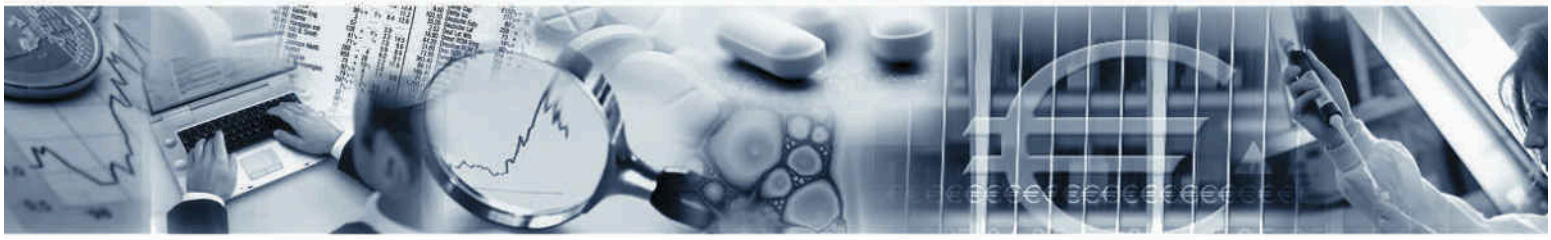

Research Note

Pharming Group

Turning the Page

Chief Research Analyst

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Date: 14 February 2017

Name:	**Pharming Group**
Country:	**The Netherlands**
Price:	**EUR 0.32**
ISIN Code:	**NL0010391025**
Reuters Code:	**PHAR.AS**
Market Cap (EUR m):	**151.7**
EV (EUR m):	**-6.7**
Cash & cash eq. (EUR m):	**34.3**
Shares outstanding (m):	**474.0**
Volume:	**18.5 million**
Free float:	**98%**
52-week Range:	**0.17-0.32**

EUR million	2015A	2016E	2017E
Total Revenues	10.9	12.5	57.9
Net (Loss)/Profit	(10.0)	(15.0)	5.8
Net (loss)/profit ps (cents)	(2.4)	(3.5)	1.2
R&D costs	14.2	15.0	18.0
Cash increase/(decrease)	(2.6)	3.2	(10.0)
Cash and marketable sec.	31.8	33.0	23.0

Executive Summary

- Pharming Group is a Dutch based biopharmaceutical company and one of the oldest publicly traded biotech companies in Europe. The company is focused on the development of recombinant proteins for therapeutic use. Pharming's main platform is the development of human recombinant proteins through the generation of transgenic animals which express the human protein in their milk.

- Its lead product is RUCONEST, a recombinant human C1 esterase inhibitor approved for the treatment of angioedema attacks in patients with Hereditary Angioedema (HAE) in the USA (approved 2014) and Europe (approved in 2010). In Europe Pharming commercializes RUCONEST in Austria, France, Germany, Netherlands and UK and has a distribution agreement with Swedish Oprhan Biovitrum (Sobi) for Scandinavia, Eastern Europe and Italy. In the US, Pharming now distributes the product itself.

- End of last year, Pharming struck a deal with Valeant to acquire all North American commercialisation rights for its own product, RUCONEST, including all rights in the US, Mexico and Canada. The deal had a total value of USD 125 million with an upfront fee paid to Valeant of USD 60 million, and future self- funding sales milestone payments up to a further USD 65 million in total.

- We feel that with the acquisition of the rights for RUCONEST in North America, the company will propel into profitability much quicker than previously expected. Already in 2017 we expect Pharming to reach a net profit due to an acceleration of sales of RUCONEST. Pharming has taken over the complete sales force for RUCONEST from Valeant and will invest in extending the sales force and medical science liaison (MSL) personnel, both of which are key for a strong uptake of the sales of RUCONEST. MSLs are needed to establish and maintain peer-peer relationships with leading physicians,

referred to as Key Opinion Leaders (KOL's), at major academic institutions and clinics.

- Following closing of the Valeant deal (08 December 2016), the Company's cash position was EUR 34 million which should be sufficient to carry out the further development of its pipeline and the further strengthening of its sales and marketing force. Last December, Pharming successfully raised EUR 104 million with the help of top tier investors from the US and Europe through a combination of new equity, straight debt and convertible bonds. With regaining full commercial rights for RUCONEST in North America, the cash flow for Pharming will dramatically improve and we estimate that Pharming will be profitable as of this year.

- **Based on NPV based valuation, we believe that Pharming is substantially undervalued at the current share price of EUR 0.32. Using our valuation model and taking into account the future revenues from RUCONEST for both acute and prophylactic use, the company's current total value should be EUR 660 million, which translates, based on an expected number of outstanding shares of approximately 700 million following conversion of the EUR 45 million 18 months amortising bond and the EUR 12.5 million 5 year convertible bond, into EUR 0.94 per share. This represents a substantial upside from the current share price.**

Company Profile & Technology Platform

Pharming Group is a Dutch based biopharmaceutical company and one of the oldest publicly traded biotech companies in Europe. Pharming became public in 1998 and is developing innovative products, focusing on the treatment of diseases with significant unmet medical needs. These products are developed utilising Pharming's proprietary transgenic production technology.

Pharming currently has a product portfolio which focuses on the commercialisation and further development of RUCONEST® (recombinant human C1-esterase inhibitor) for Hereditary Angioedema (HAE), a genetic disorder. The Company is also evaluating RUCONEST® in other potential indications in the area of ischemia reperfusion injury (e.g. Delayed Graft Function) to generate value both in the short-term and long-term. Furthermore, Pharming has other recombinant protein assets (e.g. α-glucosidase and α-galactosidase) but these have not yet entered formal clinical trials. In addition, Pharming is seeking various partnerships to generate additional income from leveraging its rare diseases commercial infrastructure in Western Europe and the USA and through expanding its pipeline further and to maximise the value of its transgenic platform.

Transgenic Production Technology Platform

Pharming's technologies include innovative platforms for the production of protein therapeutics, as well as technology and processes for the purification and formulation of these products. It has developed methods and technology for breeding transgenic animals for subsequent isolation and purification of protein products from the milk which offers the ability to produce complex proteins at high volume and low cost and for a variety of applications. Conventional methods used for producing recombinant proteins, such as cell culture using animal, bacterial, or yeast cells, are often disadvantaged in that they are either expensive for large-scale production, are immunogenetic or are unable to produce proteins with required complex posttranscriptional modifications. Transgenic technology, using either microinjection or nuclear transfer, enables the

expression of human proteins in a system that is relatively easy to scale up and can be established and operated at a relatively low cost.

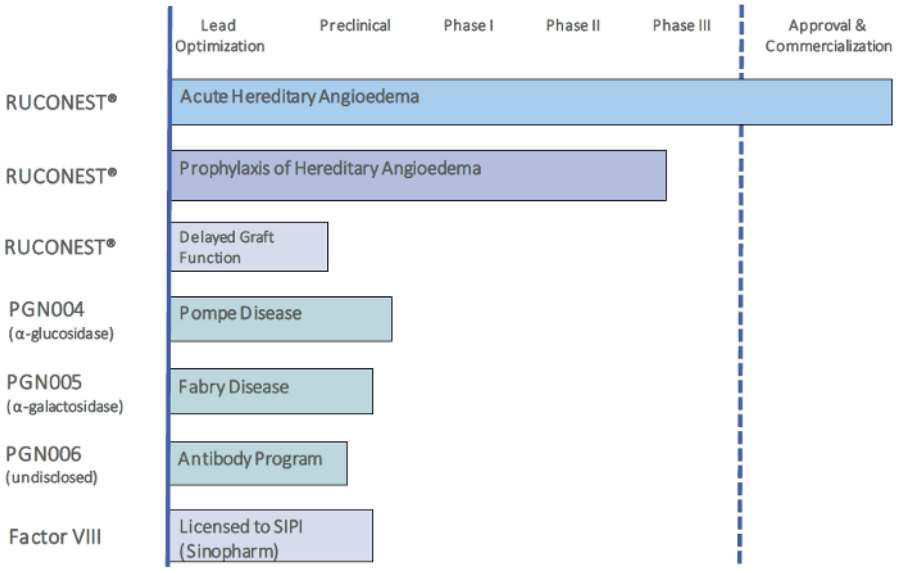
One requirement of transgenic animals used for pharmaceutical production is that the animal is able to produce the desired drug at high levels without endangering its own health. The second requirement of transgenic animals is the ability to produce a drug, an ability that can be passed onto its offspring. The current strategy to achieve these objectives is to couple the DNA gene for the protein drug with a DNA coder directing production in the mammary gland. The new gene, while present in every cell of the animal, functions only in the mammary gland so the protein drug is made only in the milk. Since the mammary gland and milk are essentially "outside" the main life support systems of the animal, there is not much danger of disease or harm to the animal in making the "foreign" protein drug. However, leakage of proteins can occur from mammary gland back into bloodstream. This is a potential threat. When comparing the different methods of producing pharmaceuticals, transgenic technology looks like to be one of the best choices, especially for more complex and highly glycosylated proteins. When analyzing the expenses of the different methods, animal housing and production operations are much cheaper than that of fermentation and cell culture facilities. In general, cell culture production is 5-10 times less economical and 2-3 times more expensive than transgenic technology. Besides lower costs, transgenic technology provides an ideal route for bulk production since very high expression levels of fully bioactive protein are obtained. However, one has to bear in mind that the purification of proteins from milk under GMP conditions is as costly as with other production methods.

Rabbits produce significant quantities of milk, in the range of 100-250ml/day (approx 10-15l of milk per rabbit per year). Their prolific nature reduces the time required to establish a transgenic line and so accelerates time-to-market for the recombinant protein. Large numbers of transgenic rabbits can be generated in a relatively short time

Pipeline: Focus on Recombinant Human Proteins

Pharming currently has a product portfolio which focuses on the commercialisation and further development of RUCONEST® (recombinant human C1-esterase inhibitor) for HAE, a genetic disorder. The Company is also evaluating RUCONEST® in other potential indications in the area of ischemia reperfusion injury (e.g. Delayed Graft Function) to generate value both in the short-term and long-term. Furthermore, Pharming has other recombinant protein assets (e.g. α-glucosidase and α-galactosidase) but these have not yet entered formal clinical trials.

In June 2010, RUCONEST® was the first recombinant C1-esterase inhibitor replacement therapy authorized by the EMA. In 2015 the marketing authorization for RUCONEST® was renewed for an unlimited period. In July 2014, the FDA approved RUCONEST for Acute HAE in the US. In October 2015, the FDA granted 12 years of exclusivity to RUCONEST® 50 IU/kg. The determination of exclusivity ensures that prior to 16 July 2026 the FDA will not approve any applications for biosimilars of RUCONEST®.



RUCONEST (approved in Acute HAE, clinical development Prophylaxis HAE)

End of last year, Pharming and its former US partner Valeant reached agreement for Pharming to acquire all North American commercialization rights to RUCONEST®, including all rights in the USA, Mexico and Canada. We feel that with the acquisition of the rights for RUCONEST in North America, the company will propel into profitability much quicker than previous expected. Already in 2017 we expect Pharming to reach a net profit due to an acceleration of sales of RUCONEST. Pharming has taken over the complete sales force for RUCONEST from Valeant and will invest in extending the sales force and medical science liaison (MSL) personnel, both of which are key for a strong uptake of the sales of RUCONEST. MSLs are needed to establish and maintain peer-peer relationships with leading physicians, referred to as Key Opinion Leaders (KOL's), at major academic institutions and clinics.

In July 2016 Pharming announced positive results from a Phase II clinical study of RUCONEST® for prophylaxis in patients with HAE. In the study, RUCONEST® showed a clinically relevant and statistically significant reduction in attack frequency for both the twice-weekly and once-weekly treatment regimens as compared with placebo. In the study, RUCONEST® showed a clinically relevant and statistically significant reduction in attack frequency for both the twice-weekly and once-weekly treatment regimens as compared with placebo.

Thirty-two HAE patients deficient in C1 esterase inhibitor and with a history of at least four attacks per month were enrolled in the randomized, double-blind, placebo-controlled study. The patients received RUCONEST® once and twice weekly and placebo in each of three four-week treatment periods in a cross-over design. The primary efficacy endpoint was the number of HAE attacks per 28 day treatment period and the secondary endpoint was clinical response, defined as a ≥ 50% reduction in the number of attacks from treatment with placebo to treatment with RUCONEST®.

In the intent-to-treat analysis (ITT), the study found a statistically significant difference in the mean number of HAE attacks that patients experienced during treatment with both the twice-weekly (p-value <0.0001) and once-weekly (p-value =0.0004) RUCONEST® regimen as compared with placebo.Patients on placebo had a mean of 7.2 attacks (95% confidence interval[CI]: 5.8-8.6) per four week treatment period which was reduced to a mean of 2.7 attacks on RUCONEST® twice weekly (95% CI: 1.8-3.7) and a mean of 4.4 attacks on RUCONEST® once-weekly (95% CI: 3.1-5.6). For the analysis of the secondary endpoint in the ITT population, 74% of patients (95% CI: 57-86) on the twice-weekly RUCONEST® regimen had at least a 50% reduction in their attack frequency. This was confirmed in the per-protocol population of patients, which included patients who completed the study without any major deviations (n=23), where 96% of patients (95% CI: 79-99) on the twice-weekly RUCONEST® regimen and 57% (95%CI: 37-74) on the once weekly RUCONEST® regimen had at least a 50% reduction in their attack frequency. Furthermore, in this group, twice weekly RUCONEST® treatment reduced the attack frequency by 72% (95% CI: 63-81) and once weekly RUCONEST® treatment reduced attack frequency by 44% (95% CI: 27-62) as compared with placebo.

		Placebo	RUCONEST®	RUCONEST®
Intent-to-Treat Analysis			Once/week	Twice/week
(n=32)	Primary: Mean number of attacks	7.2	4.4	2.7
	Confidence Interval (95%)	5.8-8.6	3.1-5.6	1.8-3.7
	p-value		*0.0004*	*p<0.0001*
(n=31)	Secondary: % Patients with more than 50% reduction in attack frequency		42%	74%
	Confidence Interval (95%)		26-59	57-86
Per Protocol Analysis				
(n=23)	Mean number of attacks	7.5	3.8	2
	Confidence Interval (95%)	6.0-9.0	2.5-5.1	1.3-2.7
	p-value		*p<0.0001*	*p<0.0001*
(n=23)	% Patients with more than 50% reduction in attack frequency		57%	96%
	Confidence Interval (95%)		37-74	79-99

If approved in this indication, RUCONEST® will be able to enter this additional market, currently worth approximately USD 700 million. RUCONEST® therefore has the potential to be the only recombinant C1 esterase inhibitor approved to target both the acute market and the prophylaxis market in HAE. The US HAE acute therapy market is worth USD 800-850 million.

Overview Current Treatments HAE

Names		Recombinant C1 Inhibitor	Plasma-derived C1 Inhibitor concentrates		Bradykinin receptor antagonist	Kallikrein inhibitor	Clinical Trial: Kallikrein inhibitor antibody
		RUCONEST® ^	Cinryze^^^	Berinert	Firazyr**	Kalbitor^^^^	DX 2930 (a.k.a. SHP643)
Owner		Pharming	Shire	CSL Behring	Shire	Shire	Shire
Sales†		$33m	$550m	$200m	$500m	$83m	Entering Phase III
Efficacy		Good & consistent	Good	Good	Good	Good	
	Dosing (C1INH)	50 U/kg*	~ 12 U/kg	20 U/kg	N/A	N/A	N/A
	Treatment type	Acute^^	Prophylaxis	Acute****	Acute	Acute	Prophylaxis
	Response < 4h	89%	~52%	70%	58-74%	73%	??
Safety concerns		Very low risk of allergic reaction	Warning: Risk of blood clots	Warning: Risk of blood clots	97% injection site reactions	Black box warning: 3.9% Anaphylaxis	Data is in mild patients only
	Plasma risk	NO	YES	YES	N/A	N/A	N/A
Purity (C1INH)		>99.9%	±80%	±95%			
Relapse / worsening		Uncommon	Uncommon	Uncommon	11-31%***	17%	??
Administration		IV (SC, IM coming)	Twice weekly IV	IV (SC coming)	SC	SC (Hospital only)	SC

† Sales figures are Pharming estimates based on relevant selling company's releases and financial reports as well as IMS data and other proprietary databases
*Optimal efficacy of C1INH therapy is achieved at doses ≥50 U/kg ("Target levels of functional C1-inhibitor in hereditary angioedema". Allergy. C. E. Hack, A. Relan, E. S. van Amersfoort & M. Cicardi)
**Icatibant Clinical Briefing Document, CDER, FDA, 2011./ Aberer, et al. Ann Allergy Asthma Immunol 2010; 105(5):P238
** *Cicardi et al, N Engl J Med 2010;363:532-41.; Aberer, et al. Ann Allergy Asthma Immunol 2010; 105(5):P238; Lumry, et al. Ann Allergy Asthma Immunol. 2011;107:529 - 537.
****Berinert not licensed for peripheral attacks in the US.
^Ruconest approved in US, EU and Israel, ^^Ruconest filed for laryngeal attacks (US), ^^^Cinryze not licensed for acute therapy in US. ^^^^Kalbitor not approved in EU.
?? Kalbitor moderate response rate is likely to be pathway-related, at least in part. Relapse rate is also likely to be pathway-related in part. Accordingly DX 2930 may also have these issues. In addition, the safety consequences of chronically inhibiting the contact pathway have not been studied, and this may also be a factor. Antibodies tend not to have large (>75%) response rates.
Note: New forms of products for different routes of administration may require clinical development and regulatory approval.

Source: Pharming, Company Reports

rC1INH and Hereditary Angioedema (HAE)

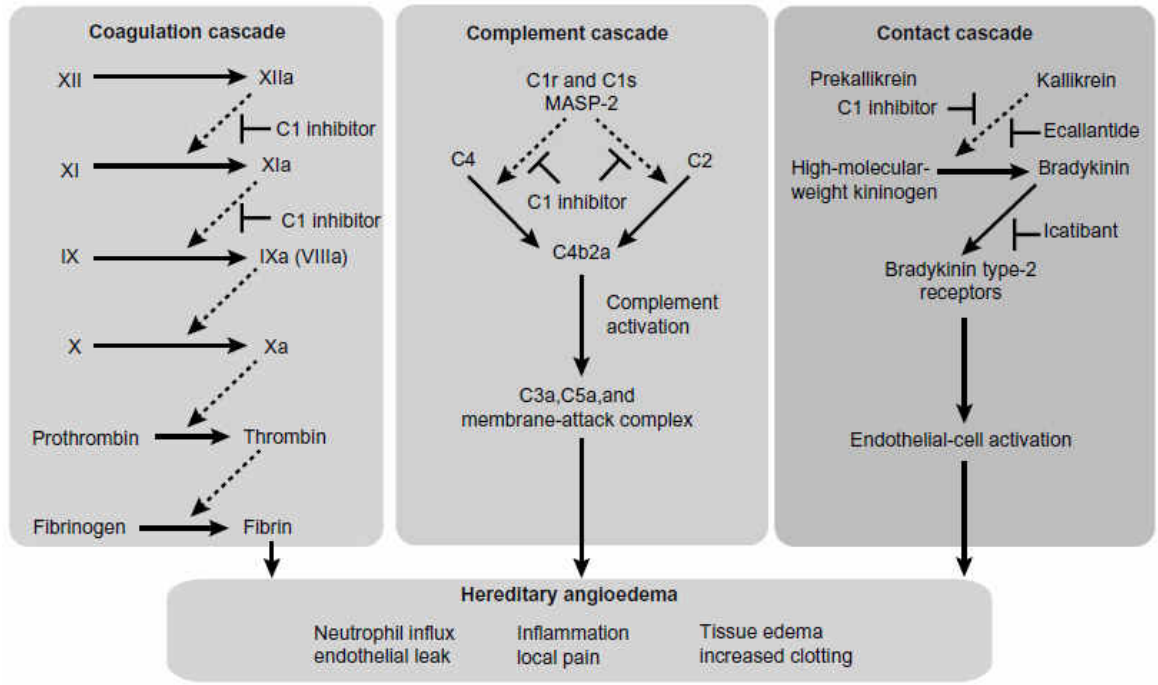
Hereditary angioedema is a genetic disease caused by the deficiency of C1-inhibitor (C1INH). C1INH is a plasma protein that is involved in the regulation of the complement system and the contact system. Both systems are part of our immune system. The disease is characterized by acute attacks of localized swellings or bruising of soft tissue. These attacks can be caused by minor trauma as well as emotional stress.

Hereditary angioedema (HAE) seriously affects the quality of life and, in those cases where the gastrointestinal tract or upper airways are affected, can lead to life-threatening complications. Most commonly, the respiratory and gastrointestinal systems are involved. Patients with gastrointestinal involvement generally experience severe abdominal pain, nausea and vomiting. Those with respiratory complication develop partial to complete obstruction, which can lead to suffocation. Onset is quick, often unprovoked, and generally persists for a duration of 24-72 hours. It is estimated that between 10,000 and 50,000 people in the western world suffer from HAE. Current therapeutic options consist of the administration of plasma derived C1INH for acute attacks and prophylactic treatment with attenuated androgens or anti-fibrinolytics.

There are however insufficient supplies of plasma-derived C1INH available to fulfil the demand. Production costs are very high and plasma-derived products always carry the risk of transferring blood-borne diseases from the donor to the recipient. The use of androgens is associated with a variety of side-effects, especially in pregnant women and children. Attenuated androgens or anti-fibrinolytics are not effective in case of an acute attack.

There are two recognized and accepted types of HAE: I and II. Type I is more common, found in 85% of the HAE population. It is characterized by a significant decrease or total absence of C1INH. Type II HAE, presenting in only 15% of those with the disorder, displays normal to increased levels of dysfunctional C1INH.

The role of C1INH, HAE and the Immune System



Dysregulation of coagulation, complement, and contact cascades in hereditary angioedema. C1 inhibitor controls activation in the complement, coagulation, and contact cascades, and all three cascades are dysregulated in hereditary angioedema. Replacement of C1 inhibitor restores homeostasis. Ecallantide (KALBITOR) and icatibant (FIRAZYR) specifically inhibit the contact cascade but have no direct effect on the complement or coagulation cascades. Dashed arrows indicate enzyme cleavage steps; T bars indicate points of inhibition.

The immune system protects the body against infections by bacteria, viruses and other parasites. It is really a collection of responses that the body makes to infection. So it is sometimes called the 'immune response'. The white blood cells involved in the acquired immune response are called 'lymphocytes'. There are two main types of lymphocytes - B cells and T cells. B and T lymphocytes are made in the bone marrow, like the other blood cells. They have to fully mature before they can help in the immune response. B cells mature in the bone marrow. But the

immature T cells travel through the blood stream to the thymus gland where they become fully developed. B cells react against invading bacteria or viruses by making proteins called antibodies. The antibody made is different for each different bug. The antibody locks onto the surface of the invading bacteria or virus. The invader is then marked with the antibody so that the body knows it is dangerous and it can be killed off. The binding of antibody to its antigen often triggers the complement system through the so-called classical pathway. The complement system is part of the immune system, like antibodies are part of the immune system. It is a defense mechanism that uses at least 30 proteins in the blood. It is named complement, because it helps antibodies to kill invaders or antigens. Another part of the immune system is the contact system. Activation of both the contact and complement system has been demonstrated in a variety of human diseases. A typical feature of both systems is that when activated, they give rise to several proteins, such as bradykinin, which influences endothelial cells. Layers of endothelial cells are lining the inside surface of blood vessels and lymph vessels. C1INH is a major inhibitor of both the complement and the contact system and is therefore endowed with anti-inflammatory properties. Several studies proved that patients with HAE had significantly decreased levels of C1INH. C1INH is the only known inhibitor of the activated proteins C1s and C1r of the complement system and is a major inhibitor of activated factor XII (pre-kallikrein = inactivated form of kallikrein) and XI (responsible for blood clotting) of the contact system. Kallikrein has been shown to release the protein bradykinin. In intact blood vessels, bradykinin has a function in keeping the blood flowing and keeping the vessels healthy. It stimulates the repair of vessels and is thought to play a major role in the symptomatology of acute attacks in patients with HAE. The large quantity of bradykinin releases during acute attacks of HAE is thought to be responsible for most symptoms by directly causing increased vascular permeability (edema and swelling). So the major function of C1INH within the human body includes the prevention of C1 complement auto activation.

Valuation

Based on NPV based valuation, we believe that Pharming is substantially undervalued at the current share price of EUR 0.32. Using our valuation model and taking into account the future revenues from RUCONEST for both acute and prophylactic use, the company's current total value should be EUR 660 million, which translates, based on an expected number of outstanding shares of approximately 700 million following conversion of the EUR 45 million 18 months amortising bond and the EUR 12.5 million 5 year convertible bond into EUR 0.94 per share. At this moment we do not address value to other programs in Pharming's pipeline.

Valuation RUCONEST in Acute and Prophylactic HAE

In estimating a value for RUCONEST, we took into account potential markets in the US and Europe with the US market calculated to be 75-85% of the total market. We calculate a Risk adjusted Discount Rate of 10%. For RUCONEST for prophylactic use we go with a LOA of 80% and a market launch in 2020 in the US. Pricing per attack is set at USD 10,000 with an average of 25 attacks per year. We calculate a net margin rising to 50% within a few years. We estimate that a peak market share of 20% for acute HAE and 10% for prophylactic HAE should be possible.

Year	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028
Market Size US Acute HAE	750	773	796	828	869	912	958	1006	1056	1109	1164	1223	1284
Penetration	5.3%	7.5%	9.5%	12%	14%	16%	18%	20%	20%	20%	20%	19%	18%
Market Size US Prophylactic	850	900	902	955	967	979	990	1001	1011	1020	1028	1036	1042
Penetration	0.0%	0.0%	0.0%	0.0%	0.5%	1.5%	2.5%	3.5%	4.5%	6.0%	7.5%	9.0%	10.0%
Total Revenues (USD m)	40.0	57.9	75.6	99.3	126.5	160.7	197.2	236.2	256.7	283.0	310.0	325.5	335.3
Margin up to 50%	-14.0	5.8	18.9	39.7	50.2	63.1	96.1	114.6	123.8	135.4	147.3	153.4	157.2
WACC 10%	1.00	0.91	0.83	0.75	0.68	0.62	0.56	0.51	0.47	0.42	0.39	0.35	0.32
NPV (million)	-14.0	5.3	15.6	14.8	34.3	26.8	40.1	58.8	57.8	57.4	56.8	53.8	50.1
Total NPV (million)													661.0
Value per share (EUR)													0.94

Near Term Milestones

In the coming 12 months we expect a number of important mile stones that can drive the stock price upwards. These are:

- 2017H1: End of Phase II meeting for prophylactic use RUCONEST
- 17 May 2017: First quarterly figures (Q1-2017) for RUCONEST wholly owned by Pharming
- 2017Q2: Disclosure other programs in the R&D Pipeline
- 2017Q3: Potential filing of BLA for RUCONEST for prophylactic use depending on outcome of FDA meeting

Analyst: Marcel Wijma MSc
Marcel Wijma, Chief Research Officer and managing partner, has a longstanding history in financial biotech research. After selling Van Leeuwenhoeck Research (VLR) to SNS Securities in 2006, he established an award winning analyst team in biotech/life sciences at SNS Securities. In 2009, Marcel was awarded by Financial Times/Starmine as being one of the Top-3 biotech analysts in Europe. Later that year, Marcel purchased VLR from SNS Securities after which the company was reconstituted. At VLR, he leads the professional VLR research organisation, which is augmented by selected external financial researchers with a specialisation in Life Sciences. Mr. Wijma has a Masters degree in Financial Economics from Erasmus University in Rotterdam.

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