



# UNIVERSITÀ DEGLI STUDI DI MILANO

TO MAGNIFICO RETTORE OF UNIVERSITA' DEGLI STUDI DI MILANO

I the undersigned asks to participate in the public selection, for qualifications and examinations, for the awarding of a type A post-doc fellowship

[Matteo Vecellio]

## CURRICULUM VITAE

### PERSONAL INFORMATION

Surname	Vecellio
Name	Matteo
Date of birth	[21, 10, 1982]

### PRESENT OCCUPATION

Appointment	Structure
Type A Research Fellowship	Medical Biotechnology and Translational Medicine Dept (BIOMETRA) University of Milan (Italy) - Humanitas Research Centre

### EDUCATION AND TRAINING

Degree	Course of studies	University	year of achievement of the degree
Degree	Biotechnology (BSc)	Bicocca - Milan	2005
Specialization	Medical Biotech. (MSc)	Bicocca - Milan	2008
PhD	DIMET - Molecular and Translational Medicine	Bicocca - Milan	2012

### FOREIGN LANGUAGES

Languages	level of knowledge
French	C1
English	C2

### AWARDS, ACKNOWLEDGEMENTS, SCHOLARSHIPS

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Year	Description of award
2016	Fondation de France - Recherche Medicale 2016 (grant number 67138) Role: Co-Investigator
2016	“EULAR Abstract Awards 2016 in Basic Science” - EULAR 2016 Congress (London, UK)
2017	Versus Arthritis Career Development Award 2017 (grant number 21428) Role: PI
2018	Versus Arthritis PhD Scholarship 2018 (grant number PhD22198) Role: PI
2018	Arthritis Research UK Travel support” - SpA Meeting 2018 (Ghent, Belgium)
2018	“EULAR Travel Bursary 2018” - EULAR 2018 Congress (Amsterdam, The Netherlands)

## TRAINING OR RESEARCH ACTIVITY

Dr. Vecellio received his PhD degree in Translational and Molecular Medicine in 2012 from the University of Milan - Bicocca (Italy). After the PhD, he spent one year after at the Goethe University, in Frankfurt-am-Main (Germany), as junior Post-Doc.

He joined the Lab of Professor Wordsworth at NDORMS - University of Oxford, in June 2013. In 2016 he has been awarded with the Arthritis Research UK Career Development Fellowship as Junior Principal Investigator. He has recently started a collaboration with the Rheumatology and Clinical Immunology Lab of Professor Selmi at HUMANITAS Research Institute (Milan, Italy).

Dr. Vecellio's research goal is to investigate the epigenetic mechanisms by which single nucleotide polymorphisms (SNPs) genetically associated with the inflammatory arthritis Ankylosing Spondylitis (AS), contribute to disease pathogenesis. He is interested in evaluate the impact of disrupted transcriptional regulation (i.e. RUNX3, NOS2 and CD8-related genes) in AS and gain new insight into disease molecular mechanisms.

## PROJECT ACTIVITY

Year	Project
2013 ongoing	The role of RUNX3 in Ankylosing spondylitis
2018 ongoing	A functional investigation of the inducible nitric oxide synthase (NOS2) association with Ankylosing Spondylitis

## CONGRESSES AND SEMINARS

Date	Title	Place
2011	AHA Scientific Sessions	Orlando, florida, US
2013	Epigenetics & system biology in	Berlin, Germany



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	cardiovascular diseases	
2013	Keystone Symposia on Epigenomics and DNA methylation	Keystone, Colorado, US
2014	IGAS, International Genetics of Ankylosing Spondylitis Consortium	Shanghai, China
2016	EULAR	London, UK
2017	IGAS, International Genetics of Ankylosing Spondylitis Consortium	Palermo, Italy
2018	EULAR	Amsterdam, The Netherlands
2018	SpA Meeting	Ghent, Belgium
2019	IGAS, International Genetics of Ankylosing Spondylitis Consortium	Versailles, France

## PUBLICATIONS

Articles in reviews
See Publication List attached below
A single nucleotide polymorphism in a RUNX3 enhancer strongly associated with ankylosing spondylitis influences recruitment of factors to the Nucleosome Remodelling Deacetylase Complex Cohen CJ, Cortes A, Knight JC, Fischer R, Selmi C, Wordsworth BP and Vecellio M*.
Under Review (Ann Rheum Dis)
*corresponding author
The pathologic role of DNA methylation in autoimmune diseases Vecellio* M, Wu h, Lu Q, Selmi C. Under Review (Mol Diagn Ther) *corresponding author

Declarations given in the present curriculum must be considered released according to art. 46 and 47 of DPR n. 445/2000.

The present curriculum does not contain confidential and legal information according to art. 4, paragraph 1, points d) and e) of D.Lgs. 30.06.2003 n. 196.

Place and date: \_\_\_\_\_ Milan \_\_\_\_\_, \_\_\_\_\_ December the 5<sup>th</sup> 2019 \_\_\_\_\_

SIGNATURE

Matteo Vecellio

*Matteo Vecellio*



## Publication List - Vecellio

19. The role of epigenetics and immunological imbalance in the etiopathogenesis of psoriasis and psoriatic arthritis  
Frischknecht L, **Vecellio M** and Selmi C.  
*Ther Adv Musculoskelet Dis.* 2019 Nov 6;11:1759720X19886505.
- 18 A single nucleotide polymorphism in a RUNX3 enhancer strongly associated with ankylosing spondylitis influences recruitment of factors to the Nucleosome Remodelling Deacetylase Complex  
Cohen CJ, Cortes A, Knight JC, Fischer R, Selmi C, Wordsworth BP and **Vecellio M\***.  
*Under Review (Ann Rheum Dis)*  
\*corresponding author
17. RUNX3 and T-Bet in Immunopathogenesis of Ankylosing Spondylitis-Novel Targets for Therapy?  
**Vecellio M\***, Cohen CJ, Roberts AR, Wordsworth BP, and Kenna T.  
*Front Immunol.* 2019 Jan 10;9:3132.  
\*corresponding author
16. Quantifying the genetic risk for the development of axial SpA - could this become a diagnostic tool?  
Wordsworth BP, Cohen CJ and **Vecellio M**  
*Curr Opin Rheumatol* 2018 Apr 26. doi: 10.1097/BOR.0000000000000517
15. Evidence for a second ankylosing spondylitis associated RUNX3 regulatory polymorphism  
**Vecellio M**, Cortes A, Roberts AR, Ellis J, Cohen CJ, Knight JC, Brown MA, Bowness P and Wordsworth BP  
*RMD Open* 2018 Feb 8;4(1):e000628. doi: 10.1136/rmdopen-2017-000628)
14. The severity of ankylosing spondylitis and responses to anti-tumour necrosis factor biologics are not influenced by the TNF receptor polymorphism incriminated in multiple sclerosis  
Watts L, Karaderi T, Roberts AR, Appleton L, Wordsworth T, Cohen C, Wordsworth BP and **Vecellio M\***  
*Genes Immun* 2018 DOI : 10.1038/s41435-018-0017-0  
\*corresponding author
13. Investigation of a possible extended risk haplotype in the IL23R region associated with ankylosing spondylitis.  
Roberts AR, **Vecellio M**, Cortes A, Knight JC, Cohen CJ, Wordsworth BP.  
*Genes Immun.* 2017 Mar;18(2):105-108.
12. Reply to Reeves et al.: No evidence for rare ERAP1 haplotypes and haplotype combinations in ankylosing spondylitis.  
Wordsworth BP, **Vecellio M**, Roberts AR.  
*Proc Natl Acad Sci U S A.* 2017 Feb 28;114(9):E1577.
11. ERAP1 association with ankylosing spondylitis is attributable to common genotypes rather than rare haplotype combinations.  
Roberts AR, Appleton LH, Cortes A, **Vecellio M**, Lau J, Watts L, Brown MA, Wordsworth P.  
*Proc Natl Acad Sci U S A.* 2017 Jan 17;114(3):558-561.
10. An ankylosing spondylitis-associated genetic variant in the IL23R-IL12RB2 intergenic region modulates enhancer activity and is associated with increased Th1-cell differentiation.  
Roberts AR, **Vecellio M**, Chen L, Ridley A, Cortes A, Knight JC, Bowness P, Cohen CJ, Wordsworth BP.  
*Ann Rheum Dis.* 2016 Dec;75(12):2150-2156.
9. Generation of a Selective Small Molecule Inhibitor of the CBP/p300 Bromodomain for Leukemia Therapy.  
Picaud S, Fedorov O, Thanasioupolou A, Leonards K, Jones K, Meier J, Olzscha H, Monteiro O, Martin S, Philpott M, Tumber A, Filippakopoulos P, Yapp C, Wells C, Che KH, Bannister A, Robson S, Kumar U, Parr



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N, Lee K, Lugo D, Jeffrey P, Taylor S, **Vecellio M**, Bountra C, Brennan PE, O'Mahony A, Velichko S, Müller S, Hay D, Daniels DL, Urh M, La Thangue NB, Kouzarides T, Prinjha R, Schwaller J, Knapp S.  
*Cancer Res.* 2015 Dec 1;75(23):5106-5119.

8. The genetic association of RUNX3 with ankylosing spondylitis can be explained by allele-specific effects on IRF4 recruitment that alter gene expression.  
**Vecellio M**, Roberts AR, Cohen CJ, Cortes A, Knight JC, Bowness P, Wordsworth BP.  
*Ann Rheum Dis.* 2016 Aug;75(8):1534-40.

7. CBP30, a selective CBP/p300 bromodomain inhibitor, suppresses human Th17 responses.  
Hammitzsch A, Tallant C, Fedorov O, O'Mahony A, Brennan PE, Hay DA, Martinez FO, Al-Mossawi MH, de Wit J, **Vecellio M**, Wells C, Wordsworth P, Müller S, Knapp S, Bowness P.  
*Proc Natl Acad Sci U S A.* 2015 Aug 25;112(34):10768-73.

6. The histone acetylase activator pentadecylenemalonate 1b rescues proliferation and differentiation in the human cardiac mesenchymal cells of type 2 diabetic patients.  
**Vecellio M**, Spallotta F, Nanni S, Colussi C, Cencioni C, Derlet A, Bassetti B, Tilenni M, Carena MC, Farsetti A, Sbardella G, Castellano S, Mai A, Martelli F, Pompilio G, Capogrossi MC, Rossini A, Dimmeler S, Zeiher A, Gaetano C.  
*Diabetes.* 2014 Jun;63(6):2132-47.

5. Detrimental effect of class-selective histone deacetylase inhibitors during tissue regeneration following hindlimb ischemia.  
Spallotta F, Tardivo S, Nanni S, Rosati JD, Straino S, Mai A, **Vecellio M**, Valente S, Capogrossi MC, Farsetti A, Martone J, Bozzoni I, Pontecorvi A, Gaetano C, Colussi C.  
*J Biol Chem.* 2013 Aug 9;288(32):22915-29.

4. In vitro epigenetic reprogramming of human cardiac mesenchymal stromal cells into functionally competent cardiovascular precursors.  
**Vecellio M**, Meraviglia V, Nanni S, Barbuti A, Scavone A, DiFrancesco D, Farsetti A, Pompilio G, Colombo GI, Capogrossi MC, Gaetano C, Rossini A.  
*PLoS One.* 2012;7(12):e51694.

3. Human chorionic villus mesenchymal stromal cells reveal strong endothelial conversion properties.  
Meraviglia V, **Vecellio M**, Grasselli A, Baccarin M, Farsetti A, Capogrossi MC, Pompilio G, Coviello DA, Gaetano C, Di Segni M, Rossini A.  
*Differentiation.* 2012 Jun;83(5):260-70.

2. Mutations in the small GTPase gene RAB39B are responsible for X-linked mental retardation associated with autism, epilepsy, and macrocephaly.  
Giannandrea M, Bianchi V, Mignogna ML, Sirri A, Carrabino S, D'Elia E, **Vecellio M**, Russo S, Cogliati F, Larizza L, Ropers HH, Tzschach A, Kalscheuer V, Oehl-Jaschkowitz B, Skinner C, Schwartz CE, Gecz J, Van Esch H, Raynaud M, Chelly J, de Brouwer AP, Toniolo D, D'Adamo P.  
*Am J Hum Genet.* 2010 Feb 12;86(2):185-95.

1. Cognitive impairment in Gdi1-deficient mice is associated with altered synaptic vesicle pools and short-term synaptic plasticity, and can be corrected by appropriate learning training.  
Bianchi V, Farisello P, Baldelli P, Meskenaite V, Milanese M, **Vecellio M**, Mühlemann S, Lipp HP, Bonanno G, Benfenati F, Toniolo D, D'Adamo P.  
*Hum Mol Genet.* 2009 Jan 1;18(1):105