing Activities

During the three months ended July 31, 2017, additions to intangible assets (patent-related costs) and property and equipment, alongside net payments of past additions, totalled $0.1 million (2016 - $0.3 million).

Contractual Obligations

As at July 31, 2017, the Group is party to cancellable agreements with contract research organizations and central laboratories conducting the BETonMACE and other trials. Corresponding estimated aggregate expenditures over the next twelve months total approximately $20 - 25 million (2016 – $15 - 20 million).

As at July 31, 2017, the Group is also committed to expenditures over the next twelve months of $8.2 million (2016 – $2.4 million) under various research and development contracts.

The table below summarizes our contractual obligations related to operating leases for office and laboratory premises, by due date, as at July 31:

(in thousands of US dollars)	2017	2016
Less than one year	$ 701	$ 835
Between one year and five years	2,495	2,702
More than five years	562	941
	3,758	4,478

Zenith has agreed to pay us for its proportionate share of operating lease payments and operating costs for office and laboratory premises of an estimated $0.3 million and $0.1 million, respectively, for the next twelve months.

Significant Accounting Policies and Estimates

Note 4 to our consolidated financial statements for the year ended April 30, 2017 includes a summary of our significant accounting policies.

The preparation of financial statements requires management to use estimates and assumptions that they believe are reasonable based upon the information available. These estimates and assumptions affect the reported amounts of assets and liabilities at the date of the financial statements and the reported amounts of revenues and expenses during the reporting periods presented. These estimates and assumptions are subject to inherent risk of uncertainty and actual results may differ from these estimates and assumptions.

Significant estimates are used for, but not limited to, the measurement of the fair value of long-term debt and the fair value of the royalty preferred shares, share-based payment transactions, warrant liability and taxes.

Off-Balance Sheet Arrangements

As of July 31, 2017, we have not entered into any off-balance sheet arrangements, other than operating leases.

Summary of Quarterly Results

The following is a summary of selected financial information derived from our unaudited condensed interim consolidated financial statements for each of the eight most recently completed quarters.

(in thousands of US dollars except as otherwise noted)	For the Three Months Ended			
	July 31, 2017	April 30, 2017	January 31, 2017	October 31, 2016
Revenue	**-**	-	-	-
Total comprehensive (loss)	**(15,356)**	(11,587)	(12,979)	(15,165)
Net (loss) per shares ($)	**(0.14)**	(0.11)	(0.12)	(0.14)

(in thousands of US dollars except as otherwise noted)	For the Three Months Ended			
	July 31, 2016	April 30, 2016	January 31, 2016	October 31, 2015
Revenue	-	-	-	-
Total comprehensive (loss)	(6,479)	(9,678)	(8,875)	(3,805)
Net (loss) per shares ($)	(0.06)	(0.09)	(0.08)	(0.04)

Items that impact the comparability of quarterly results of operations include:

- Research and development was impacted by the particular stage of our various clinical trials during each particular quarter.
- Research and development was also impacted by the timing of costs related to our chemistry and preclinical studies.
- Liability classified warrants issued pursuant to unit offerings with an exercise price denominated in a currency other than an entity's functional currency are remeasured to reflect the change in fair value as at the end of the reporting period, with changes in fair value recognized in the statement of comprehensive loss, resulting in volatility in quarterly income (loss).
- Royalty preferred shares are remeasured to reflect the change in fair value at the end of the reporting period, with changes in fair value recognized in the statement of comprehensive loss, resulting in volatility in quarterly income (loss).
- Share-based payments fluctuate from quarter to quarter based on the timing and fair value of stock option grants. Share-based payments are a non-cash expense.
- The recognition of foreign currency gains and losses resulting from fluctuations in Canadian denominated assets and liabilities (including our Canadian-denominated debt) and Canadian / US dollar exchange rates.

Related Party Transactions

Pursuant to a Management Services Agreement dated June 3, 2013 between us and Zenith, Zenith engaged us to perform management and administrative services pertaining to Zenith as required. Zenith pays us a fee based on the cost of our personnel and the proportionate time worked on behalf of Zenith. We will also be reimbursed for general and administrative costs.

Effective January 1, 2015 we entered into a Services Agreement whereby Zenith supplies research services to us.

A description of related party transactions (specifically compensation expenses paid to key management personnel, including directors, whom are considered related parties under IFRS) can be found under "Related Party Transactions" in the MD&A for the year ended April 30, 2017. As at July 31, 2017, the transactions with related parties have not changed significantly from these descriptions.

Outstanding Equity Instruments

As at September 11, 2017, we had authorized an unlimited number of common shares and preferred shares and 75,202,620 royalty preferred shares.

	As at September 11, 2017		As at July 31, 2017	As at July 31, 2016
Common Shares	**114,624,089**		111,205,345	105,207,816
Warrants	**14,893,411**		14,504,039	9,000,237
Equity Classified Warrants	**510,424**		510,424	331,750
Stock Options	**2,944,200**	(1)	2,944,200	3,353,433
Restricted Stock Units	**534,179**	(2)	534,179	981,263
Total	**133,506,303**		129,698,187	118,874,499
Royalty Preferred Shares	**75,202,620**		75,202,620	75,202,620

(1) 2,361,878 of 2,944,200 stock options are vested and exercisable
(2) 288,421 of the 534,179 restricted stock units are vested

Additional information relating to our securities can be found in Note 7 to the condensed interim consolidated financial statements for the three months ended July 31, 2017.

Disclosure Controls and Procedures and Internal Controls Over Financial Reporting

During the three months ended July 31, 2017, there were no changes in our internal controls over financial reporting that materially affected or are reasonably likely to materially affect the effectiveness of our internal controls over financial reporting.

Outlook

During November 2015, we commenced enrollment and dosing patients in the BETonMACE trial, a Phase 3 clinical trial in high risk CVD patients with diabetes. In excess of 75% of the approximately 2,400 patients outlined in the study's protocol have been enrolled. On July 25, 2017, we announced the receipt of a positive Type C written response from the Division of Metabolism and Endocrinology Products of the FDA. In light of updated information regarding apabetalone, including: human exposure, clinical dosing and established acceptable safety margins, the FDA is allowing us to include U.S. patients in Phase 3 studies, including the global Phase 3 BETonMACE trial. We will make requested adjustments to the existing BETonMACE study protocol and to update the Investigator's Brochure and the Informed Consent Documents. As discussed under "Clinical Trial Developments" herein, the primary endpoint of the BETonMACE trial is designed to demonstrate a relative risk reduction of MACE. Treatment will continue for up to 104 weeks. The study is an event-based trial and will continue until at least 250 MACE events, defined as CV death, non-fatal MI and stroke have occurred.

On May 15, 2017, we announcd the acceptance, by the Cardiovascular and Renal Products Division of the U.S. FDA, of our IND application to commence a Phase 2a kidney dialysis trial. The primary objective of the study will be to evaluate if treatment with apabetalone in addition to SoC decreases ALP in comparison to placebo and SoC.

On May 30, 2017, we announced that we had received approval from the TPD to proceed with a clinical trial apabetalone in patients with Fabry disease.

We have continued to explore the biology surrounding BET inhibition. Using a variety of cells, cell-based models and cell lines, as well as our archived clinical trial blood samples, we have identified numerous pathways and genes that are regulated in response to BET inhibition. From this effort we have been able to explore the biological processes underlying CVD and MACE as well as have identified several potential indications based on the data observed. Peer reviewed journal publications highlighting these novel findings will be submitted during the upcoming year.

We continue to explore the preferential binding of apabetalone to the BD2 of BET family members. Using microarray data of whole blood treated ex vivo with various concentrations of apabetalone, a BD2 specific gene signature is being explored. This signature differentiates apabetalone from other BET inhibitors in pre-clinical development and may aid in the discovery of follow-on compounds in indications other than high risk vascular disease.

We continue to explore additional indications. Mechanistic evidence through analysis of in vitro and clinical data suggests that BET inhibition via apabetalone suppresses pathways and reduces protein levels associated with vascular calcification. We believe that

apabetalone and its ability to modulate pathways involved in vascular calcification has the potential to beneficially impact renal patients. With leading experts on our Renal Clinical Advisory Board ("RCAB") providing input and guidance, a pharmacokinetic clinical study in this therapeutic area was initiated (as outlined in the "Clinical Trial Developments" section above). The primary objective of the Phase 1 study was met, allowing us to proceed with more advanced renal impairment and dialysis trials. After having met with the FDA, we intend to file an official IND application and proceed in 2017 with the planned Phase 2a pharmacokinetic and efficacy / safety clinical trial in patients with end-stage renal disease hemodialysis.

In addition to apabetalone, we have commenced preclinical testing on other BET inhibitors in our library of compounds which demonstrate similar efficacy and potency on important markers of vascular inflammation, acute phase response, complement and coagulation. These compounds will be further analyzed and explored for their potential use in orphan disease indications.

Epidemiological and mechanistic evidence indicate a link between low ApoA-I/HDL, complement overactivation and neurodegenerative diseases. Based on apabetalone's ability to raise plasma ApoA-I/HDL by ApoA-I production and modulate the complement cascade we believe apabetalone has the potential to beneficially impact various neurodegenerative diseases. With leading experts on our neurodegenerative clinical and scientific advisory board providing input and guidance, we continue to consider conducting a clinical trial in this therapeutic area.

Subsequent to July 31, 2017, the maturity date of our loan was extended by four months to December 26, 2017, and we completed a private placement for gross proceeds of $4.2 million (CAD$5.1 million).

We recently received a letter of intent regarding a significant potential regional licensing arrangement and/or a significant equity investment in Resverlogix. We continue discussions for regional licensing opportunities with potential pharma partners with the goal of expanding the global development of apabetalone in high risk disease with a high level of unmet medical need.

Risks and Uncertainties

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

Risks Relating to Our Business

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

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

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

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

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

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

To date, we have not recorded any revenues from the sale of biopharmaceutical products, and have incurred significant negative cash flows in many periods since our inception. As at July 31, 2017, we had a deficit of US$331.2 million. We expect to incur substantial net losses and negative cash flow for the foreseeable future. Such losses and negative cash flow have had, and will continue to have, an adverse effect on our shareholders' equity and working capital.

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

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

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

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

Our cash as at July 31, 2017, as well as the $4.2 million (CAD$5.1 million) raised subsequently, will be insufficient to fund our contractual commitments for the next year and our planned business operations over the next year based on anticipated patient enrollment for BETonMACE, or to repay our loan. We will have to raise additional capital. If we are not able to raise sufficient capital to repay our debt and fund our operations, or are unable to further extend or replace our debt, we would also have to reduce our cash requirements by eliminating or deferring spending on research, development and corporate activities. These conditions result in a material uncertainty which casts significant doubt on our ability to continue as a going concern.

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

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

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

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

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

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

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

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

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

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

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

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

To obtain regulatory approval to market and sell any of our products, we must satisfy the 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 unfavorable pharmacology, toxicology or carcinogenicity;
- our product candidates may cause undesirable side effects; and
- the supply or quality of our drugs or other materials necessary to conduct clinical trials may be insufficient, inadequate or delayed.

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

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

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

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

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

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

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

commercialization of our products, including RVX-208, may be extended, delayed or terminated or may need to be repeated, and we may not be able to obtain regulatory approval.

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

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

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

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

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

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

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

As a result of the costs and risks associated with commercializing a product candidate, we will seek strategic partnerships in order to continue to develop and, if approved, market our products. Such strategic partnerships may require us to relinquish control over the timing and manner of clinical trials and commercialization of our product candidates. Strategic partners may experience financial difficulties or choose to terminate the arrangement or independently work on a competing product resulting in the delay or discontinuation of development or commercialization of our product candidates. Furthermore, disputes may arise between us and our strategic partners that result in the delay or termination of the research, development or commercialization of our product candidates or that result in costly litigation or arbitration that diverts management's attention and consumes resources. Strategic

partners may not properly maintain or defend our intellectual property rights or may use our proprietary information in a manner that could jeopardize or invalidate our proprietary information or expose us to potential litigation.

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

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

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

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

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

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

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

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

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

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

encounter delays or rejections based on varying regulatory interpretations or changes in regulatory agency policies, during the period in which we develop a product.

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

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

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

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

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

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

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

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

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

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

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

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

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

We anticipate that, if approved for neurodegenerative disorders, apabetalone would potentially be used in conjunction with standard of care therapies such as Aricept to improve therapeutic outcomes and/or compete with other agents and novel approaches to this disease such as small molecules, Namenda and PBT2, and monoclonal antibody technologies ("MOABs") such as Bapineuzumab.

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

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

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

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

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

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

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

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

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

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

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

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

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

Variations in interest rates could adversely affect our financial condition.

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

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

We were formed under the laws of the Province of Alberta. Some of the members of our board of directors and our officers are residents of countries other than the United States. As a result, it may be impossible for U.S. investors to affect service of process within the United States upon us or these persons or to enforce against us or these persons any judgments in civil and commercial

matters, including judgments under U.S. federal or state securities laws. In addition, a Canadian court may not permit U.S. investors to bring an original action in Canada or to enforce in Canada a judgment of a state or federal court in the United States.

Risks Relating to our Intellectual Property

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

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

Our patent positions are highly uncertain, and any future patents we receive for our potential products will be subject to this uncertainty, which may adversely affect our business, financial condition and results of operations. There can be no assurance that pending patent applications will be allowed and that we will develop additional proprietary products that are patentable, that issued patents will provide any competitive advantage or will not be challenged by any third parties, or that patents of others will not have an adverse effect on the ability to do business. Furthermore, there can be no assurance that others will not independently develop similar products, duplicate any of the products, or design around the products patented by us. In addition, we may be required to obtain licenses under patents or other proprietary rights of third parties. No assurance can be given that any licenses required under such patents or proprietary rights will be available on terms acceptable to us. If such licenses are not obtained we could encounter delays in introducing one or more of our products to the market, while we attempt to design around such patents, or could find that the development, manufacturing or sale of products requiring such licenses could be foreclosed. In addition, we could incur substantial costs in defending suits brought against us on such patents or in suits in which we attempt to enforce our own patents against other parties. Such disputes could involve arbitration, litigation or proceedings declared by the 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, including subsequent to our June 27, 2013 announcement concerning our Phase 2b ASSURE clinical trial, and could fluctuate substantially in the future. During the twelve months preceding July 31, 2017, the closing market price of our common shares ranged from CAD$1.15 to CAD$2.47 per share. In addition, the trading prices of life science and biotechnology company stocks in general have experienced extreme price fluctuations in recent years. The valuations of many life science companies without consistent product revenues and earnings are high based on conventional valuation standards, such as price-to-revenue ratios. These trading prices and valuations may not be sustained. Any negative change in the public's perception of the prospects of life science or biotechnology companies could depress our stock price regardless of our results of operations. In addition our stock may fluctuate based on a variety of factors, including actual or anticipated regulatory approvals or disapprovals of our products or competing products, actual or anticipated results and timing of our clinical trials, changes in the expected or actual timing of our development programs, changes in our operating results, conditions or trends in the life science and biotechnology industries, announcements by us or our competitors of significant acquisitions, strategic partnerships, joint ventures or capital commitments, additions or departures of key personnel, sales and distributions of our common shares by us or our shareholders, changes in general conditions in the economy or other developments affecting us, our clients, or our competitors, some of which may be unrelated to our performance.

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

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

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

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

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

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

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

Additional Information

Additional information relating to Resverlogix, including our Annual Information Form, can also be found on SEDAR at www.sedar.com.