Zacks Small-Cap Research

September 23, 2015
David Bautz, PhD
312-265-9471
dbautz@zacks.com

scr.zacks.com
10 S. Riverside Plaza, Suite 1600, Chicago, IL 60606

Can-Fite Biopharma Ltd. (CANF - NYSE)

CANF: An In-Depth Look at CF102…

Current Recommendation	**Hold**
Prior Recommendation	N/A
Date of Last Change	04/14/2015
Current Price (09/22/15)	$4.04
Target Price	**$4.00**

INITIATION

Can-Fite BioPharma is developing CF102 as a second-line treatment for hepatocellular carcinoma (HCC), for which it recently received Fast Track designation from the FDA. HCC is a serious public health problem in parts of East Asia and Africa, and affects more than 700,000 individuals worldwide. The only treatment available for advanced HCC is sorafenib, which increases overall survival by approximately 2 to 3 months and which generated approximately $770 million for the indication in 2014.

CF102 is currently in a Phase 2 clinical trial in HCC patients who have advanced liver disease and have failed first-line treatment. This report provides an in-depth look at the scientific rationale and market opportunity for CF102.

SUMMARY DATA

52-Week High	**$5.54**
52-Week Low	**$1.61**
One-Year Return (%)	**20.60**
Beta	**4.14**
Average Daily Volume (sh)	**3,508,669**
Shares Outstanding (mil)	**11**
Market Capitalization ($mil)	**$44**
Short Interest Ratio (days)	**1.88**
Institutional Ownership (%)	**4**
Insider Ownership (%)	**N/A**
Annual Cash Dividend	**$0.00**
Dividend Yield (%)	**0.00**
5-Yr. Historical Growth Rates	
Sales (%)	**N/A**
Earnings Per Share (%)	**N/A**
Dividend (%)	**N/A**
P/E using TTM EPS	**N/A**
P/E using 2015 Estimate	**N/A**
P/E using 2016 Estimate	**N/A**

Risk Level	**Above Average**
Type of Stock	**Small-Growth**
Industry	**Med-Biomed/Gene**

ZACKS ESTIMATES

Revenue

(In millions of $)

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2014	0 A	0 A	0 A	0 A	0 A
2015	0 A	0 A	0 E	0 E	0 E
2016					0 E
2017					0 E

Earnings per Share

(EPS is operating earnings before non-recurring items)

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2014	-$0.11 A	-$0.10 A	-$0.06 A	-$0.10 A	-$0.37 E
2015	-$0.02 A	-$0.07 A	-$0.05 E	-$0.06 E	-$0.20 E
2016					-$0.35 E
2017					-$0.33 E

© Copyright 2015, Zacks Investment Research. All Rights Reserved.

WHAT'S NEW

An In-Depth Look at CF102

Can-Fite BioPharma, Ltd. (CANF) is developing orally available, small molecule therapeutic agents for the treatment of inflammatory, oncological, and ophthalmic diseases. The company has three lead assets: CF101 for the treatment of rheumatoid arthritis and psoriasis; CF102 for hepatocellular carcinoma (HCC); and CF602 for the treatment of erectile dysfunction.

This report will focus on CF102, which is currently in a Phase 2 clinical trial for the treatment of HCC patients who have advanced liver disease and have failed treatment with a first line therapeutic. HCC is the most common form of primary liver cancer, with over 700,000 individuals affected worldwide, and is the third leading cause of cancer death in the world (ACS). HCC is relatively rare in the U.S. (approximately 35,000 individuals in the U.S. will be diagnosed with HCC in 2015) and other countries where hepatitis infections are not widespread, as most cases of HCC are due to hepatitis infection (both hepatitis B and C). Most cases of liver cancer in countries with low hepatitis rates are due to metastasis of other primary tumors.

The high prevalence of HCC around the world indicates that more treatment options are necessary for this condition. The story of CF102 and its development as a potential HCC treatment began almost 20 years ago with a seemingly simple yet intriguing clinical observation: why doesn't cancer ever metastasize (spread) to voluntary striated muscle?

Why Doesn't Cancer Metastasize to Muscle?

Cancer remains one of the world's top killers and accounts for one out of every four deaths in the U.S. (American Cancer Society). What makes cancer so lethal is not the primary tumor, but when the primary tumor disseminates to other areas of the body through the highly complex and still poorly understood process of metastasis. Interestingly, its been known for over a 100 years that almost no tumors, no matter the organ of origin, metastasize to muscle (Willis, 1973).

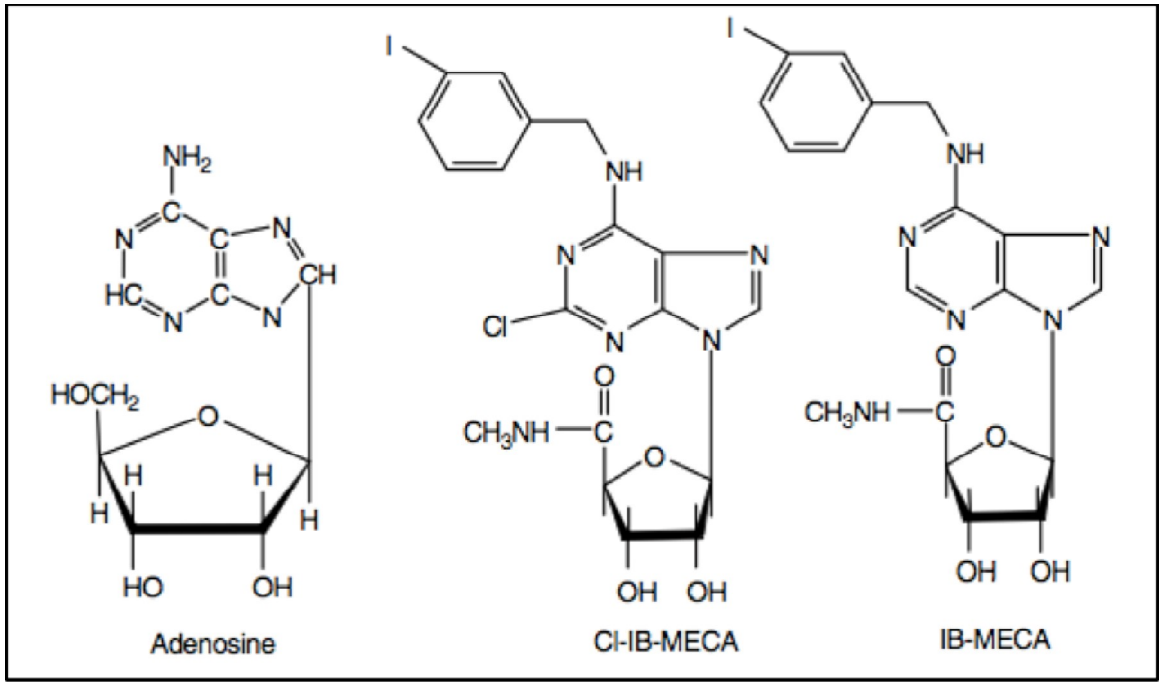
To begin to gain a better understanding of this phenomenon, studies were undertaken where researchers co-incubated various tumor cell lines with newborn rat muscle cells and noted an altered morphology of the tumor cells (Djaldetti *et al*., 1996). In addition, when the tumor cells were incubated in medium that had been used to grow the muscle cells, there was a significant dose-dependent decrease in the uptake of thymidine (a marker of proliferation) by the tumor cells. This implied that something was being released by muscle cells that inhibited tumor cell growth. Further research revealed that muscle cells release a number of anti-proliferative molecules, one of which is adenosine (Fishman *et al*., 1999), a nucleoside involved in a number of cellular processes including energy production (through adenosine triphosphate) and signal transduction (through cyclic adenosine monophosphate).

Adenosine exerts a number of physiological effects through binding of the Adenosine Receptors (ARs). The ARs (A_1, A_{2a}, A_{2b}, A_3) are G-protein coupled receptors found on the surface of various cells throughout the body. The A_1 and A_{2a} receptors both play a role in the heart and the central nervous system, while the A_{2b} and A_3 receptors are mostly located peripherally and are associated with inflammatory and immune responses. The ARs ultimately exert their effects through inhibition (A_1 and A_3) or stimulation (A_{2a} and A_{2b}) of adenylyl cyclase, which controls formation of cyclic AMP (cAMP). cAMP controls the activity of protein kinase A and B, which in turn modulate a variety of extracellular signals (Filippa *et al*., 1999).

The anti-cancer effect of adenosine is exerted through activation of the A_3AR (Fishman *et al*., 2000). Interestingly, the A_3AR was later found in very low levels in most normal tissues, but overexpressed in a number of different tumor types including skin, colon, breast, renal, ovarian, and hepatic cancers (Fishman *et al*., 2007). Thus, the reason cancer almost never metastasizes to muscle is due to the A_3AR agonists that muscle cells release (e.g., adenosine) that in turn activate the A_3AR, which is found in large quantities on tumor cells, resulting in growth inhibition of those cells in the muscle microenvironment. Having identified the molecular basis for why cancer does not metastasize to muscle, the A_3AR became a suitable target for pharmaceutical intervention.

A_3AR Agonists

Since its discovery in the early 1990's, a number of selective A_3AR agonists have been synthesized by modifying the structure of adenosine. N^6-(3-iodobenzyl)-adenosine-5'-N-methyluronamide (IB-MECA) was the first potent and selective agonist when tested against the rat A_3AR (Gallo-Rodriguez *et al.*, 1994). Since IB-MECA binds to the human A_1AR with a K_i of approximately 50 nM, a more potent and selective A_3AR agonist was produced through substitution at the C2 position in the form of 2-chloro-N^6-(3-iodobenzyl)-adenosine-5'-N-methyluronamide (Cl-IB-MECA). Can-Fite is developing both IB-MECA (CF101) and 2-Cl-IB-MECA (CF102) as therapeutic agents.



***Source*: Fishman *et al.*, 2002**

The affinity of each molecule for the human ARs is shown in the following table, clearly showing that they are selective agonists for the A_3AR:

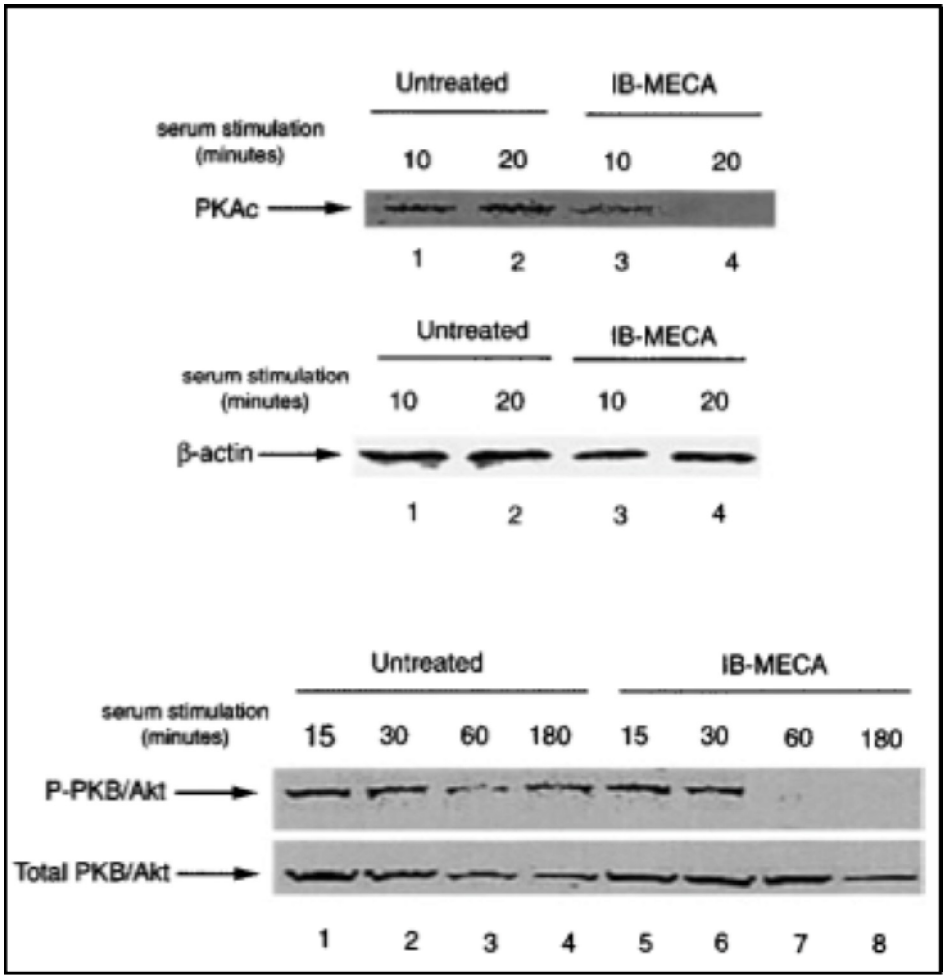
A_3AR Agonist	A_1	A_{2a}	A_{2b}	A_3
CF101 (IB-MECA)	51	2,900	11,000	1.8
CF102 (Cl-IB-MECA)	220	5,360	>100,000	1.4

K_i values (nM), *Adapted from*: Jacobson *et al.*, 2006

A_3AR Agonists Mechanism of Action

Some of the preclinical studies examining the mechanism of action with A_3AR agonists were performed with CF101, however since both CF101 and CF102 bind the same receptor the data generated using CF101 is relevant to CF102.

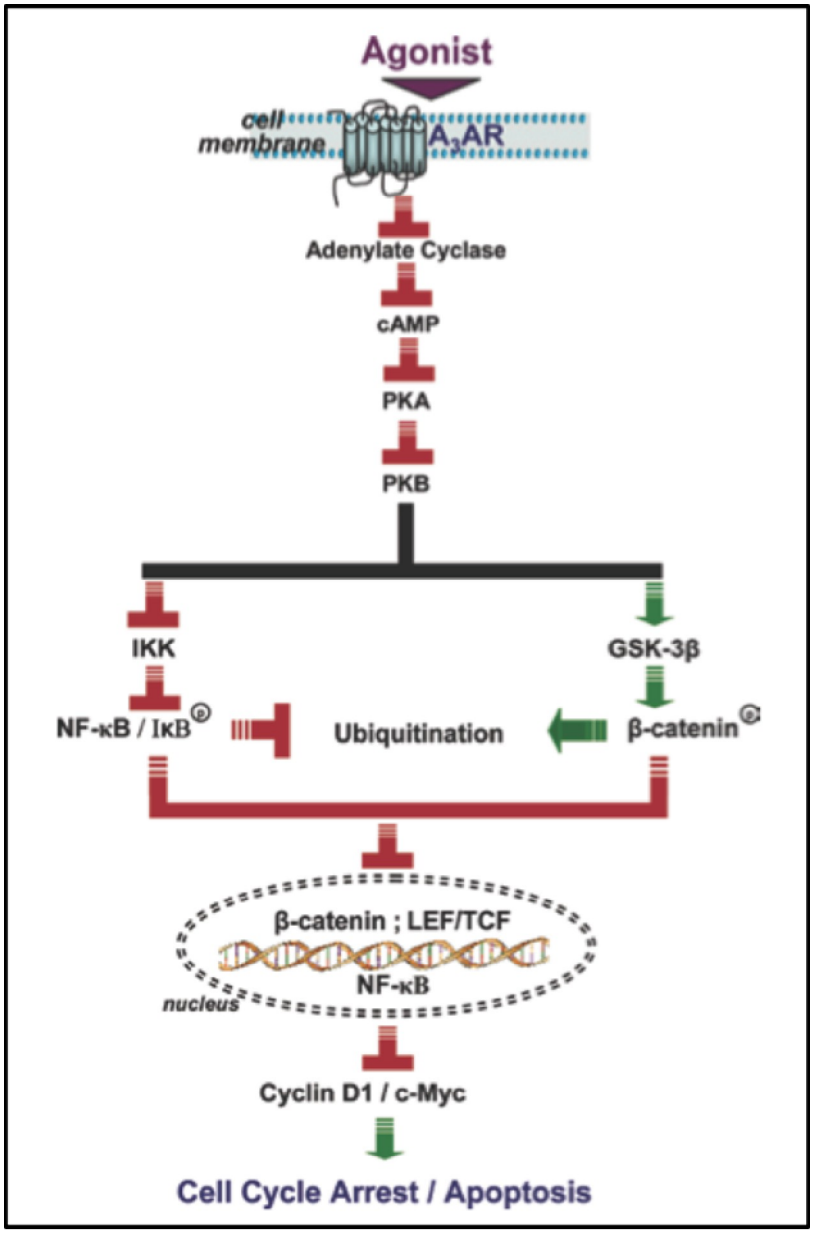
As mentioned previously, activation of A_3AR is known to decrease production of cyclic AMP (cAMP), which in turn controls expression and activity of Protein Kinas A (PKA). Levels of PKA drop dramatically after treating melanoma cells with CF101 for 20 min. Evaluation of PKB/Akt showed no dramatic decrease in protein levels after 20 min, but at 60 min there was a significant decrease in the phosphorylated (activated) form of PKB. β-actin was utilized as a loading control as the levels of it do not change following treatment with CF101. These results are shown in the following figure.



***Source*: Fishman *et al*., 2002**

The Wnt and NF-κB signaling pathways are interconnected at the PKB/Akt junction and are both involved in a number of cellular processes including differentiation, growth, motility, and apoptosis (Darnell, 2002; Forde *et al*., 2007). The Wnt signaling pathway controls cell fate during embryogenesis and is also involved in tumorigenesis. Glycogen synthase kinase 3β (GSK-3β) is a key element of the Wnt pathway and is responsible for phosphorylating β-catenin. This is a signal for the ubiquitination and subsequent degradation of β-catenin. Activation of the Wnt signaling pathway leads to the deactivation of GSK-3β. This in turn causes β-catenin to not be phosphorylated and to eventually translocate to the nucleus where it activates transcription of cell cycle progression genes such as cyclin D1 and c-myc (Clevers *et al*., 2012).

The NF-κB signaling pathway is composed of a family of proteins that control transcription of a number of genes involved in cell proliferation and cell survival, which like the Wnt signaling pathway also includes cyclin D1 and c-myc (Hoesel *et al*., 2013). The fact that cyclin D1 and c-myc are down-regulated by activation of A_3AR is important since a vast amount of literature exists showing those two genes are upregulated in cancers (Chen *et al*. 2005; Liao *et al*., 2007). The following figure shows a model to explain the mechanism of action for A_3AR agonists.



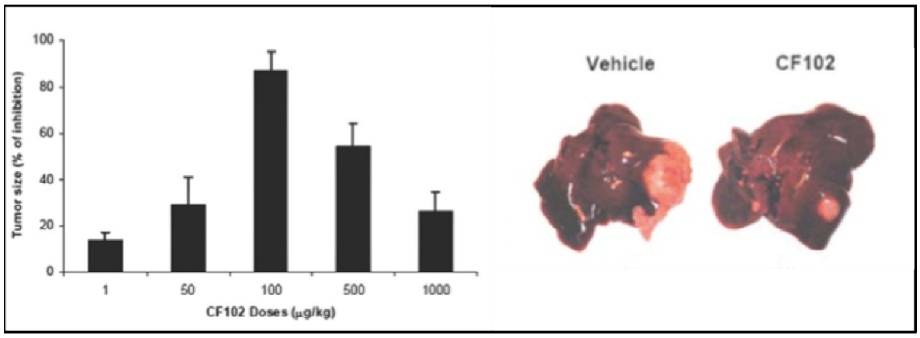
***Source*: Fishman *et al*., 2010**

CF102 Pre-Clinical Data

Can-Fite has generated a plethora of data showing that A_3AR agonists have anti-cancer activity in a wide range of tumor types, including melanoma (Fishman *et al*., 2002; Madi *et al*., 2003), prostate cancer (Fishman *et al*., 2003), colon cancer (Fishman *et al*., 2004), and HCC (Bar-Yehuda *et al*., 2008; Cohen *et al*., 2011). Interestingly, HCC was the most responsive to treatment with CF102 among all the tumor types tested. In addition, a number of important conclusions were drawn from the two studies of CF102 in HCC that set the stage for moving the compound into clinical testing for that indication:

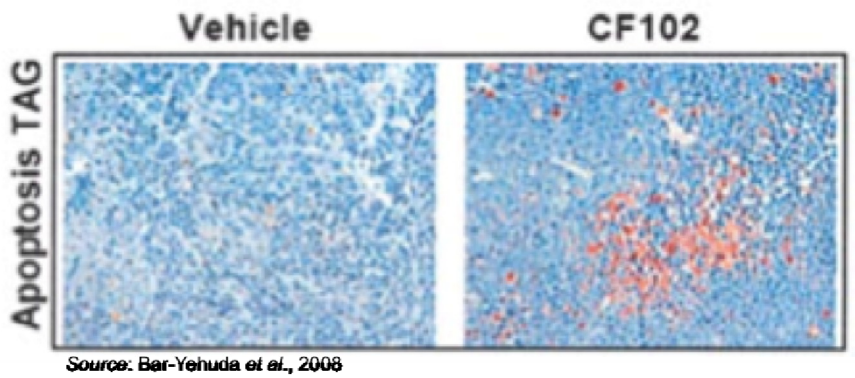
- **A_3AR is highly expressed in liver cancer**: A_3AR mRNA levels were assessed from tumor tissues derived from HCC patients as well as from tumor tissue derived from N1S1 HCC bearing rats. A_3AR expression level was elevated in both the rat and human tumor samples compared to normal liver tissue.

- **CF102 inhibited the growth of liver cancer tumors in a rat orthotopic model**: N1S1 cells were injected into the right hepatic lobe of Sprague-Dawley rats and CF102 dosing was initiated three days later and continued for 15 days. CF102 exhibited a bell shaped dosing curve and resulted in a dramatic reduction of tumor size as shown in the following figure.



***Source*: Bar-Yehuda *et al*., 2008**

- **CF102 treatment induced apoptosis in liver cancer cells**: Up-regulation of the pro-apoptotic proteins Bax and Bad was noted in tumors derived from CF102-treated animals, while no expression of those proteins was found in tumors from control-treated animals. In addition, a much larger number of apoptotic cells were noted in tumors derived from CF102-treated animals compared with tumors from control-treated animals, as shown in the following figure.



***Source*: Bar-Yehuda *et al*., 2008**

CF102 Phase 1/2 Clinical Trial in HCC

The encouraging preclinical results noted above with CF102 in HCC were followed up by a Phase 1/2 trial in HCC patients (NCT00790218). The open label, dose escalation study was undertaken in 18 HCC patients to test the safety, tolerability, and pharmacokinetics of CF102 at doses of 1 mg, 5 mg, and 25 mg twice a day. The compound exhibited good bioavailability and had linear pharmacokinetics. Median overall survival for all 18 patients was 7.8 months (Stemmer *et al*., 2013). In a subpopulation of patients (n=5) who were Child-Pugh B (liver disease staging system; discussed further below), median overall survival was 8.1 months. This compares to historical median overall survival rates in this patient subpopulation of approximately 4 months (Pressiani *et al*., 2013). Interestingly, one of the patients who presented with skin metastases showed complete metastasis regression during the three months of treatment with CF102. Perhaps most importantly, CF102 had no adverse effect on routine measures of liver function over a six-month period in 12 patients treated for at least that duration, suggesting that CF102 could be utilized safely in patients with cirrhosis or decreased liver function.

Can-Fite Targeting HCC Patients with Child-Pugh B Liver Disease

The Child-Turcotte score was originally proposed over 50 years ago as a way to predict the outcome after liver surgery in patients with cirrhosis (Child, 1964). It included two continuous variables (bilirubin and albumin) and three discrete variables (ascites, encephalopathy, nutritional status). A modified version named Child-Pugh was published approximately 10 years later that included one alteration, the replacement of nutritional status with prothrombin time (Pugh *et al*., 1974). Each of the five categories is assigned a score of 1, 2, or 3 and the final score then corresponds to a Child-Pugh grade A (5 to 6 points), B (7 to 9 points) or C (10 to 15 points).

	1 point	2 points	3 points
Total bilirubin (µmol/L)	<34	34-50	>50
Serum albumin (g/L)	>35	28-35	<28
Prothrombin time (s)	<1.7	1.71-2.30	>2.30
Ascites	None	Mild	Moderate to Severe
Hepatic encephalopathy	None	Grade I-II (or suppressed with medication)	Grade III-IV (or refractory)

	Class A	Class B	Class C
Total points	5-6	7-9	10-15

Source: 2minutemedicine.com/Zacks SCR

Numerous studies have shown that the Child-Pugh score is an independent prognostic marker in a wide range of diseases, including alcoholic cirrhosis, hepatitis C related cirrhosis, ruptured esophageal varices, and primary biliary cirrhosis. In patients with liver cancer, Child-Pugh score is correlated with overall survival, as patients with Child-Pugh A consistently show increased survival over those with Child-Pugh B or C (Cabibbo *et al*., 2012).

Clinical experience with patients who are Child-Pugh B is somewhat limited. Nexavar® (sorafenib), the only currently approved systemic therapeutic for HCC, was approved on the basis of two randomized, double blind, placebo controlled clinical trials that showed an increased median overall survival of approximately 3 months for patients treated with Nexavar® compared to placebo (Llovet *et al*., 2008; Cheng *et al*., 2009). However, those trials were mostly restricted to patients who were Child-Pugh A (≥95%). Treating patients who were Child-Pugh B with Nexavar® has consistently shown decreased median overall survival compared to those who were Child-Pugh A (Hollebecque *et al*., 2011; Marrero *et al*., 2013).

Can-Fite is targeting HCC patients who have failed first line therapy (a majority of the patients will likely have taken sorafenib) and are Child-Pugh B, and thus far no clinical trials have been identified that have specifically examined this patient population. There have been a number of recent clinical failures in second-line HCC therapy with a focus on Child-Pugh A, including:

- **Brivanib**: This is a dual tyrosine kinase inhibitor that potently inhibits vascular endothelial growth factor receptors (VEGFR)-1, 2, and 3 as well as fibroblast growth factor receptor (FGFR). In the BRISK-PS study it was tested in 395 HCC patients who had either progressed on/after or were intolerant to sorafenib therapy, with patients randomized 2:1 to receive brivanib or placebo (Llovet *et al*., 2013). 92% of patients were Child-Pugh A. Results showed no difference in overall survival between brivanib and placebo (9.4 months vs. 8.2 months, $P = 0.3307$). Exploratory endpoints showed a median time to progression of 4.2 months for brivanib and 2.7 months for placebo ($P < 0.001$) and an overall response rate of 10% for brivanib and 2% for placebo.

- **Everolimus (Afinitor™)**: This is an mTOR inhibitor that has been approved for the treatment of renal cell carcinoma. In the EVOLVE-1 trial it was tested in 546 HCC patients who had progressed on/after or were intolerant to sorafenib therapy, with patients randomized 2:1 to receive everolimus or placebo (Zhu *et al*., 2014). 97% of patients were Child-Pugh A. Results showed no significant difference in overall survival between everolimus or placebo (7.6 months vs. 7.3 months, $P = 0.68$). Median time to progression for everolimus was 3.0 months and for placebo was 2.6 months.

HCC patients are a very difficult group to treat based on the usual advanced stage of the disease when it is diagnosed and the underlying liver damage that typically accompanies the condition. This is another reason so few therapeutics are available for these patients, as they typically have advanced liver disease and are less able to tolerate harsh chemotherapeutic agents. Patients with Child-Pugh B cirrhosis are even more difficult to treat, which is exemplified by the fact that most pharmaceutical companies won't even test a drug in that cohort. Can-Fite's strategy to test CF102 in HCC patients who are Child-Pugh B and have failed sorafenib is very risky, however it is warranted based on the positive preclinical data and Phase 1/2 data showing the drug to be both safe and effective in that cohort. The reward could be immense if the strategy pays off, as there is no current treatment option for advanced liver cancer patients.

Opportunity in Liver Cancer

Can-Fite has initiated a multi-center, randomized, double blind, placebo controlled, Phase 2 trial of CF102 in patients with advanced HCC and Child-Pugh B cirrhosis (NCT02128958). A total of 78 patients are expected to be randomized 2:1 to receive 25 mg of CF102 or placebo twice a day for consecutive 28-day cycles. The study is currently enrolling patients in Israel and Europe. The primary outcome of the study is overall survival, which will not be evaluated until after 75 deaths have occurred. The first patient was dosed in December 2014 and we anticipate data from the study in mid-2016. Can-Fite recently announced that CF102 was granted Orphan Drug Status as well as Fast Track designation by the U.S. Food and Drug Administration (FDA) for the treatment of advanced liver cancer.

There are a large number of other medications currently in development for the treatment of liver cancer, including antiangiogenic agents, mTOR inhibitors, and growth factor receptor inhibitors. However, with only one drug approved for treating liver cancer, there is ample opportunity for other therapies that show efficacy in treating the disease. Can-Fite has chosen to focus on liver cancer patients who also have Child-Pugh B cirrhosis, which is indicative of more advanced liver disease, as CF102 showed somewhat better efficacy in treating those patients in the Phase 1/2 trial and did not appear to have toxic effects on the liver. With a benign side effect profile and positive proof-of-concept data, CF102 has the potential to stand out as a therapy for this hard-to-treat patient population.

Conclusion and Recommendation

The recent news that CF102 was granted Fast Track designation for the treatment of liver cancer was well received by investors, as the stock climbed from under $2 per share to an intraday high of $7.85 two days later! The company took advantage of the higher share price by raising $9 million in a registered direct offering, which will fund the company until after the data becomes available from the on-going Phase 2 trial of CF102 in mid-2016.

In addition to CF102, Can-Fite is also developing CF101 for the treatment of psoriasis and rheumatoid arthritis (RA) and CF602 for the treatment of sexual dysfunction. The company is currently developing the Phase 3 clinical trial protocol for CF101 in psoriasis, discussing the Phase 3 clinical trial plan for RA with regulators in the EU in order to clarify the path forward, and finishing up IND-enabling studies for CF602 such that an IND can be filed with the FDA and clinical studies can begin in 2016.

We value Can-Fite using a risk-adjusted discounted cash flow model that takes into account the potential for CF101 in RA and psoriasis and CF102 in liver cancer. For all indications, we estimate that Can-Fite will sign partnership agreements that will generate royalty income based on revenues of each product, with a 15% royalty assumed for each indication. For CF102, we have slightly increased our probability of approval based upon Fast Track designation, as this removes some of the regulatory risk. However, given the difficulty in successfully treating liver cancer patients, the probability of approval is still low (25%). If the company were to report positive results from the Phase 2 trial next year, it would cause our probability of approval to increase significantly. For CF101, while the market opportunity is quite large ($22 billion for RA and $10 billion for psoriasis), given the immense competition in both the RA and psoriasis markets we forecast peak market share of approximately 1% in both indications. Based on our model, we believe the shares are worth $4 and we are maintaining a ‘Hold’ rating on the stock pending further insight into the path forward for CF101 in RA and psoriasis. Upcoming milestones for the company include the finalization of the psoriasis Phase 3 clinical trial protocol in the second half of 2015, the initiation of the CF101 Phase 3 clinical trial in RA in the first half of 2016, and the data from the CF102 clinical trial in HCC in mid-2016.

PROJECTED FINANCIALS

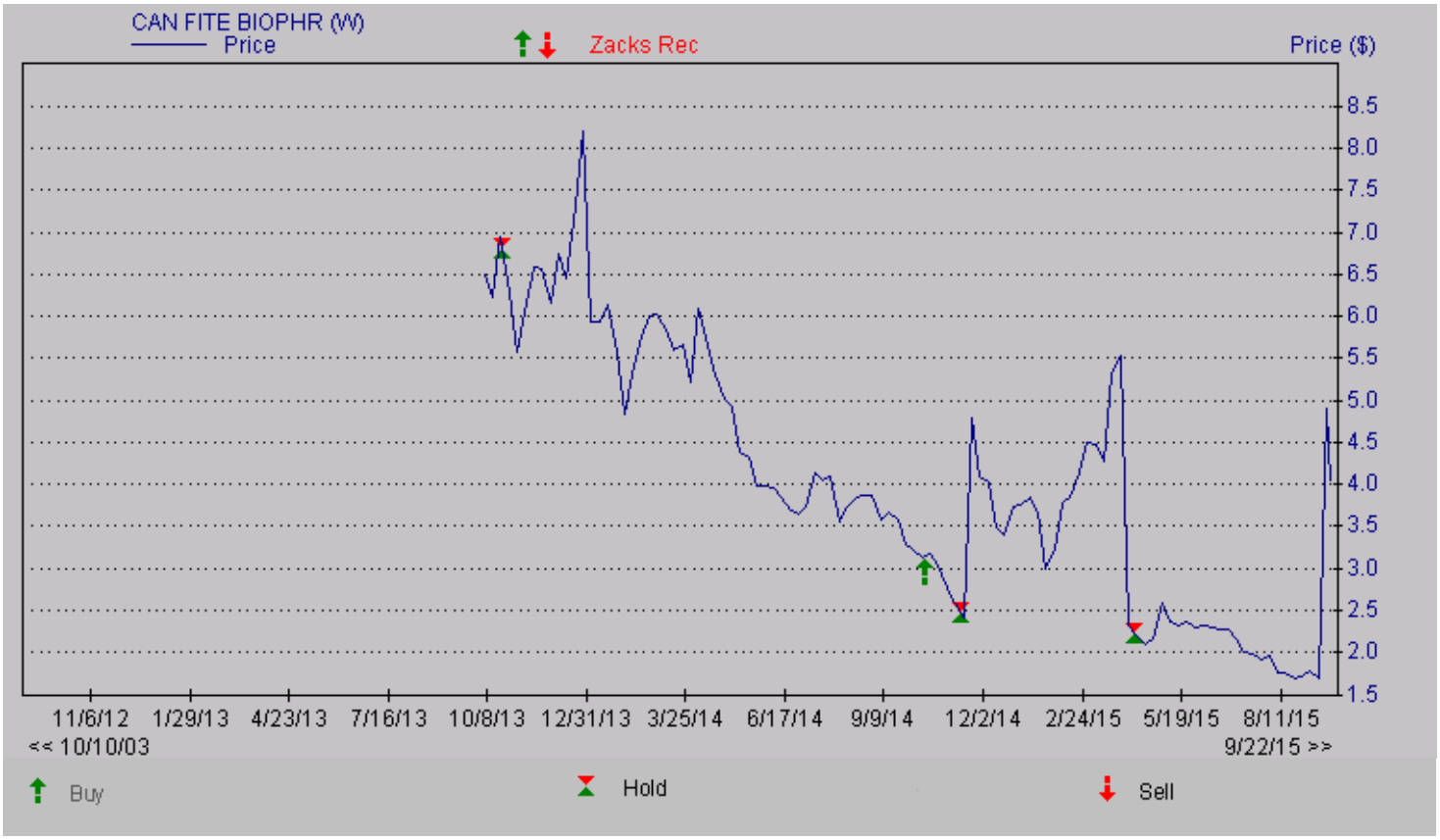
Can-Fite Biopharma Ltd.
Income Statement

Can-Fite Biopharma Ltd.	2014 A	Q1 A	Q2 A	Q3 E	Q4 E	2015 E	2016 E
CF101 (Psoriasis)	$0	$0	$0	$0	$0	$0	$0
YOY Growth	-	-	-	-	-	-	-
CF101 (RA)	$0	$0	$0	$0	$0	$0	$0
YOY Growth	-	-	-	-	-	-	-
CF101 (Glaucoma)	$0	$0	$0	$0	$0	$0	$0
YOY Growth	-	-	-	-	-	-	-
CF102 (HCC)	$0	$0	$0	$0	$0	$0	$0
YOY Growth	-	-	-	-	-	-	-
Grants & Collaborative Revenue	$0	$0	$0	$0	$0	$0	$0
YOY Growth	-	-	-	-	-	-	-
Total Revenues	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**	**$0**
YOY Growth	-	-	-	-	-	-	-
Cost of Sales	$0	$0	$0	$0	$0	$0	$0
Product Gross Margin	-	-	-	-	-	-	-
Research & Development	$4.2	$0.6	$0.9	$0.7	$0.8	$3.0	$6.0
General & Administrative	$3.0	$0.6	$0.6	$0.9	$1.0	$3.1	$5.0
Other Expenses	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.5
Operating Income	($7.1)	($1.2)	($1.5)	($1.6)	($1.8)	($6.1)	($11.5)
Operating Margin	-	-	-	-	-	-	-
Non-Operating Expenses (Net)	$0.6	$0.8	($0.2)	$0.3	$0.3	$1.2	($0.1)
Pre-Tax Income	($6.6)	($0.4)	($1.7)	($1.3)	($1.5)	($4.9)	($11.6)
Income Taxes Paid	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Tax Rate	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*	*0%*
Net Income	**($6.6)**	**($0.4)**	**($1.7)**	**($1.3)**	**($1.5)**	**($4.9)**	**($11.6)**
Net Margin	-	-	-	-	-	-	-
Reported EPS	**($0.37)**	**($0.02)**	**($0.07)**	**($0.05)**	**($0.06)**	**($0.20)**	**($0.35)**
YOY Growth	-	-	-	-	-	-	-
Basic Shares Outstanding	17.546	21.850	23.280	25.000	27.000	24.283	33.000

Source: Zacks Investment Research, Inc. *David Bautz, PhD*

© Copyright 2015, Zacks Investment Research. All Rights Reserved.

HISTORICAL ZACKS RECOMMENDATIONS



© Copyright 2015, Zacks Investment Research. All Rights Reserved.

DISCLOSURES

The following disclosures relate to relationships between Zacks Small-Cap Research ("Zacks SCR"), a division of Zacks Investment Research ("ZIR"), and the issuers covered by the Zacks SCR Analysts in the Small-Cap Universe.

ANALYST DISCLOSURES

I, David Bautz, PhD, hereby certify that the view expressed in this research report accurately reflect my personal views about the subject securities and issuers. I also certify that no part of my compensation was, is, or will be, directly or indirectly, related to the recommendations or views expressed in this research report. I believe the information used for the creation of this report has been obtained from sources I considered to be reliable, but I can neither guarantee nor represent the completeness or accuracy of the information herewith. Such information and the opinions expressed are subject to change without notice.

INVESMENT BANKING, REFERRALS, AND FEES FOR SERVICE

Zacks SCR does not provide nor has received compensation for investment banking services on the securities covered in this report. Zacks SCR does not expect to receive compensation for investment banking services on the Small-Cap Universe. Zacks SCR may seek to provide referrals for a fee to investment banks. Zacks & Co., a separate legal entity from ZIR, is, among others, one of these investment banks. Referrals may include securities and issuers noted in this report. Zacks & Co. may have paid referral fees to Zacks SCR related to some of the securities and issuers noted in this report. From time to time, Zacks SCR pays investment banks, including Zacks & Co., a referral fee for research coverage.

Zacks SCR has received compensation for non-investment banking services on the Small-Cap Universe, and expects to receive additional compensation for non-investment banking services on the Small-Cap Universe, paid by issuers of securities covered by Zacks SCR Analysts. Non-investment banking services include investor relations services and software, financial database analysis, advertising services, brokerage services, advisory services, investment research, investment management, non-deal road shows, and attendance fees for conferences sponsored or co-sponsored by Zacks SCR. The fees for these services vary on a per client basis and are subject to the number of services contracted. Fees typically range between ten thousand and fifty thousand per annum.

POLICY DISCLOSURES

Zacks SCR Analysts are restricted from holding or trading securities in the issuers which they cover. ZIR and Zacks SCR do not make a market in any security nor do they act as dealers in securities. Each Zacks SCR Analyst has full discretion on the rating and price target based on his or her own due diligence. Analysts are paid in part based on the overall profitability of Zacks SCR. Such profitability is derived from a variety of sources and includes payments received from issuers of securities covered by Zacks SCR for services described above. No part of analyst compensation was, is or will be, directly or indirectly, related to the specific recommendations or views expressed in any report or article.

ADDITIONAL INFORMATION

Additional information is available upon request. Zacks SCR reports are based on data obtained from sources we believe to be reliable, but are not guaranteed as to be accurate nor do we purport to be complete. Because of individual objectives, this report should not be construed as advice designed to meet the particular investment needs of any investor. Any opinions expressed by Zacks SCR Analysts are subject to change without notice. Reports are not to be construed as an offer or solicitation of an offer to buy or sell the securities herein mentioned.

ZACKS RATING & RECOMMENDATION

ZIR uses the following rating system for the 1170 companies whose securities it covers, including securities covered by Zacks SCR:
Buy/Outperform: The analyst expects that the subject company will outperform the broader U.S. equity market over the next one to two quarters.
Hold/Neutral: The analyst expects that the company will perform in line with the broader U.S. equity market over the next one to two quarters.
Sell/Underperform: The analyst expects the company will underperform the broader U.S. Equity market over the next one to two quarters.

The current distribution is as follows: Buy/Outperform- 25.0%, Hold/Neutral- 57.4%, Sell/Underperform – 14.6%. Data is as of midnight on the business day immediately prior to this publication.