William Blair

ZS Pharma, Inc.

A K+(O) in the Large Market of Hyperkalemia, Initiating Coverage With Outperform Rating and $75 Price Target

ZS Pharma is a specialty pharmaceutical company developing lead product ZS-9, which is in a broad Phase III program for the treatment of hyperkalemia, a condition in which there are elevated levels of potassium in a patient's blood. Hyperkalemia affects a large patient population with limited treatment options. Elevated potassium levels are observed in many disease states, including congestive heart failure, chronic kidney disease, and diabetes, and it is often observed as a side effect in patients on renin-angiotensin-aldosterone system (RAAS) inhibitor therapy. RAAS inhibitor treatment is one of the largest classes of therapies, prescribed to roughly 80 million people in the United States.

Given the well-documented prevalence and growing patient populations within these disease categories, the management of hyperkalemia has the potential to become a significant market. There is an acute need for the management of hyperkalemia and a growing awareness for the chronic management of potassium levels in various at-risk patient populations. To date, ZS Pharma has completed a 14-day Phase III clinical trial (ZS003) of ZS-9 in 753 patients with elevated potassium levels regardless of etiology. Given this data, ZS-9 looks to be a best-in-class product for the management of elevated potassium levels. Response rates of ZS-9 have approached 100% with a relatively benign side effect profile. ZS Pharma is set to report results from the company's second Phase III study, ZS004, late in the third quarter or early in the fourth quarter. Given the consistency of data in the ZS003 study, we believe ZS004 should reinforce a best-in-class profile for ZS-9 with high response rates and a reduced side effect profile versus competitive agents in development.

We estimate roughly 3.6 million patients may be currently addressed with an effective and safe therapy for hyperkalemia, and there are a greater number of patients not adequately treated with RAAS inhibitors due to the risk of becoming hyperkalemic. Following an approval in 2016, we anticipate peak sales for ZS-9 of $1.17 billion by penetrating 10% to 13% of the available patient populations within select markets. We are initiating coverage with a $75 price target, derived from our net-present-value model for ZS-9 and applying a 75% probability of success. Swing factors in our peak-year estimates include patient duration, which we estimate will reach six months; however, if ZS Pharma is successful in penetrating the chronic therapy market, this duration might hold upside. Given the many players marketing therapies for congestive heart failure, chronic kidney disease, and RAAS inhibitors, we believe ZS Pharma will likely attract significant interest from strategic partners if the development of ZS-9 is successful. Risks to an investment in ZS Pharma include the normal clinical, regulatory, and commercial risks in development-stage therapeutics companies.

ZS Pharma is a specialty pharmaceutical company located in San Mateo, California, focused on developing therapies based on highly selective ion trap chemistry.

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Basic Report (14-090)

Stock Rating:	**Outperform**
Company Profile:	**Aggressive Growth**
Price Target:	$75
Symbol:	ZSPH (NASDAQ)
Price:	$29.49 (52-Wk.: $26–$31)
Market Value (mil.):	$581.5
Fiscal Year End:	December

Estimates	**2013A**	**2014E**	**2015E**
EPS FY	$-8.52	$-0.78	$-1.08
EBITDA (mil.)	-32.2	-22.7	-29.0

Valuation			
P/E	NM	NM	NM

Trading Data	
Shares Outstanding (mil.)	19.7
Float	42%
Average Daily Volume	352,577

Financial Data	
Long-Term Debt/Total Capital	NA
Book Value Per Share	NM
Enterprise Value (mil.)	$424
EBITDA (mil.)	$-32.2
Enterprise Value/EBITDA	NM

Please refer to important disclosures on pages 34 and 35. Analyst certification is on page 34.
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Company Overview

ZS Pharma, Inc. is a biopharmaceutical company that develops and commercializes insoluble, unabsorbable drugs to treat renal, metabolic, and cardiovascular diseases. Its lead pipeline candidate, ZS-9, is a proprietary zirconium silicate–based selective trap for potassium ions. To date, ZS-9 has shown to be effective in reducing circulating blood levels of potassium without affecting the balance of other electrolytes. ZS Pharma is developing ZS-9 for the treatment of hyperkalemia and maintenance of normokalemia in chronic disease states. Hyperkalemia is a life-threatening condition wherein elevated levels of potassium can increase the risk of cardiac arrest and death.

In the human body, potassium is a cation that is responsible for maintaining a concentration gradient across the cell membrane. Potassium homeostasis (or the stable levels of potassium) is critical to preventing adverse events in patients with cardiovascular and renal diseases. The physicians' consensus on extracellular fluid potassium concentrations are in the 4.0 to 5.0 mEq/L range. Going above this range in plasma potassium concentration or above 5.0 mEq/L serum potassium concentration is diagnosed as hyperkalemia. The presence of hyperkalemia is significant in populations with cardiovascular and renal dysfunction (i.e., chronic kidney disease, heart failure, and diabetes) and can result in cardiac arrhythmias and sudden cardiac death.

The company's management team has experience in pharmaceutical early- and late-stage development, regulatory affairs, and commercialization. It plans to submit a new drug application (NDA) in the United States and marketing authorization application (MAA) in Europe in the first half of 2015; if approved, it will commercialize ZS-9 for the treatment of hyperkalemia in the United States with its own specialty salesforce. In addition, the company plans to partner for sales outside the United States. To date, ZS Pharma has completed two clinical studies with ZS-9 in 843 patients with hyperkalemia that met its primary and secondary efficacy endpoints with clinically significant results. The company is completing its second Phase III study for ZS-9 in hyperkalemia with results expected to read out by the end of 2014, as early as September. In addition, ZS Pharma began enrolling an ongoing open-label safety study in second quarter 2014, which will produce longer-term data to supplement the company's NDA submission.

Exhibit 1
ZS Pharma, Inc.
Timeline and Events

Date	Product	Event	Description/Comments
2014			
Q2 2014	ZS-9	Clinical	Initiate open-label safety study
H2 2014	ZS-9	Manufacturing	Expected commercial scale manufacturing
Q3 2014	ZS-9	Clinical	ZS004 Phase III data
Q4 2014	Patiromer (Relypsa)	Regulatory	ZS-9 Competitor (Relypsa) NDA filing with FDA
2015			
H1 2015	ZS-9	Regulatory	NDA filing with FDA
H1 2015	ZS-9	Regulatory	MAA filing with EMA

Sources: Company reports and William Blair & Company, L.L.C. estimates

Exhibit 2
ZS Pharma, Inc.
Current Product Pipeline

Drug Candidate	PRE-IND	Phase I	Phase II	Phase III/ Pivotal	NDA/MAA filing	Comments/Timing
ZS-9						For the indication of hyperkalemia, ZS004 readout in Q4 2014
ZS-1						Ammonia-binding, potential indication: hepactic encephalopathy

Source: Company reports and William Blair & Company, L.L.C.

Key Risks

The company's valuation is heavily tied to one product, ZS-9. Similar to many development-stage companies with a key late-stage program, the valuation and share price performance of ZS Pharma are heavily reliant on the successful development, regulatory approval, and commercialization of the company's lead product, ZS-9. Any clinical or regulatory setbacks for the program would weigh heavily on company shares. While the company has treated more than 840 patients to date, ZS Pharma is just now initiating a long-term safety study; the lack of a long-term safety study is a risk.

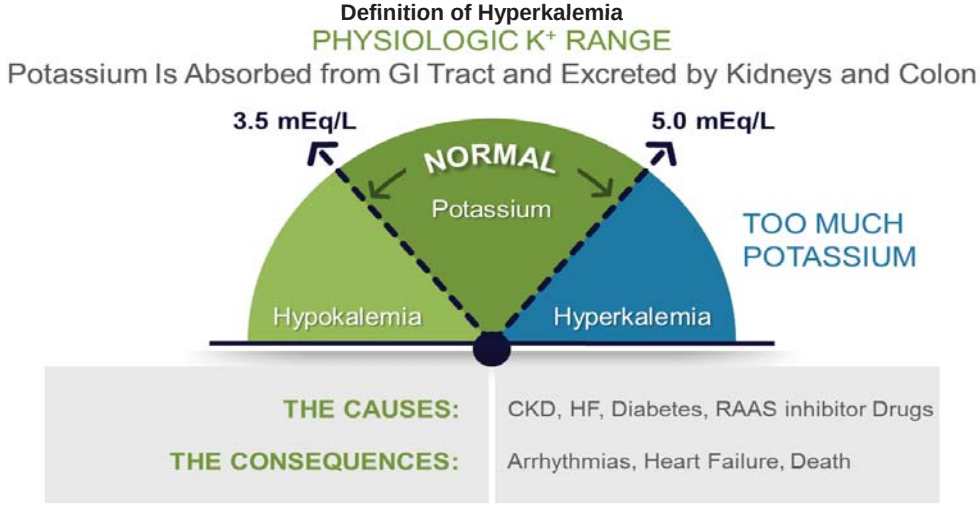
As the company ramps up manufacturing, commercial-scale manufacturing risk remains. The company has successfully manufactured ZS-9 at one-fourth of the anticipated scale, but it has yet to produce commercial scale. It expects to install a 2,000-liter commercial-scale reactor in the near term to develop its ability to internally manufacture ZS-9. While the establishment of facilities in compliance with cGMP (current good manufacturing practice) regulations and maintenance of quality control remain risks, we also believe the control of manufacturing is a key strategic asset. Specifically, we believe the ease in manufacturing and providing ZS-9 through a relatively straightforward supply chain differentiates ZS Pharma from its main competitor, Relypsa.

The company faces the clinical and regulatory risks typical of a development-stage therapeutics company. Although ZS Pharma's studies to date suggest ZS-9 is a safe and effective therapy, the company still needs to complete a second Phase III and a long-term safety study, so clinical risk remains. The company is also on track to file with the FDA during the first half of 2015, and regulatory risk remains. Specifically, ZS Pharma will be using a surrogate endpoint as its primary endpoint (potassium levels), and while we believe this endpoint has been derisked through the special protocol assessment (SPA) obtained by hyperkalemia competitor Relypsa, ZS Pharma conducted its clinical program outside of an SPA. Cardiovascular and Renal Advisory committees have also been a difficult path for drug approvals in recent history so some risk for the class could remain, depending on how the endpoint is viewed during the regulatory process.

Hyperkalemia Background

As shown in exhibit 3, the normal range for serum potassium concentration is between 3.5 mEq/L and 5.0 mEq/L. Potassium concentration can dip above or below the normal ranges in individuals with diseases that affect the glomerular filtration rate (GFR) of the kidneys, the organ responsible for the regulation of ion concentrations, or individuals that are on medication to modulate the RAAS. In hypokalemia, the range is below 3.5 mEq/L, while hyperkalemia is defined as an increased serum potassium concentration above 5.0 mEq/L. Several chronic diseases, such as chronic kidney disease, heart failure, and diabetes, involve a reduction of the GFR whereby the average dietary intake of potassium (120 mEq per day) exceeds the renal clearance of a dysfunctional kidney. In the following section we detail the mechanisms for potassium regulation, chronic diseases associated with potassium imbalance, and clinical outcomes that have shown to be associated with hyperkalemia.

Exhibit 3
ZS Pharma, Inc.
Definition of Hyperkalemia



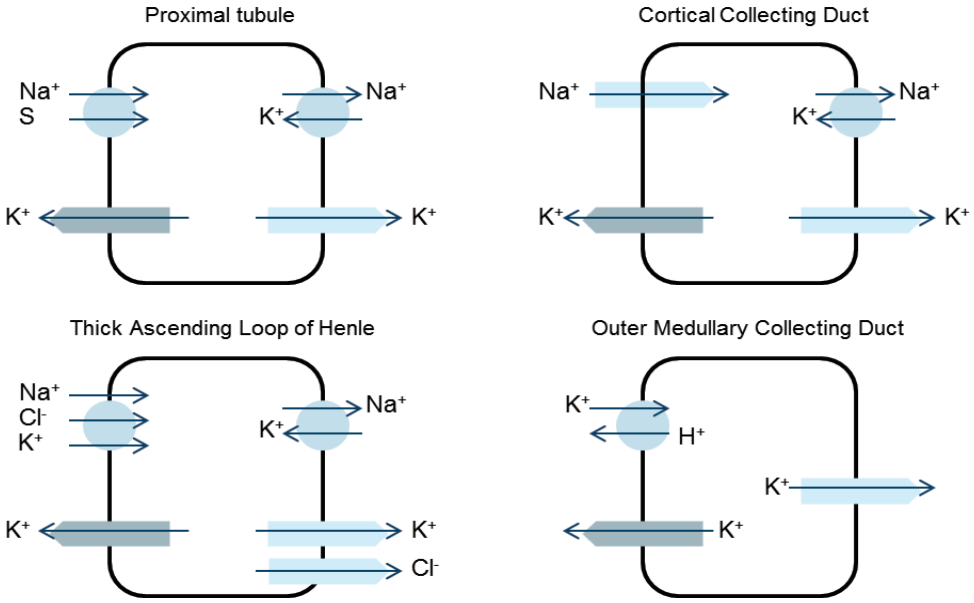
Source: Company reports

Ion Channels Regulate Potassium Homeostasis

To regulate electrolyte levels, the cell membrane has several ion channels. For potassium, the main regulator of ion flux, the Na^+/K^+ ATPase pump, is located in almost all cells and moves three sodium ions out of the cell and two potassium ions inside the cell to maintain a negative electrochemical gradient. Exhibit 4, on the following page, shows four locations in the nephron of potassium channels used to regulate intracellular and extracellular concentrations: the proximal tubule, the thick ascending limb in the loop of Henle, the cortical collecting duct, and the outer medullary collecting duct.

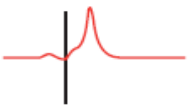
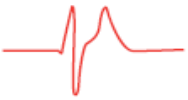
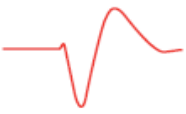
The regulation of ions in the nephrons of the kidney creates a concentration gradient that is responsible for the resting potential of the cellular membrane. When stimulation occurs, voltage-gated Na^+ channels open, which leads to an influx of Na^+ into the cell and membrane depolarization, reaching an action potential. When the action potential reaches a positive value, voltage-gated K^+ channels open, leading to an efflux of K^+ and a restoration of resting potential. This is important when considering the impact of abnormal ion concentrations on muscle firing, particularly in the heart. Exhibit 5 shows the potential electrocardiogram (ECG) abnormalities and appearance that can be seen due to increased serum potassium.

Exhibit 4
ZS Pharma, Inc.
Ion Channels Involving Potassium Concentrations in Nephron



Sources: Hebert et al., *Physiological Reviews* 2005 and William Blair & Company, L.L.C.

Exhibit 5
ZS Pharma, Inc.
Typical ECG Abnormalities Associated With Hyperkalemia

Serum Potassium	Typical ECG Appearance	Possible ECG Abnormalities
Mild (5.5-6.5 mEq/L)		Peaked T waves Prolonged PR segment
Moderate (6.5-8.0 mEq/L)		Loss of P wave Prolonged QRS complex ST-segment elevation Ectopic beats and escape rhythms
Severe (> 8.0 mEq/L)		Progressive widening of QRS complex Sine wave Ventricular fibrillation Asystole Axis deviations Bundle branch blocks Fascicular blocks

Source: McCullough et al. 2014

Physiological Mechanisms of Potassium Excretion and Reabsorption

From a physiological standpoint, potassium regulation primarily takes place in the distal nephron of the kidneys. Potassium is freely filtered in the glomerulus of the nephron and is primarily reabsorbed in the proximal tubule and thick ascending limb of the loop of Henle via the Na^+K^+-$2Cl^-$ co-transporter.

Roughly 10% of the filtered load reaches the distal nephron and cortical collecting duct, where it is excreted. Then the RAAS provides hormonal control of blood pressure and fluid balance. Within the RAAS system the concentration of K^+ is mainly due to aldosterone. Aldosterone is secreted by the adrenal glands and increases K^+ excretion in three ways. One is by increasing the likelihood of sodium reabsorption, thereby increasing potassium excretion. Another way is by promoting Na^+-K^+-ATPase activity to increase intracellular potassium concentration. Lastly, it can increase the membrane permeability to K^+. Insulin has the ability to induce the reabsorption of potassium. Postprandial secretion of insulin is the mechanism by which hyperkalemia is prevented after a meal. Insulin is secreted in response to an increase in blood glucose and subsequently, increases Na^+-K^+-ATPase pump activity to increase intracellular potassium. This is one reason insulin is often given to patients in an acute hyperkalemic state.

Exhibit 6 explains some of the potential causes of hyperkalemia based on dysfunctional physiological mechanisms due to disease and maintenance therapies.

Exhibit 6
ZS Pharma, Inc.
Causes of Hyperkalemia

Possible Causes of Hyperkalemia
Decreased excretion of potassium by kidneys (common in patients with CKD and HF patients)
Imbalances of potassium between extracellular and intracellular fluid compartments (common in diabetes patients)
Use of a number of commonly-used drugs that cause elvated potassium levels (RAAS inhibitors, transplant medicines, nonstreoidal anti-inflammatories)

Source: Company reports and William Blair & Company, L.L.C.

Exhibit 7
ZS Pharma, Inc.
Recommendations for Treatment of Degrees of Hyperkalemia

Degree of Hyperkalemia	Blood Potassium Level (mmol/L)	Recommendations
Mild elevation	5.5-5.9	· Remove potassium from body using potassium exchange resins · Address cause of hyperkalemia to correct and avoid further rise in potassium levels
Moderate elevation	6-6.4 w/o ECG changes	· Shift potassium intracellularly with glucose/insulin · Remove potassium with exchange resins · Consider hemodialysis
Severe elevation	≥6.5 w/o ECG changes	· Shift potassium intracellularly with glucose/insulin · Salbutamol 5 mg nebulized · Sodium bicarbonate if metabolic acidosis is present · Exchange resins
Severe elevation	≥6.5 w/toxic ECG changes	· Protect the heart with calcium chloride · Use glucose/insulin to shift potassium levels · Use resins to remove potassium

Source: Soar et al. 2010

Chronic hyperkalemia is treated based on the level of elevation, through a variety of mechanisms. Exhibit 7, on the previous page, shows recommendations for the treatment of different stages of hyperkalemia (Soar et al., *Resuscitation* 2010).

Potassium Imbalance and Chronic Diseases

Chronic diseases such as chronic kidney disease, congestive heart failure, and diabetes are some of the most-common disorders found in the U.S. population. However, the treatment of these conditions with standard-of-care measures can also result in side effects and secondary complications that must be monitored for the patient's health. Hyperkalemia is one of these secondary effects associated with either physiological dysfunction or treatment modalities and must be monitored due to its well-established correlation with adverse outcomes. Given the size of these patient populations the addressable market for effective therapies in not only treating hyperkalemia but also maintaining normokalemia is significant. The number of patients in each of these disease groups is also expected to increase over the next 10-20 years. We include the estimated patient populations of the largest diseases where abnormal potassium levels are observed in the following exhibit.

Exhibit 8
ZS Pharma, Inc.
Chronic Diseases Associated With Development of Hyperkalemia

Chronic Disease	No. of People Diagnosed (2009)	Projected No. of People Diagnosed (Year)
Chronic Kidney Disease (CKD)	26 million	46 million (2022)
Congestive Heart Failure (CHF)	5.7 million	8 million (2030)
Diabetes	23.4 million	44.1 million (2034)

Sources: Company reports and William Blair & Company, L.L.C.

Exhibit 9
ZS Pharma, Inc.
Selected Patient Characteristics in Goyal et al. 2012 Stratified by Mean Serum Potassium Level Postadmission

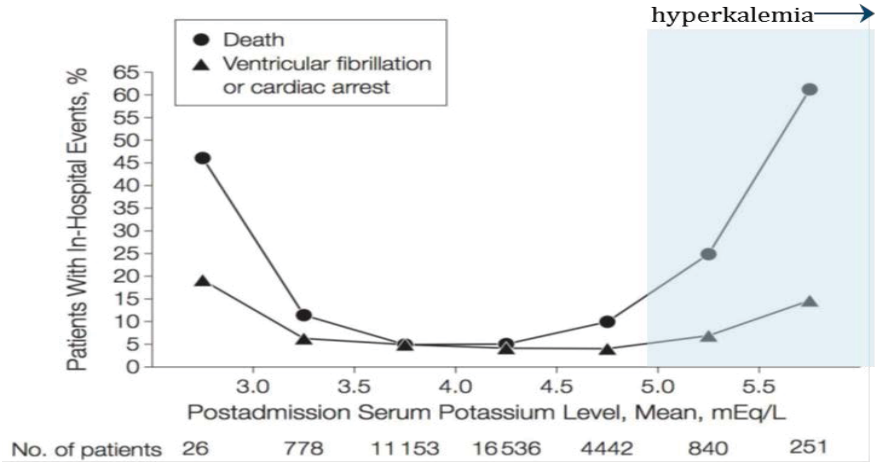
Baseline Characteristic	Postadmission Serum Potassium Level, Mean, mEq/L							
	<3.0	3.0-<3.5	3.5-<4.0	4.0-<4.5	4.5-<5.0	5.0-<5.5	≥5.5	P value
No. of Patients	26	778	11153	16536	4442	840	251	-
% of Patients on ACE Inhibitor or ARB	30.8%	55.9%	63.7%	65.5%	62.7%	46.0%	25.1%	<0.001
Hospital Length of Stay, h	48	89	108	118	125	100	41	<0.001
In-hospital Mortality	46.2%	11.4%	4.8%	5.0%	10.0%	24.8%	61.4%	-
In-hospital Cardiac Events	19.2%	6.3%	4.9%	4.1%	4.1%	6.8%	14.7%	-
Mortality Odds Ratio (Unadjusted)	17	2.38	1 (ref)	1.06	2.27	6.64	32.7	-
Events Odds Ratio (Unadjusted)	5.08	1.3	1 (ref)	0.84	0.85	1.53	3.59	-
Selected Patient History:								
Heart Failure	19.2%	36.6%	32.1%	30.8%	37.9%	46.8%	47.8%	<0.001
Hypertension	34.6%	59.4%	55.6%	53.7%	55.9%	54.4%	45.4%	<0.001
Prior MI	3.8%	5.0%	5.8%	5.8%	6.3%	5.6%	2.0%	0.9
Diabetes	30.8%	27.9%	28.8%	31.3%	39.5%	40.8%	30.3%	<0.001
Dialysis	0.0%	2.1%	1.4%	1.9%	5.4%	10.8%	14.3%	<0.001

Source: Goyal et al. 2012

The correlation between potassium blood levels and poor outcomes in the above patient populations was well defined in a multi-center, retrospective study published in the *Journal of the American Medical Association.* The retrospective study of patients who were admitted into a hospital with acute myocardial infarction (AMI) was conducted in 2012, with more than 38,000 subjects (Goyal et al., *JAMA* 2012). The exhibit above summarizes selected characteristics from the patients analyzed in this study. It is interesting to note that the percentage of patients on an angiotensin converting enzyme (ACE) inhibitor or angiotensin II receptor blocker (ARB) decreases as the serum potassium level increases. This is most likely due to the current standard-of-care to take patients off of these drugs in response to hyperkalemia, which we detail further below. We highlight that in-hospital cardiac evens versus prior myocardial infarctions (MI) increase from 5.6% to 6.8% in the 5.0 mEq/L-<5.5 mEq/L group and 2.0% to 14.7% in the ≥5.5 mEq/L group, the opposite of trends seen in the normokalemic range. Lastly, the mortality and adverse events odds ratios are highlighted as they significantly increase in the hyperkalemic range.

In exhibit 10, we include the results of the study that highlight the increase in death and cardiac events associated with serum potassium levels greater than 4.5 mEq/L, which is still considered in the normal range. Furthermore, the study noted that hyperkalemia patients were 3.2% of the total patient population, but accounted for around 16% of the total deaths. This study has been cited as an indicator of the association of hyperkalemia with adverse outcomes in the disease population with cardiovascular and renal issues.

Exhibit 10
ZS Pharma, Inc.
Relationship Between Cardiac Events and Mean Serum Potassium Levels



Source: Goyal et al. *JAMA* 2012

In addition to the Goyal et al. study, a retrospective study found that hyperkalemia (>5.0 mEq/L) was prevalent in 24.5% of 15,803 patients with cardiovascular disease treated with antihypertensive drugs and 1.7% of total hospital admissions. Furthermore, compared with patients with normokalemia, those with hyperkalemia had a higher percentage of death at 6.25% versus 2.92% (p= 0.0001) and hospital admissions at 7.80% versus 5.04% (p=0.0001) (Jain et al., *Am J Cardiol* 2012).

Chronic Kidney Disease and Hyperkalemia

Patients with chronic kidney disease (CKD) experience a progressive loss in renal function. As stated earlier, kidney function is important in regulating fluid and electrolyte balance. CKD can be caused by diabetes and hypertension, and in turn, can increase the risk of cardiovascular disease. As described above the number of patients with CKD is significant. Based on the National Health

and Nutrition Examination Survey (Nhanes), the number of stage III CKD in patients 60 years and older was roughly 10 million in 2008 and expected to increase given the aging population. Some estimates have the number of patients with CKD in the United States reaching 46 million by 2022.

Clinically, CKD is defined by the U.S. National Kidney Foundation in five stages based on severity of the disease. In patients with advanced CKD, which is associated with a glomerular filtration rate ≤ 30 ml/min/1.73 m^2, the risk of clinically significant hyperkalemia is common. These patients require monitoring, dietary advice, strict avoidance of NSAIDs (nonsteroidal anti-inflammatory drugs), adjustment of the renin-angiotensin-aldosterone system (RAAS) inhibitor dose, and use of loop diuretics. This will facilitate greater exchange of sodium for potassium in the distal nephron.

Roughly 300,000 people in the United States receive some form of dialysis. The NKF-DOQI (National Kidney Foundation's Dialysis Outcomes Quality Initiative) Steering Committee produced guidelines called the Kidney Disease Outcomes Quality Initiative (K/DOQI) to address methodological process and implementation strategies related to kidney disorder. These guidelines involve hemodialysis adequacy, peritoneal dialysis adequacy, vascular access, and treatment of anemia in CKD.

In patients with reduced renal function and particularly a reduced glomerular filtration rate (GFR), RAAS inhibitors are prescribed to delay the progression of renal disease and decrease morbidity and mortality according to KDOQI as well as guidelines from the American Heart Association and American Diabetes Association. Current treatment guidelines advise patients at risk for hyperkalemia (particularly chronic hyperkalemia) to discontinue the RAAS inhibitors that typically provide significant benefits (Palmer BF, *NEJM* 2004). The potential patient population of CKD patients with hyperkalemia in the United States is estimated to be roughly 3 million (company reports and Einhorn et al. *Arch Internal Med* 2009). The ability to reduce potassium levels and continue RAAS inhibitor therapy would most likely be a driving force for ZS-9 and other therapies able to maintain normal potassium levels despite RAAS inhibitor therapy.

Heart Failure and Hyperkalemia

Heart failure is a progressive condition in which heart muscle weakens after an injury (i.e., from a heart attack or high blood pressure) and gradually loses its ability to pump enough blood to supply the rest of the body. According to the Heart Failure Society of America, heart failure affects nearly 5 million Americans; this number could reach 8 million by 2030. Further, an estimated 400,000 to 700,000 new cases of heart failure are diagnosed each year, and the number of deaths in the United States from heart failure has doubled since 1979, to an average of 250,000 annually. The current prognosis for heart failure is that less than 50% of patients are still alive five years after being diagnosed and less than 25% are still alive 10 years after diagnosis.

In patients with heart failure, particularly those with reduced GFR, the risk of hyperkalemia is substantial. The primary approach for reducing potassium levels are dietary changes, avoidance of NSAIDs; adjusting the dose of the RAAS inhibitor, particularly if the drug is excreted; and use of loop diuretics. Patients might require potassium-binding resins as well. Similar to chronic kidney disease, if heart failure patients receiving dual RAAS blocker therapy have recurrent hyperkalemia, one RAAS inhibitor is often discontinued. The potential patient population of heart failure patients with hyperkalemia in the United States is estimated to be roughly 400,000 to 700,000 (Desai et al. *Heart Failure Rep.* 2009 and company reports).

Diabetes and Hyperkalemia

Diabetes is another chronic disease that has shown to be associated with the development of hyperkalemia. Briefly, diabetes is a condition that can be separated into type 1 (juvenile) or type 2 (adult onset). It is primarily characterized by high blood sugar levels that result from defects in insulin secretion or action. Transmembrane potassium distribution can be influenced by hormones such as insulin.

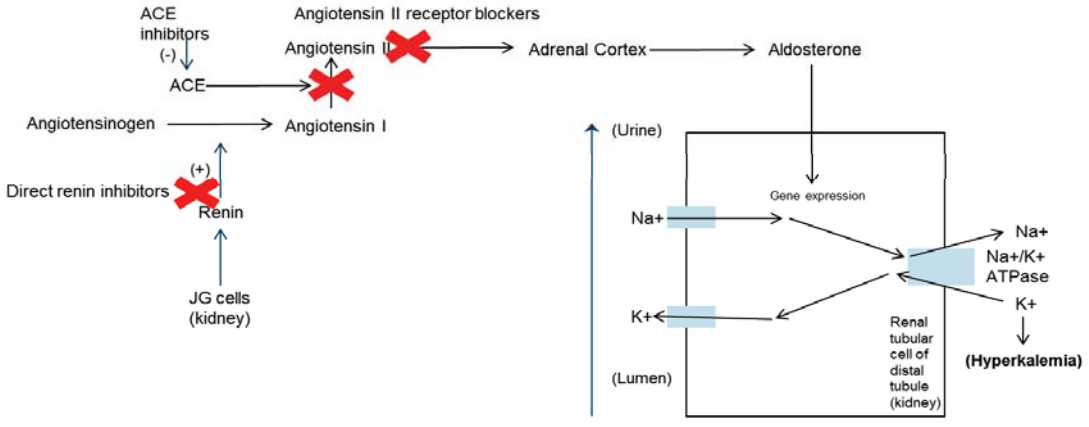
Chronic hyperkalemia in diabetic patients usually occurs due to acid-base equilibrium imbalance and hyporeninemic hypoaldosteronism. The use of RAAS inhibitors is common in the treatment of diabetic patients; it is normally prescribed to slow the progression of renal failure. A study in diabetic patients without end-stage renal disease initiating RAAS inhibitor therapy showed hyperkalemia-associated events occurring at an incidence rate of 10.2 per 1,000 patients per year (Raebel et al., *J Gen Intern Med* 2010). The number of patients in the United States with diabetes was roughly 23.4 million in 2009, and is projected to increase to 44.1 million diagnosed in 2034. The potential patient population of individuals with diabetes and hyperkalemia is roughly 250,000 (Raebel et al. *J Gen Int Med.* 2010 and company reports).

RAAS Inhibitor Therapy and Hyperkalemia

Maintenance of normal potassium homeostasis is an important factor in the treatment of the diseases detailed above. Many pharmacological agents that have a positive impact on the reduction of morbidity and mortality in patients with diseases such as congestive heart failure and myocardial infarction, such as direct renin inhibitors (DRIs), angiotensin-converting enzyme (ACE) inhibitors, β-blockers, aldosterone receptor antagonists (ARAs), and angiotensin-receptor blockers (ARBs), also raise serum potassium levels and increase the risk of developing hyperkalemia, another life-threatening outcome.

As shown in exhibit 11, ACE inhibitors act by stopping the conversion of angiotensin I to angiotensin II, ARBs act by blocking the angiotensin II receptors, and renin inhibitors act on renin receptors to stop the conversion of angiotensinogen to angiotensin I. Roughly 10% of outpatients treated with ACE inhibitors develop hyperkalemia within a year after these drugs are prescribed (Palmer, *NEJM* 2004). In addition, a study in patients with serum creatinine levels of 1.5 to 6.0 mg/dL found an incidence of hyperkalemia of 55%. Further, the authors suggested that the prevalence of hyperkalemia in patients with renal insufficiency might exceed 50%. (Gennari and Segal, *Kidney Int* 2002). Depending on the severity of their disease, patients might be prescribed multiple RAAS inhibitors which would only exacerbate the problems with potassium homestasis.

Exhibit 11
ZS Pharma, Inc.
Mechanism of Action of Renin-Angiotensin-Aldosterone (RAAS) Pathway Inhibitors



Source: William Blair & Company, L.L.C.

Of note, rates of hyperkalemia are often higher in the real world setting versus highly controlled clinical trials. Following the publication of the Randomized Aldactone Evaluation Study (RALES), which suggested use of spironolactone improves outcomes in patients with severe heart failure

(a population often on ACE inhibitors), rates of hyperkalemia rose from 2.4 per 1,000 patients to 11 per 1,000 patients, (Juurlink, *NEJM* 2004). This result reflects the effect of RAAS inhibitor use leading to potassium retention. Furthermore, the rate of in-hospital deaths from hyperkalemia increased from approximately 0.3 per 1000 patients to 2 per 1000 patients ($p<0.001$) in RALES, which is consistent with the study from Goyal et al. detailed above.

In exhibit 12, we highlight the increases in potassium concentration with RAAS inhibitor use stratified by disease type. Hyperkalemia has been associated with RAAS inhibitor use in elderly patients. In a study by Reardon and Macpherson in *JAMA Internal Medicine* (formerly *Archives of Internal Medicine),* the authors noted that 11% of the 1,818 patients from 12 randomized clinical trials developed further increases in potassium after ACE inhibitor treatment, and the most-relevant factor in predicting hyperkalemia was a baseline serum creatinine level of 144 umol/L or greater. After the one-year follow-up, 10% of the 146 case patients remaining on a regimen of ACE inhibitor developed severe hyperkalemia (>6.0 mmol/L).

Exhibit 12
ZS Pharma, Inc.
Studies in Chronic Diseases, Dose of RAAS Inhibitor Used, and Increase in Potassium Levels

Disease Type	Dose	Type of RAAS inhibitor	Increase in K^+ levels
Hypertension	20 mg/day	lisniopril	0.2 mmol/L from baseline
		losartan (ARB)	1.5% increased rate of patients with >5.5 mmol/L
		ACE inhibitor	1.3% increased rate of patients with >5.5 mmol/L
	2.5 to 80 mg/day	olmesartan	similar to placebo
	20 to 160 mg/day	telmisartan (ARB)	0.131 mmol/L from baseline
	up to 200 mg/day	epelerenone (ARA)	≤0.2 mmol/L from baseline
	200 mg/day	epelerenone (ARA)	0.2 mmol/L from baseline
	150 mg/day	aliskiren (DRI)	similar to placebo
	300 mg/day	aliskiren (DRI)	similar to placebo
Heart Failure	5 to 20 mg 2x/day	enalapril (ACEi)	4.0 increased rate of patients with >5.5 mmol/L
		losartan (ARB)	20% of pateints increased elevations ≥ 0.5 mmol/L
CKD	4 to 32 mg	candesartan	odds ratio: 2.7
	10 mg/day	lisinopril	increases ≤ 0.12 mmol/L
	80 mg/day	valsartan	
Diabetes-associated CKD	75 to 300 mg/day	irbesartan	1.9% discontinuation due to hyperkalemia
	2.5 to 10 mg/day	amlodipine	0.5% discontinuation due to hyperkalemia

Source: Soar et al., 2010, and William Blair & Company, L.L.C.

For those individuals with more-developed chronic diseases, RAAS inhibitor combination therapy is the preferred standard of care. However, a drawback to combination therapy is that it increases serum potassium levels above individual RAAS inhibitor therapy (exhibit 13). This leads to the abandonment of one of the combination drugs in response to a bout of hyperkalemia. One of the goals of ZS-9 would be to decrease serum potassium concentrations in the population where RAAS inhibitor combination therapy would be the top line of care, thereby allowing patients to be on a regimen consistent with the best clinical outcome.

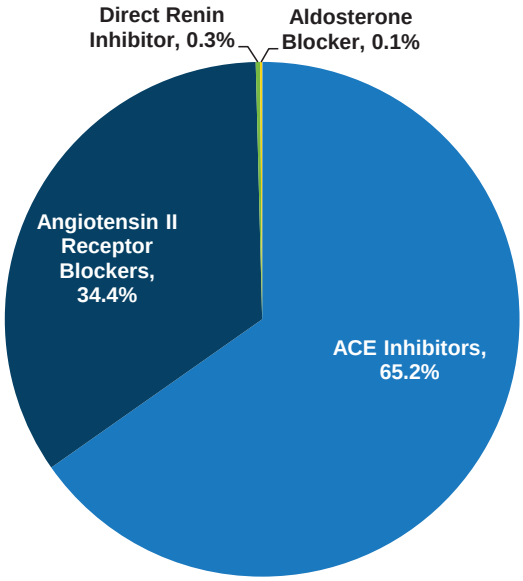
Exhibit 13
ZS Pharma, Inc.
Studies Showing RAAS Inhibitor Combinations and Assciated Increase in Potassium

Citation	Type of RAAS inhibitors (dose in mg/d)	Increase in K^+ levels
Jennings et al.	ACEi and ARB vs. ACEi	ACEI/ARB +0.2 vs. ACEi
MacKinnon et al.	ACEi and ARB vs. ACEi	ACEI/ARB +0.11 vs. ACEi
CALM study	Candesartan (16) and Lisinopril (20) vs. Candesartan (16) vs. Lisinopril (20)	Can and Lis +0.30
AVOID	Aliskiren (150 to 300) and Losartan (100) vs. Losartan (100)	Ali and Los +0.06
Tylicki et al.	Spironolactone (25)/ Telmisartan (5)/ Cilazapril (80)	+0.31 vs. baseline
	Telmisartan (5)/Cilazapril (80)	+0.16
Furumatsu et al.	Sprinolactone (25)/ Losartan (50)/ Enalapril (5)	+0.15
	Losartan (50) / Enalapril (5)	-0.06
Chrysotomou et al.	Spironolactone (25) Irbesartan (150)/ Ramipril (5)	+0.1
Chrysotomou et al.	Spironolactone (25) /Ramipril (5) Irebesartan (150) / Ramipril (5) Ramipril (5)	+0.4 +0.2 0

Sources: Weir and Rolfe 2010 and William Blair & Company

As measured by IMS Health and shown in exhibit 14, ACE inhibitors were the most prescribed RAAS inhibitors over the last 12 months, accounting for roughly 65% of total prescriptions, followed by ARBs with roughly 34%. DRIs and aldosterone blockers accounted for 0.3% and 0.1%, respectively.

Exhibit 14
ZS Pharma, Inc.
Percent Market Share of Total RAAS Inhibitor Therapy Prescriptions



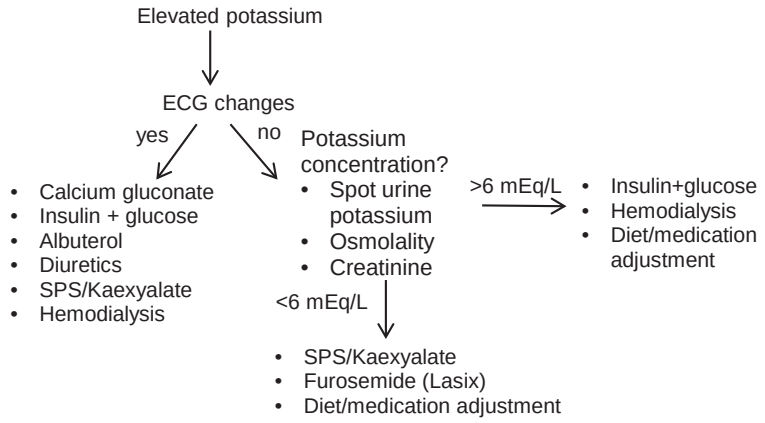
Sources: IMS Health and William Blair & Company, L.L.C.

The large numbers of patients with chronic diseases, such as CKD, heart failure, and diabetes, who are prescribed individual and combinations of RAAS inhibitors for treatment, have the potential to develop hyperkalemia, which has been shown to be associated with an increased risk of cardiovascular adverse events and mortality. The current standard of care, in our opinion, creates a large market need for a product such as ZS-9, which is specific for decreasing potassium concentrations without the need for reduction of RAAS therapy or disturbing the homeostatic balance of other electrolytes. The blockade of the renin-angiotensin system with ACE inhibitors, ARBs, DRIs, and ARAs (or a combination of two or more these drugs) are best-in-class therapies used to slow the progression of chronic cardiovascular and renal diseases.

Acute Hyperkalemia

As shown in the Goyal et al. exhibits 9 and 10 above, the association of serum potassium levels with a cardiovascular adverse event in a clinical setting makes the treatment of hyperkalemia a chronic and acute issue. In the chronic setting, patients with CKD, HF, and diabetes develop hyperkalemia primarily through a reduced glomerular filtration rate and are unable to sufficiently regulate potassium ions. In the acute setting, potassium concentrations need to be reduced into the normokalemic range in the short term, and several publications have detailed algorithms on the steps taken by clinicians to diagnose and treat acute hyperkalemia (exhibit 15).

Exhibit 15
ZS Pharma Inc.
Acute Hyperkalemia Management Algorithm



Sources: Mushiyakh et al. *J Community Hosp Intern Med Perspect* 2011, andWilliam Blair & Company, L.L.C.

If ECG abnormalities are present due to hyperkalemia, the steps are to increase the protection of cardiomyocytes and shift potassium intracellularly. Exhibit 16 lists therapies used to treat acute hyperkalemia, their onset and duration of action, and response type. The calcium salts are intended to protect the electrical activity of cardiomyocytes and glucose + insulin-and-albuterol function to increase intracellular potassium in the short term. The drawback of glucose + insulin and albuterol is that their effects begin to wear off at the two- to four-hour point and are almost completely gone by four hours after treatment. SPS/Kayexalate is the only approved resin to treat acute hyperkalemia (with up to 90 grams per treatment) and has several issues, including its adverse effect profile when delivered with sorbitol and significant percentage of nonresponders. The need for a nontoxic therapy with an onset of action that can be maintained in the longer term would provide an unmet need for this population to supplement the existing therapies of calcium salts, insulin + glucose, and albuterol.

Exhibit 16
ZS Pharma, Inc.
Therapies Used for Treatment of Acute Hyperkalemia and Expected Impact

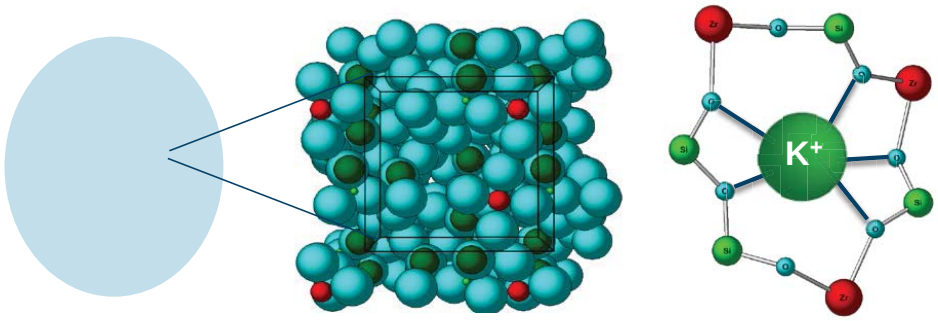
Medication	Response type	Onset of action	Duration of action	Mechanism of action	Expected decrease in potassium level
Calcium salts	rapid	1–2 min	30–60 min	Protect cardiomyocytes	0.5–1.5 mEq/L
Glucose + insulin	intermediate	10–20min	2–6 hours	Shift potassium intracellularly	0.5–1.5 mEq/L (dose dependent)
Beta-agonists	intermediate	3–5min	1–4 hours	Shift potassium intracellularly	
Sodium bicarbonate (only in patients with metabolic acidosis, bicarbonate < 22mEq/L)	intermediate	30–60min	2–6 hours	Shift potassium intracellularly (questionable effect)	
Exchange resin	delayed	2–6hours	4–6 hours	Elimination of potassium from the body	
Furosemide	delayed	5–30 min	2–6 hours	Elimination of potassium from the body	
Hemodialysis	delayed	immediate		Elimination of potassium from the body	1mmol/L in first 60 min. and total of 2mmol/l by 180 min.

Source: Mushiyakh et al. J Community Hosp Intern Med Perspect 2011

ZS-9 Overview

ZS-9, being developed by ZS Pharma, holds the potential to become the best-in-class therapy for acute hyperkalemia as well as the maintenance of normal potassium levels in chronic diseases. ZS-9 has a crystal structure that is specific to the binding of potassium and can subsequently remove it from the body, thereby making it an ideal treatment for hyperkalemia. Exhibit 17 diagrams the chemical structure of ZS-9 and its ability to bind to potassium.

Exhibit 17
ZS Pharma, Inc.
ZS-9 Structure and Potassium Binding



Average ZS-9 Particle Size 20-30 | **Pore Width 3 Å (0.0003 uM)** | **2-D Illustration of Potassium Binding**

Source: Company reports

According to the company, the oxygen atoms in the crystal structure are arranged in a manner such that they do not bind as efficiently to smaller and larger ions, whereas organic polymers (such as Kayexalate) are nonselective ion traps. Therefore, these organic polymers will absorb potassium

only in areas where there are high concentrations of the ion. ZS-9's structure and specificity for potassium ions allow it to absorb potassium immediately on ingestion and throughout the gastrointestinal tract, which is particularly important for the treatment of acute hyperkalemia.

ZS-9's clinical program has two completed studies that have dosed more than 800 patients. We expect data from another Phase III study as early as September 2014, which will be included in the company's NDA to be filed in early 2015, while an open-label, long-term safety study is expected to report out in mid-2015 and be added to the NDA during the year. The Phase II study of ZS-9 (ZS002) enrolled 90 patients, with the primary endpoint of the rate of change in serum potassium from baseline to 48 hours with patients also tested after a seven-day follow-up. Exhibit 18 shows the results of ZS002 where the 10 g dose of ZS-9 was highly significant with a rapid onset of action.

Response rates for the study were high, with 100% of patients in the 10 gram dose arm achieving serum potassium levels below the hyperkalemic threshold (5.0 mEq/L), while roughly 88% had serum potassium levels below 4.5 mEq/L. Results were largely consistent within the heterogeneous patient population; patients on RAAS inhibitor therapy showed similar response rates as those not on RAAS inhibitor therapy. In the seven-day follow-up period without therapy, potassium levels rose to near baseline levels, requiring maintenance therapy, which serves as a basis for the company's following clinical trial, ZS003, which included a longer duration but not a chronic duration of treatment.

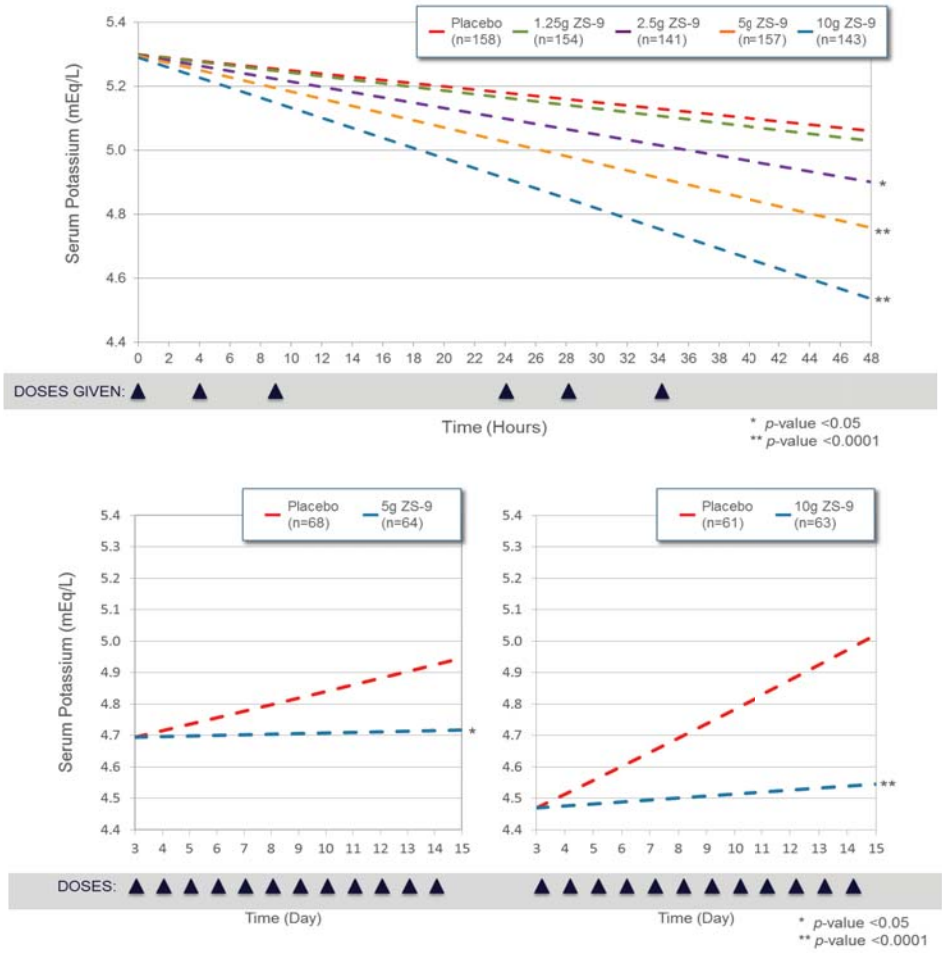
Exhibit 18
ZS Pharma, Inc.
ZS002 (Phase II Proof of Concept in Humans) Results for ZS-9

	Placebo	0.3 g ZS-9 t.i.d.	3 g ZS-9 t.i.d.	10 g ZS-9 t.i.d.
Primary Endpoint (p-value)	NA	0.4203	0.048	<0.0001
Mean K+ Change at 48 hours (mEq/L) (14 h after last dose)	-0.2	-0.3	-0.35	-0.68
Max K+ Change at 38 hours (mEq/L) (4h after last dose)	-0.26	-0.39	-0.42	-0.92

Source: ZS Pharma S-1

The company's first Phase III trial, ZS003, enrolled 753 patients with hyperkalemia, irrespective of their disease state (CKD, HF, and diabetes, as well as those on RAAS inhibitor therapy), in a double-blind, randomized, placebo-controlled study. Patients were randomized to receive one of four doses of ZS-9 or placebo with a primary endpoint similar to ZS002 (rate of change in serum potassium at 48 hours), which provides data for the acute phase of the disease and included an extended treatment phase that measured the rate of change in serum potassium from 48 hours to day 14. Exhibit 19 shows the induction phase and extended treatment phase of the study. ZS-9 showed dose-dependent decreases in serum potassium levels over the first 48 hours that were highly significant ($p<0.0001$) at the 5 g and 10 g doses. In addition, the 5 g and 10 g maintenance dosing from day 2 through day 14 kept the serum potassium levels in the normokalemic range with an average increase of 0.11 mEq/L in patients receiving 5 g of ZS-9 daily, compared with 0.25 mEq/L in placebo ($p=0.0075$), and 0.06 mEq/L in patients receiving 10 g of ZS-9 daily, compared with an increase of 0.58 Eq/L in placebo ($p<0.0001$).

Exhibit 19
ZS Pharma, Inc.
ZS003 Primary (0-48 Hours) and Secondary (48-Hour to 14-Day) Endpoints



Source: Company reports, S-1

As shown in exhibit 20, significant mean serum potassium decreases occurred in each of the individual subpopulations, showing that ZS-9 achieved its desired effect in all potential populations that might develop hyperkalemia.

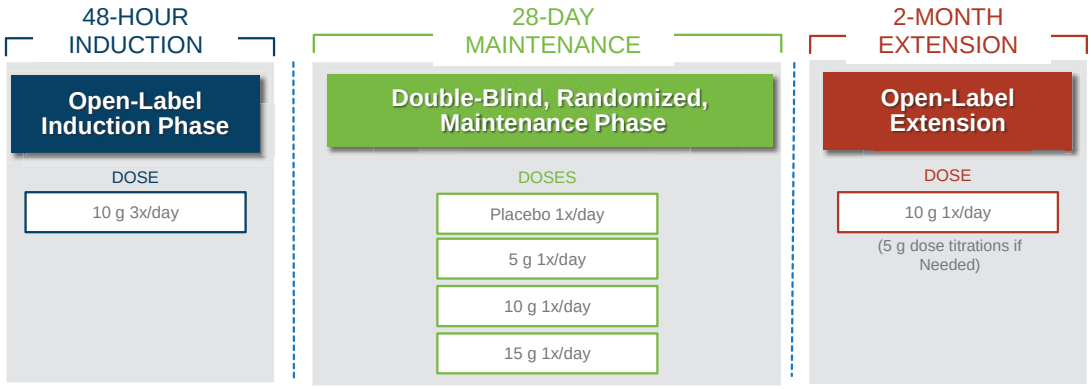
Exhibit 20
ZS Pharma, Inc.
Mean Serum Potassium Levels Stratified by Patient Subgroups in ZS003

	Placebo	ZS-9 1.25g	ZS-9 2.5g	ZS-9 5.0g	ZS-9 10.0g
Overall ITT Populations	-0.25	-0.3	-0.46	-0.54	-0.73
Disease Subsets (n; %)					
CKD (463; 61.5%)	-0.22	-0.31	-0.43	-0.58	-0.83
HF (300; 39.8%)	-0.24	-0.27	-0.46	-0.52	-0.78
RAAS inhibitors (491; 65.2%)	-0.24	-0.28	-0.48	-0.53	-0.73
Diabetes (451; 60%)	-0.25	-0.25	-0.47	-0.52	-0.74
Starting Serum K+					
Baseline S-K < 5.3 (n=427)	-0.15	-0.23	-0.39	-0.39	-0.57
Baseline S-K 5.4-5.5 (n=152)	-0.37	-0.37	-0.49	-0.65	-0.99
Baseline S-K > 5.5 (n=174)	-0.42	-0.34	-0.55	-0.87	-1.1

Source: Company reports

In addition to the two completed clinical trials, ZS002 and ZS003, which have dosed more than 800 hyperkalemic chronic disease patients with ZS-9 who had significant effects regardless of etiology, the company has two more clinical trials underway: ZS004 and ZS005. ZS004 is a one-month, randomized, double-blind withdrawal study designed to confirm the dosing for chronic administration of ZS-9. Exhibit 21 shows the trial design, which will consist of an open-label induction phase with 10 g ZS-9 three times per day, followed by a 28-day maintenance phase during which patients will receive 5 g, 10 g, or 15 g of ZS-9, or placebo, to be followed by a two-month open-label extension study in which patients will receive 10 g once per day with 5 g titrations if needed. The primary endpoint of the study will be a comparison of mean potassium levels from day 8 to day 28.

Exhibit 21
ZS Pharma, Inc.
ZS004 Study Design

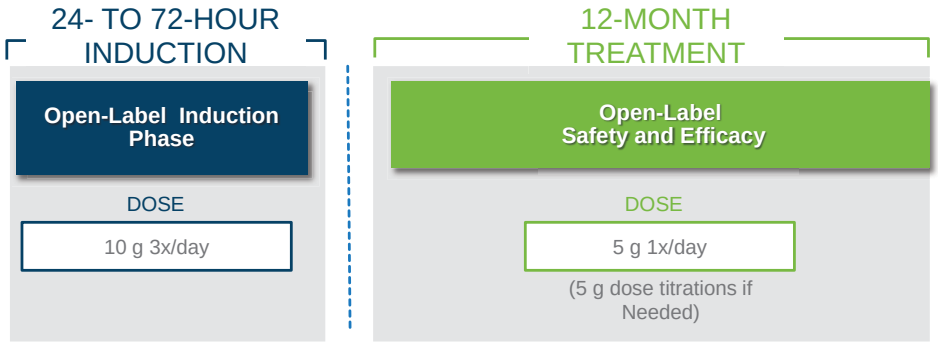


Primary Endpoint: Comparison of mean serum K^+ levels from day 8 to day 28

Secondary Endpoints: Proportion of patients normokalemic after induction phase
Proportion of patients normokalemic during 28-day maintenance period

Source: Company reports

Exhibit 22
ZS Pharma, Inc.
ZS005 Trial Design



Primary Endpoint: Safety and tolerability over 12-month treatment period

Secondary Endpoints: Proportion of patients normokalemic after induction phase
Proportion of patients normokalemic during 12-month treatment period

Source: Company reports

ZS005, the company's planned open-label safety exposure study, is designed to demonstrate the tolerability and efficacy of ZS-9 over 12 months. This trial will consist of an open-label induction phase similar to the previous trials with 10 g ZS-9 three times per day. Patients that become

normokalemic at the end of the induction phase will be eligible to enroll in the open-label treatment phase, where they will receive 5 g of ZS-9 once per day, with 5 g dose titrations taken as necessary (exhibit 22). The primary endpoint of the study will be the safety and tolerability of ZS-9 over the 12-month treatment period. The company expects to start this trial in the second quarter of 2014.

ZS Pharma expects to file an NDA for ZS-9 during the first half of 2015. Management plans to supplement the ZS-9 NDA with results from the ZS004 extension study and data from ZS005, which should be available during third quarter 2015. We believe the entirety of the data set initially provided to the FDA will be substantial with ZS002, ZS003, and the induction phase of ZS004. At the 120-day post-NDA submission point, it should have ZS004 extension data as well as roughly six months of data from the ZS005 study to supplement the initial data set.

Competitive Landscape

While we believe ZS-9 has the potential to be a best-in-class therapy, there are other innovative binders in late- and early-stage development, as well as several existing therapies used by physicians to manage hyperkalemia in the acute setting. Specifically, we believe ZS-9 will likely have an improved efficacy and tolerability profile versus the latest-stage innovative program to treat hyperkalemia, patiromer (exhibit 23). Beyond patiromer, we do not view the currently used therapies for the management of hyperkalemia such as sodium polystyrene sulfonate (SPS) as adequate in comparison to ZS-9 in the acute setting, given their tolerability profile and questionable efficacy. Ardelyx is also developing a program for hyperkalemia; however, this program has yet to enter the clinic and it is too early to determine if the program will be a significant competitor.

Exhibit 23
ZS Pharma, Inc.
Advantages of ZS-9 Over Patiromer

Differentiation Parameter
Better efficacy profile/faster onset of action
Less frequent dosing (QD versus BID)
Room temperature versus cold chain storage
Cost of goods
Better safety profile (lower GI AEs)
Supply chain flexibility

Sources: William Blair & Company, L.L.C., Company reports

Kayexalate, or SPS, is an ion exchange resin that was approved by the FDA in 1958 to bind potassium in the colon as the first treatment for hyperkalemia. SPS was approved before the implementation of the modern-day FDA—during the period before drug manufacturers were required to provide high-quality data on the efficacy and safety of a given drug prior to its approval and marketing. While SPS was commonly dosed along with sorbitol for the treatment of hyperkalemia in September 2009, the FDA required label changes for Kayexalate powder, including warnings about colonic necrosis and other gastrointestinal adverse events, particularly that "concomitant administration of sorbitol is not recommended." In a commentary published in the *Journal of the American Society of Nephrology* in 2012, Sterns et al. wrote that, "If Kayexalate or SPS in sorbitol were presented to the FDA as new drugs with data available today, it is doubtful that either would pass muster."

Aside from the currently approved therapies in use for the management of hyperkalemia, Relypsa is developing patiromer, which is being filed with the FDA and will compete with ZS-9 if both agents are approved. We compare the type of polymer, chemistry, and ionic binding of SPS, patiromer, and ZS-9 in exhibit 24. Of particular note is the specificity (>96%) of ZS-9 for potassium over other ions, calcium, and magnesium. While ZS-9 looks to be very specific, Kayexalate looks to be a relatively

nonspecific binder with a higher affinity for calcium and magnesium versus potassium. Comparatively, Kayexalate binds to 59% of calcium and 24% of magnesium versus only 18% of potassium, which is likely the source of off-target effects and tolerability issues for the therapy when used for the treatment of hyperkalemia. Patiromer has been stated to have twice the binding capacity of Kayexalate, which is approximately 36%.

Exhibit 24
ZS Pharma, Inc.
Chemical Properties of Compounds Used to Treat Hyperkalemia

Property	ZS-9	Kayexalate	Patiromer
Type	Inorganic polymer	Organic polymer	Organic polymer
Chemistry	Octahedral [ZrO6]2- units confers negative charge to the framework, enabling cation exchange	Large MW K binding units synthesized by bulk polymerization process, moderate K+ binding capacity (in vitro), Na-loaded	Small MW K binding units, synthesized by suspension polymerization process, significant K binding capacity (in vitro), Ca2+/sorbitol-loaded

Ion Binding	ZS-9	Kayexalate	Patiromer
K^+	>96%	18%	2x Kaexylate (~36%)
Ca^{2+}	<2%	59%	
Mg^{2+}	<2%	24%	

Sources: McCullough et al. 2014 and company reports

In exhibit 25, on the following page, we compare several characteristics from published studies on SPS, patiromer, and ZS-9. The gastrointestinal side effects of patiromer are higher than that of ZS-9 and hypomagnesemia has been observed in past trials. The dose of ZS-9 used in the post-acute setting is lower than both SPS and patiromer as well. Furthermore, Kayexalate is sodium-loaded and patiromer is calcium and sorbitol-loaded, whereas ZS-9 does not include additional ions.

While patiromer has been in development much longer than ZS-9, the two compounds are facing a similar approval time frame, with patiromer only one to two quarters ahead of ZS-9. Relypsa will complete its Phase I onset-of-action study prior to filing its NDA with the FDA, and patiromer is on track for a fourth quarter 2014 NDA submission. We believe ZS-9 is on track for a first quarter 2015 NDA submission after the company announces results from its second Phase III study in September 2014. ZS Pharma has been able to catch up with patiromer through an aggressive Phase III development program, which holds some key differences from Relypsa's development of patiromer.

In exhibit 26, on the following page, we examine ZS Pharma's and Relypsa's anticipated sample size, patient types, and trial type as well as the design of each study to be used in their potential applications to the FDA. Relypsa has already completed a 52-week study and recently reported interim results from its Phase I onset-of-action study. ZS Pharma has initiated a long-term, open-label study; however, it intends to report results in 2015 with roughly 750 patients. Appendix A, at the end of this report, offers a more-detailed view of the study designs of each ZS Pharma and Relypsa trial.

Exhibit 25
ZS Pharma, Inc.
Information From Major Studies on Cation Exchange Therapies for CKD and HF

	Sodium Polystyrene Sulfonate (SPS)	Patiromer Calcium	ZS-9 ZS003
Drug Class	Organic polymer	Organic polymer	Inorganic selective potassium binder
Dose	15-30g PO single dose in 20g sorbitol (33% sorbitol) or 30-40 g p.r. i	15 g PO BID (powder in water)	10g PO t.i.d. (powder or table) x 48h then 10g q.d. thereafter
Route of Administration	Oral or colonic enema (in 100 mL of warm aqueous vehicle)	Oral	Oral
Trial Population	Baseline K >5.1 mEq/L	Prior hyperkalemia or HF with eGFR < 60 mL/min/1.73 m^2	Baseline potassium 5-6.5 mEq/L with HF, CKD, DM or ACEI, ARB, MRA
Citation	Kessler 2011	Pitt 2011	Ash 2013
N	140	104	753
Study Length	days	4 weeks	7 days
Baseline GFR	NR	84 ± 35	AP
Baseline Potassium	5.40 ± 0.18	4.69	AP
Within Group Reduction in Potassium	Dose-dependent	-0.22 mEq/L by Day 28	-0.73 mEq/L at 48 h (14 h after last dose)
Hyperkalemic Events (K > 5.5 mEq/L vs. Placebo)	NR	7% vs. 25%	AP
Decongestion in HF	NR	NR	AP
Hypokalemia (< 3.5 mEq/L)	NR	6%	0.30%
Hypocalcemia	NR	No	NS
Hypomagnesemia	NR	24%	NS
GI AEs	NR	21%	3.50%

ACEI: Angiotensin-Converting Enzyme Inhibitors, AP: Awaiting Publication, ARB: Angiotensin Receptor Blockers, CKD: Chronic Kidney Disease, DM: Diabetes Mellitus, GI: Gastrointestinal, HF: Heart Failure, MRA: Mineralocorticoid Receptor Antagonists, NR: Not Reported, NS: Not Significant

Source: McCullough et al. 2014 *Rev Cardiovascular Med, and* company reports

Exhibit 26
ZS Pharma, Inc.
ZS-9 and Patiromer Clinical Trial Programs

Patiromer			
Completed/Timeline	**Patient Types**	**N**	**Trial**
Completed	HK, CKD, Type II Diabetes, Hypertension	306	Phase IIb: 52-week safety & efficacy
Completed	HK with CKD	301	Phase III pivotal Part A/B
2014	HK with CKD	103	Phase I onset-of-action
Total Anticipated Size of Safety Database at NDA Submission = Roughly 726 Total Patients			
ZS-9			
Date	**Patient Types**	**N**	**Trial**
Completed	Hyperkalemia, CKD	90	Phase II
Completed	Hyperkalemia regardless of etiology	753	Phase III pivotal
2014	Hyperkalemia regardless of etiology	230	Phase III pivotal
2015	Hyperkalemia	750	Open-label long term
Total Anticipated Size of Safety Database at NDA Submission = Roughly 1,073 Total Patients			

Source: Relypsa and ZS Pharma Company Reports

As shown in exhibit 27, the relative adverse event profile for patiromer seems to show a greater percentage of adverse events, and particularly GI events in comparison to ZS-9. We believe these data show that ZS-9 has a best-in-class safety profile. In addition, ZS Pharma enrolled a broader patient population with the clinical trials recruiting patients with hyperkalemia regardless of etiology. Efficacy in reducing potassium levels regardless of etiology further suggests a potentially best-in-class profile with possible use in a broad patient population.

Exhibit 27
ZS Pharma, Inc.
Safety Measures From ZS-9 and Patiromer (RLY5016)

	Induction Phase (48 h)		Maintenance Phase (2 weeks)	
Safety Measurement	Placebo	ZS-9	Placebo	ZS-9
All Adverse Events	10.8%	12.9%	24.5%	25.1%
GI Events	5.2%	3.5%	3.7%	5.5%

Safety Measurement	Placebo	RLY5016
All Adverse Events	31%	54%
GI Events	6%	21%

Sources: Pitt et al. Eur Heart J 2011, Company Reports, William Blair & Company, L.L.C.

Head-to-head studies of development compounds are rarely available, and cross-trial comparison is always difficult due to different patient populations. However, in an attempt to at least directionally compare the efficacy and side effect profiles of ZS-9 and patiromer, we estimated the baseline and post-treatment mean serum potassium levels for Relypsa's studies using company reports. In the early-stage study comparison, at the 48-hour period, ZS-9 patients (especially at doses above 2.5 grams three times per day or TID) are in the normokalemic range after treatment, whereas patiromer patients are still above the range (though significantly reduced from baseline) for normal potassium concentration.

In addition, in the comparison of data after two weeks of treatment, ZS-9 in the maintenance phase showed lower mean serum potassium concentration (4.71 mEq/L for 5g QD, and 4.55 mEq/L for 10g QD), while our best estimate for 14-day data for patiromer suggests end-values of 4.8 mEq/L for both the mild and moderate-to-severe hyperkalemia patients. Baseline values of about 5.2 mEq/L in the mild hyperkalemia patients and roughly 5.6 mEq/L in the moderate-to-severe hyperkalemia patients suggest likely higher median baseline levels for the patiromer-treated patients, given the median 5.3 mEq/L baseline in the ZS-9 14-day maintenance/induction study, ZS003.

While baseline values and differing patient populations suggest a best-in-class profile for ZS-9, we will continue to monitor the data coming out of both programs, specifically as data from the patiromer onset-of-action study as well as potentially long-term data is reported—likely at the American Society of Nephrology meeting November 11-16, 2014 in Philadelphia. For ZS Pharma, we expect additional 14-day treatment data from ZS003, top-line results from ZS004 during the fourth quarter, and long-term results in mid-2015.

Exhibit 28
ZS Pharma, Inc.
Change in Serum Potassium After Treatment With ZS-9 and Patiromer

Time Point	Patiromer	Dose	RLYP Study	ZS-9	Dose	ZSPH Study
48 hours	5.83 mEq/L -> 5.1 mEq/L	8.4g b.i.d.	Phase I Onset-of-Action	5.3 mEq/L -> 5 mEq/L	1.25g t.i.d.	ZS-003 Induction Phase
				5.3 mEq/L -> 4.84 mEq/L	2.5g t.i.d.	
				5.3 mEq/L -> 4.76 mEq/L	5g t.i.d.	
				5.3 mEq/L -> 4.57 mEq/L	10g t.i.d.	
2 weeks	Mild HK: ~5.2 mEq/L -> ~4.8 mEq/L Moderate/Severe HK ~5.6 mEq/L -> 4.8 mEq/L	10g t.i.d	RLY5016-205 (AMETHYST-DN)	5.3 mEq/L ->4.71 mEq/L	5g q.d.	ZS-003
				5.3 mEq/L -> 4.55 mEq/L	10g q.i.d.	Maint. Phase
4 weeks	Mild HK: ~5.1 mEq/L -> ~4.65 mEq/L Moderate/Severe HK: ~5.8 mEq/L -> 4.5 mEq/L	Mild: 8.4 q.d. Mod/Sev HK: 8.4g b.i.d.	Phase III	Study Results in Q4 2014		ZS-004
52 weeks	Mild HK: ~5.2 mEq/L -> ~4.6 mEq/L Moderate/Severe HK: ~5.65 mEq/L -> ~4.6 mEq/L	10-40g q.d.	RLY5016-205 (AMETHYST-DN)	Study Initiated in Q2 2014		ZS-005

Sources: ZS Pharma and Relypsa company reports

ZS-9 Metal Backbone Likely to Be a Focus for Competition

We acknowledge that if both ZS-9 and patiromer are approved during the 2015-2016 period, Relypsa will likely seek to differentiate patiromer from ZS-9 by focusing on the metal basis of the therapy given the zirconium silicate backbone. This strategy was used by Genzyme during the marketing of Renagel (sevelamer hydrochloride) during the life cycle of that franchise, which faced competition from Shire and the rare earth, metal-based phosphate binder, Fosrenol (lanthanum carbonate). The marketing message was very effective during the Shire and Genzyme competition for share of the phosphate binder market; Fosrenol sales in 2013 approximated $183 million, while sales of Renvela/Renagel exceeded $1 billion in sales.

However, the Renagel/Fosrenol market dynamics hold many differences versus the hyperkalemia markets. Primarily, we believe Genzyme's Renagel had entered the market with a significant lead versus Shire's Fosrenol. Renagel was first approved in 1998, while Fosrenol entered the market six years later, following an approval in October 2004. The chewable Fosrenol was also likely an unpleasant formulation for ingestion in doses up to 1.5 to 3.0 grams with every meal. While the two to four 400 mg or 800 mg tablets of sevelamer are not pleasant, either, they were often mentioned by physicians as having a taste preferred over the metal-based Fosrenol. For ZS-9, we believe the bolus of drug that might be stirred into a glass of water with no taste is an improved formulation. However, patiromer, which is also a dry, odorless powder, is dosed with calcium and sorbitol, which could have an effect on the binding of potassium. Calcium-based binders are also used as the relatively inexpensive alternative to Renagel and Fosrenol; however, both companies have incorporated into their marketing message for years the potential negative cardiovascular outcomes of calcium-based products used in patients with poor cardiovascular health. This message might linger in the chronic kidney disease markets and could be a liability for patiromer.

Lastly, zirconium has a long history of use in biomedical applications and consumer products. The daily zirconium intake of a normal U.S. citizen is likely 1 to 9 milligrams. Experimental toxicity studies of zirconium demonstrate that the metal is inert and has low toxicity. We list some common products with zirconium and a history of safe use in humans in exhibits 29 and 30, on the following page. However, the most applicable use for zirconium, considering the hyperkalemia patient population, is its use in millions of hemodialysis columns since the 1970s as well as in knee and hip replacements.

Exhibit 29
ZS Pharma, Inc.
Zirconium-Containing Products

Product	Amount	Relative to ZS-9
85g Antiperspirant Stick	2295 mg	8,196,429x
Soil	300 mg/L	1,071,429x
Human Body Content	300 mg	1,071,429x
Daily Food Content	3.65 mg	13,036x
Zr From Four-Hour Sorbent Hemodialysis	0.758 mg	2,707x
Daily Drinking Water Content	0.65 mg	2,321x
Sea Water	0.004 mg/L	14x
Soluble From 10 g ZS-9	0.00028 mg	1x

Source: Company reports

Exhibit 30
ZS Pharma, Inc.
History of Safe Zirconium Uses in Humans

Most comprehensive study of Zr was conducted by Schroeder and Balassa, Lee recently reviewed current biomedical uses
Daily Zr intake is estimated to be 1-9 mg per day
Experimental toxicity studies demonstrate that Zr is inert and has low toxicity, making it well suited for biomedical applications
Zirconium-containing compounds safely used in many biomedical applications
Nephrology (hemodialysis, peritoneal dialysis, hemofiltration)
Dental implants and other restorative practices
Middle ear ossicular chain reconstruction
Safely used in Patients with CKD
Millions of dialysis treatments with REDY and SORB columns since 1970s
Fresenius developing new Zr based DIALISORB column
Fresenius developing Zr based wearable artificial kidney

Sources: Company reports, Schroeder *J. Chron. Dis.* 1966, Lee *ASAIO Journal* 2010 Ash *Seminars in Dialysis* 2009.

Other Pipeline Compounds

In addition to ZS-9 for hyperkalemia, ZS Pharma is in preclinical examination for the company's other pipeline candidate, ZS-1, an ammonium-specific crystal for the potential indication of hepatic encephalopathy. Briefly, hepatic encephalopathy (HE) is an altered level of consciousness resulting from liver failure; it could include coma. The patient's liver dysfunction leads to the buildup of toxins in the bloodstream; in HE, ammonia is the key toxin. A study examined the correlation between ammonia levels and HE in 121 patients with cirrhosis and found that four types of ammonia measurements were correlated with HE: arterial total ammonia (r^2=0.61, p≤0.001), venous total ammonia (r^2=0.56, p≤0.001), arterial partial pressure of ammonia (r^2=0.55, p≤0.001), and venous partial pressure of ammonia (r^2=0.52, p≤0.001) (Ong et al. *Am J Med* 2003).

Given the key role of ammonia in hepatic encephalopathy, an ammonia binder is likely to show efficacy in management of the disease. The market for therapies for HE treatment is significant with obesity and hepatitis all leading to liver disease and potentially HE. Salix received approval of Xifaxan for the treatment of overt hepatic encephalopathy recurrence in 2010 and has described the market as a potential $1 billion opportunity for the company. As the HE market is heterogeneous and likely difficult to conduct trials in given the patient population and the growth of Xifaxan in the indication, we believe the company will look to advance ZS-1 in the clinic during 2015. While we are not including the compound in our valuation, we believe ammonia depletion is a validated mechanism of action in hepatic encephalopathy and that there is likely a role for the therapy given concerns over widespread antibiotic use for chronic indications such as hepatic encephalopathy.

Key Management

The ZS Pharma management team brings a diverse set of experience, ranging from venture capital to pharmaceutical development, regulation, and commercialization in the biotech industry. The company's CEO and CFO worked together at SARcode Bioscience, which was acquired by Shire Pharmaceuticals. Founder Alvaro Guillem, Ph.D. has significant experience in binders from his past role as an executive at Bone Care, which was acquired by Genzyme. We include key management members in the following section.

Robert Alexander, Ph.D., chief executive officer. Dr. Alexander has served as a member of the board of directors for ZS Pharma since October 2012 and was named CEO in December 2013. Prior to becoming CEO, he was a director at Alta Partners, which was an investor in ZS Pharma. Prior to Alta, he worked in business development at Genentech Inc. Dr. Alexander received his Ph.D. in immunology from the University of North Carolina, completed a postdoctoral fellowship at Stanford University, and has a B.A. in zoology from Miami University in Ohio.

Alvaro Guillem, Ph.D., co-founder and president. Dr. Guillem founded ZS Pharma and has served as president and a member of the board since 2008. Prior to Dr. Alexander's appointment, Dr. Guillem also served as the company's CEO. He has served in various roles in the pharmaceutical industry, including vice president of quality and scientific affairs at Ash Access Technology, Genzyme/Bone Care, and Adams Respiratory Therapeutics. Dr. Guillem received his Ph.D. in chemistry from Virginia Commonwealth University and a B.S. in chemistry from the University of Mary Washington.

Todd A. Creech, chief financial officer and treasurer. Mr. Creech has served as CFO since August 2013 and as treasurer since February 2014. Prior to his appointment at ZS Pharma, Mr. Creech was CFO of SARcode Bioscience and Sirion Therapeutics. At SARcode Bioscience, he led financing, accounting, and corporate development before the company's sale to Shire plc in 2013. At Sirion Therapeutics, he raised debt and equity financing to support six clinical programs and two NDA approvals. In addition, Mr. Creech co-founded Centice, an optical sensor start-up from Duke University, and worked with NovaQuest (Quintiles's investment group). Mr. Creech holds an M.B.A. from Duke University and B.S. in finance and accounting from Miami University of Ohio.

Henrik Sandvad Rasmussen, M.D., Ph.D., chief medical officer and chief scientific officer. Dr. Rasmussen has served as CMO and CSO of ZS Pharma since October 2012. He brings his experience leading global development programs and regulatory filings in the biotech industry. Previously he held positions as corporate vice president and head of clinical development and medical and regulatory affairs at Novo Nordisk, and served as chief medical officer of Babi Biopharmaceuticals. Dr. Rasmussen received his M.D. and Ph.D. from the University of Copenhagen and has training in cardiology and internal medicine.

D. Jeffrey Keyser, J.D., Ph.D., co-founder, secretary, and chief operating officer. Dr. Keyser has served in all of his roles and has been a member of the board of directors since 2008. Dr. Keyser has several years of experience in various aspects of the pharmaceutical industry in regulatory, clinical, and product development. He previously served as chief compliance officer and vice president of regulatory affairs at Encysive Pharmaceuticals. He has also held several senior positions at Adams Respiratory Therapeutics, Medava Americas, Marion Merrell Dow, Marion Laboratories, and Abbott Laboratories. Dr. Keyser received his Ph.D. in economics from the University of Texas at Dallas, an M.P.A. from the University of Missouri–Kansas City, a J.D. from Creighton University, and a B.S. in pharmacy from Creighton University.

Cynthia Smith, chief commercial officer. Ms. Smith became the CCO of ZS Pharma in June 2013. Prior to her role at ZS Pharma, she served as the vice president of market access and commercial development at Affymax, Inc. For eight years, she held various positions at Merck & Co., Inc. in

several areas related to public affairs and corporate strategy. Ms. Smith earned her M.B.A. from the Wharton School at the University of Pennsylvania, an M.S. in public policy from the Eagleton Institute of Politics at Rutgers University, and a B.A. from the University of North Carolina at Chapel Hill.

Adam Tomasi, Ph.D., vice president of corporate development. Dr. Tomasi joined ZS Pharma from Alta Partners. Prior to Alta, he spent seven years in early-stage drug discovery with Gilead Sciences and Cytokinetics. In his last technical position, he founded and led the Anti-Infectives Medicinal Chemistry Program at Cytokinetics. Dr. Tomasi was a Kauffman fellow; holds a Ph.D. in chemistry from the University of California, Irvine; earned a B.S. in chemistry from the University of California, Berkeley; and an M.B.A. from the MIT Sloan School of Management.

Financial Overview

Income Statement

The performance of ZS Pharma's shares will largely be driven in the near term by the development of the company's main pipeline asset, ZS-9. However, pending an approval and launch of ZS-9 in 2016, we believe the company will break into profitability in 2017, posting $2.29 in earnings during the year. Between 2014 and profitability in 2017, we estimate the company will use the approximately $140 million in cash post-offering as it advances ZS-9 in clinical development and through the regulatory process, then prepares it for market entry in 2017. We estimate profitably for ZS Pharma in 2017, one year before consensus estimates suggest hyperkalemia competitor Relypsa will achieve profitability. While we understand the difficulties estimating earnings several years prior to profitability, we believe the significant gross margin advantage ZS Pharma should hold over patiromer suggests an ultimate advantage in the company's structure.

Exhibit 31
ZS Pharma, Inc.
Per Patient Cost for ZS-9

Induction Phase Dosing		
Grams/day	10	
Doses/day	3	
Days	2	
Maintenance and Long-term Extension Dosing		
Days	363	
Grams/day	10	
Doses/day	1	
Grams/year	3,690	
COGS/gram (initially, at peak)	$0.30	$0.20
COGS	$1,107	$738
Cost of therapy/month	$600	
Cost of therapy/year	$7,200	
Gross Margin (initially)	84%	
Gross Margin (at peak sales)	90%	

Sources: Company reports & William Blair & Company, L.L.C.

Gross margins for ZS Pharma's ZS-9 should be high as zirconium silicate is a material commonly used in industrial processes. Because ZS Pharma will be controlling manufacturing with its Coppell, Texas, and Denver facilities, management estimates the cost of goods at $0.30 per gram at launch, which will eventually fall to $0.20 per gram as production efficiency increases with increased volumes. Given our pricing estimate for ZS-9 of roughly $600 per month, even with significant discounting, we believe the company should be able to obtain gross margins that will begin around 84% and reach better than 90% by peak sales. The company will owe the academic developer of ZS-9 a flat

5% annual royalty on all worldwide net sales of ZS-9, which will end in 2024. However, the recently allowed IP for ZS-9 pushes composition of matter protection out to 2032. We include our gross margin assumptions in exhibit 31.

We believe selling, general, and administrative (SG&A) expense will ramp up from about $15 million in 2014 to peak levels of $30 million, as ZS Pharma completes the company's pivotal program for ZS-9, builds the necessary medical science liaison support for ZS-9, and executes on post-marketing studies. While this R&D spending is restrained, compared with that for other compounds targeting large indications such as diabetes and oncology, the company will need to invest heavily in SG&A as the company launches ZS-9 into the cardiovascular and renal specialist settings after approval in 2016. We estimate SG&A expense will ramp up from roughly $10 million to over $120 million in 2019, or roughly 20% of product sales, which is in line with the company's specialty peers. In discussions with management, we believe the company believes peak-year SG&A spend as a percentage of product sales will likely approximate 17.5% to 18%. The company's salesforce will likely include 150 representatives, which we assume should cost $37.5 million to $40 million in salary alone. In addition to sales representatives, ZS Pharma will need to build out at least 45 additional senior sales managers and reimbursement specialists. In addition, the company's intellectual property portfolio on ZS-9 includes composition of matter and methods patents through 2032. We include our income statement estimates in exhibit 32.

Balance Sheet and Cash Flow

We estimate that ZS Pharma holds about $140 million in cash on its balance sheet, following a successful public offering in which the company priced shares at $18, above the original $15-$17 range, and netted $112 million in cash. We believe the company will need roughly $200 million in cash prior to breaking into profitability during 2017. Although additional nondilutive capital might be available through a global or Japanese partnership, we would not rule out the company executing another opportunistic raise following positive Phase III data or long-term safety data in 2015.

Throughout its operating history, ZS Pharma has accumulated net losses of only $25 million to date after running a very capital-efficient Phase II program for ZS-9. Cash used in operating activities during 2013 approximated $26.5 million. We believe that ZS Pharma will use approximately $20 to 30 million in cash per year from 2014 through 2016 as the company's breaks into profitability in 2017. This spending will be heavily influenced on the continued development of ZS-9 and the need to build a sales infrastructure pending approval of the product in 2016. We estimate that once the company achieves its first year of profitability in 2017, ZS Pharma will generate more than $50 million in cash, which should ramp up to $100 million in 2018 and likely exceed $500 million in out-years.

Valuation and Stock Thoughts

Shares of ZS Pharma have performed well since the company priced its initial public offering at $18, above the offering range. Shares have appreciated around 63% since the IPO, which we believe is based on investor appetite for a best-in-class, late-stage asset entering a large market with potentially very attractive margins. Despite the stock appreciation, we believe shares of ZS Pharma continue to hold a strong risk/reward profile, given the large blockbuster potential of the hyperkalemia market.

We are establishing a price target of $75, based on a net present value of the company's lead development program, ZS-9. In this calculation, we assume a launch of ZS-9 in 2016 following an approval early that same year. We believe peak-year sales of $1 billion are possible with our estimates, assuming only a 13% penetration into the CKD market and a 10% penetration into the congestive heart failure market. Appendices B and C include the potential markets for ZS-9 in acute and chronic hyperkalemia, as well as our market build for ZS-9. We believe that ZS-9 has the potential to become a blockbuster, given the lack of good treatment options for the treatment of hyperkalemia and what

Exhibit 32
ZS Pharma, Inc.
Income Statement
($ in millions except EPS data)

	2012(A)	2013(A)	Q1(A)	Q2(E)	Q3(E)	Q4(A)	2014(E)	2015(E)	2016(E)	2017(E)	2018(E)	2019(E)
ZS-9	-	-	-	-	-	-	-	-	36,767	218,357	445,814	740,445
Total Revenue	-	-	-	-	-	-	-	-	36,767	218,357	445,814	740,445
yr/yr growth	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	104.2%	66.1%
Cost of Goods Sold	-	-	-	-	-	-	-	-	3,677	21,836	44,581	74,044
Gross Profit	-	-	-	-	-	-	-	-	33,090	196,521	401,233	666,400
SG&A	1,148	7,686	1,053	2,498	2,998	2,998	9,992	11,990	23,980	47,961	102,537	148,089
Growth							30%	20%	100%	100%	76%	15%
R&D	6,989	24,508	1,394	3,750	4,500	4,500	15,000	18,000	21,600	24,840	27,324	28,690
Growth		251%	-	-	-	-	-39%	20%	20%	15%	10%	5%
Total Operating Expenses	8,137	32,194	2,447	6,248	7,498	7,498	23,690	29,990	45,580	72,801	129,861	176,779
growth			NA	NA	NA	NA	-26%	27%	52%	60%	78%	36%
Operating Income	(8,137)	(32,194)	(2,447)	(6,248)	(7,498)	(7,498)	(23,690)	(29,990)	(12,490)	123,720	271,371	489,621
EBIT Margin							NM	NM	NM	NM	61%	66%
growth y/y (%)			NA	NA	NA	NA	NM	NM	NM	NM	NM	NM
Depreciation and Amortization	-		250	250	250	250	1,000	1,000	1,000	1,000	1,000	1,000
EBITDA	(8,137)	(32,194)	(2,197)	(5,998)	(7,248)	(7,248)	(22,690.0)	(28,990.2)	(11,490.0)	124,720	272,371	490,621
							NM	NM	NM	NM	61%	66%
Interest income	(17)	(31)										
Interest expense	2,099	9	(366)	750.0	750.0	750.0	3,000	2,000	1,500	1,500	1,000	1,000
Change in fair value of warrants	62	1,424										
Other	-	1										
Income Before Taxes	(10,281)	(33,597)	(2,813)	(5,498)	(6,748)	(6,748)	(21,806)	(27,990)	(10,990)	125,220	272,371	490,621
Income Tax Provision	-	-	(3,652)	275	225	225	(2,927)	1,000	1,000	45,079	92,606	166,811
Effective Tax Rate	0.0%	0.0%	NA	5.0%	NA	NA	NM	NA	NA	34%	34%	34%
Preferred stock accretion	(174)	(689)										
Net Income (loss) Attributable to Common	(10,455)	(34,286)	839	(5,773)	(6,972)	(6,973)	(18,879)	(28,990)	(11,990)	80,141	179,765	323,810
Net income to common per share (diluted)	$ (2.63)	$ (8.52)	0.02	(0.24)	(0.29)	(0.29)	(0.78)	(1.08)	(0.43)	2.82	6.30	11.13
Basic avg. number of shares used in computing net income	3,981	4,025	75,953	22,777	22,877	22,977	22,877	25,477	26,627	27,027	28,250	28,250
Diluted avg. number of shares used in computing net income	3,981	4,025	587,270	24,200	24,300	24,400	24,300	26,900	28,050	28,450	28,550	29,094
Key Ratios (GAAP unless noted)												
Gross Margin	NM	NM	NM	NM	NM	NM	NM	NM	NM	90.0%	90.0%	90.0%
R&D (% Total Rev.)	NM	NM	NM	NM	NM	NM	NM	NM	NM	11.4%	6.1%	3.9%
SG&A (% Total Rev.)	NM	NM	NM	NM	NM	NM	NM	NM	NM	22.0%	**23.0%**	**20.0%**
Operating Margin	NM	NM	NM	NM	NM	NM	NM	NM	NM	56.7%	60.9%	66.1%
Net Income Margin	NM	NM	NM	NM	NM	NM	NM	NM	NM	36.7%	40.3%	43.7%
Revenue Growth												
Growth Yr/Yr	NM	NM	NM	NM	NM	NM	NM	NM	NM	494%	104%	66%
Growth Q/Q	NM		NM	NM	NM	NM						
SG&A Growth												
Growth Yr/Yr	NM	570%	NM	NM	NM	NM	30%	20%	100%	100%	114%	44%
Growth Q/Q	NM		NM	NM	NM	NM						
R&D Growth												
Growth Yr/Yr	NM	251%	NM	NM	NM	NM	-39%	20%	20%	15%	10%	5%
Growth Q/Q	NM		NM	NM	NM	NM						

Sources: Company reports and William Blair & Company, L.L.C. estimates

we view as the best efficacy and safety profile for the therapy to date. Our valuation excludes other pipeline compounds, such as ZS-1, which is a binder of ammonia and could be developed for the treatment of hepatic encephalopathy. We include our risk-adjusted net-present-value breakdown for ZS Pharma shares in exhibit 33.

Exhibit 33
ZS Pharma, Inc.
Sum of the Parts Valuation

	Peak Sales	Discount Rate	Probability of Success	Peak Sales	Value Per Share
ZS-9	$1,170	11%	75%	2021	$ 72.40
Cash Per Share					$ 4.71
NPV of Future Losses Per Share					$ (2.41)
NPV Value					$ 1,807,728
NPV Value Per Share					$ 74.70

Source: William Blair & Company L.L.C. estimates
For per share numbers we use fully diluted share count of 24.2 million

In exhibit 34, we provide the enterprise values of other development companies with either Phase III data in hand or recently approved therapies. While the closest comparable is Relypsa with an enterprise value of about $730 million, we still view the current $424 million enterprise value of ZS Pharma as attractive. Mean comparable enterprise values, which we include in our comparable analysis (exhibit 34), suggest a potential enterprise value of around $864 million for late-stage assets. This implies an approximately 104% discount to this group alone. Given this discount to Relypsa and the company's peer group, we believe shares should trade well as the company executes on its Phase III development program, with top-line data from ZS004 reporting out in September 2014 as well as long-term safety and efficacy data, which is expected to report during 2015.

Exhibit 34
ZS Pharma, Inc.
Brand Specialty Pharmaceutical Comparable Companies

				Market				Revenue Multiples			
Company	Ticker	Price	Rating	Cap.	Debt	Cash	EV	2013A	2014E	2015E	2016E
Pacira Pharmaceuticals	PCRX	$88.94	Not Rated	$2,998	$101	$65	$3,035	76.7	16.7	9.5	6.4
Horizon Pharma	HZNP	$15.73	Not Rated	$1,056	$426	$104	$1,378	53.8	3.9	2.7	2.3
Keryx Biopharmaceuticals	KERX	$15.61	Not Rated	$1,382	$0	$155	$1,227	197.4	40.5	9.8	5.9
Insys Therapeutics	INSY	$29.99	Not Rated	$1,107	$0	$47	$1,059	71.4	5.4	5.4	4.2
Depomed	DEPO	$13.26	Not Rated	$795	$227	$208	$813	8.7	3.8	3.8	NM
Relypsa	**RLYP**	**$25.00**	**Not Rated**	**$846**	**$13**	**$79**	**$780**	**NM**	**NM**	**NM**	**9.4**
Kythera	KYTH	$36.80	Not Rated	$804	$32	$153	$683	NM	NM	NM	5.8
Revance	RVNC	$32.25	Not Rated	$731	$15	$88	$659	NM	NM	NM	NM
BioDelivery Sciences	BDSI	$13.06	Outperform	$575	$17	$89	$503	10.6	12.0	6.6	4.2
Versartis	VSAR	$27.79	Not Rated	$672	$0	$205	$467	NM	NM	NM	13.4
Auspex	ASPX	$23.57	Outperform	$559	$14	$120	$454	NM	NM	NM	10.6
ZS Pharma	**ZSPH**	**$28.14**	**Outperform**	**$555**	**$9**	**$140**	**$424**	**NA**	**NM**	**NM**	**15.4**
Acelryx	ACRX	$11.16	Not Rated	$482	$14	$93	$404	NM	NM	NM	4.5
Adamas	ADMS	$17.30	Outperform	$289	$0	$80	$209	NM	NM	9.3	216.8
Averages				$918	$62	$116	$864	69.8	13.7	6.7	7.5

Values on 7/7/14
Sources: FactSet, William Blair & Company, L.L.C.

Stock Performance
The performance of ZS Pharma since the IPO on June 18 is an increase of 8.38% which compares with the S&P 500 increase of 1.3%, the Nasdaq Biotechnology Index increase of 3.8%, and the Russell 2000 decline of 1.4%. Despite a recent sell-off in the sector, likely due to a more-risk-averse overall market, year-to-date performance for biotechnology and development-stage pharmaceutical companies continues to be strong, with the Nasdaq Biotechnology Index up 13.5% versus the S&P 500, which is up 6.45% year-to-date. Based on our belief that ZS-9 has the potential to become a best-in-class compound for the treatment of hyperkalemia and the maintenance of normokalemic levels, we expect shares to continue outperforming the broad market over the next 12 months. Over that period, we believe several catalysts should drive share performance, including top-line data from the Phase III study, ZS004; competitive data from hyperkalemia patiromer, which should strengthen our view that ZS-9 is the best-in-class candidate for the indication; NDA submission for ZS-9 in early 2015; and long-term safety and efficacy data in mid-2015.

Conclusion

ZS Pharma is developing what we believe is the best-in-class compound for the treatment of hyperkalemia and the maintenance of normokalemia. With hyperkalemia being associated with some of the most-prevalent diseases in the U.S. healthcare system, including chronic kidney disease, congestive heart failure, and diabetes, as well as the use of one of the most-prescribed classes of therapies in the United States, RAAS inhibitors, we believe the market for ZS-9 is significant. We estimate the total addressable market for ZS-9 at more than 3 million patients, and given reasonable penetration, pricing, and duration-of-use assumptions, we easily derive the potential for ZS-9 to become a blockbuster therapy.

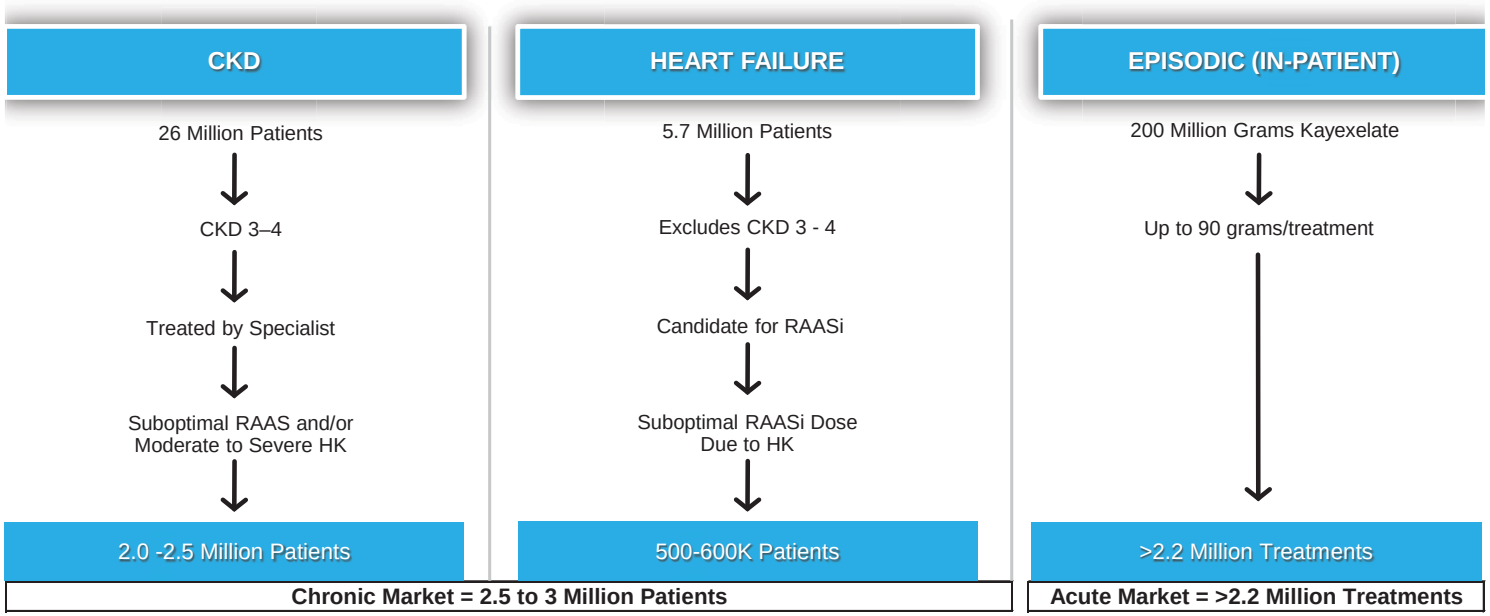
Based on our belief that ZS-9 represents a significant opportunity for ZS Pharma with a best-in-class profile in a large patient population with few good options for treatment, we are initiating coverage with a $75 price target, Outperform rating, and Aggressive Growth company profile.

Appendix A
ZS-9 and Patiromer Phase II and Phase III Study Design Details

Trial Name	Study Design		
ZS002 (Phase II)	**48 h induction:** Placebo t.i.d. 0.3 g ZS-9 t.i.d. 3 g ZS-9 t.i.d. 10g ZS-9 t.i.d.	**7 day follow up** observation	
ZS003 (Phase III)	**48 h induction:** Placebo t.i.d. 1.25 g ZS-9 t.i.d. 2.5 g ZS-9 t.i.d. 5 g ZS-9 t.i.d. 10 g ZS-9 t.i.d.	**12 day maintenance:** 1.25g and 2.5 g ZS-9 q.d. placebo and 1.25 ZS-9 q.d. placebo and 2.5 g ZS-9 q.d. placebo and 5 g ZS-9 q.d. placebo and 10 g ZS-9 q.d.	
ZS004 Phase III (Phase III)	**48h induction:** 10 g ZS-9 t.i.d.	**28 day maintenance:** placebo q.d. ZS-9 5g q.d. ZS-9 10g q.d. ZS-9 15g q.d.	**Open-label Extension:** ZS-9 10g q.d. (5g ZS-9 titrations if nec.)
ZS005 Open-label, long-term study	**Open-label 12-month treatment** 10 g ZS-9 t.i.d. for 3 days, 10 g ZS-9 q.d. up to 12 months	**1-month withdrawal (substudy in 120 patients after 6 months)** 10 g ZS-9 q.d. placebo q.d.	
RLY5016-205 (Phase IIb)	**8-week initiation:**	**44-week maintenance:**	**Follow-up:**
	Patients divided into two strata based on baseline serum potassium levels and given one of three different starting doses, Stratum 1: K+ >5.0-5.5, Stratum 2: K+ > 5.5-6.0 **Stratum 1 Starting Doses** 10 g patiromer q.d. 20 g patiromer q.d. 30 g patiromer q.d. **Stratum 2 Starting Doses** 20 g patiromer q.d. 30 g patiromer q.d. 40 g patiromer q.d. **Titration Schedule** Day 3 and weekly patiromer dose titrations, as needed Stable patients: montly follow-up, Patients req patiromer dose adjustment: weekly follow-up K<5.0 discontinue patiromer, K>5.0 discontinue patiromer and RAASi		
RLYP Phase III	**Treatment Phase (Part A):** Single arm 4 weeks starting dose: Group 1- 8.4 g patiromer q.d. if initial K was 5.1 to <5.5 or Group 2- 8.4 g patiromer b.i.d. if initial K was 5.5 to <6.5	**Withdrawal Phase (Part B):** 8 weeks, placebo-controlled if recurrent hyperkalemia was developed, patiromer dose as increased, placebo arm decreased RAASi. If K continued to increase in patiromer-treated, RAASi therapy was withdrawn	
RLYP Phase I onset-of action	**Open-label single-arm trial** 3 day restricted potassium diet run-in period, 8.4 g patiromer b.i.d. for 48 h followed by 7-day post-treatment safety follow-up period		

Source: Relypsa and ZS Pharma Company Reports

Appendix B
ZS Pharma, Inc.
Potential U.S. Market for ZS-9



Source: Company reports

Appendix C
ZS Pharma, Inc.
Hyperkalemia Market Model

CKD Stage 2/3 Seeing Specialists	2013	2014	2015	2016	2017	2018	2019	2020	2021	2022	2023
U.S. stage 3/ CKD patients (000)	19,200	19,392	19,586	19,782	19,980	20,179	20,381	20,585	20,791	20,999	21,209
% of stage 3/4 seeing nephrologists or cardiologists	30%	30%	30%	30%	30%	30%	30%	30%	30%	30%	30%
CKD patients stage 3/4 seeing neph or cardiologists	5,760	5,818	5,876	5,935	5,994	6,054	6,114	6,175	6,237	6,300	6,363
Percent of treated CKD 3/4 on RAAS	66%	66%	66%	66%	66%	66%	66%	66%	66%	66%	66%
Total patients with stage 3/4 CKD patients on RAAS therapy	3,802	3,840	3,878	3,917	3,956	3,996	4,035	4,076	4,117	4,158	4,199
Sub-populations of stage 3/4 CKD patients on RAAS therapy											
% of treated CKD 3/4 patients on optimal dose RAAS w/HK	4%	4%	4%	4%	4%	4%	4%	4%	4%	4%	4%
% of treated CKD 3/4 on suboptimal dose RAAS due to HK	20%	20%	20%	20%	20%	20%	20%	20%	20%	20%	20%
% of treated CKD 3/4 indicated but not on RAAS due to HK	17%	17%	17%	17%	17%	17%	17%	17%	17%	17%	17%
% of not on RAAS with HK (K+>5.5)	15%	15%	15%	15%	15%	15%	15%	15%	15%	15%	15%
Total CKD patients addressable for ZS-9 (%)	56%	56%	56%	56%	56%	56%	56%	56%	56%	56%	56%
Total CKD patients addressable for ZS-9 (000)	2,129	2,150	2,172	2,193	2,215	2,237	2,260	2,282	2,305	2,328	2,352
% of ZS-9 into addressable CKD population	-	-	-	0.5%	1.3%	7.5%	10.0%	11.0%	12.2%	13.0%	13.0%
CKD Stage 3/4 Patients on ZS-9	-	-	-	44	28	168	226	251	281	303	306
U.S. Patients with congested heart failure (000)	5,700	5,757	5,815	5,873	5,931	5,991	6,051	6,111	6,172	6,234	6,296
% of CHF patients excluding CKD stage 3 or higher	45%	45%	45%	45%	45%	45%	45%	45%	45%	45%	45%
Total U.S. CHF patients excluding CKD stage 3 or higher	2,565	2,591	2,617	2,643	2,669	2,696	2,723	2,750	2,778	2,805	2,833
Sub-populations of CHF patients excluding CKD stage 3 or higher											
% of treated CHF on optimal dose RAAS w/HK (>5.5)	4%	4%	4%	4%	4%	4%	4.0%	4.0%	4.0%	4.0%	4.0%
% of treated CHF on suboptimal dose RAAS due to HK	20%	20%	20%	20%	20%	20%	20.0%	20.0%	20.0%	20.0%	20.0%
% of treated CHF indicated but not on RAAS due to HK	17%	17%	17%	17%	17%	17%	17.0%	17.0%	17.0%	17.0%	17.0%
Total CHF patients addressable for ZS-9 (%)	41%	41%	41%	41%	41%	41%	41%	41%	41%	41%	41%
Total CHF patients addressable for ZS-9 (000)	1,052	1,062	1,073	1,084	1,094	1,105	1,116	1,128	1,139	1,150	1,162
% of ZS-9 into addressable CHF population	-	-	-	0.5%	1.4%	5.5%	8.0%	9.0%	10.0%	10.0%	10.0%
CHF Patients on ZS-9	-	-	-	5	15	61	89	101	114	115	116
Chronic Kidney Disease											
Treatment Duration (months, assumes 70% compliance)	-	-	-	1	2.2	3.5	4.2	5.0	5.0	5.0	5.0
Monthly cost ($net)	600	600	600	600	600	600	600	600	600	600	600
(gross to net adjustment)		15%	15%	15%	15%	15%	15%	15%	15%	15%	15%
ZS-9 sales in CKD	-	-	-	24,610	148,871	337,302	549,147	730,617	818,424	880,812	889,620
Chronic Heart Failure											
Treatment Duration (months)	-	-	-	1	2.2	3.5	4.2	5.0	5.0	5.0	5.0
Monthly cost ($net)	600	600	600	600	600	600	600	600	600	600	600
(gross to net adjustment)		15%	15%	15%	15%	15%	15%	15%	15%	15%	15%
ZS-9 sales in CHF	-	-	-	12,157	69,486	108,512	191,297	258,764	290,390	293,294	296,227
Total ZS-9 Patients				49.3	42.7	228.6	315.3	352.5	395.1	417.7	421.9
Total ZS-9 Sales	-	-	-	36,767	218,357	445,814	740,445	989,381	1,108,814	1,174,106	1,185,847
Growth Yr/Yr	NA	NA	NA	NA	505%	127%	63%	33%	12%	8%	1%
Growth Q/Q	NA	NA	NA	NA							

Sources: Company reports and William Blair & Company, L.L.C. estimates

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DJIA:	16,943.81
S&P 500:	1,967.57
NASDAQ:	4,415.49

The prices of the common stock of other public companies mentioned in this report follow:

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Affymax, Inc.	$0.10
Cytokinetics, Inc.	$4.59
Gilead Sciences, Inc. (Outperform)	$88.64
Merck & Co., Inc.	$58.46
Novo Nordisk A/S	$247.80
Quintiles Transnational Holdings Inc. (Outperform)	$55.38
Shire plc (Market Perform)	$248.99
Relypsa	$23.50

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