NOVARTIS AG
Form 6-K
October 20, 2009

SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

FORM 6-K

REPORT OF FOREIGN PRIVATE ISSUER

PURSUANT TO RULE 13a-16 or 15d-16 OF

THE SECURITIES EXCHANGE ACT OF 1934

Report on Form 6-K dated October 20, 2009

(Commission File No. 1-15024)

Novartis AG

(Name of Registrant)

Lichtstrasse 35

4056 Basel

Switzerland

(Address of Principal Executive Offices)

Indicate by check mark whether the registrant files or will file annual reports under cover of Form 20-F or Form 40-F:

Form 20-F: x Form 40-F: o

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(1):

Yes: o **No**: x

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(7):

Yes: o **No**: x

Indicate by check mark whether the registrant by furnishing the information contained in this form is also thereby furnishing the information to the Commission pursuant to Rule 12g3-2(b) under the Securities Exchange Act of 1934.

Yes: o **No**: x

Novartis International AG

Novartis Global Communications

CH-4002 Basel

Switzerland

http://www.novartis.com

- Investor Relations Release -

New Phase II data show ACZ885 gave better pain relief and flare prevention for patients with chronic gout than an injectable corticosteroid

- *ACZ885 provided pain relief and reduced risk of flares by 94% versus an injectable corticosteroid in hard-to-treat patients unable to use common gout medicines(1)*

- *Gout is one of the most painful forms of arthritis involving acute attacks of inflammation that can damage joints and bone(2),(3)*

- *ACZ885 blocks action of inflammatory protein interleukin-1 beta which plays key role in causing painful gout flares(4),(5),(6)*

Basel, October 20, 2009 New Phase II results show that the novel biological therapy ACZ885 (canakinumab) is significantly more effective than an injectable corticosteroid at reducing pain and preventing recurrent attacks or flares in patients with hard-to-treat gout, one of the most painful forms of arthritis(1).

The study met its primary endpoint by showing that during acute gout flares, ACZ885 reduced pain faster and more effectively than the injectable corticosteroid triamcinolone acetonide(1), a potent steroid with sustained effect used to treat severe inflammatory conditions ($p<0.05$)(7).

At the end of the eight-week study, the risk of flare recurrence was 94% less for patients on ACZ885 than on the steroid ($p=0.006$)(1). The results were presented today at the American College of Rheumatology (ACR) Annual Scientific Meeting in Philadelphia, USA.

If not appropriately treated, gout can be a devastating condition. Current therapies can have limited efficacy and tolerability, and may be unsuitable for some patients(8),(9), said Professor Alexander So, MD, Department of Rheumatology at the University of Lausanne, Switzerland. These results are important as they indicate that canakinumab may provide significant benefit in both the prevention and treatment of painful

acute flares in these hard-to-treat patients.

ACZ885 is a fully human monoclonal antibody which blocks the action of the inflammatory protein interleukin-1 beta (IL-1 beta). It has already been approved under the brand name Ilaris® in a number of countries for treating cryopyrin-associated periodic syndrome (CAPS), a rare life-long auto-inflammatory disease with debilitating symptoms and few treatment options.

Studies with ACZ885 are ongoing in other diseases in which IL-1 beta plays an important role, such as chronic obstructive pulmonary disease (COPD), type 2 diabetes and systemic juvenile idiopathic arthritis (SJIA). Not all potential patients with these diseases would be eligible for treatment with ACZ885, if approved.

Gout, also known as gouty arthritis, affects more than 1% of adults in Western countries(10),(11). It is more common in older people, with around 10% of men and 6% of women over 70 years old

suffering from the disease(6),(12),(13). Approximately one in 10 patients have poorly controlled gout resulting in more frequent flares(14). They are regarded as being hard-to-treat as they are often intolerant or unresponsive to standard medications such as colchicine or non-steroidal anti-inflammatory drugs (NSAIDs)(15). Corticosteroids have traditionally been given to these patients as a last resort to treat acute pain, but they may have significant side effects(7),(16).

Gout is caused by the accumulation of uric acid crystals leading to severe inflammation in the joints and surrounding tissue(2),(3). Recent advances in the understanding of the disease have shown that uric acid crystals activate production of IL-1 beta, which is responsible for the inflammatory symptoms experienced by gout patients(6). ACZ885 provides a potent and selective blockade of IL-1 beta for a sustained period, neutralizing it and reducing inflammation(4),(17),(18).

The devastating impact of chronic gout is often underestimated by both healthcare professionals and the general public, and there is a real unmet need among patients, said Trevor Mundel, MD, Head of Global Development at Novartis Pharma AG. The latest data are encouraging for patients with hard-to-treat gout in whom the disease is not effectively managed, resulting in chronic pain. These findings also reinforce the potential of ACZ885 in a number of inflammatory diseases where IL-1 beta plays a key role.

The results presented at ACR were from a randomized, single-blind, double-dummy Phase II study involving 200 patients aged 18-80 years old with chronic gout, designed to assess the efficacy and optimum dose of ACZ885 in patients for whom current treatments (colchicine and/or NSAIDs) are ineffective or contraindicated(1).

The study showed that patients given ACZ885 150 mg experienced faster and more effective pain relief than those given the corticosteroid triamcinolone acetonide from 24 hours up to seven days(1). ACZ885 was given by subcutaneous injection, i.e. under the skin, whereas the steroid was given by intramuscular injection, i.e. into the muscle.

No pattern of adverse events was seen in any of the treatment groups and the incidence of adverse events was similar for both medicines(1). Serious adverse events occurred in two patients receiving ACZ885 and one receiving triamcinolone acetonide. Investigators reported that these events were not related to the study drug(1). There were no discontinuations due to adverse events(1).

Ilaris has been launched in the US and Switzerland, and received a positive opinion recommending approval in the EU in July 2009 to treat adults and children over four years old with CAPS. In the US, Ilaris is approved to treat adults and children four years of age and older with CAPS, including Familial Cold Autoinflammatory Syndrome (FCAS) and Muckle-Wells Syndrome (MWS). Reviews are ongoing in other countries including priority review in Australia, Brazil and Canada. Ilaris has been designated as an orphan drug for treating SJIA in the US, EU and Switzerland, and has fast-track status for SJIA in the US.

Disclaimer

The foregoing release contains forward-looking statements that can be identified by terminology such as can , may, potential, priority review, fast-track status, or similar expressions, or by express or implied discussions regarding potential additional indications for Ilaris, or regarding potential future revenues from Ilaris. You should not place undue reliance on these statements. Such forward-looking statements reflect the current views of the Company regarding future events, and involve known and unknown risks, uncertainties and other factors that may cause

actual results with Ilaris to be materially different from any future results, performance or achievements expressed or implied by such statements. There can be no guarantee that Ilaris will be approved for any additional indication. Nor can there be any guarantee that Ilaris will achieve any levels of revenue in the future. In particular, management s expectations regarding Ilaris could be affected by, among other things, unexpected clinical trial results, including unexpected

new clinical data and unexpected additional analysis of existing clinical data; unexpected regulatory actions or delays or government regulation generally; the company s ability to obtain or maintain patent or other proprietary intellectual property protection; competition in general; government, industry and general public pricing pressures; the impact that the foregoing factors could have on the values attributed to the Group s assets and liabilities as recorded in the Group s consolidated balance sheet, and other risks and factors referred to in Novartis AG s current Form 20-F on file with the US Securities and Exchange Commission. Should one or more of these risks or uncertainties materialize, or should underlying assumptions prove incorrect, actual results may vary materially from those anticipated, believed, estimated or expected. Novartis is providing the information in this press release as of this date and does not undertake any obligation to update any forward-looking statements contained in this press release as a result of new information, future events or otherwise.

About Novartis

Novartis provides healthcare solutions that address the evolving needs of patients and societies. Focused solely on healthcare, Novartis offers a diversified portfolio to best meet these needs: innovative medicines, cost-saving generic pharmaceuticals, preventive vaccines, diagnostic tools and consumer health products. Novartis is the only company with leading positions in these areas. In 2008, the Group s continuing operations achieved net sales of USD 41.5 billion and net income of USD 8.2 billion. Approximately USD 7.2 billion was invested in R&D activities throughout the Group. Headquartered in Basel, Switzerland, Novartis Group companies employ approximately 99,000 full-time-equivalent associates and operate in more than 140 countries around the world. For more information, please visit http://www.novartis.com.

References

(1) So A, De Meulemeester M, Shamim T et al. Canakinumab (ACZ885) vs. triamcinolone acetonide for treatment of acute flares and prevention of recurrent flares in gouty arthritis patients refractory to or contraindicated to NSAIDs and/or colchicine. Abstract presented at the American College of Rheumatology Annual Meeting, Philadelphia, United States of America (US), October 17-21, 2009.

(2) Martinon F, Glimcher LH. Gout: new insights into an old disease. *J Clin Invest*, 2006;116: 2073 5.

(3) Choi HK, Curhan G. Gout: epidemiology and lifestyle choices. *Curr Opin Rheumatol*, 2005; 17: 341 5.

(4) Alten R, Gram H, Joosten LA, et al. The human anti-interleukin-1ß (IL-1ß) monoclonal antibody ACZ885 is effective in joint inflammation models in mice and in a proof of concept study in rheumatoid arthritis patients. *Arthritis Res Ther*, 2008; 10: R67.

(5) Carneiro LA, Magalhaes JG, Tattoli I et al. Nod-like proteins in inflammation and disease. *J Pathol*, 2008 Jan; 214(2): 136-48.

(6) So A. Developments in the scientific and clinical understanding of gout. *Arthritis Res Ther*, 2008; 10: 221-226.

(7) KENALOG-10. Triamcinolone acetonide injection, suspension. US FDA label. Available at: http://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?id=6526 Accessed: 13 October 2009.

(8) Harrold LR, Andrade SE, Briesacher BA et al. Adherence with urate-lowering therapies for the treatment of gout. *Arthritis Res Ther*, 2009; 11: R46.

(9) Ganson NJ, Kelly SJ, Scarlett E et al. Control of hyperuricemia in subjects with refractory gout, and induction of antibody against poly(ethylene glycol) (PEG), in a phase I trial of subcutaneous PEGylated urate oxidase. *Arthritis Res Ther,* 2006; 8: R12.

(10) Terkeltaub RA. Learning how and when to employ uricase as bridge therapy in refractory gout. *J Rheumatol,* 2007; 34: 1955 8.

(11) Bieber JD, Terkeltaub RA. Gout: on the brink of novel therapeutic options for an ancient disease. *Arthritis and Rheumatism*, 2004; 50: 2400-2414.

(12) Kramer HM, Curhan G. The association between gout and nephrolithiasis: The National Health and Nutrition Examination Survey III, 1988-1994. *Am J Kidney Dis,* 2002; 40: 37 42.

(13) Mikuls TR, Farrar JT, Bilker WB et al. Gout epidemiology: results from the UK General Practice Research Database, 1990 1999*Ann Rheum Dis*, 2005; 64; 267-272.

(14) Halpern R, Fuldeore MJ, Mody RR et al. The Effect of Serum Urate on Gout Flares and Their Associated Costs. An Administrative Claims Analysis. *J Clin Rheumatol*, 2009; 15(1): 3-7.

(15) Strand V et al. Oral presentation at the European League Against Rheumatism (EULAR) 2009 (Abstract 291).

(16) Pascual E, Sivera F. Therapeutic advances in gout. *Curr Opin Rheumatol*, 2007; 19: 122 7.

(17)

National Horizon Scanning Centre. Canakinumab for cryopyrin associated periodic syndrome. November 2008. Available at: http://www.pcpoh.bham.ac.uk/publichealth/horizon/outputs/documents/2008/sept-dec/Canakinumab.pdf Accessed: 02 October 2009.

(18) Lachmann HJ, Lowe P, Felix SD et al. In vivo regulation of interleukin 1 in patients with cryopyrin-associated periodic syndromes. *J. Exp. Med*, 2009. Published online April 13 2009. Available at: www.jem.org/cgi/doi/10.1084/jem.20082481. Accessed: 02 October 2009.

Novartis Media Relations

Eric Althoff

Novartis Global Media Relations

+41 61 324 7999 (direct)

+41 79 593 4202 (mobile)

eric.althoff@novartis.com

Irina Ferluga

Novartis Pharma Communications

+41 61 324 2422 (direct)

+41 79 824 1121 (mobile)

irina.ferluga@novartis.com

e-mail: media.relations@novartis.com

Novartis Investor Relations

Central phone:	+41 61 324 7944		
Ruth Metzler-Arnold	+41 61 324 9980	**North America:**	
Pierre-Michel Bringer	+41 61 324 1065	Richard Jarvis	+1 212 830 2433
John Gilardi	+41 61 324 3018	Jill Pozarek	+1 212 830 2445
Thomas Hungerbuehler	+41 61 324 8425	Edwin Valeriano	+1 212 830 2456
Isabella Zinck	+41 61 324 7188		

e-mail: investor.relations@novartis.com

e-mail: investor.relations@novartis.com

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized.

Novartis AG

Date: October 20, 2009

By: /s/ MALCOLM B. CHEETHAM

Name: Malcolm B. Cheetham
Title: Head Group Financial Reporting and Accounting